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Mathematical Modelling of Epidemics with account for Population Awareness

By

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Declaration

I hereby declare that this thesis has not been and will not be, submitted in whole or in part to another University for the award of any other degree.

Signature .....

Grace Omeche Agaba

Dedication

To God Almighty, the creator of heaven and earth, who has always being my loving, faithful and caring father, my saviour, redeemer, protector, provider and divine guidance.

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Abstract

In this thesis I developed and analysed several mathematical models that describe the dynamics of infectious diseases spreading in a population simultaneously with people becoming aware of the presence of the disease and thus modifying their behaviour. This is achieved using compartmental models, with further extensions to models with time delays and the administration of vaccines. Resulting mathematical models were analysed using the techniques of dynamical systems and bifurcations theory, complemented by direct numerical simulations. Design of optimal strategies maximising the reduction of infection rates subject to logistical constraints were studied within the new modelling framework and with a view to be used in realistic contexts. Of particular interest is the design and analysis of the impact of local and global awareness campaigns, as well as the administration of vaccines to minimise the spread of infections.

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Preface

Epidemic models are often employed to analyse the dynamics of infectious diseases. Whilst some situations require the use of detailed network-based models, significant insights can be obtained using mean-field compartmental models, such as the susceptible-infected-recovered (SIR), susceptible-infected-susceptible (SIS), or the susceptible-infected-recovered-susceptible (SIRS) models and their multiple modifications. When developing epidemic models for the dynamics of infectious disease with account for human behavioural changes, the propagation of awareness about a disease has been shown to play a significant role in curtailing the spread of infectious diseases. In this context, delay in the circulation of information about the disease locally (through direct interactions between individuals) and globally (through the media and other sources of awareness) can influence the rate of spread of the disease within human population. Significant delays in the spread of awareness tend to facilitate the endemic state of the disease outbreak, thus highlighting the need for a prompt dissemination of information to the population. These effects are particularly important when one considers different strategies for administration of vaccines against infections, especially if the vaccines are leaky, i.e. when they do not provide perfect immunity.

In the first part of the thesis I develop an SIRS-type epidemic model to explore the dynamics of infectious diseases spreading in a population simultaneously with people becoming aware of the presence of the disease, and thus modifying their behaviour. The model is studied analytically and numerically to evaluate the impact of both local and global awareness on the spread of disease. The main underlying assumption of the model is that the susceptible, the infected and the recovered individuals can all access information regarding the outbreak through global sources, such as media campaigns, in addition to becoming aware through contacts or in-

teractions with their aware neighbours. The results of the model show how the availability of local, as well as global awareness to the population can act as a control measure for the spread of infectious diseases.

In order to better understand the role of time delays associated with behavioural changes in the population in response to awareness, in the second part of the thesis I develop and analyse an SIS epidemic model that explicitly takes into account a time delay representing the time required for the dissemination of information and subsequent changes in human behaviour. Analytical and numerical studies yield stability results for the disease-free and endemic equilibria, together with a detailed information about the characteristic eigenvalues of the model. The outcome of this analysis suggests that while stability of the disease-free steady state is independent of the delay in the dissemination of awareness, this delay can cause Hopf bifurcation of the endemic equilibrium, thus resulting in periodic oscillations. The results show that the propagation of awareness tends to curtail the spread of infectious diseases, and the delay fosters the spread of infectious diseases within a human population. This provides useful information about requirements on the speed of circulation of awareness and human responses needed to minimise the spread of infectious diseases.

In the last part of the thesis I develop and study a time-delayed model with vital dynamics to understand the effects of vaccination on the dynamics of infectious diseases in the presence of disease awareness. Having established well-posedness of the model, conditions for stability of the disease-free and endemic equilibria are derived in terms of system parameters, and conditions for a Hopf bifurcation of the endemic steady state are obtained. To gain a better insight into the dynamics, characteristic eigenvalues are computed numerically using the traceDDE suite, and numerical simulations are performed to illustrate the behaviour of the model in different dynamical regimes. DDE-BIFTOOL numerical bifurcation software is used to continue periodic solutions of the model and to obtain information about the dependence of their amplitudes and periods on the time delay.

Chapter 1

Introduction

Infectious diseases are known to have caused huge devastation and loss of human life throughout the ages, and most recently they pose a great threat, especially to developing countries [19, 49]. The last two decades have witnessed a number of major outbreaks of infectious diseases, including avian and swine flu, SARS, Ebola, and, most recently, the Zika virus. Due to the globalised travel and significant advances in social media, information about these outbreaks is now spreading quite quickly, and this, in turn, can have a profound effect on the actual epidemic dynamics [15, 25, 43, 45].

Interestingly, awareness can have very complex and sometimes unexpected effects on the dynamics of the disease spread. It can have a clearly positive influence, where disease propagation is minimised or fully stopped by various disease control measures. These include among others the use of face masks, condoms or other tools appropriate for specific diseases, as well as vaccination and even quarantine, with examples ranging from the plague outbreak in the English village of Eyam in 1665-1666 [42], where the village completely sealed itself off to prevent further transmission of plague, to more recent outbreaks of swine influenza [25] and Ebola [45].

On the other hand, the spread of information about a disease can also result in anxiety and panic, which can lead to undesired consequences, such as the uncontrolled spread of plague during the 1994 outbreak in one of the states in India, where by fleeing the endemic area the people carried the disease with them, thus infecting other parts of the country [46], or the failure of HPV vaccination campaign

in Romania due to the negative press coverage [44]. In light of this complexity of behavioural changes in the population in the presence of awareness, it is important to understand how the concurrent spread of disease and awareness affects disease dynamics.

1.1 Literature review

1.1.1 Epidemic models

The general study of the causes of epidemic outbreaks and the spread of epidemics has led to the development of a variety of mathematical models [42]. These models are considered very useful in describing the dynamics of infectious diseases and for predicting how a disease can be contained or eradicated from a given geographical location.

Epidemic dynamics are often represented mathematically through compartmental models, such as the SIS (susceptible-infected-susceptible) model [10, 30, 35, 56, 62] and the SIR (susceptible-infected-recovered) model [1, 5, 17, 29, 31, 34, 53, 59]. The SIS and SIR models form the bases for developing other, more advanced epidemic models, for instance, the SAIS model with 'A' denoting the aware population [26, 48], the SIRS model [16, 66] for cases that involve a loss of immunity, the SEIR model with 'E' representing the exposed population (that is, those infected but not infectious as the disease is latent) [43, 45]. Similarly, there are epidemic models that use separate compartments for the vaccinated (V) individuals [3, 27, 54, 58], the cumulative density of awareness programs (M) [19, 39, 40, 47, 49, 68], the hospitalized (H) [36, 60] and treated (T) individuals [23, 28, 61] to analyse their respective impacts on the disease dynamics via the SIS, SIR, SEIR and SIRS epidemic models as appropriate.

A simple SIS model for a disease spreading in a closed homogeneously mixed population can be represented mathematically as

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\beta\, S\, I}{N} + r\, I, \\ \frac{dI}{dt} &= \frac{\beta\, S\, I}{N} - r\, I, \end{aligned} \tag{1.1}$$

where S and I are in proportion to the entire population, N. The parameter β is

the transmission rate of infection from the infected to the susceptible, and r denotes the rate of recovery of the infected individuals, that is, $1/r$ measures the duration of time spent in the infectious state. Introducing non-dimensional quantities $s = \frac{S}{N}$ and $i = \frac{I}{N}$, this model can be equivalently rewritten in the form

$$\begin{aligned} \frac{ds}{dt} &= -\beta\, s\, i + r\, i, \\ \frac{di}{dt} &= \beta\, s\, i - r\, i. \end{aligned}$$

Similarly, a simple SIRS epidemic model can be written as follows

$$\begin{aligned} \frac{dS}{dt} &= -\frac{\beta\, S\, I}{N} + \delta\, R, \\ \frac{dI}{dt} &= \frac{\beta\, S\, I}{N} - r\, I, \\ \frac{dR}{dt} &= r\, I - \delta\, R, \end{aligned} \tag{1.2}$$

with δ denoting the rate at which the recovered lose their immunity over time.

Models (1.1) and (1.2) represent a constant population, since the sum of the right-hand sides of all equations is equal to zero. Considering the model (1.2), we have

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0 \qquad \Rightarrow \qquad S(t) + I(t) + R(t) = N.$$

The SIRS epidemic model can be analysed using the initial conditions

$$S(0) = S_0 > 0, \quad I(0) = I_0 > 0, \quad \text{and} \quad R(0) = 0,$$

which imply

$$\left.\frac{dI}{dt}\right|_{t=0} = \frac{\beta\, S_0\, I_0}{N} - r\, I_0 = \frac{\beta\, I_0}{N}\left(S_0 - \frac{N\, r}{\beta}\right) \quad \begin{cases} > 0, \\ < 0, \end{cases} \qquad \text{if} \quad S_0 \begin{cases} > \frac{N\, r}{\beta}, \\ < \frac{N\, r}{\beta}, \end{cases}$$

and

$$\left.\frac{dS}{dt}\right|_{t=0} = -\frac{\beta\, S_0\, I_0}{N} \leq 0.$$

Hence, if $S_0 < \frac{N\, r}{\beta}$, it implies that

$$\frac{dI}{dt} = \frac{\beta\, I}{N}\left(S - \frac{N\, r}{\beta}\right) \leq 0 \qquad \text{for all} \quad t \geq 0.$$

Therefore, $I_0 > I(t) \to 0$ as $t \to \infty$, and the disease fades out, thus preventing an epidemic outbreak. On the other hand, for $S_0 > \frac{N\, r}{\beta}$, there is an epidemic outbreak,

as for sufficiently small t, we have $I(t) > I_0$, that is, the infected population $I(t)$ increases.

In this case, the basic reproductive number, R_0, which is defined as the number of secondary infections that occur when an infected individual is introduced into a fully susceptible population, can be readily found as $R_0 = \frac{\beta}{r}$. Hence for $R_0 > 1$, the spread of the disease reaches an epidemic state, while for $R_0 < 1$ the infection dies out.

The study of epidemiology over the past years has witnessed much attention been focused on areas that study the dynamics of epidemic models with time delay [34]. Time delay models are often used to evaluate or analyse the incubation period of infectious diseases, the periods of infection of the infected individuals, the immunity period of recovery of the disease, and in the case of awareness, the delay in individual responses to available information and/or the delay in awareness circulation [1, 18, 34, 59, 63]. Consequently, delay models that describe situations as aforementioned are best represented by delay differential equations. A delay differential equation can be written in the following general form

$$\frac{dz(t)}{dt} = f\big(t, z(t), z_\tau\big), \qquad z(t) \in \mathbb{R}^n, \qquad \text{and} \qquad z_\tau = \{z(\tau) : \tau \leq t\},$$

where z_τ represents the solution of the system in the past, and τ is the representation for time delay. Delay differential equations can take the form of a continuous delay equation

$$\frac{dz(t)}{dt} = f\left(t, z(t), \int_{-\infty}^{0} z(t+\tau)e^{k\tau}d\tau\right),$$

or discrete delay equation

$$\frac{dz(t)}{dt} = f\big(t, z(t), z(t-\tau_1), \ldots, z(t-\tau_m)\big), \qquad \text{for} \qquad \tau_1 > \cdots > \tau_m \geq 0.$$

In the case of a single delay term, the discrete equation is expressed as

$$\frac{dz(t)}{dt} = f\big(t, z(t), z(t-\tau)\big), \qquad \text{for} \qquad \tau \geq 0. \tag{1.3}$$

The solutions of delay differential equation are often obtained using the method of steps [13]. For instance, considering (1.3) with the initial condition $\tilde{z} : [-\tau, 0] \to \mathbb{R}^n$, it implies that the solution $z(t)$ over the interval $[0, \tau]$ satisfies the inhomogeneous initial value problem

$$\frac{dz(t)}{dt} = f\big(z(t), \tilde{z}(t-\tau)\big), \qquad z(0) = \tilde{z}(0).$$

Linear discrete delay equations have the form

$$\frac{dz(t)}{dt} = A_0\, z(t) + A_1\, z(t-\tau_1) + \cdots + A_m\, z(t-\tau_m), \tag{1.4}$$

where $A_i \in \mathbb{R}^{n\times n}$, $i = 0, 1, 2, \ldots, m$. Just like the case of ordinary differential equation, the linear discrete delay equation can be analysed using the characteristic equation. In the case of (1.4), the characteristic equation has the form,

$$\det\left(-k\,\mathbb{I} + A_0 + A_1\, e^{-k\,\tau_1} + \cdots + A_m\, e^{-k\,\tau_m}\right) = 0, \tag{1.5}$$

where k denotes the eigenvalue, and $\mathbb{I}$ is the $n \times n$ identity matrix. Due to the presence of the exponentials in the characteristic equation, the characteristic equation (1.5) has an infinite number of eigenvalues unlike the case of ordinary differential equations. However, despite the infinite number of eigenvalues, only a finite number of them are in the right complex half-plane [13, 38].

Delay differential equations differ from ordinary differential equations in the fact that, with delay differential equations, past history influences present dynamics. Considering the following linear first-order ordinary differential equation,

$$\frac{dz(t)}{dt} = x_{_0}\, z(t), \qquad z(0) = 1, \tag{1.6}$$

with $x_{_0}$ being a positive constant. The solution of this equation can be derived using the method of separation of variables which implies

$$\frac{dz(t)}{z(t)} = x_{_0}\, dt,$$

and integrating both sides of this equation gives

$$\int \frac{dz(t)}{z(t)} = \int x_{_0}\, dt \qquad \Rightarrow \qquad \ln z(t) = x_{_0}\, t + c,$$

where c is the constant of integration. Hence, we obtain

$$z(t) = e^{x_{_0} t\, +\, c} = y_{_0}\, e^{x_{_0} t}, \qquad \text{with} \quad y_{_0} = e^{c}.$$

Using the initial condition, $z(0) = 1$, the solution of (1.6) has the form

$$z(t) = e^{x_{_0} t}. \tag{1.7}$$

Consequently, the present value of z, that is, $z(0) = 1$, determines the future value of z at any given time, t. An equivalent delay differential equation would have a form

$$\frac{dz(t)}{dt} = x_{_0}\, z(t-\tau), \qquad z(s) = 1, \qquad \text{with} \quad s \in [-\tau, 0). \tag{1.8}$$

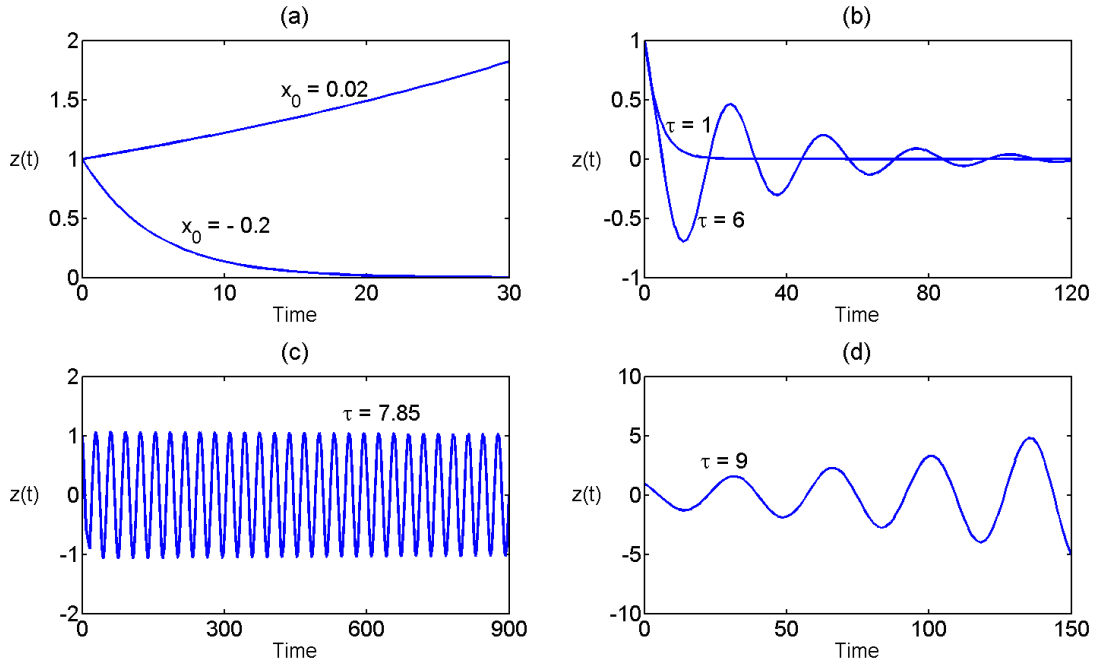

Figure 1.1: Dynamics of solutions representing ordinary differential equation (1.6) in plot (a) and delay differential equation (1.8) with $x_0 = -0.2$ in plots (b) - (d).

In contrast to (1.6), the initial condition is given by a function defined over a finite time interval [13], and by substituting $z(t) = y_0\, e^{k\,t}$ and its derivative into (1.8) we obtain

$$k\, y_0\, e^{k\,t} = x_0\, y_0\, e^{k\,(t-\tau)} \qquad \Rightarrow \qquad k\, e^{k\,t} = x_0\, e^{k\,t}\, e^{-k\,\tau},$$

multiplying both sides of this equation with the inverse of $e^{k\,t}$ gives

$$k = x_0\, e^{-k\,\tau}.$$

Hence, the solution of the delay differential equation (1.8) takes the form

$$z(t) = y_0\, e^{k\,t}, \qquad \text{where} \quad k = x_0\, e^{-k\,\tau}. \tag{1.9}$$

Considering (1.9) for $\tau = 0$, we have $k = x_0$, which implies that the system (1.8) is linearly asymptotically stable whenever $x_0 < 0$ and unstable for $x_0 > 0$. These results coincide with the dynamics of the ordinary differential equation (1.6). For $\tau > 0$, we note that $k = 0$ is not a solution of (1.8), therefore considering the solution in the form $k = i\,\mu$ gives

$$\begin{aligned} i\,\mu &= x_0\, e^{-i\,\mu\,\tau} \\ &= x_0\, \big[\cos(\mu\,\tau) - i\, \sin(\mu\,\tau)\big]. \end{aligned}$$

Equating the real and imaginary parts give the conditions for oscillatory solution as

$$\cos(\mu\,\tau) = 0 \qquad \text{and} \qquad \mu = -x_{_0}\,\sin(\mu\,\tau),$$

and for $\cos(\mu\,\tau) = 0$ we have infinite number of solutions for $\mu\,\tau$ but since cosine function has a period of $2\,\pi$, it implies that $\cos(\mu\,\tau) = 0$ yields $\mu\,\tau = \frac{\pi}{2}$ or $\mu\,\tau = \frac{3\,\pi}{2}$ within the interval $(0,\, 2\,\pi)$. Hence, from $\mu = -x_{_0}\,\sin(\mu\,\tau)$ we obtain

$$x_{_0}\,\tau = -\frac{\pi}{2} \qquad \text{when} \qquad \mu\,\tau = \frac{\pi}{2},$$

and

$$x_{_0}\,\tau = \frac{3\,\pi}{2} \qquad \text{when} \qquad \mu\,\tau = \frac{3\,\pi}{2}.$$

Therefore, the following values of the time delay can give rise to periodic solutions:

$$i)\quad \mu\,\tau = \frac{\pi}{2} \quad \text{and} \quad x_{_0}\,\tau = -\frac{\pi}{2}, \qquad ii)\quad \mu\,\tau = \frac{3\,\pi}{2} \quad \text{and} \quad x_{_0}\,\tau = \frac{3\,\pi}{2}.$$

Figure 1.1 illustrates the respective dynamics of the steady states as regard the ordinary differential equation (1.6) and the delay differential equation (1.8).

1.1.2 Epidemic models with population awareness

A number of mathematical models have looked into the effects of information and awareness on the spread of epidemics. These models can be roughly divided into two major classes: network-based models, where individuals are represented as nodes of a network, and edges correspond to possible connections along which a disease can be transmitted [17, 18, 20, 23, 26, 48, 62, 65], and mean-field models that assume global mixing between individuals in the population [9, 10, 35, 36, 56, 58].

Funk *et al.* [17] investigated how the spread of awareness prompted by a first-hand contact with the disease affects the disease dynamics. They showed that in a social network, the spread of awareness and the resulting reduction in susceptibility does not only lower the incidence of the disease, but in some cases can even prevent onset of epidemics, thus implying that awareness can act as an effective measure of disease control. Furthermore, their results suggest that in the presence of an infectious disease, social distancing should be considered not only from the perspective of some centrally controlled action, but also in terms of self-initiated behavioural

changes of individuals. This is further supported by Kleczkowski *et al.* [29] who analysed two dimensions of behavioural changes: reduction in the number of contacts (staying at home) and reduction in the likelihood of contacts resulting in infections (washing hands). Their results revealed that "washing hands" appeared more effective for short-lived diseases, while "staying at home" was better for long-lived diseases.

Mean-field models have provided an alternative approach for modelling the effects of awareness on disease transmission. One possibility is to represent awareness as the reduction of the transmission (contact) rate by some factor that grows with the number of infected individuals, with the common choices being either a saturated [10, 35, 56, 58] or an exponential [9, 36, 57] growth of the reduction factor. In the specific context of STIs, most individuals are actually aware of the spreading infection but they may still choose to not respond to the threat. Kiss *et al.* [28] have considered the effects of disease awareness in the case of STIs, where the rate of information transmission has the form of a saturating function of the number of infected individuals, and the value of information is allowed to decay over time. The authors have shown that whilst the population-wide (global) awareness does not affect the epidemic threshold, it reduces the infection prevalence at endemic equilibrium.

Another way to include awareness in mean-field models is by introducing a separate compartment for the "media" variable that effectively represents the level of awareness in the population, and the populations move from the unaware to aware compartments at rates proportional to this level of awareness [39, 40, 41, 49]. Mean-field models have highlighted a number of important features of dynamics associated with the simultaneous spread of disease and awareness, such as the occurrence of multiple disease outbreaks due to the spread of information [36], co-existence of multiple feasible equilibria [9, 36], as well as helped to analyse optimal disease control programs [35, 47, 58, 61] and the role of time delay in the response to awareness campaigns on disease dynamics [19, 66, 67, 68].

1.1.3 Time delayed models with population awareness

The significance of awareness creation cannot be overemphasized, as both local and global dissemination of information play very important roles in influencing the behavioural changes in humans [16, 23, 39, 49, 60, 62]. These behavioural changes often generate practices that protect individuals from infection, and, consequently, produce an impact on the dynamics of the spread of infectious diseases [17, 48, 49, 68]. Wu *et al.* [65] noted that local dissemination of awareness is effective at stopping the spread of infectious diseases, while the global circulation of awareness helps in curtailing the prevalence of the disease.

Public education at an early stage of an outbreak is considered very important in controlling the spread of infectious diseases. This is particularly important in the cases when the pharmaceutical intervention seems to be delayed as a result of prolonged processes of either discovering the nature of the pathogen or developing the required vaccine, or determining the treatment procedures [40, 56]. In such cases, the population could employ other preventive and/or control measures when informed of the infectious disease. According to Misra *et al.* [40], people largely depend on the information released by the media and other global sources in order to plan their respective movements and behaviour in the case of an infectious disease outbreak. They act on available information to control and prevent further spread of the pathogen in circulation. Consequently, the delay in disseminating such valuable information or awareness could eventually cause more harm.

In the past, early dissemination of information concerning an outbreak yielded positive results and assisted in providing detailed records of the prevalence of infectious diseases, controlling subsequent spread of infection, enhancing psychological impact on individual behaviour and response to treatment and preventative measures [56, 60]. Recent examples include the cases of SARS outbreak in 2003, H1N1 influenza pandemic in 2009 [17, 49, 62], and the spread of HIV/AIDS in Bangladesh among married couples [49]. The delay in the impact of awareness could be attributed to a number of factors that include the circulation of misleading information and the misinterpretation of accessed information [17, 56, 58, 67], which often leads to devastating consequences. For instance, the spread of rumors, which according to [16, 17] is similar to the spread of infectious diseases, is often a result of the circula-

tion of information from wrong channels (sources) or the spreading of misinterpreted information about the spread of the disease.

Time delays in epidemic models often represent the waiting periods at different stages, such as the incubation period of infectious diseases (that is the elapsed time between when a susceptible is infected by a pathogen and when they become infective), the duration of infection of patients, the period of immunity to the disease (recovery period), the time of response of individuals to available information (alertness to awareness) [1, 34, 59, 63]. Dynamical systems based on ordinary differential equation assume implicitly that all these periods are exponentially distributed. 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 individuals' 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.* [68] included time delay in the equation for the "media" variable M to account for a delayed reporting of cases of infections, while Misra *et al.* [41] have also included some degree of global awareness. Zhao *et al.* [66] incorporated delayed reporting into the reduced disease transmission rate. Zuo and Liu [67] focused on the analysis of the time delay between reports of infection and changes in the behaviour. In all these models, the disease-free steady state is stable when some 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 a zero time delay, and in the models of Zuo *et al.* [68], Zhao *et al.* [66], and Misra *et al.* [41] it can undergo Hopf bifurcation at certain value of the time delay, whereas in the model of Zuo and Liu [67], the endemic steady state is globally asymptotically stable independent of the time delay, provided it is biologically feasible. Greenhalgh *et al.* [19] 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 number of infected individuals, and both time delays can lead to a destabilisation of the endemic equilibrium and onset of oscillations.

1.1.4 Time delayed models with population awareness and vaccination

Vaccines are known to be effective means of disease control and prevention [14, 21, 27, 53], having led to a complete eradication of smallpox [4, 35] and a substantial reduction in the cases of polio, measles, mumps, rubella. Latest WHO forecasts suggest expected eradication of measles and mumps in Europe in the next few years [64]. Depending on a particular disease and each individual vaccine, the vaccine-induced immunity may be life-long, or individuals may require subsequent vaccinations to improve their immunity status. Another relevant aspect is vaccine "leakiness" [21, 22], which describes the situation where even with a complete population coverage, some individuals are unable to develop protective antibodies, which makes them susceptible to further disease outbreaks.

In order to achieve maximum impact, every vaccination campaign should be accompanied by appropriate information campaigns that educate individuals about the need of vaccination to prevent the spread of infection and achieve the desired level of herd immunity [27]. In some cases, negative press coverage has led to a reduction in vaccine uptake or even complete disruption of the vaccination campaign, as has been the case with an HPV vaccine in Romania [44] and the MMR vaccine in the UK [8]. Furthermore, fears associated with possible side effects or incorrect perceptions about vaccine efficiency may also be detrimental to the vaccine uptake and subsequent success [3, 54].

A number of mathematical models have looked into the dynamics of vaccination [2, 3, 11, 24, 27, 30, 50, 55] focusing on different types of vaccination schedules, various scenarios of vaccine uptake and efficiency, and the resulting control of epidemics. Some work has also been done on developing techniques for assessment and quantifying of vaccine efficacy and efficiency [14, 21, 53]. More recently, attention has turned to vaccination models that include different types of population awareness [35, 52, 58] and/or time delays due to either epidemiological properties of infection, such as latency or temporary immunity, or time delay in individuals' responses to information about the disease [1, 32, 37, 51].

1.2 Thesis outline

The research in this thesis focuses on the mathematical modelling of epidemics with account for population awareness.

In Chapter 2, I present and analyse an epidemic model where the unaware susceptible, infected and recovered populations can all become aware of an invading disease through various means, such as the media, health campaign programme, etc. in addition to direct interactions between individuals. Feasibility and stability of different steady states are established in terms of disease parameters, and numerical simulations are performed to illustrate the behaviour of the model in different dynamical regimes.

Chapter 3 derives a time-delayed SIS epidemic model with population awareness to evaluate the impact of time delay in the response of unaware individuals to available information on the epidemic dynamics. Conditions for stability and the Hopf bifurcation of the endemic steady state are found in terms of system parameters and the time delay. Numerical bifurcation analysis reveal how the amplitude and the period of the periodic solutions vary with the response time delay.

In Chapter 4, an SIRS time delayed model is developed to investigate the effects of vaccination on the dynamics of the infectious disease, which is spreading in a population concurrently with awareness. The model considers contributions to the overall awareness from a global information campaign, direct contacts between unaware and aware individuals, and reported cases of infection. It is assumed that there is some time delay between individuals becoming aware and modifying their behaviour. Vaccination is administered to newborns, as well as to aware individuals, and it is further assumed that vaccine-induced immunity may wane with time. I show how different types of awareness and vaccination rates affect the disease dynamics, and how the time delay in individuals' response can lead to the onset of oscillations around the endemic equilibrium.

Chapter 5 contains the summary and discussion of the main results of thesis, as well as directions for further research.

Chapter 2

Mathematical model for the impact of awareness on the dynamics of infectious diseases

In an outbreak of infectious disease, people sometimes change their behaviour not necessarily because they were infected by the disease but often as a result of the acquired information from the media or awareness campaigns. However, information from global sources such as the media and health campaign usually pay attention to high-profile diseases and large statistics of reported cases of infection which in most cases generates low amount of awareness among the people. Consequently, awareness prompt by individuals hearing of others being infected as they interact or by other forms of local information transmission tends to increase in the advent of disease outbreak not covered by global sources of awareness [16].

Earlier work has highlighted the fact that the spread of awareness from both local and global sources of information influence changes in human behaviour, which in turn, affects the spread of infectious diseases within the population. This chapter focuses on how the dissemination of local awareness arising from direct contacts between unaware and aware individuals, and global awareness by information campaigns affect the dynamics of the disease spread. The model includes the possibility of direct contacts between unaware and aware individuals regardless of their disease status, and it also takes into account the global spread of awareness through various media and information campaigns.

2.1 Model derivation

In order to analyse the effects of awareness on the dynamics of a directly transmitted disease, we use an SIRS-type model similar to a model considered in Funk *et al.* [16], and divide the overall population into two major groups of compartments: unaware susceptible, infected and recovered individuals (denoted by S_n, I_n and R_n) and aware susceptible, infected and recovered individuals (denoted by S_a, I_a and R_a). A disease is characterised by a transmission rate β for unaware population, which is reduced by the factors $0 < \sigma_i < 1$ and $0 < \sigma_s < 1$ that represent the decrease in infectivity and susceptibility, respectively. A reduction in infectivity occurs due to infected individuals taking treatment or possibly staying at home (quarantine) to reduce their contacts, while a reduction in susceptibility is associated with susceptible individuals taking measures for disease prevention, such as face masks, vaccination or tablets etc. Infected individuals recover at a rate r, which is further amplified by a factor $\varepsilon > 1$ for aware individuals. Upon recovery, it is assumed that individuals remain immune to the disease for an average period of $1/\delta$, after which they return to their respective class of susceptibles. The duration of temporary immunity for aware individuals is taken to be longer by a factor of $1/\phi$ [16].

$$
\begin{aligned}
\frac{dS_n}{dt} &= -\frac{(I_n + \sigma_i I_a)\,\beta\, S_n}{N} - \frac{\alpha\,(S_a + I_a + R_a)\,S_n}{N} + \lambda\, S_a + \delta\, R_n,\\
\frac{dI_n}{dt} &= \frac{(I_n + \sigma_i I_a)\,\beta\, S_n}{N} - \frac{\alpha\,(S_a + I_a + R_a)\,I_n}{N} + \lambda\, I_a - r\, I_n - \omega\, I_n,\\
\frac{dR_n}{dt} &= -\frac{\alpha\,(S_a + I_a + R_a)\,R_n}{N} + \lambda\, R_a - \delta\, R_n + r\, I_n,\\
\frac{dS_a}{dt} &= -\frac{(I_n + \sigma_i I_a)\,\sigma_s\,\beta\, S_a}{N} + \frac{\alpha\,(S_a + I_a + R_a)\,S_n}{N} - \lambda\, S_a + \phi\,\delta\, R_a,\\
\frac{dI_a}{dt} &= \frac{(I_n + \sigma_i I_a)\,\sigma_s\,\beta\, S_a}{N} + \frac{\alpha\,(S_a + I_a + R_a)\,I_n}{N} - \lambda\, I_a - \varepsilon\, r\, I_a + \omega\, I_n,\\
\frac{dR_a}{dt} &= \frac{\alpha\,(S_a + I_a + R_a)\,R_n}{N} - \lambda\, R_a - \phi\,\delta\, R_a + \varepsilon\, r\, I_a.
\end{aligned}
\tag{2.1}
$$

Awareness is assumed to spread from the aware section of the population to the unaware at a rate α and to be lost at a rate λ. Besides this 'local' awareness associated with direct contacts between unaware and aware individuals, we also include a possibility of a general population-wide campaign aimed at reducing the impact of the disease by distributing information about this disease. Formally, this is represented in the model by direct transitions from each unaware population to

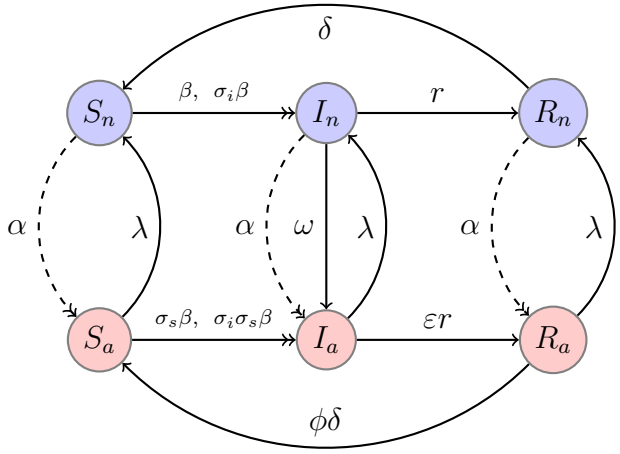

Figure 2.1: Diagram of transitions in model (2.1). Solid lines represent transitions associated with individuals. Arrow stands for "possible transition": doubled arrow head indicating processes subject to contacts on the disease (solid lines) or awareness (dash lines) networks, single head arrows indicate processes that are not subject to contact.

an associated aware population (e.g., from S_n to S_a) at a rate ω. With the above assumptions, the model for the simultaneous spread of disease and awareness takes the form

$$\begin{aligned}
\frac{dS_n}{dt} &= -\frac{(I_n+\sigma_i I_a)\,\beta\,S_n}{N} - \frac{\alpha\,(S_a+I_a+R_a)\,S_n}{N} + \lambda\,S_a + \delta\,R_n - \omega\,S_n,\\
\frac{dI_n}{dt} &= \frac{(I_n+\sigma_i I_a)\,\beta\,S_n}{N} - \frac{\alpha\,(S_a+I_a+R_a)\,I_n}{N} + \lambda\,I_a - r\,I_n - \omega\,I_n,\\
\frac{dR_n}{dt} &= -\frac{\alpha\,(S_a+I_a+R_a)\,R_n}{N} + \lambda\,R_a - \delta\,R_n + r\,I_n - \omega\,R_n,\\
\frac{dS_a}{dt} &= -\frac{(I_n+\sigma_i I_a)\,\sigma_s\,\beta\,S_a}{N} + \frac{\alpha\,(S_a+I_a+R_a)\,S_n}{N} - \lambda\,S_a + \phi\,\delta\,R_a + \omega\,S_n,\\
\frac{dI_a}{dt} &= \frac{(I_n+\sigma_i I_a)\,\sigma_s\,\beta\,S_a}{N} + \frac{\alpha\,(S_a+I_a+R_a)\,I_n}{N} - \lambda\,I_a - \varepsilon\,r\,I_a + \omega\,I_n,\\
\frac{dR_a}{dt} &= \frac{\alpha\,(S_a+I_a+R_a)\,R_n}{N} - \lambda\,R_a - \phi\,\delta\,R_a + \varepsilon\,r\,I_a + \omega\,R_n.
\end{aligned} \tag{2.2}$$

This model generalises model (2.1), by allowing the unaware susceptible and recovered populations to acquire information through a global awareness programme without the need for contacts with aware individuals. This provides a very important practical difference, since preventing the disease through an appropriate information programme is very effective and more economical than treating the disease once it has started spreading in the population. The system of equations (2.1) is represented by the model diagram shown in Figure 2.1, while Figure 2.2 shows the model diagram for (2.2) with all the transitions between different compartments.

Since model (2.2) does not include vital dynamics and there are no disease-

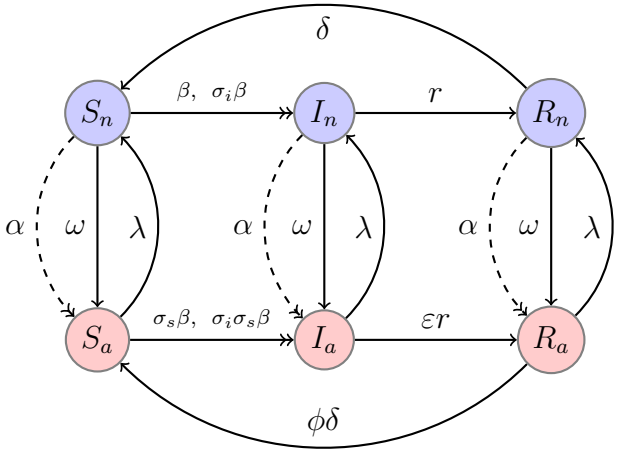

Figure 2.2: Model diagram: dynamics of transitions in model (2.2). Solid lines represent transitions associated with individuals. Arrow stands for "possible transition": doubled arrow head indicating processes subject to contacts on the disease (solid lines) or awareness (dash lines) networks, single head arrows indicate processes that are not subject to contact.

induced deaths, it implies that the total population $N(t) = N_n(t) + N_a(t) = N$ is constant, where $N_n(t) = S_n(t) + I_n(t) + R_n(t)$ and $N_a(t) = S_a(t) + I_a(t) + R_a(t)$ are total populations of unaware and aware individuals, respectively. It is easy to show that model (2.2) is well-posed, i.e. its solutions are non-negative for all $t \geq 0$.

Summing up the last three equations in (2.2) gives

$$\frac{dN_a}{dt} = \frac{\alpha\, N_a\, N_n}{N} - \lambda\, N_a + \omega\, N_n, \tag{2.3}$$

and using the fact that $N_n(t) = N - N_a(t)$ yields the following logistic-type equation for the total aware population

$$\begin{aligned} \frac{dN_a}{dt} &= \frac{\alpha\, N_a\, (N - N_a)}{N} - \lambda\, N_a + \omega\, (N - N_a) \\ &= b_v\, N_a \left(1 - \frac{N_a}{K}\right) + \omega\, N, \end{aligned} \tag{2.4}$$

where $b_v = \alpha - \lambda - \omega$ and $K = \frac{b_v\, N}{\alpha}$. Introducing a rescaled population $x_a = \frac{N_a}{K}$, it follows that

$$\frac{dx_a}{dt} = b_v\, x_a\, (1 - x_a) + b_s, \tag{2.5}$$

where $b_s = \frac{\omega\, N}{K} = \frac{\alpha\, \omega}{b_v}$. Solving this equation gives

$$x_a\,(t) = \frac{1}{2\, b_v} \left[b_v + \tanh\left(\frac{t\, \sqrt{4\, b_s\, b_v + b_v^2}}{2} + \frac{c_1\, \sqrt{4\, b_s\, b_v + b_v^2}}{2} \right) \sqrt{4\, b_s\, b_v + b_v^2} \right],$$

or, in terms of original variables,

$$N_a\left(t\right)=\frac{N}{2\,\alpha}\left[b_v+\tanh\left(\frac{t\,\sqrt{4\,b_s\,b_v+b_v^2}}{2}+\frac{c_1\,\sqrt{4\,b_s\,b_v+b_v^2}}{2}\right)\,\sqrt{4\,b_s\,b_v+b_v^2}\right].$$

At time, $t=0$, $N_a(0)=0$, therefore $c_1=\dfrac{-2\,\tanh^{-1}\left(\frac{b_v}{\sqrt{4\,b_s\,b_v+b_v^2}}\right)}{\sqrt{4\,b_s\,b_v+b_v^2}}$, and

$$N_a\left(t\right)=\frac{N}{2\,\alpha}\left[b_v+\tanh\left(\frac{t\sqrt{4\,b_s\,b_v+b_v^2}}{2}-\tanh^{-1}\left(\frac{b_v}{\sqrt{4\,b_s\,b_v+b_v^2}}\right)\right)\sqrt{4\,b_s\,b_v+b_v^2}\right],$$

and as $t\to\infty$, $N_a(t)\to N_a(\infty)=\frac{N}{2\,\alpha}\left(b_v+\sqrt{4\,b_s\,b_v+b_v^2}\right)$, since $\tanh(\infty)=1$. Equivalently, one can rewrite this as

$$\begin{aligned}\frac{N}{2\,\alpha}\left(b_v+\sqrt{4\,b_s\,b_v+b_v^2}\right)&=\frac{N}{2\,\alpha}\left(\alpha-\lambda-\omega+\sqrt{4\,\alpha\,\omega+(\alpha-\lambda-\omega)^2}\right)\\&=N\left[\frac{1}{2}\left(1-\frac{\lambda+\omega}{\alpha}\right)+\sqrt{\frac{1}{4}\left(1-\frac{\lambda+\omega}{\alpha}\right)^2+\frac{\omega}{\alpha}}\right]\\&=N\,h,\end{aligned}$$

where

$$h=\frac{1}{2}\left(1-\frac{\lambda+\omega}{\alpha}\right)+\sqrt{\frac{1}{4}\left(1-\frac{\lambda+\omega}{\alpha}\right)^2+\frac{\omega}{\alpha}}. \tag{2.6}$$

Hence, one can write

$$N_a(\infty)=N\,h\qquad\text{and}\qquad N_n(\infty)=N-N_a(\infty)=N\,(1-h),\qquad 0<h<1.$$

This implies that as $t\to\infty$, $N_n(t)$ and $N_a(t)$ tend to some constant values that only depend on the rates of gain of local and global awareness (α and ω) and the loss rate of awareness λ, but are independent of the initial conditions for individual populations or the characteristics of the disease, such as transmission rate, or durations of recovery or temporary immunity.

2.2 Steady states

Steady states with the absence of global awareness

As a first step in the analysis, we look at possible steady states of model (2.3). In the case when $\omega=0$, one possibility is a trivial case $N_a=S_a+I_a+R_a=0$, in

which case all state variables are zero except for $S_n = N$. Therefore, we have an equilibrium state $E_0 = (S_n^\circ, 0, 0, 0, 0, 0)$. This is a steady state free of disease and awareness.

Staying with the case $N_a = 0$ we consider next the second and the third equations of (2.2) for the case when $I_n \neq 0$, which gives

$$S_n = \frac{r\,N}{\beta} \qquad \text{and} \qquad R_n = \frac{r\,I_n}{\delta}.$$

Since $S_n + I_n + R_n = N - N_a = N \quad \Rightarrow \quad I_n + R_n = N - S_n,$ it follows therefore that

$$I_n = \frac{\delta\,N}{\delta + r}\left(1 - \frac{r}{\beta}\right), \qquad \text{and} \qquad R_n = \frac{r\,N}{\delta + r}\left(1 - \frac{r}{\beta}\right).$$

This gives a disease equilibrium state $E_1 = (S_n^*, I_n^*, R_n^*, 0, 0, 0)$ independent of awareness in the case when the global awareness is zero. We have

$$S_n^* = \frac{r\,N}{\beta}, \qquad I_n^* = \frac{\delta\,N}{\delta + r}\left(1 - \frac{r}{\beta}\right) \qquad \text{and} \qquad R_n^* = \frac{r\,N}{\delta + r}\left(1 - \frac{r}{\beta}\right),$$

and this steady state is biologically feasible provided $R_0^d = \frac{\beta}{r} > 1$.

Next, considering the case when $I_n = 0$ but $N_a > 0$, we have from the fifth equation in (2.2)

$$[\sigma_i\,\sigma_s\,\beta\,S_a - N\,(\lambda + \varepsilon\,r)]\,I_a = 0,$$

$$\Rightarrow \quad I_a = 0 \qquad \text{or} \qquad \sigma_i\,\sigma_s\,\beta\,S_a - N\,(\lambda + \varepsilon\,r) = 0,$$

and from the second equation

$$I_a = 0 \qquad \text{or} \qquad \frac{\sigma_i\,\beta\,S_n}{N} + \lambda = 0.$$

Since $\frac{\sigma_i\,\beta\,S_n}{N} + \lambda > 0$, hence $I_a = 0$, which implies $R_n = 0$ and $R_a = 0$. Now, the first and fourth equations in (2.2) reduce to

$$\frac{\alpha\,S_a\,S_n}{N} - \lambda\,S_a = 0,$$

which implies

$$S_a = 0 \qquad \text{or} \qquad S_n = N\,\frac{\lambda}{\alpha},$$

but, since in this case, $S_a > 0$, this yields

$$S_n = N\,\frac{\lambda}{\alpha} \qquad \text{and} \qquad S_a = N\left(1 - \frac{\lambda}{\alpha}\right) \qquad \text{with} \quad R_0^a = \frac{\alpha}{\lambda} > 1.$$

Therefore we have an awareness-endemic steady state $E_0^0 = (S_n^*, 0, 0, S_a^*, 0, 0)$ established only by local awareness (through contacts) in a disease-free population with $\omega = 0$,

$$S_n^* = N\,\frac{\lambda}{\alpha}, \qquad S_a^* = N\,\left(1 - \frac{\lambda}{\alpha}\right) \qquad \text{and} \qquad R_0^a > 1.$$

Steady states with global awareness

In the case where global awareness is present, i.e. $\omega > 0$, the disease-free steady state is actually an awareness-endemic equilibrium which involves both unaware and aware populations. Now, the first and the fourth equations of (2.2) reduce to

$$\frac{\alpha\, S_a\, S_n}{N} - \lambda\, S_a + \omega\, S_n = 0.$$

Substituting $S_n = N - S_a$ gives the following quadratic equation

$$S_a^2 - N\,\left(1 - \frac{\lambda + \omega}{\alpha}\right)\, S_a - \frac{\omega\, N^2}{\alpha} = 0,$$

with a single positive root

$$S_a = N\,\left[\frac{1}{2}\,\left(1 - \frac{\lambda+\omega}{\alpha}\right) + \sqrt{\frac{1}{4}\,\left(1 - \frac{\lambda+\omega}{\alpha}\right)^2 + \frac{\omega}{\alpha}}\right] = N\,h,$$

$$\Rightarrow \qquad S_a = N\,h \qquad \text{and} \qquad S_n = N - N\,h = N\,(1-h), \qquad 0 < h < 1.$$

Hence, we have an awareness-endemic equilibrium state $E_0^\omega = (S_n^*, 0, 0, S_a^*, 0, 0)$ with $\omega > 0$,

$$I_n^* = I_a^* = R_n^* = R_a^* = 0, \qquad S_a^* = N\,h, \qquad \text{and} \qquad S_n^* = N\,(1-h).$$

Earlier analysis has shown that as $t \to \infty$, N_a tends to a steady state value of $N\,h$. Thus, the steady states of (2.2) are determined from the following system of equations

$$0 = -\alpha\, h\, S_n - \frac{(I_n + \sigma_i\, I_a)\,\beta\, S_n}{N} + \lambda\, S_a + \delta\, R_n - \omega\, S_n, \tag{2.7}$$

$$0 = -\alpha\, h\, I_n + \frac{(I_n + \sigma_i\, I_a)\,\beta\, S_n}{N} + \lambda\, I_a - r\, I_n - \omega\, I_n, \tag{2.8}$$

$$0 = -\alpha\, h\, R_n + \lambda\, R_a - \delta\, R_n + r\, I_n - \omega\, R_n, \tag{2.9}$$

$$0 = \alpha\, h\, S_n - \frac{(I_n + \sigma_i\, I_a)\,\sigma_s\,\beta\, S_a}{N} - \lambda\, S_a + \phi\,\delta\, R_a + \omega\, S_n, \tag{2.10}$$

$$0 = \alpha\, h\, I_n + \frac{(I_n + \sigma_i\, I_a)\,\sigma_s\,\beta\, S_a}{N} - \lambda\, I_a - \varepsilon\, r\, I_a + \omega\, I_n, \tag{2.11}$$

$$0 = \alpha\, h\, R_n - \lambda\, R_a - \phi\,\delta\, R_a + \varepsilon\, r\, I_a + \omega\, R_n. \tag{2.12}$$

Adding equations (2.7) and (2.10), (2.8) and (2.11), (2.9) and (2.12) gives

$$\beta\,(I_n + \sigma_i\,I_a)\,(S_n + \sigma_i\,S_a) = N\,(\delta\,R_n + \phi\,\delta\,R_a), \tag{2.13}$$

$$\beta\,(I_n + \sigma_i\,I_a)\,(S_n + \sigma_i\,S_a) = N\,(r\,I_n + \varepsilon\,r\,I_a), \tag{2.14}$$

and

$$\delta\,R_n + \phi\,\delta\,R_a = r\,I_n + \varepsilon\,r\,I_a. \tag{2.15}$$

One can notice that, the equation (2.13) follows from (2.14) and (2.15). Solving equation (2.8) for I_a gives

$$I_a = \frac{[N\,(\alpha\,h + r + \omega) - \beta\,S_n]\;I_n}{N\,\lambda + \sigma_i\,\beta\,S_n},$$

and similarly from equation (2.11) one finds

$$I_a = \frac{[N\,(\alpha\,h + \omega) + \sigma_s\,\beta\,S_a]\;I_n}{N\,(\lambda + \varepsilon\,r) - \sigma_i\,\sigma_s\,\beta\,S_a}.$$

Equating these two expressions for I_a gives

$$\frac{[N\,(\alpha\,h + r + \omega) - \beta\,S_n]\;I_n}{N\,\lambda + \sigma_i\,\beta\,S_n} = \frac{[N\,(\alpha\,h + \omega) + \sigma_s\,\beta\,S_a]\;I_n}{N\,(\lambda + \varepsilon\,r) - \sigma_i\,\sigma_s\,\beta\,S_a},$$

which implies

$$S_a = \frac{N\,[\lambda\,r + \varepsilon\,r\,(\alpha\,h + r + \omega)] - \beta\,[\sigma_i\,(\alpha\,h + \omega) + \lambda + \varepsilon\,r]\,S_n}{\beta\,\sigma_s\,[\sigma_i\,(\alpha\,h + r + \omega) + \lambda]}.$$

In a similar way, we solve equations (2.9) and (2.12) to find

$$R_a = \frac{(\alpha\,h + \delta + \omega)\,R_n - r\,I_n}{\lambda} \qquad \text{and} \qquad R_n = \frac{\lambda\,R_a + r\,I_n}{\alpha\,h + \delta + \omega},$$

and

$$R_a = \frac{(\alpha\,h + \omega)\,R_n + \varepsilon\,r\,I_a}{\lambda + \phi\,\delta} \qquad \text{and} \qquad R_n = \frac{(\lambda + \phi\,\delta)\,R_a - \varepsilon\,r\,I_a}{\alpha\,h + \omega}.$$

Equating the two expressions for R_n yields

$$R_a = \frac{r\,(\alpha\,h + \omega)\,I_n + \varepsilon\,r\,(\alpha\,h + \delta + \omega)\,I_a}{\lambda\,\delta + \phi\,\delta\,(\alpha\,h + \delta + \omega)},$$

whereas, equating the expressions for R_a gives

$$R_n = \frac{r\,(\lambda + \phi\,\delta)\,I_n + \lambda\,\varepsilon\,r\,I_a}{\lambda\,\delta + \phi\,\delta\,(\alpha\,h + \delta + \omega)}.$$

Introducing the auxiliary parameters

$m_1 = \varepsilon\,r\,(\alpha\,h + \delta + \omega)$, $m_2 = \lambda\,\delta + \phi\,\delta\,(\alpha\,h + \delta + \omega)$, $m_3 = \lambda\,r + \varepsilon\,r\,(\alpha\,h + r + \omega)$,

$m_4 = \beta\,[\sigma_i\,(\alpha\,h + \omega) + \lambda + \varepsilon\,r], \quad m_5 = \beta\,\sigma_s\,[\sigma_i\,(\alpha\,h + r + \omega) + \lambda],$

$m_6 = N\,(\alpha\,h + r + \omega) - \beta\,S_n, \quad m_7 = N\,\lambda + \sigma_i\,\beta\,S_n,$

we have

$$I_a = \frac{m_6\,I_n}{m_7} \qquad \Rightarrow \qquad I_n = \frac{m_7\,I_a}{m_6},$$

$$S_a = \frac{N\,m_3 - m_4\,S_n}{m_5}, \tag{2.16}$$

$$R_a = \frac{[r\,(\alpha\,h + \omega)\,m_7 + m_1\,m_6]\,I_a}{m_2\,m_6}, \tag{2.17}$$

$$R_n = \frac{[r\,(\lambda + \phi\,\delta)\,m_7 + \lambda\,\varepsilon\,r\,m_6]\,I_n}{m_2\,m_7}, \tag{2.18}$$

this implies

$$S_n < \min\left\{\frac{N\,m_3}{m_4}, \quad \frac{N\,(\alpha\,h + r + \omega)}{\beta}\right\}.$$

We recall that $S_a + I_a + R_a = N_a = N\,h$ and $S_n + I_n + R_n = N_n = N - N_a = N(1-h)$, which gives

$$I_a + R_a = N\,h - S_a,$$

$$\Rightarrow \qquad I_a = \frac{m_2\,m_6\,[N\,(m_5\,h - m_3) + m_4\,S_n]}{m_5\,[m_6\,(m_1 + m_2) + r\,(\alpha\,h + \omega)\,m_7]}. \tag{2.19}$$

Hence,

$$S_n > \frac{N\,(m_3 - m_5\,h)}{m_4},$$

and

$$I_n = \frac{m_2\,m_7\,[N\,(1-h) - S_n]}{m_2\,m_7 + r\,(\lambda + \phi\,\delta)\,m_7 + \lambda\,\varepsilon\,r\,m_6}. \tag{2.20}$$

Consequently,

$$\frac{N\,(m_3 - m_5\,h)}{m_4} < S_n < \min\left\{\frac{N\,m_3}{m_4}, \quad \frac{N\,(\alpha\,h + r + \omega)}{\beta}, \quad N\,(1-h)\right\}.$$

Equating (2.20) with

$$I_n = \frac{m_7\,I_a}{m_6} = \frac{m_2\,m_7\,[N\,(m_5\,h - m_3) + m_4\,S_n]}{m_5\,[m_6\,(m_1 + m_2) + r\,(\alpha\,h + \omega)\,m_7]}$$

gives the following quadratic equation

$$x_1\,S_n^2 - x_2\,S_n + x_3 = 0,$$

where

$x_1 = \beta\,[m_4\,\lambda\,\varepsilon\,r + m_5\,(m_1 + m_2)] - \beta\,\sigma_i\,[m_4\,(m_2 + \lambda\,r + r\,\phi\,\delta) + r\,(\alpha\,h + \omega)\,m_5],$

$$x_2 = N\Big(m_4\,\lambda\,(m_2+m_3+r\,\phi\,\delta)+m_5\,(m_1+m_2)\,[\beta\,(1-h)+\alpha\,h+r+\omega]+$$
$$r\,(\alpha\,h+\omega)\,m_5\,[\lambda-\sigma_i\,\beta\,(1-h)]-\beta\,(m_3-m_5\,h)\,[\sigma_i\,(m_2+\lambda\,r+r\,\phi\,\delta)-\lambda\,\varepsilon\,r]\Big),$$
$$x_3 = N^2\Big[m_5\,(1-h)\,[(\alpha\,h+r+\omega)\,(m_1+m_2)+\lambda\,r\,(\alpha\,h+\omega)]+$$
$$\lambda\,(m_3-m_5\,h)\,(m_2+m_3+r\,\phi\,\delta)\Big].$$

Thus, the solution to the quadratic equation is

$$S_n = \frac{x_2 \pm \sqrt{x_2^2-4\,x_1\,x_3}}{2\,x_1}, \tag{2.21}$$

and it must lie within the interval

$$\frac{N\,(m_3-m_5\,h)}{m_4} < S_n < \min\left\{\frac{N\,m_3}{m_4},\quad \frac{N\,(\alpha\,h+r+\omega)}{\beta},\quad N\,(1-h)\right\}.$$

Therefore, we obtain an endemic equilibrium state $E_2^{\omega}=(S_n^*,I_n^*,R_n^*,S_a^*,I_a^*,R_a^*)$, with $\omega\geq 0$ and

$$S_n^* = \frac{x_2 \pm \sqrt{x_2^2-4\,x_1\,x_3}}{2\,x_1}, \qquad I_n^* = \frac{m_2\,m_7\,[N\,(1-h)-S_n^*]}{m_2\,m_7+r\,(\lambda+\phi\,\delta)\,m_7+\lambda\,\varepsilon\,r\,m_6},$$
$$R_n^* = \frac{[r\,(\lambda+\phi\,\delta)\,m_7+\lambda\,\varepsilon\,r\,m_6]\,[N\,(1-h)-S_n^*]}{m_2\,m_7+r\,(\lambda+\phi\,\delta)\,m_7+\lambda\,\varepsilon\,r\,m_6},$$
$$S_a^* = \frac{N\,m_3-m_4\,S_n^*}{m_5}, \qquad I_a^* = \frac{m_2\,m_6\,[N\,(m_5\,h-m_3)+m_4\,S_n^*]}{m_5\,[m_6\,(m_1+m_2)+r\,(\alpha\,h+\omega)\,m_7]},$$
$$R_a^* = \frac{[r\,(\alpha\,h+\omega)\,m_7+m_1\,m_6]\,[N\,(m_5\,h-m_3)+m_4\,S_n^*]}{m_5\,[m_6\,(m_1+m_2)+r\,(\alpha\,h+\omega)\,m_7]}.$$

This steady state is only feasible when the value(s) of S_n^* lie within the interval

$$\frac{N\,(m_3-m_5\,h)}{m_4} < S_n^* < \min\left\{\frac{N\,m_3}{m_4},\frac{N\,(\alpha\,h+r+\omega)}{\beta},N\,(1-h)\right\}.$$

In conclusion, the model (2.2) has the following steady states

$$E_0=(S_n^\circ,0,0,0,0,0),\quad E_1=(S_n^*,I_n^*,R_n^*,0,0,0),\quad \text{for}\quad \omega=0,$$

$$E_0^{\omega}=(S_n^*,0,0,S_a^*,0,0)\quad\text{and}\quad E_2^{\omega}=(S_n^*,I_n^*,R_n^*,S_a^*,I_a^*,R_a^*),\quad\text{for}\quad\omega\geq 0.$$

2.3 Stability analysis

Stability of steady states independent of global awareness

To analyse the stability of the different steady states, we again start by considering the case with $\omega = 0$ and linearise the system (2.2) near the disease-free steady state E_0. This gives the Jacobian matrix

$$J_0 = \begin{pmatrix} -\omega & -\beta & \delta & -\alpha+\lambda & -\alpha-\sigma_i\,\beta & -\alpha \\ 0 & \beta-r-\omega & 0 & 0 & \sigma_i\,\beta+\lambda & 0 \\ 0 & r & -\delta-\omega & 0 & 0 & \lambda \\ \omega & 0 & 0 & \alpha-\lambda & \alpha & \alpha+\phi\,\delta \\ 0 & \omega & 0 & 0 & -\lambda-\varepsilon\,r & 0 \\ 0 & 0 & \omega & 0 & \varepsilon\,r & -\lambda-\phi\,\delta \end{pmatrix},$$

with the characteristic equation for eigenvalues k, which can be factorised as follows

$$k\,(k-\beta+r)\,(k+\delta)\,(k-\alpha+\lambda)\,(k+\lambda+\varepsilon\,r)\,(k+\lambda+\phi\,\delta) = 0.$$

Therefore,

$k_1 = 0, \quad k_2 = \beta - r, \quad k_3 = -\delta, \quad k_4 = \alpha - \lambda, \quad k_5 = -\lambda - \varepsilon\,r, \quad k_6 = -\lambda - \phi\,\delta,$

suggesting that the steady state E_0 is linearly asymptotically stable, provided

$$\beta < r \qquad \text{and} \qquad \alpha < \lambda,$$

or, equivalently if

$$R_0^d = \frac{\beta}{r} < 1, \qquad R_0^a = \frac{\alpha}{\lambda} < 1.$$

Similarly, one can show that the endemic steady state $E_1 = (S_n^*, I_n^*, R_n^*, 0, 0, 0)$, with $S_a^* = I_a^* = R_a^* = 0$ and $S_n^* > 0, I_n^* > 0, R_n^* > 0$ is linearly stable whenever $R_0^a < 1$ and $R_0^d > 1$, which coincides with the conditions for feasibility of this steady state. Since this steady state is independent of awareness, one can substitute $\sigma_s = \sigma_i = \varepsilon = \phi = 1$ [16], which simplifies the Jacobian to

$$J_n = \begin{pmatrix} -a_1 & -a_2 & \delta & -\tilde{a}_2+\lambda & -\tilde{a}_2-a_2 & -\tilde{a}_2 \\ a_1 & a_2-r & 0 & -\tilde{a}_1 & -\tilde{a}_1+a_2+\lambda & -\tilde{a}_1 \\ 0 & r & -\delta & -a_3 & -a_3 & -a_3+\lambda \\ 0 & 0 & 0 & \tilde{a}_2-a_1-\lambda & \tilde{a}_2 & \tilde{a}_2+\delta \\ 0 & 0 & 0 & \tilde{a}_1+a_1 & \tilde{a}_1-\lambda-r & \tilde{a}_1 \\ 0 & 0 & 0 & a_3 & a_3+r & a_3-\lambda-\delta \end{pmatrix},$$

with $a_1 = \frac{\beta I_n^*}{N}$, $\tilde{a}_1 = \frac{\alpha I_n^*}{N}$, $a_2 = \frac{\beta S_n^*}{N}$, $\tilde{a}_2 = \frac{\alpha S_n^*}{N}$, $a_3 = \frac{\alpha R_n^*}{N}$. The characteristic equation has the form

$$k\,(k+\lambda-\tilde{a}_2-\tilde{a}_1-a_3)\big[k^2+k\,(a_1+\delta+2\,\lambda+r)+(a_1+\lambda)(\delta+r+\lambda)+\delta\,r\big]\times$$
$$\big[k^2+k\,(\delta+r+a_1-a_2)+\delta\,(a_1-a_2)+r\,(\delta+a_1)\big]=0,$$

with the eigenvalues,

$k_1 = 0$,

$k_2 = \tilde{a}_2+\tilde{a}_1+a_3-\lambda \quad \Rightarrow \quad k_2 = \frac{\alpha S_n^*}{N}+\frac{\alpha I_n^*}{N}+\frac{\alpha R_n^*}{N}-\lambda = \alpha-\lambda$, and

$$k_2<0 \quad \text{if} \quad \alpha<\lambda \qquad \Rightarrow \quad R_0^a<1.$$

From $k^2+k\,(a_1+\delta+2\,\lambda+r)+(a_1+\lambda)\,(\delta+r+\lambda)+\delta\,r=0$ we have $k_{3,4}<0$, and from $k^2+k\,(\delta+r+a_1-a_2)+\delta\,(a_1-a_2)+r\,(\delta+a_1)=0$ we obtain

$$k^2+k\,(\delta+a_1)+a_1\,(\delta+r)=0,$$

since

$$S_n^*=\frac{r\,N}{\beta} \quad \text{and} \quad I_n^*=\frac{\delta\,N}{\delta+r}\left(1-\frac{r}{\beta}\right),$$
$$\Rightarrow \qquad a_1=\frac{\beta\,I_n^*}{N}=\frac{\beta\,\delta}{\delta+r}\left(1-\frac{r}{\beta}\right) \qquad \text{and} \qquad a_2=\frac{\beta\,S_n^*}{N}=r,$$

hence $k_{5,6}<0$ if $a_1>0$, which is equivalent to $R_0^d>1$.

In the case where the spread of the disease is dependent on the spread of only local awareness, i.e. in the absence of global awareness ($\omega=0$), one has the equilibrium state $E_0^0=(S_n^*,0,0,S_a^*,0,0)$ with $I_n^*=I_a^*=R_n^*=R_a^*=0$ and $S_n^*=N\,\frac{\lambda}{\alpha}$, $S_a^*=N\,\left(1-\frac{\lambda}{\alpha}\right)$. Linearisation near this steady state yields the Jacobian

$$J_s^\circ=\begin{pmatrix} -a_0 & -a_2 & \delta & -\tilde{a}_2+\lambda & -\tilde{a}_2-\hat{a}_2 & -\tilde{a}_2 \\ 0 & -a_0+a_2-r & 0 & 0 & \hat{a}_2+\lambda & 0 \\ 0 & r & -a_0-\delta & 0 & 0 & \lambda \\ a_0 & -\tilde{a}_0 & 0 & \tilde{a}_2-\lambda & \tilde{a}_2-\hat{a}_0 & \tilde{a}_2+\phi\,\delta \\ 0 & a_0+\tilde{a}_0 & 0 & 0 & \hat{a}_0-\lambda-\varepsilon\,r & 0 \\ 0 & 0 & a_0 & 0 & \varepsilon\,r & -\lambda-\phi\,\delta \end{pmatrix},$$

with the characteristic equation

$$k\,(a_0-\tilde{a}_2+\lambda+k)[k^2+k\,(\lambda+\phi\,\delta+a_0+\delta)+\lambda\,\delta+\phi\,\delta\,(a_0+\delta)]\times$$
$$[k^2+k\,(\lambda+\varepsilon\,r-\hat{a}_0+a_0+r-a_2)+(\lambda+\varepsilon\,r-\hat{a}_0)\,(a_0+r-a_2)-(a_0+\tilde{a}_0)\,(\hat{a}_2+\lambda)]=0.$$

where $a_0 = \frac{\alpha S_a^*}{N}$, $\tilde{a}_0 = \frac{\sigma_s \beta S_a^*}{N}$, $\hat{a}_0 = \frac{\sigma_i \sigma_s \beta S_a^*}{N}$, $a_2 = \frac{\beta S_n^*}{N}$, $\tilde{a}_2 = \frac{\alpha S_n^*}{N}$, $\hat{a}_2 = \frac{\sigma_i \beta S_n^*}{N}$.

Hence, we obtain

$k_1 = 0,$

$k_2 = \tilde{a}_2 - a_0 - \lambda,$

which is negative as long as $\frac{\alpha}{\lambda} > 1$, and since $R_0^a = \frac{\alpha}{\lambda} > 1$ is the feasibility condition of this steady state, this means that $k_2 < 0$.

The roots $k_{3,4}$ of the equation $k^2 + k\,(\lambda + \phi\,\delta + a_0 + \delta) + \lambda\,\delta + \phi\,\delta\,(a_0 + \delta) = 0$ always have a negative real part, while from the equation

$$k^2 + k\,(\lambda + \varepsilon\,r - \hat{a}_0 + a_0 + r - a_2) + (\lambda + \varepsilon\,r - \hat{a}_0)\,(a_0 + r - a_2) - (a_0 + \tilde{a}_0)\,(\hat{a}_2 + \lambda) = 0$$

we have $k_{5,6} < 0$ if

$$\lambda + \varepsilon\,r - \hat{a}_0 + a_0 + r - a_2 > 0 \quad \text{and} \quad (\lambda + \varepsilon\,r - \hat{a}_0)\,(a_0 + r - a_2) > (a_0 + \tilde{a}_0)\,(\hat{a}_2 + \lambda).$$

Since $(a_0 + \tilde{a}_0)\,(\hat{a}_2 + \lambda) > 0$ it follows that $(\lambda + \varepsilon\,r - \hat{a}_0)\,(a_0 + r - a_2) > 0$. Therefore, $\lambda + \varepsilon\,r - \hat{a}_0 + a_0 + r - a_2 > 0$ if and only if $\lambda + \varepsilon\,r - \hat{a}_0 > 0$ and $a_0 + r - a_2 > 0$. Consequently, we have the conditions for stability as:

i) $\lambda + \varepsilon\,r - \hat{a}_0 > 0 \quad \Rightarrow \quad \alpha\,(\lambda + \varepsilon\,r) + \lambda\,\sigma_i\,\sigma_s\,\beta > \alpha\,\sigma_i\,\sigma_s\,\beta,$

ii) $a_0 + r - a_2 > 0 \quad \Rightarrow \quad \alpha\,(\alpha + r) > \lambda\,(\beta + \alpha),$

iii) $(\lambda + \varepsilon\,r - \hat{a}_0)\,(a_0 + r - a_2) > (a_0 + \tilde{a}_0)\,(\hat{a}_2 + \lambda)$

$$\Rightarrow \quad \alpha\,(\lambda + \varepsilon\,r)\,(\alpha - \lambda + r) > \sigma_i\,\sigma_s\,\beta\,(\alpha - \lambda)(\alpha - \lambda + r) + \beta\,\lambda\,(\lambda + \varepsilon\,r) + \lambda\,(\alpha - \lambda)\,(\alpha + \sigma_s\,\beta + \sigma_i\,\beta).$$

This implies

$$\alpha\,(\lambda + \varepsilon\,r)\,(\alpha - \lambda + r) > \sigma_i\,\sigma_s\,\beta\,(\alpha - \lambda)(\alpha - \lambda + r),$$

which can be recast as

$$\alpha\,(\lambda + \varepsilon\,r) + \lambda\,\sigma_i\,\sigma_s\,\beta > \alpha\,\sigma_i\,\sigma_s\,\beta.$$

Similarly, we have

$$\alpha\,(\lambda + \varepsilon\,r)\,(\alpha - \lambda + r) > \beta\,\lambda\,(\lambda + \varepsilon\,r),$$

$$\Rightarrow \qquad \alpha\,(\alpha + r) > \lambda\,(\beta + \alpha).$$

This shows that conditions (i) and (ii) are always satisfied when the condition (iii) is satisfied. Consequently, the single condition for stability is

$$\alpha\, r\,[\lambda+\varepsilon\,(\alpha-\lambda+r)] > \beta\,(\alpha-\lambda)\,[\sigma_i\,\sigma_s\,(\alpha-\lambda+r)+\lambda\,(\sigma_s+\sigma_i)]+\beta\,\lambda\,(\lambda+\varepsilon\,r),$$

which can be rewritten as

$$\frac{\beta}{r} < \frac{\alpha\,[\lambda+\varepsilon\,(\alpha-\lambda+r)]}{(\alpha-\lambda)\,[\sigma_i\,\sigma_s\,(\alpha-\lambda+r)+\lambda\,(\sigma_s+\sigma_i)]+\lambda\,(\lambda+\varepsilon\,r)},$$

or $R_0^d < \varphi_\circ$ with

$$\varphi_\circ = \frac{\alpha\,[\lambda+\varepsilon\,(\alpha-\lambda+r)]}{(\alpha-\lambda)\,[\sigma_i\,\sigma_s\,(\alpha-\lambda+r)+\lambda\,(\sigma_s+\sigma_i)]+\lambda\,(\lambda+\varepsilon\,r)}.$$

Therefore, the awareness-endemic state E_0^0 is linearly stable provided

$$R_{\varphi_\circ}^d = \frac{1}{\varphi_\circ}\,R_0^d < 1.$$

Stability of steady states with global awareness

Considering stability of the awareness-endemic steady state $E_0^\omega = (S_n^*, 0, 0, S_a^*, 0, 0)$ with $I_n^* = I_a^* = R_n^* = R_a^* = 0$, $S_a^* = N\,h$, and $S_n^* = N(1-h)$, we introduce an auxiliary parameter $a_s = \frac{\alpha\,S_a^*}{N} + \omega$, and obtain the Jacobian matrix

$$J_s = \begin{pmatrix} -a_s & -a_2 & \delta & -\tilde{a}_2+\lambda & -\tilde{a}_2-\hat{a}_2 & -\tilde{a}_2 \\ 0 & -a_s+a_2-r & 0 & 0 & \hat{a}_2+\lambda & 0 \\ 0 & r & -a_s-\delta & 0 & 0 & \lambda \\ a_s & -\tilde{a}_0 & 0 & \tilde{a}_2-\lambda & \tilde{a}_2-\hat{a}_0 & \tilde{a}_2+\phi\,\delta \\ 0 & a_s+\tilde{a}_0 & 0 & 0 & \hat{a}_0-\lambda-\varepsilon\,r & 0 \\ 0 & 0 & a_s & 0 & \varepsilon\,r & -\lambda-\phi\,\delta \end{pmatrix},$$

with the characteristics equation

$$k\,(a_s-\tilde{a}_2+\lambda+k)\left[k^2+k\,(\lambda+\phi\,\delta+a_s+\delta)+\lambda\,\delta+\phi\,\delta\,(a_s+\delta)\right]\times$$
$$\left[k^2+k\,(\lambda+\varepsilon\,r-\hat{a}_0+a_s+r-a_2)+(\lambda+\varepsilon\,r-\hat{a}_0)\,(a_s+r-a_2)-(a_s+\tilde{a}_0)\,(\hat{a}_2+\lambda)\right]=0,$$

which gives,

$k_1 = 0$,

$k_2 = \tilde{a}_2 - a_s - \lambda$,

which is negative, provided $\tilde{a}_2 < a_s+\lambda$, or equivalently, $\frac{\alpha\,S_n^*}{N} < \frac{\alpha\,S_a^*}{N} + \omega + \lambda$,

but since $S_a^* = N\,h$ and $S_n^* = N\,(1-h)$, the above inequality takes the form $\alpha\,(1-h) < \alpha\,h + \omega + \lambda$, which means

$$\frac{\lambda+\omega}{\alpha} > 1 - 2\,h \qquad \Rightarrow \qquad h > \tfrac{1}{2}\,\left(1 - \tfrac{\lambda+\omega}{\alpha}\right).$$

From the expression for h in (2.6), it follows that this condition is always satisfied, hence $k_2 < 0$.

By the Routh-Hurwitz criterion, the roots of the equation

$$k^2 + k\,(\lambda + \phi\,\delta + a_s + \delta) + \lambda\,\delta + \phi\,\delta\,(a_s + \delta) = 0$$

always have negative real part, and from

$$k^2 + k\,(\lambda + \varepsilon\,r - \hat{a}_0 + a_s + r - a_2) + (\lambda + \varepsilon\,r - \hat{a}_0)\,(a_s + r - a_2) - (a_s + \tilde{a}_0)\,(\hat{a}_2 + \lambda) = 0$$

we obtain $k_{5,6} < 0$ if

$$\lambda + \varepsilon\,r - \hat{a}_0 + a_s + r - a_2 > 0 \quad \text{and} \quad (\lambda + \varepsilon\,r - \hat{a}_0)\,(a_s + r - a_2) > (a_s + \tilde{a}_0)\,(\hat{a}_2 + \lambda).$$

Since $(a_s + \tilde{a}_0)\,(\hat{a}_2 + \lambda) > 0$, this implies that $(\lambda + \varepsilon\,r - \hat{a}_0)\,(a_s + r - a_2) > 0$. Hence, $\lambda + \varepsilon\,r - \hat{a}_0 + a_s + r - a_2 > 0$ if and only if $\lambda + \varepsilon\,r - \hat{a}_0 > 0$ and $a_s + r - a_2 > 0$.

Therefore, we have the conditions for stability as follows:

i) $\lambda + \varepsilon\,r - \hat{a}_0 > 0 \quad \Rightarrow \quad \lambda + \varepsilon\,r > \sigma_i\,\sigma_s\,\beta\,h,$

ii) $a_s + r - a_2 > 0 \quad \Rightarrow \quad \alpha\,h + \omega + r > \beta\,(1-h),$

iii) $(\lambda + \varepsilon\,r - \hat{a}_0)\,(a_s + r - a_2) > (a_s + \tilde{a}_0)\,(\hat{a}_2 + \lambda),$

$$\Rightarrow \quad (\lambda + \varepsilon\,r)\,(\alpha\,h + \omega + r) > \sigma_i\,\sigma_s\,\beta\,h\,(\alpha\,h + \omega + r) + \beta\,(1-h)\,(\lambda + \varepsilon\,r) + \lambda\,(\alpha\,h + \omega + \sigma_s\,\beta\,h) + \sigma_i\,\beta\,(1-h)\,(\alpha\,h + \omega). \quad (2.22)$$

If the inequality (2.22) holds, it implies

$$(\lambda + \varepsilon\,r)\,(\alpha\,h + \omega + r) > \sigma_i\,\sigma_s\,\beta\,h\,(\alpha\,h + \omega + r),$$

$$\Rightarrow \qquad \lambda + \varepsilon\,r > \sigma_i\,\sigma_s\,\beta\,h,$$

and similarly,

$$(\lambda + \varepsilon\,r)\,(\alpha\,h + \omega + r) > \beta\,(1-h)\,(\lambda + \varepsilon\,r),$$

$$\Rightarrow \qquad \alpha\,h + \omega + r > \beta\,(1-h).$$

Hence, conditions (i) and (ii) are always satisfied if the condition (iii) is satisfied. The condition for stability is thus given by a single inequality (2.22) which can be rewritten as follows

$$\frac{\beta}{r} < \frac{\lambda + \varepsilon\,(\alpha\,h + r + \omega)}{(1-h)\,[\sigma_i\,(\alpha\,h + \omega) + \lambda + \varepsilon\,r] + h\,\sigma_s\,[\sigma_i\,(\alpha\,h + r + \omega) + \lambda]}.$$

Let

$$\varphi = \frac{\lambda + \varepsilon\,(\alpha\,h + r + \omega)}{(1-h)\,[\sigma_i\,(\alpha\,h + \omega) + \lambda + \varepsilon\,r] + h\,\sigma_s\,[\sigma_i\,(\alpha\,h + r + \omega) + \lambda]} \quad \Rightarrow \quad R_0^d < \varphi.$$

Therefore, the awareness-endemic state $E_0^\omega = (S_n^*, 0, 0, S_a^*, 0, 0)$ with $\omega > 0$ is linearly stable if

$$R_\varphi^d = \frac{1}{\varphi}\,R_0^d < 1.$$

To determine the stability of the steady state $E_2^\omega = (S_n^*, I_n^*, R_n^*, S_a^*, I_a^*, R_a^*)$ with $S_n^* > 0,\ I_n^* > 0,\ R_n^* > 0,\ S_a^* > 0,\ I_a^* > 0,\ R_a^* > 0$ for $\omega \geq 0$ we introduce auxiliary parameters $\tilde{a}_s = \alpha\,h + \omega,\ a_4 = \frac{(I_n^* + \sigma_i\,I_a^*)\,\beta}{N},\ \tilde{a}_4 = \frac{(I_n^* + \sigma_i\,I_a^*)\,\sigma_s\,\beta}{N}$, and obtain the Jacobian matrix

$$J_a = \begin{pmatrix} -\tilde{a}_s - a_4 & -a_2 & \delta & \lambda & -\hat{a}_2 & 0 \\ a_4 & -\tilde{a}_s + a_2 - r & 0 & 0 & \hat{a}_2 + \lambda & 0 \\ 0 & r & -\tilde{a}_s - \delta & 0 & 0 & \lambda \\ \tilde{a}_s & -\tilde{a}_0 & 0 & -\tilde{a}_4 - \lambda & -\hat{a}_0 & \phi\,\delta \\ 0 & \tilde{a}_s + \tilde{a}_0 & 0 & \tilde{a}_4 & \hat{a}_0 - \lambda - \varepsilon\,r & 0 \\ 0 & 0 & \tilde{a}_s & 0 & \varepsilon\,r & -\lambda - \phi\,\delta \end{pmatrix},$$

with the associated characteristic equation

$$k\,(k + \tilde{a}_s + \lambda)(k^4 + P_1\,k^3 + P_2\,k^2 + P_3\,k + P_4) = 0, \tag{2.23}$$

where

$$P_1 = x_4 + x_5 + x_6 + \lambda,$$

$$P_2 = \delta(\phi\delta + \phi\tilde{a}_s + \lambda) + (x_4 + x_5)(\lambda + x_6) + r(a_4 + \tilde{a}_4\varepsilon) + x_4\,x_5 - (\tilde{a}_s + \tilde{a}_0)(\lambda + \hat{a}_2),$$

$$P_3 = \delta\,(\phi\,\delta + \phi\,\tilde{a}_s + \lambda)\,(x_4 + x_5) + (\lambda + x_6)\,(x_4\,x_5 - (\tilde{a}_s + \tilde{a}_0)\,(\lambda + \hat{a}_2)) + r\,a_4\,(x_4 + \phi\,\delta + \lambda) + \tilde{a}_4\,\varepsilon\,r\,(x_5 + \tilde{a}_s + \delta),$$

$$P_4 = \delta(\phi\,\delta + \phi\,\tilde{a}_s + \lambda)[x_4\,x_5 - (\tilde{a}_s + \tilde{a}_0)(\lambda + \hat{a}_2)] + r\,[a_4\,x_4(\lambda + \phi\,\delta) + \tilde{a}_4\,\tilde{a}_s(\lambda + \hat{a}_2)] + \varepsilon\,r\,(a_4\,\tilde{a}_4\,r + a_4\,\lambda\,(\tilde{a}_s + \tilde{a}_0) + x_5\,\tilde{a}_4\,(\delta + \tilde{a}_s)),$$

and

$$x_4 = \varepsilon\, r + \tilde{a}_4 + \lambda - \hat{a}_0, \quad x_5 = \tilde{a}_s + a_4 + r - a_2 \quad \text{and} \quad x_6 = \phi\,\delta + \delta + \tilde{a}_s.$$

Two of the eigenvalues of the characteristic equation (2.23) can be readily found as $k = 0$ and $k = -(\alpha h + \omega + \lambda)$, so the stability of the endemic steady state E_2^ω is determined by the roots of the quartic

$$k^4 + P_1\, k^3 + P_2\, k^2 + P_3\, k + P_4 = 0.$$

Using the Routh-Hurwitz criterion, one can conclude that the steady state E_2^ω with $\omega \geq 0$ is linearly asymptotically stable if and only if the following conditions hold [6, 12, 33].

$$P_4 > 0, \quad P_1 > 0, \quad P_2 > 0 \quad \text{and} \quad P_3\,(P_1\,P_2 - P_3) > P_1^2\,P_4. \tag{2.24}$$

Figures 2.3, 2.4 and 2.5 illustrate how the stability of different steady states varies with parameters. All of these Figures indicate that the endemic steady state is only biologically feasible and stable in the parameter region where the disease-free steady state is unstable. The region of stability of the disease-free steady state increases with α and ω, implying that increasing awareness allows disease eradication and prevents establishment of some steady levels of disease even for higher values of the disease transmission rate β. Similar effect is observed by increasing the recovery rate r, whereby disease is eradicated not so much through the spread of awareness, as due to the fact that infected individuals recover faster than they are able to spread the infection. Increasing the rate λ of awareness loss naturally has the opposite effect of increasing the parameter region where the endemic steady state is biologically feasible and stable.

2.4 Effects of awareness on system dynamics

In order to get a better understanding of relative effects of different aspects of awareness on determining the stability of different steady states and eventual evolution of the system, we fix three of the four parameters, σ_s, σ_i, ε and ϕ, to be equal to one, and allow one of them to vary to individually investigate the effect it has on the disease propagation. Qualitative behaviour is similar in all cases considered below

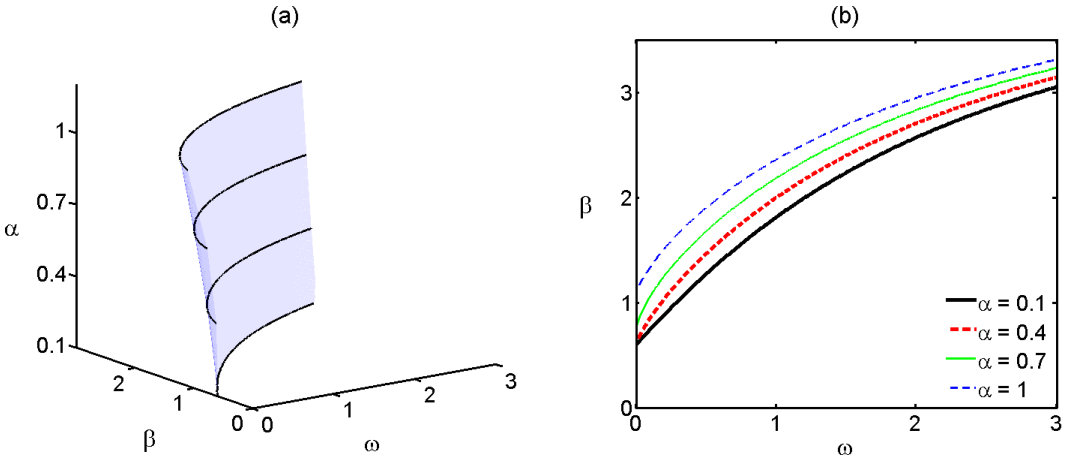

Figure 2.3: Existence and stability of different steady states. The disease-free steady state is stable to the right of the surface in (a) and below each curve in (b), and in these parameter regions the endemic steady state is not feasible. To the left of the surface in (a) and above each curve in (b), the disease-free steady state is unstable, while the endemic steady state exists and is stable. Parameter values are $\lambda = 0.6$, $r = 0.6$, $\sigma_i = 0.5$, $\sigma_s = 0.5$, $\phi = 0.3$, $\varepsilon = 2$, $\delta = 0.4$.

in that in the absence of global awareness ($\omega = 0$), the epidemic threshold is $R_0^d > 1$ for $R_0^a < 1$, and $R_0^d > \varphi_\circ$ with $\varphi_\circ = \varphi(\omega = 0)$ for $R_0^a > 1$, whereas for $\omega > 0$, it is given by $R_0^d > \varphi$ regardless of the value of R_0^a. In the case of $\omega = 0$ and $R_0^a < 1$, the disease is established in the form of a stable disease-endemic steady state E_1, while for $R_0^a > 1$, and for $\omega > 0$ and any value of R_0^a, the system settles on the stable endemic equilibrium E_2^ω.

2.4.1 Reduced susceptibility

In the case of reduced susceptibility, where $\sigma_i = \varepsilon = \phi = 1$ and $0 \leq \sigma_s < 1$, the epidemic threshold is given by

$$\begin{aligned} \varphi &= \frac{\lambda + \alpha h + r + \omega}{(1-h)(\alpha h + \omega + \lambda + r) + h\sigma_s(\alpha h + r + \omega + \lambda)} \\ &= 1 + \frac{h(1-\sigma_s)}{1 - h(1-\sigma_s)}, \end{aligned} \tag{2.25}$$

where h was introduced in (2.6) and can be equivalently rewritten as

$$h = \frac{1}{2}\left(1 - \frac{1}{R_0^a} - \frac{\omega}{\alpha}\right) + \sqrt{\frac{1}{4}\left(1 - \frac{1}{R_0^a} - \frac{\omega}{\alpha}\right)^2 + \frac{\omega}{\alpha}}.$$

For $\omega = 0$, the expression for epidemic threshold reduces to

$$\varphi_\circ = 1 + \frac{(R_0^a - 1)(1-\sigma_s)}{1 + (R_0^a - 1)\sigma_s}. \tag{2.26}$$

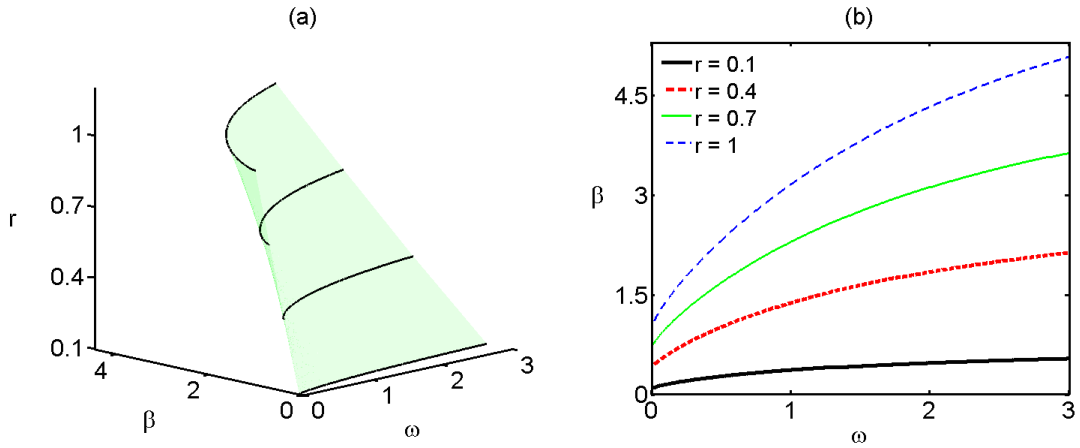

Figure 2.4: Existence and stability of different steady states. The disease-free steady state is stable to the right of the surface in (a) and below each curve in (b), and in these parameter regions the endemic steady state is not feasible. To the left of the surface in (a) and above each curve in (b), the disease-free steady state is unstable, while the endemic steady state exists and is stable. Parameter values are $\alpha = 0.4$, $\lambda = 0.6$, $\sigma_i = 0.5$, $\sigma_s = 0.5$, $\phi = 0.3$, $\varepsilon = 2$, $\delta = 0.4$.

When $\alpha \to \infty$, this threshold tends to the same limit of $1/\sigma_s$ as the epidemic threshold in a model of Funk *et al.* [16], thus suggesting that when the level of local awareness is much higher than that of global awareness, it is this local awareness that dominates the dynamics, and then it does not really matter whether global awareness extends only to susceptible individuals or to the whole population. However, for intermediate values of α, the epidemic threshold in our model depends not only on R_0^a and σ_s, but also on the ratio of the global (ω) and local (α) awareness rates as shown in (2.25), whereas in Funk *et al.* [16], the epidemic threshold was given by (2.26) for any value of ω.

2.4.2 Reduced infectivity

When one considers reduced infectivity where $\sigma_s = \varepsilon = \phi = 1$, and the infective population has its infectivity reduced by a factor $0 \leq \sigma_i < 1$, the epidemic threshold is given by

$$\begin{aligned}\varphi &= \frac{\lambda + \alpha\, h + r + \omega}{(1-h)\left[\sigma_i\left(\alpha\, h + \omega\right) + \lambda + r\right] + h\left[\sigma_i\left(\alpha\, h + r + \omega\right) + \lambda\right]} \\ &= \frac{\lambda + \alpha\, h + r + \omega}{\sigma_i\left[h\left(\alpha + r\right) + \omega\right] + \lambda + r\left(1-h\right)} = 1 + \frac{\left[h\left(\alpha + r\right) + \omega\right]\left(1 - \sigma_i\right)}{\sigma_i\left[h\left(\alpha + r\right) + \omega\right] + \lambda + r\left(1-h\right)},\end{aligned}$$

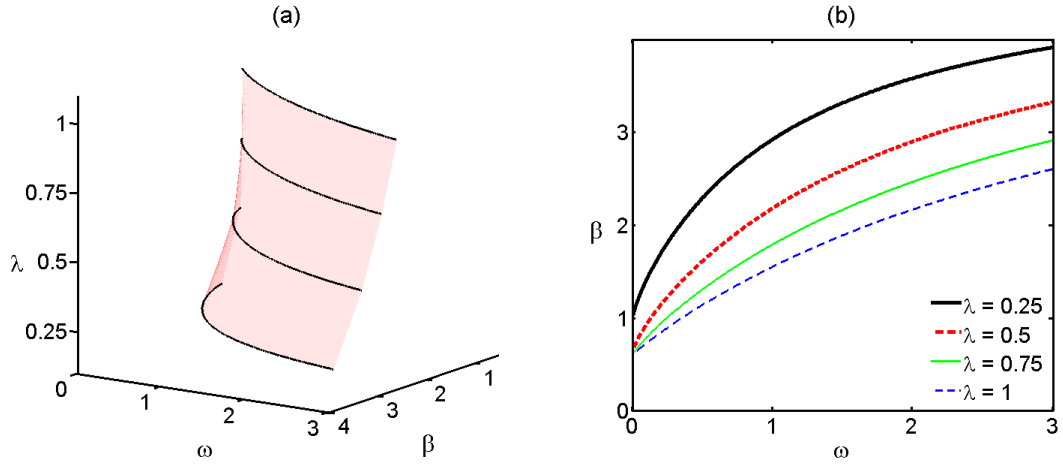

Figure 2.5: Existence and stability of different steady states. The disease-free steady state is stable to the right of the surface in (a) and below each curve in (b), and in these parameter regions the endemic steady state is not feasible. To the left of the surface in (a) and above each curve in (b), the disease-free steady state is unstable, while the endemic steady state exists and is stable. Parameter values are $\alpha = 0.4$, $r = 0.6$, $\sigma_i = 0.5$, $\sigma_s = 0.5$, $\phi = 0.3$, $\varepsilon = 2$, $\delta = 0.4$.

and similarly to the previous case, it now depends on both types of awareness and, in fact, it increases with both α and ω.

2.4.3 Faster recovery

In the case of faster recovery with $\sigma_s = \sigma_i = \phi = 1$ and $\varepsilon > 1$, the epidemic threshold becomes

$$\varphi = \frac{\lambda + \varepsilon\,(\alpha\,h + r + \omega)}{(1-h)\,(\alpha\,h + \omega + \lambda + \varepsilon\,r) + h\,(\alpha\,h + r + \omega + \lambda)} = \frac{\lambda + \varepsilon\,(\alpha\,h + r + \omega)}{h\,(\alpha + r) + \omega + \lambda + \varepsilon\,r\,(1-h)} = 1 + \frac{[h\,(\alpha + r) + \omega]\,(\varepsilon - 1)}{h\,(\alpha + r) + \omega + \lambda + \varepsilon\,r\,(1-h)},$$

and this shows that the threshold depends on both types of awareness and increases with both α and ω. Hence, the period of infectivity is shorten as a result of information dissemination.

2.4.4 Longer preservation of immunity

For longer temporary immunity with $\sigma_s = \sigma_i = \varepsilon = 1$ and $0 \leq \phi < 1$ (average duration of immunity is given by $1/\phi$), the epidemic threshold remains unchanged

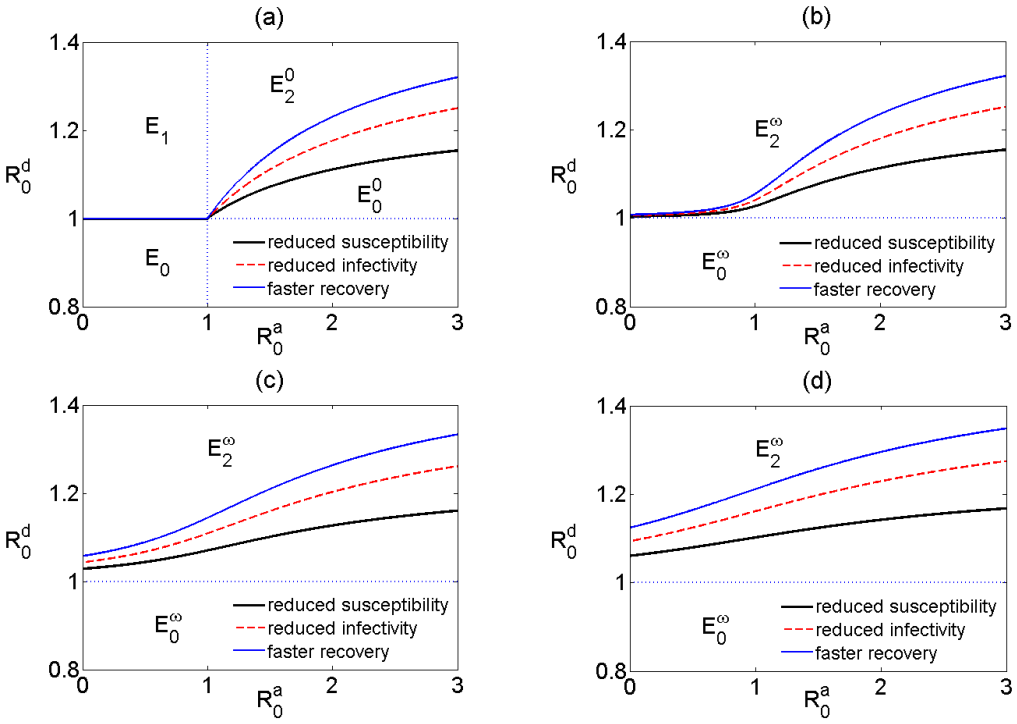

Figure 2.6: Effects of local and global awareness on the spread of infectious diseases for (a) $\omega = 0$ (b) $\omega = 0.01$ (c) $\omega = 0.08$ (d) $\omega = 0.2$. Other parameters are $\lambda = 0.5, r = 0.5, \sigma_i = 0.7, \sigma_s = 0.8, \varepsilon = 1.5$, with varied value of α.

at $R_0^d > \varphi = 1$, since

$$\varphi = \frac{\lambda + \varepsilon\,(\alpha\,h + r + \omega)}{(1-h)\,[\sigma_i\,(\alpha\,h + \omega) + \lambda + \varepsilon\,r] + h\,\sigma_s\,[\sigma_i\,(\alpha\,h + r + \omega) + \lambda]}$$
$$= \frac{\lambda + \alpha\,h + r + \omega}{(1-h)\,(\alpha\,h + \omega + \lambda + r) + h\,(\alpha\,h + r + \omega + \lambda)} = 1.$$

However, if the awareness of an individual population influences the duration of its immunity ϕ^{-1}, the fractions of infected and recovered populations in the endemic state can also change [16].

Figure 2.6 illustrates the dependence of epidemic threshold on the values of R_0^d and R_0^a for reduced susceptibility, reduced infectivity and faster recovery. As suggested by the earlier analysis, in the absence of global awareness (ω), depending on the values of R_0^d and R_0^a the system can settle on one of the four stable steady states, namely, a disease-free E_0, a disease-endemic E_1, an awareness-endemic E_0^0, or endemic equilibrium E_2^0. When the global awareness is present, i.e. $\omega > 0$, the options are limited to either an awareness-endemic equilibrium E_0^ω, which in this case also plays a role of a disease-free state, and an endemic steady state E_2^ω. As $\omega \to 0$, the results shown in plots (b) and (c) illustrate that the steady states in plot

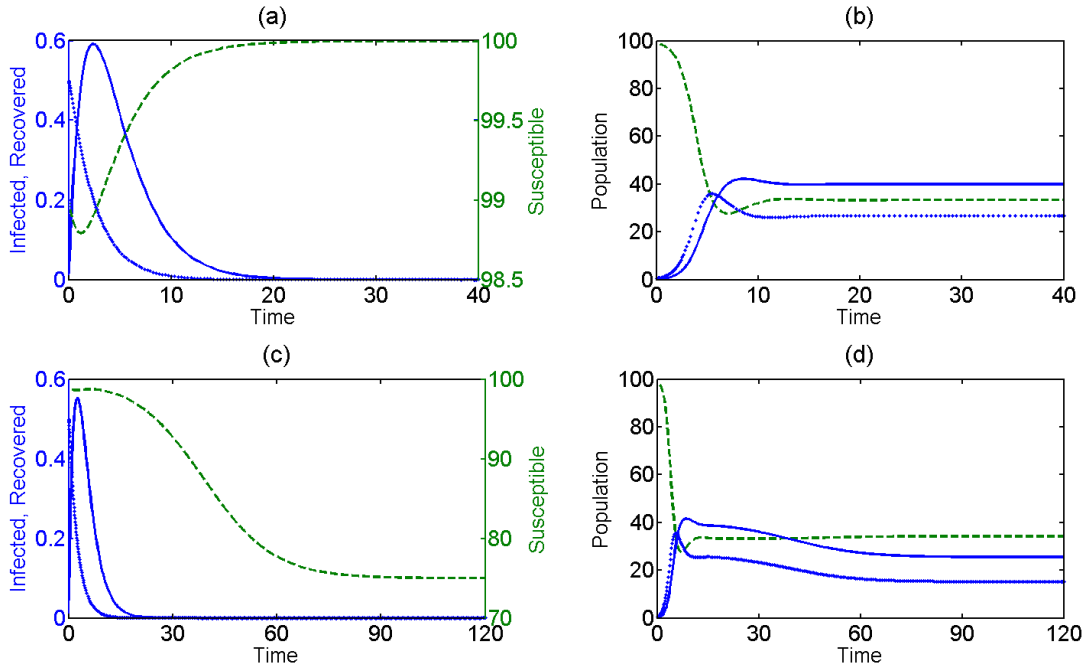

Figure 2.7: Steady states in the absence of global awareness ($\omega = 0$), (a) disease-free state E_0 with $R_0^d < 1, R_0^a < 1$ ($r = 1, \beta = 0.6, \lambda = 0.6$) (b) disease-endemic state, E_1 with $R_0^d > 1, R_0^a < 1$ ($r = 0.6, \beta = 1.8, \lambda = 0.6$) (c) awareness-endemic state, E_0^0 with $R_{\varphi_\circ}^d < 1, R_0^a > 1$ ($r = 1, \beta = 0.6, \lambda = 0.3$) (d) endemic state E_2^0 with $R_{\varphi_\circ}^d > 1, R_0^a > 1$ ($r = 0.6, \beta = 1.8, \lambda = 0.3$). Dashed line denotes S_n, dotted line denotes I_n, solid line denotes R_n. Other parameters are $\alpha = 0.4, \sigma_i = 0.5, \sigma_s = 0.5, \phi = 0.3, \varepsilon = 2, \delta = 0.4, N = 100$.

(d) will eventually transmute into the same result in plot (a) when $\omega = 0$.

2.5 Numerical simulation of the model

In Figure 2.7 we show numerical solution of the system (2.2) in the absence of global awareness, i.e. for $\omega = 0$. Provided the level of local awareness is sufficiently small to ensure $R_0^a < 1$, and the transmission rate is such that $R_0^d < 1$, after the initial growth, the number of infected individuals decreases, and eventually the system approaches a disease-free steady state E_0, as illustrated in Figure 2.7(a). Once the transmission rate exceeds the critical value determined by R_0^d, even after the initial outbreak, certain level of disease is maintained in the population, however, all compartments with aware individuals approach zero, thus giving a disease-endemic steady state E_1 shown in Figure 2.7(b). Figure 2.7(c) shows that for sufficiently high local awareness rate, such that $R_0^a > 1$, as long as the disease transmission rate β is not too high, the population clears the infection, and then the system tends toward an awareness-

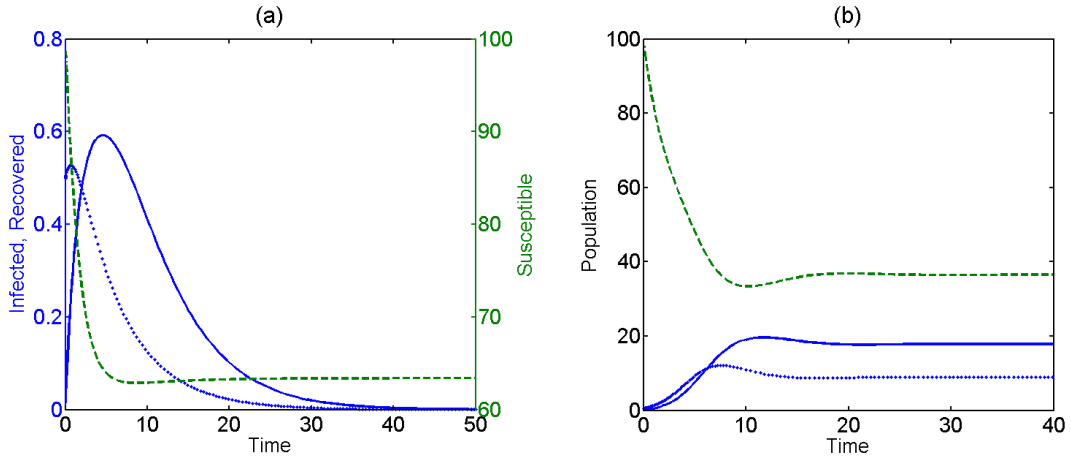

Figure 2.8: Dynamics of infectious disease with global awareness: (a) disease-free state, $\beta = 0.8, R_\varphi^d < 1$ (b) endemic state, $\beta = 1.8, R_\varphi^d > 1$. Other parameter values are $\alpha = 0.4, \lambda = 0.6, \omega = 0.2, \sigma_i = 0.5, \sigma_s = 0.5, \phi = 0.3, \varepsilon = 2, \delta = 0.4, N = 100$. Dashed line denotes S_n, dotted line denotes I_n, solid line denotes R_n. The result gave similar dynamics for the aware population.

endemic steady state E_0^0. Finally, for higher values of β, the final state of the system is given by a stable endemic steady state E_2^0, as shown in Figure 2.7(d).

In the case of $\omega > 0$ illustrated in Figure 2.8, there are just two options: the system either approaches a disease-free steady state, whose role is now played by the awareness-endemic steady state E_0^ω for $R_0^d < \varphi$, or it tends to a fully endemic steady state E_2^ω when $R_0^d > \varphi$.

Although we have not rigorously proven global stability of individual steady states, extensive numerical simulations suggest that in each parameter region only one of the steady states of the system is a global attractor, and the system approaches this steady state for arbitrary initial conditions. It is noteworthy that while stability of the disease-free, disease-endemic and awareness-endemic equilibria can change when some parameters are varied, the endemic steady state with all compartments being positive is always stable whenever it is biologically feasible.

2.6 Discussion

This chapter has analysed the impact of local and global awareness on the spread of infectious diseases in a human population. The main feature of the model is the possibility of individuals in any of the unaware compartments to become aware of

infection both through interactions with aware individuals (regardless of the disease status of the latter), and through a global awareness campaign. This assumption generalises an earlier work of Funk *et al.* [16] who only accounted for the effects of global awareness on infected individuals. Unlike the analysis presented in [16], we have been able to obtain analytical expressions for all steady states of the model together with restrictions on parameters that guarantee their biological feasibility, as well as derived analytical conditions for stability of all these equilibria.

Our results show that both local and global awareness have the capacity to reduce the spread of epidemic by increasing the threshold for onset of a stable endemic steady state characterised by persistent infection. Interestingly, unlike some of the earlier models, we have shown that there is an intricate interplay between the two aspects of awareness as illustrated by the dependence of epidemic threshold of α and ω. Quite naturally, the faster people lose awareness (i.e. the larger is the unaware population), the higher is the overall rate of infection as manifested by the disease-endemic state. Conversely, higher recovery rates due to disease awareness lead to a reduction in infected population. From a more general perspective, the presence of awareness causes corresponding behavioural change in the population, which, in turn, causes the reduction in the size of epidemic outbreaks. Hence, the spread of local awareness or global information campaigns allow one to control or minimise the spread of the disease, whilst they are also helping boost recovery rates for infected individuals. This suggests that information campaigns provide a viable complement if not a replacement for more direct intervention strategies, such as vaccination or quarantine.

Chapter 3

Time-delayed SIS epidemic model with population awareness

This chapter studies the dynamics of epidemic diseases with account for population awareness of the disease generating some human behavioural changes characterised by delay. The time delay is included as an additional parameter in an SIS epidemic model with population awareness to account for the delay in response of unaware individuals to available information often encountered during dissemination of awareness generated from global or local sources regarding the outbreak of diseases, such as, Influenza, STDs, SARS, etc.

3.1 Model derivation

We consider an SIS-type epidemic model similar to the ones in [67, 68] and divide the total population N into susceptible individuals unaware of the diseases S_n, susceptible individuals aware of the disease S_a, and infected individuals I who could become aware of the disease by virtue of being infected. The model focuses on a directly-transmitted infection with a disease transmission rate β, which is modified by a factor $0 < \sigma_s < 1$ in aware susceptibles to describe the prevention measures, such as, reduction in contact, use of vaccine etc., that they undertake in the light of 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 cumulative density of awareness in the population is denoted by M, and it contains a contribution from some global sources, such as, the level of disease awareness in the population generated by general public awareness and media campaigns represented by 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 as a proportion from the entire population at 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. These assumptions give the following system of equations

$$\begin{aligned} S_n' &= -\frac{\beta\, I\, S_n}{N} - \eta\, M\, S_n + \lambda\, S_a + r\, q\, I, \\ S_a' &= -\frac{\sigma_s\, \beta\, I\, S_a}{N} + \eta\, M\, S_n - \lambda\, S_a + r\, p\, I, \\ I' &= \frac{\beta\, I\, S_n}{N} + \frac{\sigma_s\, \beta\, I\, S_a}{N} - r\, I, \\ M' &= \omega_o + \alpha_o\, I + \frac{\alpha\, S_a}{N} - \lambda_o\, M. \end{aligned} \tag{3.1}$$

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 properly aware susceptible individuals. With this assumption, the model takes the form

$$\begin{aligned} S_n' &= -\frac{\beta\, I\, S_n}{N} - \eta\, M(t-\tau)\, S_n + \lambda\, S_a + r\, q\, I, \\ S_a' &= -\frac{\sigma_s\, \beta\, I\, S_a}{N} + \eta\, M(t-\tau)\, S_n - \lambda\, S_a + r\, p\, I, \\ I' &= \frac{\beta\, I\, S_n}{N} + \frac{\sigma_s\, \beta\, I\, S_a}{N} - r\, I, \\ M' &= \omega_o + \alpha_o\, I + \frac{\alpha\, S_a}{N} - \lambda_o\, M, \end{aligned} \tag{3.2}$$

with the initial conditions

$$\begin{aligned} S_n(0) = S_{n_0} \geq 0, \quad S_a(0) = S_{a_0} \geq 0, \quad I(0) = I_0 > 0, \quad S_{n_0} + S_{a_0} + I_0 = N, \\ M(0) = M_0 \geq 0 \quad \text{and} \quad M(s) = M_0(s) \geq 0, \quad -\tau \leq s < 0. \end{aligned} \tag{3.3}$$

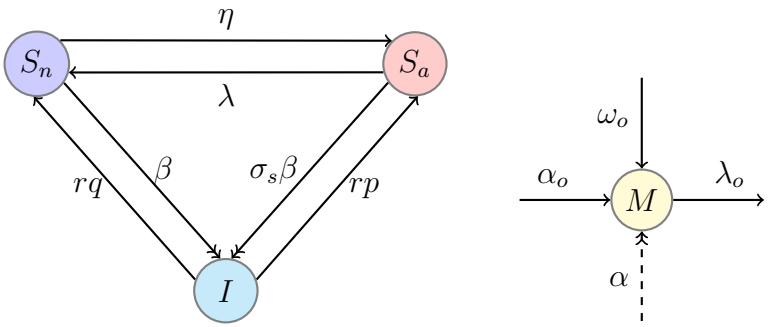

Figure 3.1: Transition diagram of model (3.2). Solid lines represent transitions associated with individuals. Arrow stands for "possible transition": doubled arrow head indicating processes subject to contacts on the disease (solid lines) or awareness (dash lines) network, single head arrows indicate processes that are not subject to contact.

Since this model has no vital dynamics or disease-induced deaths, the total population $N = S_n + S_a + I$ is constant. Figure 3.1 represents the model diagram for (3.2) with all the transitions between the compartments.

Before proceeding with the analysis, we have to ascertain that solutions of model (3.2) 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 (3.2) with the initial conditions in (3.3) are non-negative for all $t \geq 0$.*

Proof.

We begin the proof by showing that $I(t) > 0$ for all $t > 0$. Let $t_1 > 0$ be the first time when

$$\frac{\beta\, I(t_1)\, S_n(t_1)}{N} = 0.$$

Assuming $I(t_1) = 0$, we obtain

$$S_n(t) \geq 0 \qquad \text{for all} \quad t \in [0, t_1].$$

Let

$$\psi_1 = \min_{0 \leq t \leq t_1} \left\{ \frac{\beta\, S_n(t)}{N} + \frac{\sigma_s\, \beta\, S_a(t)}{N} - r \right\} \qquad \Rightarrow \qquad I' \geq \psi_1\, I.$$

Hence, for $t \in [0, t_1]$ one has

$$I'(t) \geq \psi_1\, I(t) \qquad \Rightarrow \qquad I(t_1) \geq I(0)\, e^{\psi_1\, t_1} > 0,$$

which gives a contradiction, hence $I(t) > 0$ for $t > 0$.

Next, we prove by contradiction that $S_n(t) > 0$ for all $t > 0$. Assuming there exists a first time $t_0 > 0$ such that $S_n(t_0) = 0$, implies that $S_n(t) > 0$ for $t \in [0, t_0)$, and $dS_n(t_1)/dt < 0$. On the other hand, from the first equation of the system (3.2) it follows that

$$\left.\frac{dS_n}{dt}\right|_{t=t_0} = \lambda\, S_a + r\, q\, I > 0,$$

which is a contradiction. Hence, $S_n(t) > 0$ for $t > 0$.

To prove that $S_a(t) > 0$ for all $t > 0$ we again assume that there exists a first time $t_s > 0$ such that $S_a(t_s) = 0$, and $dS_a(t_s)/dt < 0$. The third equation of the system (3.2) gives

$$\left.\frac{dS_a}{dt}\right|_{t=t_s} = \eta\, M(t-\tau)\, S_n + r\, p\, I > 0,$$

which is a contradiction, and therefore, $S_a(t) > 0$ for $t > 0$.

Finally, from the fourth equation in (3.2) we have that when $M(t) = 0$, $M'(t) > 0$, thus $M(t)$ can never become negative.

Therefore, during their evolution, the solutions of the system (3.2) with the initial conditions (3.3) will remain non-negative for all $t \in [0, \infty)$. In fact, they will be contained in the bounded set:

$$\Phi_1 = \left\{(S_n,\ S_a,\ I,\ M)\ \in\ \mathbb{R}^4_+ :\ 0\ \leq S_n,\ S_a,\ I\ \leq\ N,\ 0 \leq M \leq \widetilde{M}\right\},$$

where $\widetilde{M} = M(0) + \dfrac{\omega_o + \alpha_o N + \alpha}{\lambda_o}$. ■

3.2 Steady states

All steady states of the system (3.2) satisfy the following system of algebraic equations

$$\begin{aligned}
0 &= -\frac{\beta\, I\, S_n}{N} - \eta\, M\, S_n + \lambda\, S_a + r\, q\, I,\\
0 &= -\frac{\sigma_s\, \beta\, I\, S_a}{N} + \eta\, M\, S_n - \lambda\, S_a + r\, p\, I,\\
0 &= \frac{\beta\, I\, S_n}{N} + \frac{\sigma_s\, \beta\, I\, S_a}{N} - r\, I,\\
0 &= \omega_o + \alpha_o\, I + \frac{\alpha\, S_a}{N} - \lambda_o\, M.
\end{aligned} \tag{3.4}$$

Disease-free state

From the third equation of (3.4), we have

$$\left(\frac{\beta\, S_n}{N} + \frac{\sigma_s\, \beta\, S_a}{N} - r\right) I = 0,$$

hence, either

$$I = 0, \qquad \text{or} \qquad \frac{\beta\, S_n}{N} + \frac{\sigma_s\, \beta\, S_a}{N} - r = 0.$$

Considering $I = 0$, we obtain

$$\begin{aligned} 0 &= \eta\, M\, S_n - \lambda\, S_a, \\ 0 &= \omega_o + \frac{\alpha\, S_a}{N} - \lambda_o\, M. \end{aligned}$$

One can find M from the last equation as

$$M = \frac{N\,\omega_o + \alpha\, S_a}{N\,\lambda_o},$$

and substitute it into the first equation, which using $S_n = N - S_a$ gives

$$\frac{\eta\,(N\,\omega_o + \alpha\, S_a)}{N\,\lambda_o}\,(N - S_a) = \lambda\, S_a,$$

which can be rewritten as a single quadratic equation for S_a

$$S_a^2 - N\left(1 - \frac{\lambda\,\lambda_o + \eta\,\omega_o}{\eta\,\alpha}\right) S_a - \frac{N^2\,\omega_o}{\alpha} = 0.$$

This equation has a single positive solution

$$S_a = N\left[\frac{1}{2}\left(1 - \frac{\lambda\,\lambda_o + \eta\,\omega_o}{\eta\,\alpha}\right) + \sqrt{\frac{1}{4}\left(1 - \frac{\lambda\,\lambda_o + \eta\,\omega_o}{\eta\,\alpha}\right)^2 + \frac{\omega_o}{\alpha}}\right].$$

Introducing

$$h_o = \frac{1}{2}\left(1 - \frac{\lambda\,\lambda_o + \eta\,\omega_o}{\eta\,\alpha}\right) + \sqrt{\frac{1}{4}\left(1 - \frac{\lambda\,\lambda_o + \eta\,\omega_o}{\eta\,\alpha}\right)^2 + \frac{\omega_o}{\alpha}}, \tag{3.5}$$

then

$$S_a = N\,h_o, \qquad \text{and} \qquad S_n = N - S_a = N - N\,h_o = N\,(1 - h_o), \qquad 0 < h_o < 1.$$

This gives the disease-free equilibrium state $E_0 = (S_n^\circ, S_a^\circ, 0, M^\circ)$, where

$$S_n^\circ = N\,(1 - h_o), \qquad S_a^\circ = N\,h_o \qquad \text{and} \qquad M^\circ = \frac{\omega_o + \alpha\, h_o}{\lambda_o}. \tag{3.6}$$

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

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

Endemic state

Returning to the third equation of (3.4), we now explore the scenario when $I > 0$ and

$$\frac{\beta\, S_n}{N} + \frac{\sigma_s\, \beta\, S_a}{N} - r = 0,$$

which implies

$$S_a = \frac{N\, r - \beta\, S_n}{\sigma_s\, \beta}. \tag{3.7}$$

In order to have $S_a > 0$, it follows that S_n should satisfy the condition $S_n < \frac{N\, r}{\beta}$.

From the fourth equation of (3.4) we obtain

$$M = \frac{N\,(\omega_o + \alpha_o\, I) + \alpha\, S_a}{N\, \lambda_o}, \tag{3.8}$$

and the first equation of (3.4) gives

$$M = \frac{N\, \lambda\, S_a + N\, r\, q\, I - \beta\, I\, S_n}{N\, \eta\, S_n}. \tag{3.9}$$

Equating these two expressions for M yields

$$\frac{N\,(\omega_o + \alpha_o\, I) + \alpha\, S_a}{N\, \lambda_o} = \frac{N\, \lambda\, S_a + N\, r\, q\, I - \beta\, I\, S_n}{N\, \eta\, S_n},$$

$$[(N\, \eta\, \alpha_o + \beta\, \lambda_o)\, S_n - N\, r\, q\, \lambda_o]\, I = (N\, \lambda\, \lambda_o - \eta\, \alpha\, S_n)\, S_a - N\, \eta\, \omega_o\, S_n. \tag{3.10}$$

Substituting the value of S_a from (3.7) gives after simplification

$$I = \frac{N^2\, r\, \lambda\, \lambda_o + \beta\, \eta\, \alpha\, S_n^2 - N\,(\beta\, \lambda\, \lambda_o + \sigma_s\, \beta\, \eta\, \omega_o + r\, \eta\, \alpha)\, S_n}{\sigma_s\, \beta\, [(N\, \eta\, \alpha_o + \beta\, \lambda_o)\, S_n - N\, r\, q\, \lambda_o]}. \tag{3.11}$$

Since I must be positive, we note from (3.10) that

$$(N\, \eta\, \omega_o + \eta\, \alpha\, S_a)\, S_n = N\, \lambda\, \lambda_o\, S_a + \left[N\, r\, q\, \lambda_o - (N\, \eta\, \alpha_o + \beta\, \lambda_o)\, S_n\right] I,$$

and

$(N\, \eta\, \omega_o + \eta\, \alpha\, S_a)\, S_n > 0,$ this implies $N\, \lambda\, \lambda_o\, S_a + \left[N\, r\, q\, \lambda_o - (N\, \eta\, \alpha_o + \beta\, \lambda_o)\, S_n\right] I > 0,$

which implies

$$I < \frac{N\, \lambda\, \lambda_o\, S_a}{(N\, \eta\, \alpha_o + \beta\, \lambda_o)\, S_n - N\, r\, q\, \lambda_o}.$$

In order for I to be positive, one then has to require

$$S_n > \frac{N\,r\,q\,\lambda_o}{N\,\eta\,\alpha_o + \beta\,\lambda_o}. \tag{3.12}$$

Using the fact that $N = S_n + S_a + I$, and substituting (3.7) and (3.11) gives

$$S_n + \frac{N\,r - \beta\,S_n}{\sigma_s\,\beta} + \frac{N^2\,r\,\lambda\,\lambda_o + \beta\,\eta\,\alpha\,S_n^2 - N\,(\beta\,\lambda\,\lambda_o + \sigma_s\,\beta\,\eta\,\omega_o + r\,\eta\,\alpha)\,S_n}{\sigma_s\,\beta\,[(N\,\eta\,\alpha_o + \beta\,\lambda_o)\,S_n - N\,r\,q\,\lambda_o]} = N,$$

which can be recast as the following quadratic equation for S_n

$$y_1\,S_n^2 - y_2\,S_n + y_3 = 0,$$

where $\quad y_1 = \beta\,[(1-\sigma_s)\,(N\,\eta\,\alpha_o + \beta\,\lambda_o) - \eta\,\alpha],$

$y_2 = N\,[\beta\,r\,q\,\lambda_o\,(1-\sigma_s) + (N\,\eta\,\alpha_o + \beta\,\lambda_o)\,(r - \sigma_s\,\beta) - \beta\,(\lambda\,\lambda_o + \eta\,\sigma_s\,\omega_o) - r\,\eta\,\alpha],$

$y_3 = N^2\,r\,\lambda_o\,[q\,(r - \sigma_s\,\beta) - \lambda].$

The solution of this equation yields an endemic state $E^* = (S_n^*, S_a^*, I^*, M^*)$, where

$$\begin{aligned} S_n^* &= \frac{y_2 \pm \sqrt{y_2^2 - 4\,y_1\,y_3}}{2\,y_1}, \\ I^* &= \frac{N^2\,r\,\lambda\,\lambda_o + \beta\,\eta\,\alpha\,S_n^{*2} - N\,(\beta\,\lambda\,\lambda_o + \sigma_s\,\beta\,\eta\,\omega_o + r\,\eta\,\alpha)\,S_n^*}{\sigma_s\,\beta\,[(N\,\eta\,\alpha_o + \beta\,\lambda_o)\,S_n^* - N\,r\,q\,\lambda_o]}, \\ S_a^* &= \frac{N\,r - \beta\,S_n^*}{\sigma_s\,\beta}, \qquad \text{and} \qquad M^* = \frac{N\,(\omega_o + \alpha_o\,I^*) + \alpha\,S_a^*}{N\,\lambda_o}, \end{aligned} \tag{3.13}$$

with the value of S_n^* lying within the interval $\dfrac{N\,r\,q\,\lambda_o}{N\,\eta\,\alpha_o + \beta\,\lambda_o} < S_n^* < \dfrac{N\,r}{\beta}$ for biological feasibility.

In summary, the model (3.2) has only two steady states,

$$E_0 = (S_n^\circ, S_a^\circ, 0, M^\circ) \qquad \text{and} \qquad E^* = (S_n^*, S_a^*, I^*, M^*).$$

3.3 Stability analysis

Linearising system of equations (3.2) near each steady state $(\widehat{S}_n, \widehat{S}_a, \widehat{I}, \widehat{M})$, gives the following Jacobian matrix

$$J_p = \begin{pmatrix} -a_5 - a_6 & \lambda & -a_2 + r\,q & -a_\tau\,\tilde{a}_6 \\ a_6 & -\tilde{a}_5 - \lambda & -\tilde{a}_0 + r\,p & a_\tau\,\tilde{a}_6 \\ a_5 & \tilde{a}_5 & a_2 + \tilde{a}_0 - r & 0 \\ 0 & a_7 & \alpha_o & -\lambda_o \end{pmatrix}, \tag{3.14}$$

where $a_\tau = e^{-k\,\tau}$, $\tilde{a}_0 = \frac{\sigma_s\,\beta\,\widehat{S}_a}{N}$, $a_2 = \frac{\beta\,\widehat{S}_n}{N}$ $a_5 = \frac{\beta\,\widehat{I}}{N}$, $\tilde{a}_5 = \frac{\sigma_s\,\beta\,\widehat{I}}{N}$, $a_6 = \eta\,\widehat{M}$, $\tilde{a}_6 = \eta\,\widehat{S}_n$, $a_7 = \frac{\alpha}{N}$.

Stability of the disease-free state

Theorem 2. *The disease-free steady state E_0 of the system of equations (3.2) is linearly asymptotically stable for all $\tau \geq 0$ if the basic reproductive number, $R_0 < 1$, unstable for $R_0 > 1$, and undergoes bifurcation at $R_0 = 1$, where*

$$R_0 = \frac{\beta\,(1 + \sigma_s\,h_o - h_o)}{r}.$$

Proof.

Evaluating the Jacobian matrix (3.14) at the disease-free steady state $E_0 = (S_n^\circ,\, S_a^\circ,\, 0,\, M^\circ)$ with $S_n^\circ > 0,\ S_a^\circ > 0$ and $M^\circ > 0$ gives the following matrix

$$J_p^\circ = \begin{pmatrix} -a_6 & \lambda & -a_2 + r\,q & -a_\tau\,\tilde{a}_6 \\ a_6 & -\lambda & -\tilde{a}_0 + r\,p & a_\tau\,\tilde{a}_6 \\ 0 & 0 & a_2 + \tilde{a}_0 - r & 0 \\ 0 & a_7 & \alpha_o & -\lambda_o \end{pmatrix},$$

with the associated characteristic equation

$$k\,(k + r - a_2 - \tilde{a}_0)\,[k^2 + k\,(\lambda_o + \lambda + a_6) + \lambda_o\,(\lambda + a_6) - \tilde{a}_6\,a_7\,a_\tau] = 0.$$

One of the eigenvalues is always $k_1 = 0$, another is given by

$$k_2 = a_2 + \tilde{a}_0 - r = \frac{\beta(S_n^\circ + \sigma_s S_a^\circ)}{N} - r,$$

and the rest are determined by the roots of the transcendental equation

$$k^2 + k\,(\lambda_o + \lambda + a_6) + \lambda_o\,(\lambda + a_6) - \tilde{a}_6\,a_7\,e^{-k\,\tau} = 0. \tag{3.15}$$

The eigenvalue k_2 is negative, provided

$$\frac{\beta(S_n^\circ + \sigma_s S_a^\circ)}{N} - r < 0 \quad \Longleftrightarrow \quad \frac{\beta(S_n^\circ + \sigma_s S_a^\circ)}{Nr} < 1,$$

which, using the values of S_n° and S_a° from (3.6), can be recast as

$$R_0 = \frac{\beta\,(1 + \sigma_s\,h_o - h_o)}{r} < 1.$$

Therefore, $k_2 < 0$ if $R_0 < 1$. It is clear that when R_0 passes the value of 1, the eigenvalue k_2 goes through zero and becomes positive, thus making the disease-free steady state E_0 unstable by means of a steady state bifurcation.

For $\tau = 0$, the equation (3.15) turns into a quadratic

$$k^2 + k\,(\lambda_o + \lambda + a_6) + \lambda_o\,(\lambda + a_6) - \tilde{a}_6\,a_7 = 0 \tag{3.16}$$

whose roots are both negative if and only if

$$\lambda_o\,(\lambda + a_6) > \tilde{a}_6\,a_7 \qquad \Rightarrow \qquad \lambda_o\,(\lambda + \eta\,M^\circ) > \frac{\eta\,\alpha\,S_n^\circ}{N}. \tag{3.17}$$

Substituting the values of M° and S_n° from (3.6) shows that this condition is equivalent to

$$\lambda\,\lambda_o + \eta\,\omega_o + 2\,\eta\,\alpha\,h_o > \eta\,\alpha, \qquad \text{or} \qquad h_o > \frac{1}{2}\left(1 - \frac{\lambda\,\lambda_o + \eta\,\omega_o}{\eta\,\alpha}\right),$$

and this inequality always holds in the light of (3.5), thus implying that for $\tau = 0$, both roots of the quadratic equation (3.16) always have negative real part.

Consequently, the disease-free steady state E_0 with $\tau = 0$ is stable if the basic reproductive number satisfies

$$R_0 = \frac{\beta\,(1 + \sigma_s\,h_o - h_o)}{r} < 1. \tag{3.18}$$

To investigate whether the disease-free steady state can lose its stability for $\tau > 0$, we first note that $k = 0$ is not a solution of this equation (this follows immediately from (3.17)), so we look for solutions of the equation (3.15) in the form $k = i\mu$. This gives,

$$\begin{aligned} -\mu^2 + i\,\mu\,(\lambda_o + \lambda + a_6) + \lambda_o\,(\lambda + a_6) &= \tilde{a}_6\,a_7\,e^{-i\,\mu\,\tau} \\ &= \tilde{a}_6\,a_7\,[\cos(\mu\,\tau) - i\,\sin(\mu\,\tau)]. \end{aligned}$$

Separating real and imaginary parts we obtain

$$\begin{aligned} -\mu^2 + \lambda_o\,(\lambda + a_6) &= \tilde{a}_6\,a_7\,\cos(\mu\,\tau), \\ \mu\,(\lambda_o + \lambda + a_6) &= -\tilde{a}_6\,a_7\,\sin(\mu\,\tau). \end{aligned} \tag{3.19}$$

Squaring and adding these two equations yields the following quartic equation

$$\mu^4 + \mu^2\,[\lambda_o^2 + (\lambda + a_6)^2] + (\lambda_o\,(\lambda + a_6) + \tilde{a}_6\,a_7)\,(\lambda_o\,(\lambda + a_6) - \tilde{a}_6\,a_7) = 0.$$

Substituting $y_4 = \lambda_o^2 + (\lambda + a_6)^2$ and $y_5 = (\lambda_o\,(\lambda + a_6) + \tilde{a}_6\,a_7)\,(\lambda_o\,(\lambda + a_6) - \tilde{a}_6\,a_7)$, yields a quartic equation for μ

$$\mu^4 + y_4\,\mu^2 + y_5 = 0.$$

Since $y_4 > 0$ and $\lambda_o\,(\lambda + a_6) > \tilde{a}_6\,a_7$, which means that $y_5 > 0$, this suggests that there are no real positive roots μ^2 of the above equation, such that $k = i\mu$ would be a root of equation (3.15). Consequently, the disease-free state is always stable if $R_0 < 1$ for all $\tau \geq 0$. ■

Stability of the endemic state

Next, we investigate stability of the endemic equilibrium of system (3.2). Evaluating the Jacobian (3.14) at the endemic equilibrium state $E^* = (S_n^*, S_a^*, I^*, M^*)$ with $S_n^* > 0,\ S_a^* > 0\ I^* > 0$ and $M^* > 0$, yields the characteristic equation

$$k\left(k^3 + k^2\,(\lambda_o + y_6) + k\,(\lambda_o\,y_6 + y_7 - \tilde{a}_6\,a_7\,a_\tau) + \lambda_o\,y_7 + a_\tau\left[\alpha\,\tilde{a}_6(a_5 - \tilde{a}_5) - a_5\,\tilde{a}_6\,a_7\right]\right) = 0, \quad (3.20)$$

with $y_6 = \lambda + a_5 + \tilde{a}_5 + a_6$ and $y_7 = [\tilde{a}_5\,(a_5 + \tilde{a}_0 + a_6) - a_5\,\tilde{a}_0] + \lambda\,a_5 + r\,p\,(a_5 - \tilde{a}_5)$.

Note that

$$a_5 - \tilde{a}_5 = \frac{\beta\,I^*}{N} - \frac{\sigma_s\,\beta\,I^*}{N} = \frac{\beta\,I^*}{N}\,(1 - \sigma_s) > 0, \quad (3.21)$$

$$\begin{aligned}
&\tilde{a}_5\,(a_5 + \tilde{a}_0 + a_6) - a_5\,\tilde{a}_0 \\
&\quad = \frac{\sigma_s\,\beta\,I^*}{N}\left[\frac{\beta\,I^*}{N} + \frac{\sigma_s\,\beta\,S_a^*}{N} + \frac{N\,\eta\,(\omega_o + \alpha_o\,I^*) + \eta\,\alpha\,S_a^*}{N\,\lambda_o}\right] - \frac{\beta\,I^*}{N}\,\frac{\sigma_s\,\beta\,S_a^*}{N} \\
&\quad = \frac{\sigma_s\,\beta\,I^*}{N}\left[\frac{N\,\eta\,(\omega_o + \alpha_o\,I^*) + \eta\,\alpha\,S_a^* + \beta\,\lambda_o\,I^* + \sigma_s\,\beta\,\lambda_o\,S_a^* - \beta\,\lambda_o\,S_a^*}{N\,\lambda_o}\right],
\end{aligned}$$

but we obtain from (3.10) that

$$N\,\eta\,(\omega_o + \alpha_o\,I^*)\,S_n^* + \eta\,\alpha\,S_a^*\,S_n^* = N\,\lambda\,\lambda_o\,S_a^* + N\,r\,q\,\lambda_o\,I^* - \beta\,\lambda_o\,I^*\,S_n^*,$$

which can be rewritten as

$$[N\,\eta\,(\omega_o + \alpha_o\,I^*) + \eta\,\alpha\,S_a^* + \beta\,\lambda_o\,I^*]\,S_n^* = N\,\lambda\,\lambda_o\,S_a^* + N\,r\,q\,\lambda_o\,I^*,$$

$$\Rightarrow \qquad N\,\eta\,(\omega_o+\alpha_o\,I^*)+\eta\,\alpha\,S_a^*+\beta\,\lambda_o\,I^*=\frac{N}{S_n^*}\left[\lambda\,\lambda_o\,S_a^*+r\,q\,\lambda_o\,I^*\right]$$
$$=\frac{N\,r\left[\lambda\,\lambda_o\,S_a^*+r\,q\,\lambda_o\,I^*\right]}{r\,S_n^*}$$
$$=\frac{(\beta\,S_n^*+\sigma_s\,\beta\,S_a^*)\left(\lambda\,\lambda_o\,S_a^*+r\,q\,\lambda_o{}^*I\right)}{r\,S_n^*}$$
$$=\beta\,\lambda_o\,S_a^*\left[\frac{\lambda\,S_n^*+\sigma_s\,\lambda\,S_a^*}{r\,S_n^*}\right]+\beta\,\lambda_o\,q\,I^*\left[\frac{S_n^*+\sigma_s\,S_a^*}{S_n^*}\right].$$

Hence,

$$N\,\eta\,(\omega_o+\alpha_o\,I^*)+\eta\,\alpha\,S_a^*+\beta\,\lambda_o\,I^*+\sigma_s\,\beta\,\lambda_o\,S_a^*$$
$$=\beta\,\lambda_o\,S_a^*\left[\frac{(\lambda+\sigma_s\,r)\,S_n^*+\sigma_s\,\lambda\,S_a^*}{r\,S_n^*}\right]+\beta\,\lambda_o\,q\,I^*\left[\frac{S_n^*+\sigma_s\,S_a^*}{S_n^*}\right].$$

We also obtain from (3.10) the expression

$$(N\,\lambda\,\lambda_o-\eta\,\alpha\,S_n^*)\,S_a^*-N\,\eta\,\omega_o\,S_n^*>0,$$

which can be written as

$$N\,\lambda\,\lambda_o\,S_a^*>\eta\,\alpha\,S_a^*\,S_n^*+N\,\eta\,\omega_o\,S_n^*=(N\,\eta\,\omega_o+\eta\,\alpha\,S_a^*)\,S_n^*$$
$$\Rightarrow \qquad \sigma_s\,\lambda\,S_a^*>\frac{\sigma_s\,(N\,\eta\,\omega_o+\eta\,\alpha\,S_a^*)\,S_n^*}{N\,\lambda_o}.$$

Therefore,

$$(\lambda+\sigma_s\,r)\,S_n^*+\sigma_s\,\lambda\,S_a^*>\left[\frac{N\,\lambda_o\,(\lambda+\sigma_s\,r)+N\,\sigma_s\,\eta\,\omega_o+\sigma_s\,\eta\,\alpha\,S_a^*}{N\,\lambda_o}\right]S_n^*$$
$$=\frac{1}{N\,\lambda_o}\Big[\left[\lambda_o\,(\lambda+\sigma_s\,r)+\sigma_s\,\eta\,\omega_o\right]S_n^*+\left[\lambda_o\,(\lambda+\sigma_s\,r)+\sigma_s\,\eta\,(\omega_o+\alpha)\right]S_a^*+$$
$$\left[\lambda_o\,(\lambda+\sigma_s\,r)+\sigma_s\,\eta\,\omega_o\right]I^*\Big]S_n^*$$
$$>\left[\frac{\beta\,\lambda_o\,S_n^*+\sigma_s\,\beta\,\lambda_o\,S_a^*}{N\,\lambda_o}\right]S_n^*=r\,S_n^*,$$

since $r=\frac{\beta\,S_n^*+\sigma_s\,\beta\,S_a^*}{N}$. It follows that,

$$N\,\eta\,(\omega_o+\alpha_o\,I^*)+\eta\,\alpha\,S_a^*+\beta\,\lambda_o\,I^*+\sigma_s\,\beta\,\lambda_o\,S_a^*>\beta\,\lambda_o\,S_a^*,$$

$$\Rightarrow \qquad \tilde{a}_5\,(a_5+\tilde{a}_0+a_6)>a_5\,\tilde{a}_0. \tag{3.22}$$

Similarly, considering $(N\,\lambda\,\lambda_o-\eta\,\alpha\,S_n)\,S_a-N\,\eta\,\omega_o\,S_n>0$ gives

$$N\,\lambda\,\lambda_o\,S_a>\eta\,\alpha\,S_n\,S_a+N\,\eta\,\omega_o\,S_n>\eta\,\alpha\,S_n\,S_a,$$

$$\lambda\,\lambda_o > \frac{\eta\,\alpha\,S_n}{N} = \tilde{a}_6\,a_7 \qquad \Rightarrow \qquad \lambda\,\lambda_o > \tilde{a}_6\,a_7. \tag{3.23}$$

Hence, the characteristic equation (3.20) gives the eigenvalue $k_1 = 0$, and the rest of the spectrum being given by the roots of the transcendental equation

$$k^3 + k^2(\lambda_o + y_6) + k(\lambda_o y_6 + y_7) + \lambda_o y_7 = [\tilde{a}_6 a_7(k + a_5) - \alpha\tilde{a}_6(a_5 - \tilde{a}_5)]e^{-k\tau}. \tag{3.24}$$

For $\tau = 0$, the equation (3.24) turns into a cubic equation

$$k^3 + k^2\,(\lambda_o + y_6) + k\,(\lambda_o\,y_6 + y_7 - \tilde{a}_6\,a_7) + \lambda_o\,y_7 + \alpha\,\tilde{a}_6\,(a_5 - \tilde{a}_5) - a_5\,\tilde{a}_6\,a_7 = 0. \tag{3.25}$$

By the Routh-Hurwitz criterion, the roots of this cubic equation have negative real part if and only if the following conditions are satisfied

$$\lambda_o + y_6 > 0, \qquad \lambda_o\,y_6 + y_7 - \tilde{a}_6\,a_7 > 0, \qquad \lambda_o\,y_7 + \alpha\,\tilde{a}_6\,(a_5 - \tilde{a}_5) - a_5\,\tilde{a}_6\,a_7 > 0,$$
$$\text{and} \qquad (\lambda_o + y_6)\,(\lambda_o\,y_6 + y_7 - \tilde{a}_6\,a_7) > \lambda_o\,y_7 + \alpha\,\tilde{a}_6\,(a_5 - \tilde{a}_5) - a_5\,\tilde{a}_6\,a_7. \tag{3.26}$$

Showing that the criteria above are satisfied gives

$\rightarrow \quad \lambda_o + y_6 = \lambda_o + \lambda + a_5 + \tilde{a}_5 + a_6 > 0,$

this first condition is always satisfied.

$$\rightarrow \quad \lambda_o\,y_6 + y_7 - \tilde{a}_6\,a_7 > \lambda_o\,y_6 - \tilde{a}_6\,a_7$$
$$= \lambda_o\,(a_5 + \tilde{a}_5 + a_6) + (\lambda\,\lambda_o - \tilde{a}_6\,a_7) > 0,$$

the second condition holds based on (3.23).

$$\rightarrow \quad \lambda_o\,y_7 + \alpha\,\tilde{a}_6\,(a_5 - \tilde{a}_5) - a_5\,\tilde{a}_6\,a_7 = \lambda_o\left[\tilde{a}_5\,(a_5 + \tilde{a}_0 + a_6) - a_5\,\tilde{a}_0\right] + \lambda\,\lambda_o\,a_5 +$$
$$\lambda_o\,r\,p\,(a_5 - \tilde{a}_5) + \alpha\,\tilde{a}_6\,(a_5 - \tilde{a}_5) - a_5\,\tilde{a}_6\,a_7$$
$$= \lambda_o\,[\tilde{a}_5\,(a_5 + \tilde{a}_0 + a_6) - a_5\,\tilde{a}_0] + (\lambda_o\,r\,p + \alpha\,\tilde{a}_6)\,(a_5 - \tilde{a}_5) +$$
$$a_5\,(\lambda\,\lambda_o - \tilde{a}_6\,a_7) > 0,$$

the third condition is also satisfied since all the brackets in the last expression are positive due to (3.21), (3.22) and (3.23).

$\rightarrow \quad (\lambda_o + y_6)\,(\lambda_o\,y_6 + y_7 - \tilde{a}_6\,a_7) > \lambda_o\,y_7 + \alpha\,\tilde{a}_6\,(a_5 - \tilde{a}_5) - a_5\,\tilde{a}_6\,a_7,$

this can be expressed as

$$(\lambda_o + y_6)\left[(\lambda\,\lambda_o - \tilde{a}_6\,a_7) + \lambda_o\,(a_5 + \tilde{a}_5 + a_6)\right] + y_6\,y_7 + \tilde{a}_6\,(\alpha\,\tilde{a}_5 + a_5\,a_7) - \alpha\,a_5\,\tilde{a}_6 > 0,$$

hence, we have the following result.

Lemma 1. *If the condition*

$$(\lambda_o + y_6)\,(\lambda_o\, y_6 + y_7 - \tilde{a}_6\, a_7) > \lambda_o\, y_7 + \alpha\, \tilde{a}_6\, (a_5 - \tilde{a}_5) - a_5\, \tilde{a}_6\, a_7 \tag{3.27}$$

holds, the endemic steady state E^* is linearly asymptotically stable.

Remark 1. *Although it does not appear possible to analytically prove that the condition (3.27) always holds, numerical simulations suggest that it does indeed for any parameter value, for which the endemic steady state E^* is biologically feasible.*

Since we have now established that for $\tau = 0$ the endemic state E^* is linearly asymptotically stable, one still has to find out whether this steady state can lose stability for $\tau > 0$. First of all, one should note that in the light of the third condition for stability, $k = 0$ is not a root of the characteristic equation (3.24). Hence, the steady state E^* can only lose its stability when a pair of complex conjugate eigenvalues crosses the imaginary axis from left to right. Introducing auxiliary parameters,

$$\tilde{y}_1 = \lambda_o + y_6,\ \tilde{y}_2 = \lambda_o\, y_6 + y_7,\ \tilde{y}_3 = \lambda_o\, y_7,\ \tilde{y}_4 = \tilde{a}_6\, a_7,\ \tilde{y}_5 = \alpha\, \tilde{a}_6\, (a_5 - \tilde{a}_5) - a_5\, \tilde{a}_6\, a_7,$$

the characteristic equation (3.24) can be recast in the form

$$k^3 + \tilde{y}_1\, k^2 + \tilde{y}_2\, k + \tilde{y}_3 = (\tilde{y}_4\, k + \tilde{y}_5)\, e^{-k\,\tau}. \tag{3.28}$$

Substituting $k = i\,\mu$ gives

$$\begin{aligned} -i\,\mu^3 - \tilde{y}_1\,\mu^2 + \tilde{y}_2\, i\,\mu + \tilde{y}_3 &= (\tilde{y}_4\, i\,\mu + \tilde{y}_5)\, e^{-i\,\mu\,\tau} \\ &= (\tilde{y}_4\, i\,\mu + \tilde{y}_5)\, [\cos(\mu\,\tau) - i\, \sin(\mu\,\tau)]. \end{aligned}$$

Separating real and imaginary parts, we obtain

$$\begin{aligned} -\tilde{y}_1\,\mu^2 + \tilde{y}_3 &= \tilde{y}_5\, \cos(\mu\,\tau) + \tilde{y}_4\,\mu\, \sin(\mu\,\tau), \\ -\mu^3 + \tilde{y}_2\,\mu &= \tilde{y}_4\,\mu\, \cos(\mu\,\tau) - \tilde{y}_5\, \sin(\mu\,\tau). \end{aligned} \tag{3.29}$$

Squaring and adding these equations yield the following equation for the Hopf frequency w:

$$f(\mu) = \mu^6 + (\tilde{y}_1^2 - 2\,\tilde{y}_2)\,\mu^4 + (\tilde{y}_2^2 - \tilde{y}_4^2 - 2\,\tilde{y}_1\,\tilde{y}_3)\,\mu^2 + \tilde{y}_3^2 - \tilde{y}_5^2 = 0. \tag{3.30}$$

The derivative of the function, $f(\mu)$ can be found as

$$f'(\mu) = 2\,\mu\left[3\,\mu^4 + 2\,(\tilde{y}_1^2 - 2\,\tilde{y}_2)\,\mu^2 - 2\,\tilde{y}_1\,\tilde{y}_3 + \tilde{y}_2^2 - \tilde{y}_4^2\right].$$

Multiplying the first equation of the system (3.29) by $\tilde{y}_5$ and the second equation by $\tilde{y}_4$ and adding these two equations gives

$$\cos(\mu\,\tau) = \frac{\tilde{y}_3\,\tilde{y}_5 + (\tilde{y}_2\,\tilde{y}_4 - \tilde{y}_1\,\tilde{y}_5)\,\mu^2 - \tilde{y}_4\,\mu^4}{\tilde{y}_5^2 + \tilde{y}_4^2\,\mu^2}, \tag{3.31}$$

and similarly,

$$\sin(\mu\,\tau) = \frac{(\tilde{y}_5 - \tilde{y}_1\,\tilde{y}_4)\,\mu^3 + (\tilde{y}_3\,\tilde{y}_4 - \tilde{y}_2\,\tilde{y}_5)\,\mu}{\tilde{y}_5^2 + \tilde{y}_4^2\,\mu^2}. \tag{3.32}$$

Therefore, the value of τ can be found as follows

$$\tau_n = \frac{1}{\mu}\left[\cos^{-1}\left(\frac{\tilde{y}_3\,\tilde{y}_5 + (\tilde{y}_2\,\tilde{y}_4 - \tilde{y}_1\,\tilde{y}_5)\,\mu^2 - \tilde{y}_4\,\mu^4}{\tilde{y}_5^2 + \tilde{y}_4^2\,\mu^2}\right) + 2\,\pi\,n\right], \quad n = 0, 1, 2, \ldots$$

Without loss of generality, let us assume that equation (3.30) has six distinct positive real roots μ_i, $i = 1, \ldots, 6$. For each μ_i, we have the value of τ as

$$\tau_{j,n} = \frac{1}{\mu_j}\left[\cos^{-1}\left(\frac{\tilde{y}_3\tilde{y}_5 + (\tilde{y}_2\tilde{y}_4 - \tilde{y}_1\tilde{y}_5)\mu_j^2 - \tilde{y}_4\mu_j^4}{\tilde{y}_5^2 + \tilde{y}_4^2\mu_j^2}\right) + 2\pi\,(n-1)\right],\ j = 1, \ldots, 6,\ n \in \mathbb{N}.$$

This allows one to define

$$\tau_0 = \tau_{j_0,n_0} = \min_{1\le j\le 6, n\ge 1}\{\tau_{j,n}\}, \quad \mu_0 = \mu_{j_0}. \tag{3.33}$$

In order to establish whether the endemic steady state E^* actually undergoes Hopf bifurcation at $\tau = \tau_0$, one has to compute the sign of $d[\mathrm{Re}(k)]/d\tau$. Differentiating the characteristic equation (3.28) with respect to τ gives

$$\left[3\,k^2 + 2\,\tilde{y}_1\,k + \tilde{y}_2\right]\frac{dk}{d\tau} = \left[\tilde{y}_4\,e^{-k\,\tau} - \tau\,e^{-k\,\tau}(\tilde{y}_4\,k + \tilde{y}_5)\right]\frac{dk}{d\tau} - k\,(\tilde{y}_4\,k + \tilde{y}_5)\,e^{-k\,\tau},$$

which can be rewritten as

$$\left(\frac{dk}{d\tau}\right)^{-1} = \frac{\tilde{y}_4\,e^{-k\,\tau} - 3\,k^2 - 2\,\tilde{y}_1\,k - \tilde{y}_2}{(\tilde{y}_4\,k^2 + \tilde{y}_5\,k)\,e^{-k\,\tau}} - \frac{\tau}{k}.$$

Evaluating this at $\tau = \tau_0$ with $k = i\mu_0$ we obtain

$$\left(\frac{dk}{d\tau}\right)^{-1}\Bigg|_{\tau=\tau_0} = \frac{\tilde{y}_4\,\cos(\mu_0\tau_0) + 3\,\mu_0^2 - \tilde{y}_2 - i\left[\tilde{y}_4\,\sin(\mu_0\tau_0) + 2\,\tilde{y}_1\,\mu_0\right]}{\mu_0\left[\tilde{y}_5\sin(\mu_0\tau_0) - \tilde{y}_4\mu_0\cos(\mu_0\tau_0)\right] + i\mu_0\left[\tilde{y}_5\cos(\mu_0\tau_0) + \tilde{y}_4\mu_0\sin(\mu_0\tau_0)\right]} - \frac{\tau_0}{i\mu_0},$$

and the real part of this expression can be found as

$$\mathrm{Re}\left(\frac{dk}{d\tau}\right)^{-1}\Bigg|_{\tau=\tau_0} = \frac{1}{\mu_0\left[\tilde{y}_5^2+\tilde{y}_4^2\,\mu_0^2\right]}\Big[\big[(3\,\tilde{y}_5-2\,\tilde{y}_1\,\tilde{y}_4)\mu_0^2-\tilde{y}_2\,\tilde{y}_5\big]\sin(\mu_0\tau_0)+ \big[(\tilde{y}_2\,\tilde{y}_4-2\,\tilde{y}_1\,\tilde{y}_5)\mu_0-3\,\tilde{y}_4\,\mu_0^3\big]\cos(\mu_0\tau_0)-\tilde{y}_4^2\,\mu_0\Big].$$

Substituting the values of $\cos(\mu_0\tau_0)$ and $\sin(\mu_0\tau_0)$ from (3.31) and (3.32) gives the following after simplification,

$$\begin{aligned}\mathrm{Re}\left(\frac{dk}{d\tau}\right)^{-1}\Bigg|_{\tau=\tau_0} &= \frac{3\,\mu_0^4+(2\,\tilde{y}_1^2-4\,\tilde{y}_2)\,\mu_0^2-2\,\tilde{y}_1\,\tilde{y}_3+\tilde{y}_2^2-\tilde{y}_4^2}{\tilde{y}_5^2+\tilde{y}_4^2\,\mu_0^2}\\ &= \frac{2\,\mu_0\big[3\,\mu_0^4+2\,(\tilde{y}_1^2-2\,\tilde{y}_2)\,\mu_0^2-2\,\tilde{y}_1\,\tilde{y}_3+\tilde{y}_2^2-\tilde{y}_4^2\big]}{2\,\mu_0\big[\tilde{y}_5^2+\tilde{y}_4^2\,\mu_0^2\big]} = z_\mu\,f'(\mu_0),\end{aligned}$$

where $z_\mu = \big[2\,\mu_0(\tilde{y}_5^2+\tilde{y}_4^2\,\mu_0^2)\big]^{-1}$, and since $z_\mu > 0$, it implies

$$\mathrm{sign}\left\{\frac{d[\mathrm{Re}(k)]}{d\tau}\right\}_{\tau=\tau_0} = \mathrm{sign}\left\{\mathrm{Re}\left(\frac{dk(\tau_0)}{d\tau}\right)^{-1}\right\} = \mathrm{sign}\big\{z_\mu\,f'(\mu_0)\big\} = \mathrm{sign}\big\{f'(\mu_0)\big\}.$$

These calculations can now be summarised in the following result.

Theorem 3. *Let the condition of Lemma 1 holds and also let τ_0 and μ_0 be defined as in (3.33) and $f'(\mu_0) > 0$. Then the endemic steady state E^* of the system (3.2) is linearly asymptotically stable for $\tau < \tau_0$, unstable for $\tau > \tau_0$ and undergoes Hopf bifurcation at $\tau = \tau_0$.*

3.4 Numerical stability analysis and simulations

To get a better understanding of the effects of different parameters on the dynamics of the system (3.2), we expand the analysis presented in the previous section by numerically computing characteristic eigenvalues. This is achieved by using a pseudospectral method implemented in a traceDDE suite in MATLAB [7].

Figure 3.2 illustrates regions of stability of the endemic steady state E^* depending on the disease transmission rate β, time delay τ, awareness from global sources, ω_o, α_o, the rate of local awareness α and the rate at which unaware susceptible individuals become aware, η. This Figure shows that for sufficiently small time delays τ, the endemic steady state E^* is stable, thus providing numerical evidence to support Remark 1. As the time delay τ increases, the steady state E^* loses its stability in

accordance with Theorem 3. There are several important observations that have to be made here. First of all, one should note a qualitative difference in the effects of different types of awareness transmission. Whereas the endemic steady state E^* can be destabilised for arbitrarily small values of the general awareness campaigns ω_o, or the rate of local awareness α, in the case of awareness associated with the increasing number of reported disease cases α_o and the proportion of the unaware susceptible individuals becoming aware η, the endemic steady state remains stable for all possible values of the time delay τ.

Another counter-intuitive result is that as the level of awareness ω_o and α increases, the endemic steady state actually remains stable for longer durations of the time delay in response. When one considers the effects of the speed of disease transmission, as shown in Figure 3.2(c), it becomes clear that for sufficiently high values of the disease transmission rate β, the endemic steady state is stable for any values of the time delay τ. On the other hand, for small values of β, it has a destabilising role: as β increases, the critical time delay at which the Hopf bifurcation occurs is decreasing, but this effect reverses starting with some value of β. For sufficiently large values of α and ω_o, or for sufficiently small values of β, where $R_0 < 1$, the endemic steady state E^* is not feasible, whereas the disease-free steady state E_0 is feasible and stable for any values of τ.

Figure 3.3 shows how the stability boundary of the steady state E^* changes depending on the values of global awareness ω_o and local awareness α. One can see that increasing the level of local awareness α results in the endemic steady state losing its stability for smaller values of the global awareness ω_o for the same time delay τ. Conversely, if one fixes the value of ω_o and increases α, instability occurs for higher values of τ, suggesting that the local awareness actually helps the state of infection remain present in the population for longer durations of the individual response time.

To investigate the behaviour of the system beyond the Hopf bifurcation, we have used the continuation software DDE-BIFTOOL to numerically continue branches of periodic solutions in the parameter space, and the results are shown in Figures 3.4 - 3.7 for different values of the parameters α and τ. In Figure 3.4, with $\alpha = 0.3$, plots (a)-(c) show how the eigenvalues change with increasing time delay from the

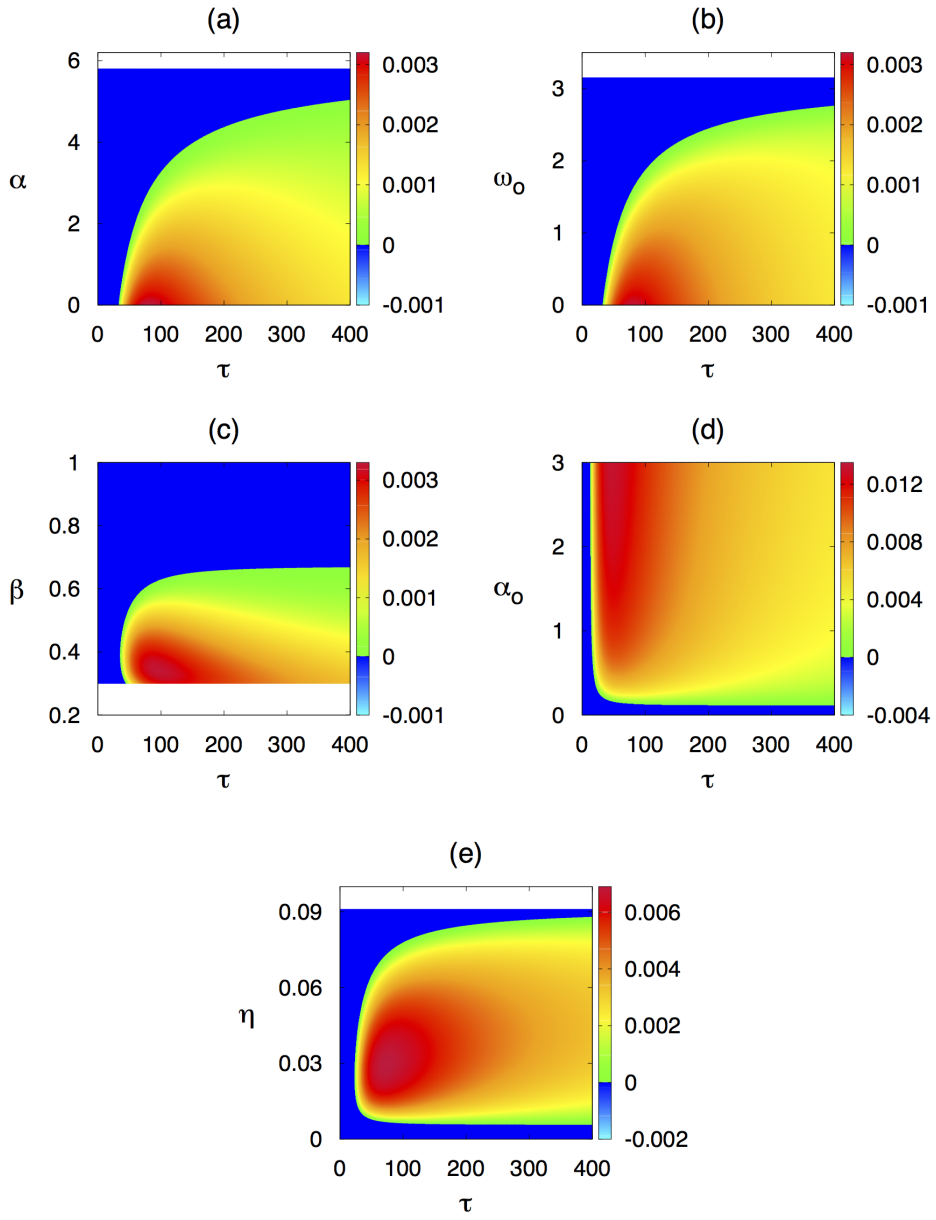

Figure 3.2: Stability of the endemic steady state E^*. Colour code denotes max[Re(k)] whenever the endemic steady state is feasible. Parameter values are, $\alpha = 0.3$ but varied in (a), $\omega_o = 0.2$ but varied in (b), $\beta = 0.4$ but varied in (c), $\alpha_o = 0.2$ but varied in (d), $\eta = 0.01$ but varied in (e), $\lambda = 0.1, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \lambda_o = 0.3, N = 100$.

stable endemic steady state to unstable steady state through a Hopf bifurcation, while plot (d) illustrates the comparison of the Hopf boundary as computed using the DDE-BIFTOOL and the traceDDE software.

The outcome for the bifurcation analysis of the endemic steady state E^* for $\alpha = 0.3$, $\tau = 45$ is presented in Figure 3.5 showing the solutions, amplitude and the period of oscillations. Similar results for $\alpha = 1.5$, $\tau = 55$ are shown in Figure 3.6, and Figure 3.7 illustrates the outcome for $\alpha = 2.7$, $\tau = 80$. The plots (b) and (c) in

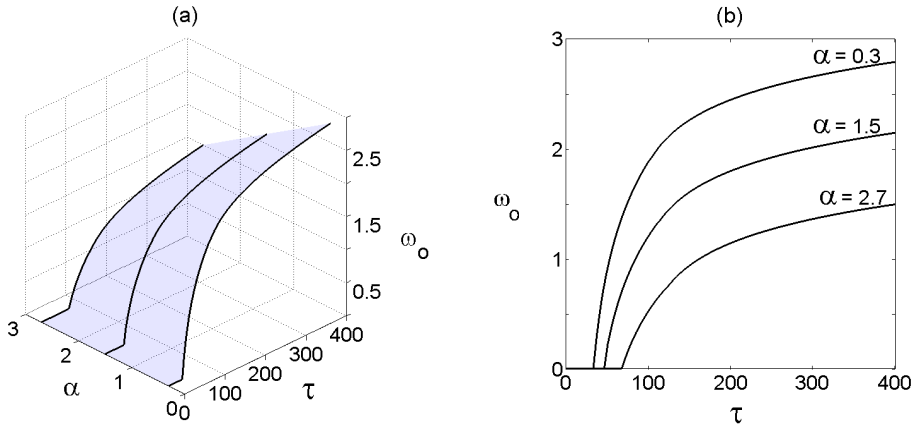

Figure 3.3: Stability boundaries of the endemic steady state E^*. The steady state is stable to the left of the surface in (a), and to the left of the lines in (b). Parameter values are $\lambda = 0.1, \beta = 0.4, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.01, N = 100$.

Figures 3.5, 3.6 and 3.7 suggest that increasing the time delay τ results in a larger amplitude and a larger period of periodic oscillations around the endemic steady state E^*. For the same time delay τ, the higher rate of local awareness α results in smaller amplitude of oscillations, but a larger period of those oscillations, provided α is not too high to ensure the existence of periodic solutions.

Figure 3.8 illustrates the dynamics of the system (3.2) in the case where $R_0 < 1$ or $R_0 > 1$ with zero time delay. In the situation where $R_0 < 1$, the recovery from infection is sufficiently fast to ensure the initial outbreak is contained, and the disease is eradicated from the population. Figure 3.9 shows a similar behaviour for $R_0 < 1$ in the case of positive time delay τ thus illustrating the conclusion of Theorem 2 that stability of the disease-free steady state is independent of the time delay. For slower recovery rates and sufficiently small delays in response to awareness, the system settles on a stable endemic steady state, as shown in Figure 3.10(a)-(c).

Increasing the time delay τ results in higher-amplitude decaying oscillations around the endemic steady state, and once τ exceeds the critical value determined by Theorem 3, the endemic steady state loses its stability, which results in the emergence of stable periodic solutions shown in Figure 3.10(d).

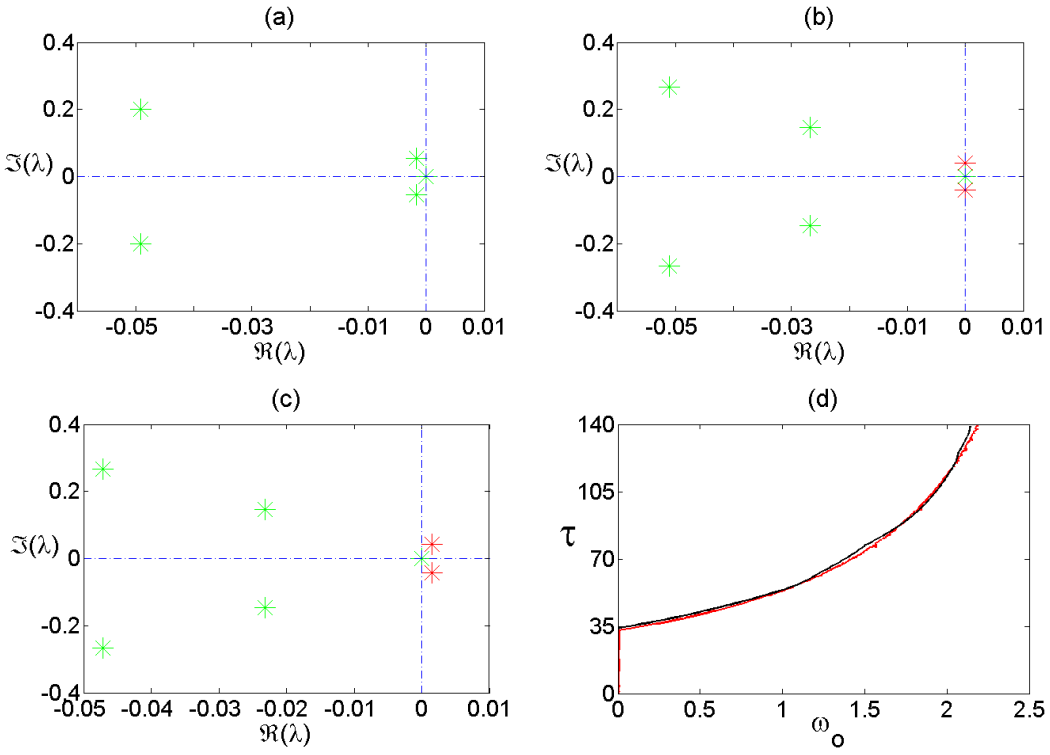

Figure 3.4: Bifurcation analysis of the endemic steady state E^*: (a) distribution of eigenvalues for $\tau = 30$, (b) distribution of eigenvalues at the Hopf point for $\tau = 45$, (c) distribution of eigenvalues for $\tau = 45$, (d) comparison of the Hopf boundary of the steady state E^* as computed by DDE-BIFTOOL (black) and traceDDE (red). The parameter values are: $\alpha = 0.3, \lambda = 0.1, \beta = 0.4, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.2, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.01, N = 100$.

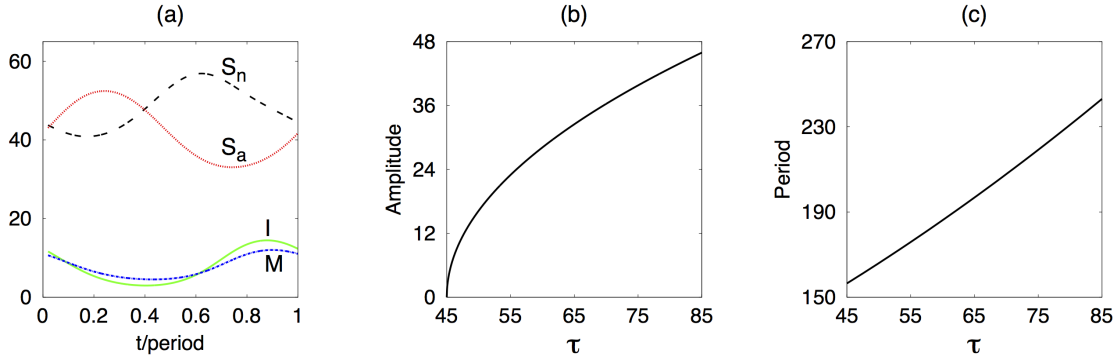

Figure 3.5: Bifurcation analysis of the endemic steady state E^* for $\alpha = 0.3, \tau = 45$: (a) periodic orbit of solutions (b) plot of amplitude against time delay, τ (c) plot of the period against time delay, τ. The parameter values include: $\lambda = 0.1, \beta = 0.4, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.2, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.01, N = 100$.

3.5 Discussion

In this chapter we have analysed the dynamics of a non-lethal infectious disease with the simultaneous spread of awareness, and a delayed response of individuals to available information. Specific emphasis was made on explicitly incorporating

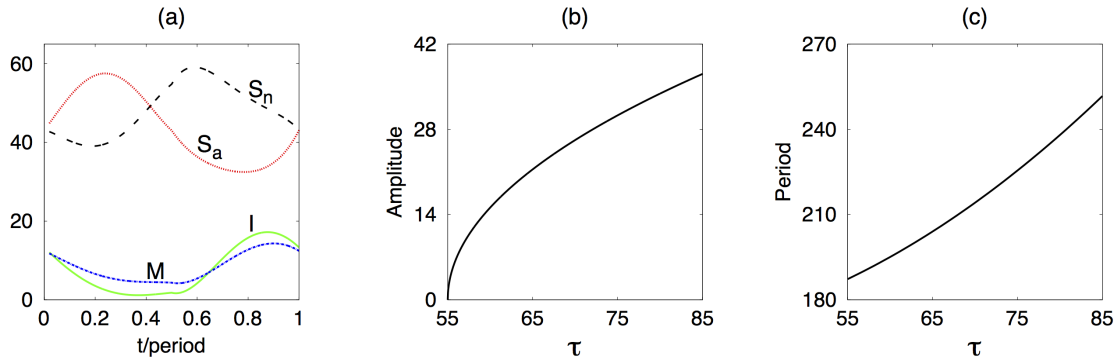

Figure 3.6: Bifurcation analysis of the endemic steady state E^* for $\alpha = 1.5, \tau = 55$: (a) periodic orbit of solutions (b) plot of amplitude against time delay, τ (c) plot of the period against time delay, τ. Value of parameters include: $\lambda = 0.1, \beta = 0.4, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.2, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.01, N = 100$.

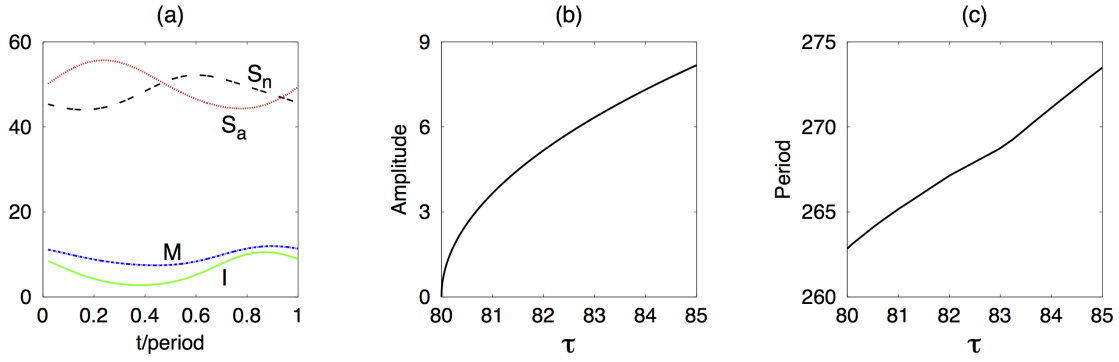

Figure 3.7: Bifurcation analysis of the endemic steady state E^* for $\alpha = 2.7, \tau = 80$: (a) periodic orbit of solutions (b) plot of amplitude against time delay, τ (c) plot of the period against time delay, τ. The parameters are: $\lambda = 0.1, \beta = 0.4, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.2, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.01, N = 100$.

different facets of disease awareness that can arise due to general public information campaigns, public reports of observed cases of disease, a word-of-mouth spread from aware to unaware individuals. We have derived conditions for feasibility and stability of the disease-free and endemic equilibria in terms of system parameters and the time delay associated with changes in individuals' behaviour. These results suggest that stability of the disease-free equilibrium is independent of the time delay but depends on the rates at which awareness is required.

An interesting result is that for sufficiently high values of the spread of global information from general campaigns or information from aware population, it is possible to eradicate the disease, whereas an increase in awareness stemming from the higher number of reported disease cases does not result in disease eradication. Another important observation is that in the presence of a delay in response of individuals to available information, increasing the values of global awareness campaign

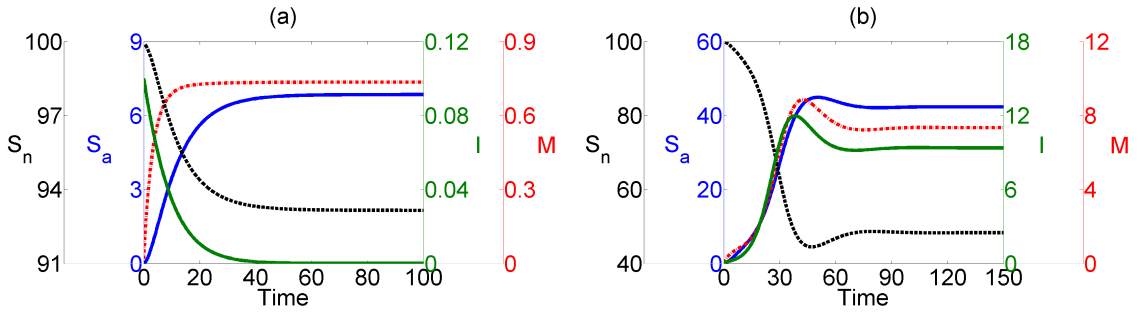

Figure 3.8: Numerical solution of the system (3.2) with $\tau = 0$ (a) disease-free state, $r = 0.5, R_0 = 0.7474$ (b) endemic state, $r = 0.2, R_0 = 1.8685$. Other parameter values are, $\alpha = 0.3, \lambda = 0.1, \beta = 0.4, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.2, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.01, N = 100$.

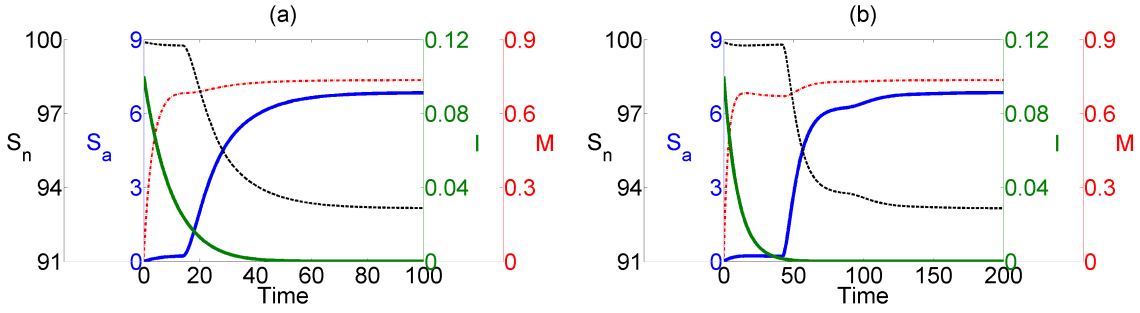

Figure 3.9: Numerical solution of the system (3.2) with $R_0 < 1$ (a) $\tau = 14$ (b) $\tau = 42$. Parameter values are, $\alpha = 0.3, \lambda = 0.1, \beta = 0.4, r = 0.5, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.2, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.01, N = 100$. In this case, $R_0 = 0.7474$, and the system settles on a stable disease-free steady state.

or local awareness actually results in stabilising the endemic equilibrium, i.e. maintaining its presence in the population, and only when these values get quite high that the disease is eradicated.

Considering the effects of time delay on the disease dynamics, we have discovered that it can destabilise the endemic steady state, thus causing periodic oscillations. Both the amplitude and the period of these oscillations increase with the time delay in the individuals' response, however, the period is also growing with the rate of local information transmission, whereas the amplitude of oscillations decreases, and the oscillations can be completely suppressed for sufficiently high rates of local information transmission. If the disease is transmitted quite quickly, i.e. the transmission rate is sufficiently high, then increasing the delay will not affect the stability of the endemic equilibrium, hence, the disease will always be present at some constant level in the population. In a narrow range of values of the time delay, increasing the disease transmission rate initially destabilises the endemic steady state, whilst fur-

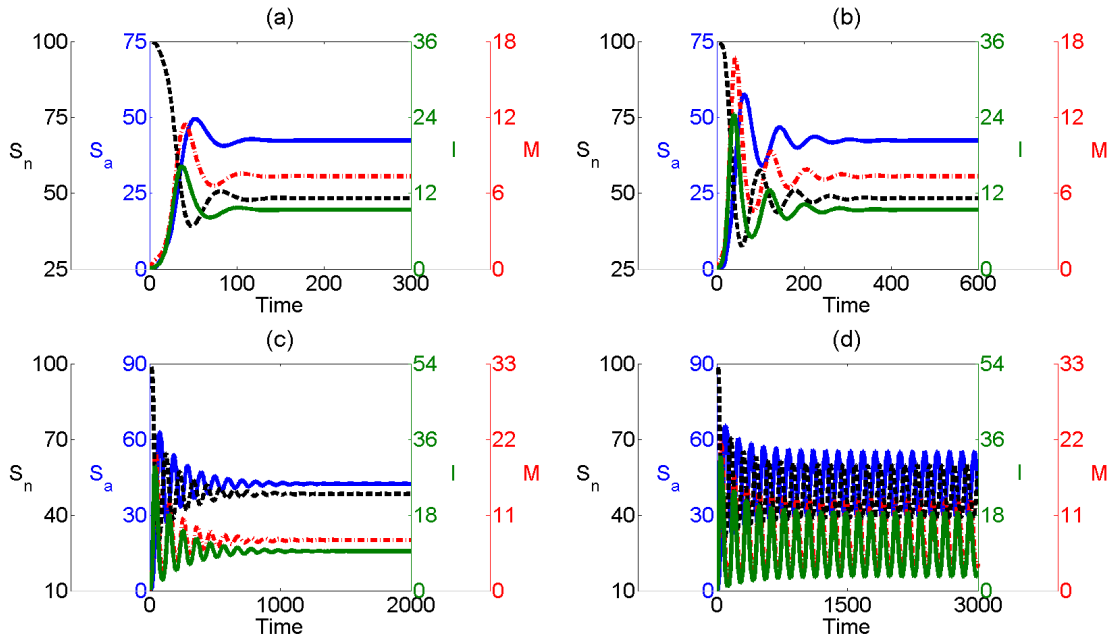

Figure 3.10: Numerical solution of the system (3.2) with $R_0 = 1.8685$, (a) $\tau = 5$, (b) $\tau = 14$, (c) $\tau = 25$, (d) $\tau = 42$. Parameter values include, $\alpha = 0.3, \lambda = 0.1, \beta = 0.4, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.2, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.01, N = 100$.

ther increase leads to stability being regained. These results provide some practical insights into the development and assessment of possible information campaigns targeted at disease control and prevention by elucidating how different routes of transmission of awareness affect the progression of the disease in the population.

In a nutshell, the results of the analysis show that the disease-free steady state is stable for all values of time delay τ provided the threshold, $R_0 < 1$ and the endemic state is stable for zero time delay, these are similar to the results in [67, 68]. Contrary to Zuo and Liu [67] but in agreement with Zuo *et al.* [68], the endemic steady state switches stability from a stable to an unstable state as the value of the time delay exceeds the critical value. Furthermore, the analysis shows that the threshold of the model is dependent on both global awareness campaigns ω_o and local awareness rate α which indicates that both ω_o and α can influence the disease dynamics. Based on the results, infectious diseases can be eradicated when the values of ω_o and α are sufficiently high. Consequently, in addition to global awareness campaigns, dissemination of local awareness also aid in the eradication process of infectious diseases and the delay in response of individuals to available information from general public awareness campaigns and local awareness can increase the rate of spread of infectious diseases within the population.

Chapter 4

Dynamics of vaccination in a time-delayed epidemic model with awareness

The chapter investigates the impact of combining awareness with vaccination on the spread of infectious diseases in human population. Vaccination targets newborn against childhood infections, such as, influenza, whooping cough, measles, etc. and then people who become aware of the spreading of infections, and similar to the last chapter we include multiple sources of disease awareness.

4.1 Derivation of the model

Similarly to the model analysed in the previous chapter, we consider a non-lethal disease, but now we include vital dynamics and assume that infection confers a temporary immunity. The population is divided into groups of susceptible individuals unaware of infection, $S_n(t)$, susceptible individuals aware of infection, $S_a(t)$, infected individuals $I(t)$, and recovered (immune) individuals $R(t)$. There is a constant birth rate b, which is taken to be the same as the death rate, so that the total population N remains constant, and it is assumed that all newborns are unaware and susceptible to infection. The disease is transmitted from infected to unaware susceptible individuals at a rate β, and this rate is reduced by a factor $0 < \sigma_s < 1$ for aware susceptibles, who take some measures to reduce their potential contact rate.

Infected individuals recover at rate r. The cumulative level of disease awareness $M(t)$ has contributions from the reported number of cases at a rate α_o, from the aware individuals at a rate α, and from some global awareness campaigns ω_o, and the awareness is lost at a rate λ_o, whereas aware susceptibles lose their awareness at a rate λ. Finally, unaware susceptibles become aware at a rate η, and it is assumed that it takes time τ for them to actually become aware or to modify their behaviour in the relation to the spreading infection. These assumptions lead to the following basic model

$$\begin{aligned}
S_n' &= b\,N - \frac{\beta\,I\,S_n}{N} - \eta\,M(t-\tau)\,S_n + \lambda\,S_a - b\,S_n,\\
S_a' &= -\frac{\sigma_s\,\beta\,I\,S_a}{N} + \eta\,M(t-\tau)\,S_n - \lambda\,S_a - b\,S_a,\\
I' &= \frac{\beta\,I\,S_n}{N} + \frac{\sigma_s\,\beta\,I\,S_a}{N} - r\,I - b\,I,\\
R' &= r\,I - b\,R,\\
M' &= \omega_o + \alpha_o\,I + \frac{\alpha\,S_a}{N} - \lambda_o\,M,
\end{aligned} \tag{4.1}$$

where $S_n(t) + S_a(t) + I(t) + R(t) = N$ a constant.

To investigate the effects of the introduction of a vaccine on the disease dynamics, we consider a situation where a proportion v of newborns and a proportion v_s of aware susceptible individuals are vaccinated [27]. With this assumption, $bvN \equiv v_iN$ newborns appear straight in the recovered (protected) class, and $b(1-v)N = (b-v_i)N$ newborns go to the class of unaware susceptibles. It is further assumed that after a period of time $1/\delta$, the individuals lose their immunity against the infection. If $\delta = 0$, this describes a perfect vaccine, while $\delta > 0$ describes a leaky vaccine resulting in temporary immunity. Similar to some earlier works [67, 68], it is assumed that upon losing immunity, a certain proportion, p, of individuals will join the aware susceptible class while the remaining proportion, $q = 1-p$, will return to the unaware susceptible class. With these assumptions, a modified model has the

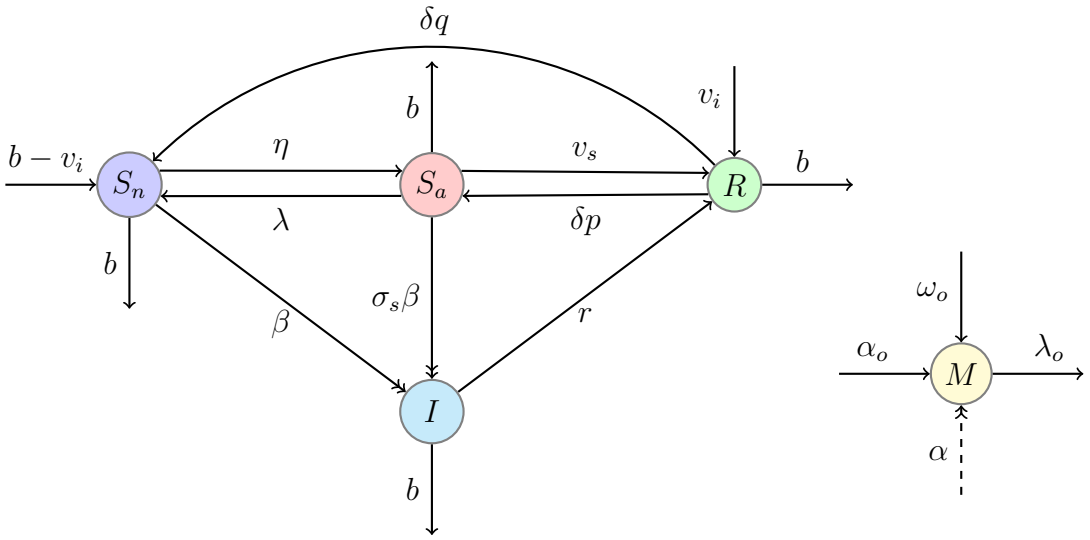

Figure 4.1: Diagram of transitions in model (4.2). Solid lines represent transitions associated with individuals. Arrow stands for "possible transition": doubled arrow head indicating processes subject to contacts on the disease (solid lines) or awareness (dash lines) network, single head arrows indicate processes that are not subject to contact.

form

$$\begin{aligned}
S_n' &= (b - v_i)\, N - \frac{\beta\, I\, S_n}{N} - \eta\, M(t-\tau)\, S_n + \lambda\, S_a + \delta\, q\, R - b\, S_n,\\
S_a' &= -\frac{\sigma_s\, \beta\, I\, S_a}{N} + \eta\, M(t-\tau)\, S_n - (\lambda + v_s + b)\, S_a + \delta\, p\, R,\\
I' &= \frac{\beta\, I\, S_n}{N} + \frac{\sigma_s\, \beta\, I\, S_a}{N} - (r + b)\, I,\\
R' &= r\, I + v_i\, N + v_s\, S_a - (\delta + b)\, R,\\
M' &= \omega_o + \alpha_o\, I + \frac{\alpha\, S_a}{N} - \lambda_o\, M,
\end{aligned} \tag{4.2}$$

with the initial conditions

$$\begin{gathered}
S_n(0) \geq 0, \quad S_a(0) \geq 0, \quad I(0) > 0, \quad R(0) \geq 0, \quad M(0) \geq 0\\
\text{and} \quad M(s) \geq 0 \quad \text{for all} \quad s \in [-\tau, 0).
\end{gathered} \tag{4.3}$$

The model diagram for (4.2) is represented by Figure 4.1 showing all the transitions between the compartments.

Positivity of solutions

Since the model (4.2) with the initial conditions (4.3) represents the dynamics of human population, it is essential to show that its solutions are positive and bounded for all $t \in [0, \infty)$.

Theorem 4. *The solutions, $S_n(t), S_a(t), I(t), R(t), M(t)$, of the system (4.2) with the initial conditions (4.3) are non-negative and bounded for all $t \geq 0$.*

Proof.

Considering the equation for $I(t)$, let $t_i > 0$ be the first time when $I(t) = 0$, and the other components are still non-negative as per initial conditions, so

$$S_n(t) \geq 0, \quad S_a(t) \geq 0 \qquad \text{for all} \quad t \in [0, t_i].$$

Introducing an auxiliary quantity

$$\psi_2 = \min_{0 \leq t \leq t_i} \left\{ \frac{\beta S_n}{N} + \frac{\sigma_s \beta S_a}{N} - (r + b) \right\},$$

we have the relation

$$I' = \frac{\beta I S_n}{N} + \frac{\sigma_s \beta I S_a}{N} - (r + b) I \geq \psi_2 I,$$

that can be readily solved to yield

$$I(t_i) \geq I(0) e^{\psi_2 t_i} > 0,$$

which gives a contradiction.

In a similar way, let us assume there exists a first time $t_n > 0$ such that $S_n(t) > 0$ for $t \in [0, t_n)$ and $S_n(t_n) = 0$, which implies $dS_n(t_n)/dt < 0$. Substituting this value of S_n into the first equation of the system (4.2) gives

$$\left. \frac{dS_n}{dt} \right|_{t=t_n} = (b - v_i)\, N + \lambda\, S_a + \delta\, q\, R > 0,$$

which contradicts the initial assumption. Consequently, $S_n(t) > 0$ for $t \geq 0$, and similar arguments can be used to establish that S_a, R and M remain non-negative for all $t \geq 0$.

Having established the positivity of all state variables, from the fact that $S_n(t) + S_a(t) + I(t) + R(t) = N = \text{const}$, it immediately follows that they are also all bounded between 0 and N. Looking at the last equation of the system (4.2), we have

$$M' = \omega_o + \alpha_o\, I + \frac{\alpha\, S_a}{N} - \lambda_o\, M \leq \omega_o + \alpha_o\, N + \alpha - \lambda_o\, M,$$

which can be solved to give

$$M(t) \leq M(0) e^{-\lambda_o t} + \frac{\omega_o + \alpha_o N + \alpha}{\lambda_o} \left(1 - e^{-\lambda_o t}\right) \leq \widehat{M},$$

where

$$\widehat{M} = M(0) + \frac{\omega_o + \alpha_o N + \alpha}{\lambda_o}. \tag{4.4}$$

This suggests that throughout the time evolution, all solutions remain within the bounded region

$$\Phi_2 = \left\{ (S_n,\, S_a,\, I,\, R,\, M) \,\in\, \mathbb{R}^5_+ :\, 0 \,< S_n,\, S_a,\, I,\, R \,\leq\, N,\, 0 \leq M \leq \widehat{M} \right\}.$$

■

4.2 Steady states

The system of equations (4.2) can have at most two steady states, a *disease-free equilibrium* and an *endemic equilibrium*. They can be found by solving the following system of equations

$$\begin{aligned}
0 &= (b - v_i)\, N - \frac{\beta\, I\, S_n}{N} - \eta\, M\, S_n + \lambda\, S_a + \delta\, q\, R - b\, S_n, \\
0 &= -\frac{\sigma_s\, \beta\, I\, S_a}{N} + \eta\, M\, S_n - (\lambda + v_s + b)\, S_a + \delta\, p\, R, \\
0 &= \frac{\beta\, I\, S_n}{N} + \frac{\sigma_s\, \beta\, I\, S_a}{N} - (r + b)\, I, \\
0 &= r\, I + v_i\, N + v_s\, S_a - (\delta + b)\, R, \\
0 &= \omega_o + \alpha_o\, I + \frac{\alpha\, S_a}{N} - \lambda_o\, M.
\end{aligned}$$

Since $N = S_n + S_a + I + R$, substituting $R = N - S_n - S_a - I$ gives

$$\begin{aligned}
0 &= (b + \delta\, q - v_i)\, N - \frac{\beta\, I\, S_n}{N} - (\eta\, M + \delta\, q + b)\, S_n + (\lambda - \delta\, q)\, S_a - \delta\, q\, I, \\
0 &= -\frac{\sigma_s\, \beta\, I\, S_a}{N} + (\eta\, M - \delta\, p)\, S_n - (\lambda + \delta\, p + v_s + b)\, S_a + \delta\, p\, (N - I), \\
0 &= \frac{\beta\, I\, S_n}{N} + \frac{\sigma_s\, \beta\, I\, S_a}{N} - (r + b)\, I, \\
0 &= \omega_o + \alpha_o\, I + \frac{\alpha\, S_a}{N} - \lambda_o\, M,
\end{aligned} \tag{4.5}$$

which reduces the total number of equations without affecting the system dynamics since the total population, N is constant.

Disease-free state

The third equation of (4.5) gives

$$\left[\frac{\beta\, S_n}{N} + \frac{\sigma_s\, \beta\, S_a}{N} - (r + b) \right] I = 0,$$

which implies

$$I = 0 \qquad \text{or} \qquad \frac{\beta\, S_n}{N} + \frac{\sigma_s\, \beta\, S_a}{N} - (r + b) = 0.$$

For $I = 0$, the other three equations reduce to

$$\begin{aligned} 0 &= (b + \delta\, q - v_i)\, N - (\eta\, M + \delta\, q + b)\, S_n + (\lambda - \delta\, q)\, S_a, \\ 0 &= (\eta\, M - \delta\, p)\, S_n - (\lambda + \delta\, p + v_s + b)\, S_a + \delta\, p\, N, \\ 0 &= \omega_o + \frac{\alpha\, S_a}{N} - \lambda_o\, M. \end{aligned} \tag{4.6}$$

Hence, we have

$$M = \frac{N\, \omega_o + \alpha\, S_a}{N\, \lambda_o},$$

and from the first equation of the system (4.6) one finds

$$S_n = \frac{N\, \lambda_o\, [N\, (b + \delta\, q - v_i) + (\lambda - \delta\, q)\, S_a]}{N\, [\eta\, \omega_o + \lambda_o\, (\delta\, q + b)] + \eta\, \alpha\, S_a}. \tag{4.7}$$

On the other hand, adding the first two equations of the system (4.6) yields

$$(b + \delta\, q + \delta\, p - v_i)\, N - (\delta\, q + \delta\, p + b)\, S_n - (\delta\, q + \delta\, p + v_s + b)\, S_a = 0,$$

$$S_n = \frac{N\, (b + \delta - v_i) - (\delta + v_s + b)\, S_a}{\delta + b}, \tag{4.8}$$

which shows that S_a must satisfy the condition $S_a < \frac{N\,(b+\delta-v_i)}{\delta+v_s+b}$ to ensure that $S_n > 0$. Equating the two expressions for S_n in (4.7) and (4.8), we obtain

$$\frac{N\, \lambda_o\, [N\, (b + \delta\, q - v_i) + (\lambda - \delta\, q)\, S_a]}{N\, [\eta\, \omega_o + \lambda_o\, (\delta\, q + b)] + \eta\, \alpha\, S_a} = \frac{N\, (b + \delta - v_i) - (\delta + v_s + b)\, S_a}{\delta + b},$$

which can be rewritten as the following quadratic equation

$$z_1\, S_a^2 + N\, z_2\, S_a - N^2\, z_3 = 0,$$

where $z_1 = \eta\, \alpha\, (\delta + v_s + b)$,

$z_2 = \lambda_o\, (\delta + b)\, (\lambda + b) + \lambda_o\, v_s\, (\delta\, q + b) + \eta\, \omega_o\, (\delta + v_s + b) - \eta\, \alpha\, (b + \delta - v_i)$,

$z_3 = \lambda_o\, \delta\, p\, v_i + \eta\, \omega_o\, (b + \delta - v_i)$,

with the roots

$$S_a = N \left[\frac{-z_2 \pm \sqrt{z_2^2 + 4\, z_1\, z_3}}{2\, z_1} \right].$$

Since we know that $b - v_i > 0$, it means that $z_3 > 0$, and $z_1 > 0$, and therefore,

$$S_a = N \left[\frac{-z_2 + \sqrt{z_2^2 + 4\, z_1\, z_3}}{2\, z_1} \right] \qquad \Rightarrow \qquad S_a = N\, h_a,$$

and with $S_a < \frac{N\,(b+\delta-v_i)}{\delta+v_s+b}$, it follows that $h_a = \frac{-z_2+\sqrt{z_2^2+4\,z_1\,z_3}}{2\,z_1} < \frac{b+\delta-v_i}{\delta+v_s+b} < 1$. Consequently,

$$M = \frac{N\,\omega_o + \alpha\,S_a}{N\,\lambda_o} = \frac{\omega_o + \alpha\,h_a}{\lambda_o},$$

and

$$S_n = \frac{N\,\lambda_o\,[b - v_i + \delta\,q\,(1-h_a) + \lambda\,h_a]}{\eta\,(\omega_o + \alpha\,h_a) + \lambda_o\,(\delta\,q + b)} = N\,h_n,$$

where $h_n = \dfrac{\lambda_o\,[b - v_i + \delta\,q\,(1-h_a) + \lambda\,h_a]}{\eta\,(\omega_o + \alpha\,h_a) + \lambda_o\,(\delta\,q + b)}$, $0 < h_n < 1$, and

$$R = N - S_n - S_a = N(1 - h_n - h_a).$$

This gives the disease-free steady state $E_0 = (S_n^\circ, S_a^\circ, 0, R^\circ, M^\circ)$ where

$$S_n^\circ = N\,h_n, \quad S_a^\circ = N\,h_a, \quad R^\circ = N\,(1-h_n-h_a), \quad \text{and} \quad M^\circ = \frac{\omega_o + \alpha\,h_a}{\lambda_o}. \tag{4.9}$$

with

$$\begin{aligned} h_n &= \frac{\lambda_o\,[b - v_i + \delta\,q\,(1-h_a) + \lambda\,h_a]}{\eta\,(\omega_o + \alpha\,h_a) + \lambda_o\,(\delta\,q + b)}, \qquad 0 < h_n < 1, \qquad \text{and} \\ h_a &= \frac{-z_2 + \sqrt{z_2^2 + 4\,z_1\,z_3}}{2\,z_1}, \qquad 0 < h_a < \frac{b+\delta-v_i}{\delta+v_s+b}. \end{aligned} \tag{4.10}$$

The steady state E_0 is biologically feasible, as long as the condition $h_a < \dfrac{b+\delta-v_i}{\delta+v_s+b}$ holds.

Endemic state

Considering $I > 0$ in the third equation gives

$$S_a = \frac{N\,(r+b) - \beta\,S_n}{\sigma_s\,\beta}, \tag{4.11}$$

and $S_a > 0$, if $S_n < \frac{N\,(r+b)}{\beta}$.

From the fourth equation of (4.5) we have

$$M = \frac{N\,(\omega_o + \alpha_o\,I) + \alpha\,S_a}{N\,\lambda_o}, \tag{4.12}$$

and substituting this into the second equation of (4.5) gives

$$\begin{aligned} N\,[\eta\,(\omega_o + \alpha_o\,I) - \lambda_o\,\delta\,p]\,S_n + N\,\lambda_o\,\delta\,p\,(N-I) = \\ [N\,\lambda_o\,(\lambda + \delta\,p + v_s + b) + \lambda_o\,\sigma_s\,\beta\,I - \eta\,\alpha\,S_n]\,S_a. \end{aligned} \tag{4.13}$$

Substituting the value of S_a from (4.11) into (4.13) and simplifying yields

$$I = \frac{\tilde{m}_1\, S_n^2 - N\,\tilde{m}_2\, S_n + N^2\,\tilde{m}_3}{\sigma_s\,\beta\,(\tilde{m}_4\, S_n - N\,\tilde{m}_5)}, \tag{4.14}$$

where $\tilde{m}_1 = \beta\,\eta\,\alpha$, $\tilde{m}_2 = \beta\,\lambda_o\,[\lambda + v_s + b + \delta\,p(1-\sigma_s)] + \eta\,\alpha\,(r+b) + \sigma_s\,\beta\,\eta\,\omega_o$,
$\tilde{m}_3 = \lambda_o\,[(\lambda + \delta\,p + v_s + b)\,(r+b) - \sigma_s\,\beta\,\delta\,p]$, $\tilde{m}_4 = N\,\eta\,\alpha_o + \beta\,\lambda_o$,
$\tilde{m}_5 = \lambda_o\,(r + \delta\,p + b)$.

Lastly, from the first equation of (4.5) we obtain

$$(b + \delta\,q - v_i)\,N - \frac{\beta\,I\,S_n}{N} - (\eta\,M + \delta\,q + b)\,S_n + (\lambda - \delta\,q)\,S_a - \delta\,q\,I = 0,$$

and again, substituting the value of M from (4.12) gives

$$N^2\lambda_o\,(b + \delta\,q - v_i) - N[\eta\,\omega_o + \lambda_o\,(\delta\,q + b)]\,S_n + [N\lambda_o\,(\lambda - \delta\,q) - \eta\,\alpha\,S_n]S_a = [N\lambda_o\,\delta\,q + (\beta\,\lambda_o + N\eta\,\alpha_o)\,S_n]I.$$

Substituting the value of S_a from (4.11) gives after simplification

$$I = \frac{\tilde{m}_1\, S_n^2 - N\,\tilde{m}_6\, S_n + N^2\,\tilde{m}_7}{\sigma_s\,\beta\,(N\,\lambda_o\,\delta\,q + \tilde{m}_4\, S_n)}, \tag{4.15}$$

where $\tilde{m}_6 = \beta\,\lambda_o\,(\lambda + \sigma_s\,\delta\,q + \sigma_s\,b - \delta\,q) + \sigma_s\,\beta\,\eta\,\omega_o + \eta\,\alpha\,(r+b)$,
$\tilde{m}_7 = \lambda_o\,[\sigma_s\,\beta\,(b + \delta\,q - v_i) + (\lambda - \delta\,q)\,(r+b)]$.

Equating the two expressions for I from (4.14) and (4.15), we have

$$z_4\, S_n^2 - N\, z_5\, S_n + N^2\, z_6 = 0,$$

where $z_4 = \tilde{m}_1\,(\lambda_o\,\delta\,q + \tilde{m}_5) + \tilde{m}_4\,(\tilde{m}_6 - \tilde{m}_2)$, $z_5 = \lambda_o\,\delta\,q\,\tilde{m}_2 + \tilde{m}_5\,\tilde{m}_6 + \tilde{m}_4\,(\tilde{m}_7 - \tilde{m}_3)$,
$z_6 = \lambda_o\,\delta\,q\,\tilde{m}_3 + \tilde{m}_5\,\tilde{m}_7$.

This equation can be readily solved to give

$$S_n = N\left[\frac{z_5 \pm \sqrt{z_5^2 - 4\,z_4\,z_6}}{2\,z_4}\right] = N\,h_{n_*},$$

with $h_{n_*} = \frac{z_5 \pm \sqrt{z_5^2 - 4\,z_4\,z_6}}{2\,z_4}$ and $0 < S_n < \frac{N\,(r+b)}{\beta} \;\Rightarrow\; 0 < h_{n_*} < \frac{r+b}{\beta}$.

Thus, we have the endemic equilibrium state $E^* = (S_n^*, S_a^*, I^*, R^*, M^*)$ where

$$\begin{aligned} S_n^* &= N\,h_{n_*} \quad S_a^* = N\,h_{a_*}, \quad I^* = N\,h_{i_*}, \\ R^* &= N\,(1 - h_{n_*} - h_{a_*} - h_{i_*}), \quad M^* = \frac{N\,h_{i_*}\,\alpha_o + \omega_o + \alpha\,h_{a_*}}{\lambda_o}, \end{aligned} \tag{4.16}$$

with

$$h_{n_*} = \frac{z_5 \pm \sqrt{z_5^2 - 4\,z_4\,z_6}}{2\,z_4}, \quad h_{a_*} = \frac{r + b - \beta\,h_{n_*}}{\sigma_s\,\beta}, \quad h_{i_*} = \frac{h_{n_*}\,(\tilde{m}_1\,h_{n_*} - \tilde{m}_6) + \tilde{m}_7}{\sigma_s\,\beta\,(\lambda_o\,\delta\,q + \tilde{m}_4\,h_{n_*})}.$$

The endemic steady state E^* is biologically feasible, provided $0 < h_{n_*} < \dfrac{r+b}{\beta}$.

Consequently, the system of equations (4.2) has the following steady states,

$$E_0 = (S_n^\circ, S_a^\circ, 0, R^\circ, M^\circ) \qquad \text{and} \qquad E^* = (S_n^*, S_a^*, I^*, R^*, M^*).$$

4.3 Stability analysis

Linearising the system of equations (4.2) near each steady state gives the Jacobian matrix

$$J_q = \begin{pmatrix} -(a_5{+}a_6{+}\delta\,q{+}b) & \lambda{-}\delta\,q & -(a_2{+}\delta\,q) & -a_\tau\,\tilde{a}_6 \\ a_6{-}\delta\,p & -(\tilde{a}_5{+}\lambda{+}\delta\,p{+}v_s{+}b) & -(\tilde{a}_0{+}\delta\,p) & a_\tau\,\tilde{a}_6 \\ a_5 & \tilde{a}_5 & a_2{+}\tilde{a}_0{-}(r{+}b) & 0 \\ 0 & a_7 & \alpha_o & -\lambda_o \end{pmatrix}, \tag{4.17}$$

where $a_\tau = e^{-k\,\tau}$, $\tilde{a}_0 = \frac{\sigma_s\,\beta\,S_a}{N}$, $a_2 = \frac{\beta\,S_n}{N}$, $a_5 = \frac{\beta\,I}{N}$, $\tilde{a}_5 = \frac{\sigma_s\,\beta\,I}{N}$, $a_6 = \eta\,M$, $\tilde{a}_6 = \eta\,S_n$, $a_7 = \frac{\alpha}{N}$.

Stability of the disease-free state

Theorem 5. *The disease-free equilibrium state E_0 of the system (4.2) is linearly asymptotically stable for all $\tau \geq 0$ if the basic reproductive number satisfies the condition $R_v^d < 1$, where*

$$R_v^d = \frac{\beta\,(h_n + \sigma_s\,h_a)}{r + b}. \tag{4.18}$$

Proof

Linearisation of the system (4.2) near its disease-free equilibrium state $E_0 = (S_n^\circ,\ S_a^\circ,\ 0,\ R^\circ,\ M^\circ)$ with $S_n^\circ > 0,\ S_a^\circ > 0,\ R^\circ > 0$ and $M^\circ > 0$ yields the following

Jacobian matrix

$$J_q^\circ = \begin{pmatrix} -(a_6+\delta\,q+b) & \lambda-\delta\,q & -(a_2+\delta\,q) & -a_\tau\,\tilde{a}_6 \\ a_6-\delta\,p & -(\lambda+\delta\,p+v_s+b) & -(\tilde{a}_0+\delta\,p) & a_\tau\,\tilde{a}_6 \\ 0 & 0 & a_2+\tilde{a}_0-(r+b) & 0 \\ 0 & a_7 & \alpha_o & -\lambda_o \end{pmatrix},$$

with the characteristic equation

$$(k+r+b-a_2-\tilde{a}_0)\,[k^3+\tilde{y}_5\,k^2+\tilde{y}_6\,k+\tilde{y}_7-a_\tau\,(\tilde{y}_8\,k+\tilde{y}_9)]=0,$$

where

$$\begin{aligned} &a_\tau = e^{-k\,\tau},\ \ \tilde{a}_0=\frac{\sigma_s\,\beta\,S_a^\circ}{N},\ \ a_2=\frac{\beta\,S_n^\circ}{N},\ \ a_6=\eta\,M^\circ,\ \ \tilde{a}_6=\eta\,S_n^\circ,\ \ a_7=\frac{\alpha}{N}, \\ &g_1=\lambda+b+a_6,\ \ g_2=\lambda_o+\delta+b,\ \ g_3=\delta+b+v_s,\ \ g_4=\delta\,q+b+a_6, \\ &\tilde{y}_5=g_1+g_2+v_s,\ \ \tilde{y}_6=g_1\,g_2+\lambda_o\,g_3+v_s\,g_4,\ \ \tilde{y}_7=\lambda_o\,[v_s\,g_4+g_1\,(\delta+b)], \\ &\tilde{y}_8=\tilde{a}_6\,a_7,\ \ \tilde{y}_9=\tilde{a}_6\,a_7\,(\delta+b). \end{aligned} \tag{4.19}$$

The first eigenvalue $k=a_2+\tilde{a}_0-(r+b)$ is negative whenever

$$a_2+\tilde{a}_0-(r+b)<0 \qquad\Longleftrightarrow\qquad \frac{\beta\,(h_n+\sigma_s\,h_a)}{r+b}<1 \qquad\Longleftrightarrow\qquad R_v^d<1,$$

with R_v^d defined in (4.18).

Other eigenvalues can be found as the roots of the transcendental equation

$$k^3+\tilde{y}_5\,k^2+\tilde{y}_6\,k+\tilde{y}_7=(\tilde{y}_8\,k+\tilde{y}_9)\,e^{-k\,\tau}. \tag{4.20}$$

For $\tau=0,$ this equation turns into a simple cubic

$$k^3+\tilde{y}_5\,k^2+(\tilde{y}_6-\tilde{y}_8)\,k+\tilde{y}_7-\tilde{y}_9=0, \tag{4.21}$$

whose roots all have a negative real part if and only if the following Routh-Hurwitz conditions hold

$$\tilde{y}_5>0,\quad \tilde{y}_6-\tilde{y}_8>0,\quad \tilde{y}_7-\tilde{y}_9>0,\quad \text{and}\quad \tilde{y}_5\,(\tilde{y}_6-\tilde{y}_8)>\tilde{y}_7-\tilde{y}_9. \tag{4.22}$$

The first of these conditions $\tilde{y}_5=g_1+g_2+v_s>0$ holds, since $g_1,g_2>0$ in accordance with (4.19). The second Routh-Hurwitz condition can be written as

$$\begin{aligned} \tilde{y}_6-\tilde{y}_8 &= g_1\,g_2+\lambda_o\,g_3+v_s\,g_4-\tilde{a}_6\,a_7 \\ &= g_1\,(\delta+b)+\lambda_o\,g_3+v_s\,g_4+(\lambda_o\,g_1-\tilde{a}_6\,a_7)>0. \end{aligned}$$

Since

$$h_n = \frac{\lambda_o\,[b - v_i + \delta\,q\,(1 - h_a) + \lambda\,h_a]}{\eta\,(\omega_o + \alpha\,h_a) + \lambda_o\,(\delta\,q + b)} < 1,$$

this relation can be equivalently rewritten as

$$\eta\,(\omega_o + \alpha\,h_a) > \lambda_o\,(\lambda\,h_a - \delta\,q\,h_a - v_i) \quad \Rightarrow \quad h_a < \frac{\eta\,\omega_o + \lambda_o\,v_i}{\lambda\,\lambda_o - \eta\,\alpha - \lambda_o\,\delta\,q},$$

which in light of $0 < h_a < 1$, gives $\lambda\,\lambda_o > \eta\,\alpha\,h_n$, and, subsequently,

$$\lambda_o\,g_1 - \tilde{a}_6\,a_7 = \lambda_o\,(\lambda + b) + \eta\,(\omega_o + \alpha\,h_a) - \eta\,\alpha\,h_n > 0, \tag{4.23}$$

thus implying

$$\tilde{y}_6 - \tilde{y}_8 > 0.$$

Hence, the second Routh-Hurwitz condition in (4.22) is also satisfied.

The third condition has the form

$$\begin{aligned}\tilde{y}_7 - \tilde{y}_9 &= \lambda_o\,[v_s\,g_4 + g_1\,(\delta + b)] - \tilde{a}_6\,a_7\,(\delta + b)\\ &= \lambda_o v_s\,g_4 + (\delta + b)(\lambda_o\,g_1 - \tilde{a}_6\,a_7) > 0,\end{aligned}$$

and it is also satisfied due to above relation $\lambda_o\,g_1 - \tilde{a}_6\,a_7 > 0$.

Similarly, the last condition

$$\tilde{y}_5\,(\tilde{y}_6 - \tilde{y}_8) > \tilde{y}_7 - \tilde{y}_9,$$

can be recast in the form

$$(g_1 + g_2 + v_s)[\lambda_o\,g_3 + g_1(\delta + b)] + v_s\,g_4(g_1 + g_3) + (g_1 + \lambda_o + v_s)(\lambda_o\,g_1 - \tilde{a}_6\,a_7) > 0,$$

which immediately shows that it is also always satisfied. Hence, one can conclude that for $\tau = 0$, the disease-free steady state E_0 is linearly asymptotically stable provided $R_v^d < 1$.

To investigate whether stability can be lost for $\tau > 0$, we look for solutions of equation (4.20) in the form $k = i\,\mu$, which gives

$$\begin{aligned}-i\,\mu^3 - \tilde{y}_5\,\mu^2 + \tilde{y}_6\,i\,\mu + \tilde{y}_7 &= (\tilde{y}_8\,i\,\mu + \tilde{y}_9)\,e^{-i\,\mu\,\tau}\\ &= (\tilde{y}_8\,i\,\mu + \tilde{y}_9)[\cos(\mu\,\tau) - i\,\sin(\mu\,\tau)].\end{aligned}$$

Equating real and imaginary parts we obtain

$$\begin{aligned}-\tilde{y}_5\,\mu^2 + \tilde{y}_7 &= \tilde{y}_8\,\mu\,\sin(\mu\,\tau) + \tilde{y}_9\,\cos(\mu\,\tau),\\ -\mu^3 + \tilde{y}_6\,\mu &= \tilde{y}_8\,\mu\,\cos(\mu\,\tau) - \tilde{y}_9\,\sin(\mu\,\tau),\end{aligned} \tag{4.24}$$

and by squaring both equations in (4.24) and summing the results give

$$(-\tilde{y}_5\,\mu^2+\tilde{y}_7)^2+(-\mu^3+\tilde{y}_6\,\mu)^2=\tilde{y}_8^2\,\mu^2+\tilde{y}_9^2,$$

$$\Rightarrow \qquad \mu^6+(\tilde{y}_5^2-2\,\tilde{y}_6)\,\mu^4+(\tilde{y}_6^2-2\,\tilde{y}_5\,\tilde{y}_7-\tilde{y}_8^2)\,\mu^2+\tilde{y}_7^2-\tilde{y}_9^2=0.$$

Substituting $\tilde{x}_1=\tilde{y}_5^2-2\,\tilde{y}_6$, $\tilde{x}_2=\tilde{y}_6^2-2\,\tilde{y}_5\,\tilde{y}_7-\tilde{y}_8^2$, $\tilde{x}_3=\tilde{y}_7^2-\tilde{y}_9^2$, yields

$$\mu_p^3+\tilde{x}_1\,\mu_p^2+\tilde{x}_2\,\mu_p+\tilde{x}_3=0, \qquad \mu_p=\mu^2, \tag{4.25}$$

so if one can show that there are no real positive roots μ_p of this equation, then no eigenvalues of the equation (4.20) can even cross the imaginary axis, thus implying the stability of the disease-free steady state. We will once again use the Routh-Hurwitz criteria to show that all roots of the cubic equation (4.25) have a negative real part, which is true if and only if

$$\tilde{x}_1>0, \qquad \tilde{x}_2>0, \qquad \tilde{x}_3>0, \qquad \text{and} \qquad \tilde{x}_1\,\tilde{x}_2>\tilde{x}_3.$$

It is straightforward to show that the first three of these conditions holds,

$$\begin{aligned}\rightarrow \quad \tilde{x}_1&=\tilde{y}_5^2-2\,\tilde{y}_6=(g_1+g_2+v_s)^2-2\,(g_1g_2+\lambda_o\,g_3+v_s\,g_4)\\&=g_1^2+\lambda_o^2+(\delta+b)^2+v_s^2+2\,v_s\,(\lambda+\delta\,p+b)>0,\end{aligned}$$

the first condition is always satisfied.

$$\begin{aligned}\rightarrow \quad \tilde{x}_2&=\tilde{y}_6^2-2\,\tilde{y}_5\,\tilde{y}_7-\tilde{y}_8^2\\&=(g_1g_2+\lambda_o\,g_3+v_s\,g_4)^2-2\,\lambda_o\,(g_1+g_2+v_s)\,[v_s\,g_4+g_1\,(\delta+b)]-(\tilde{a}_6\,a_7)^2\\&=(\lambda_o\,g_1+\tilde{a}_6\,a_7)(\lambda_o\,g_1-\tilde{a}_6\,a_7)+\lambda_o^2\,[v_s^2+(\delta+b)^2+2\,v_s\,(\lambda+\delta\,p+b)]+\\&\quad\ [v_s\,g_4+g_1\,(\delta+b)]^2>0,\end{aligned}$$

using (4.23) shows that the condition holds.

$$\rightarrow \quad \tilde{x}_3=\tilde{y}_7^2-\tilde{y}_9^2=(\tilde{y}_7+\tilde{y}_9)(\tilde{y}_7-\tilde{y}_9)>0,$$

the third condition holds since $\tilde{y}_7-\tilde{y}_9>0$ as shown earlier.

$$\rightarrow \quad \tilde{x}_1\,\tilde{x}_2>\tilde{x}_3 \qquad \Rightarrow \qquad (\tilde{y}_5^2-2\,\tilde{y}_6)\,(\tilde{y}_6^2-2\,\tilde{y}_5\,\tilde{y}_7-\tilde{y}_8^2)>\tilde{y}_7^2-\tilde{y}_9^2,$$

which can be transformed into

$$
\begin{aligned}
&(\tilde{y}_5^2 - 2\,\tilde{y}_6)\,(\tilde{y}_6^2 - 2\,\tilde{y}_5\,\tilde{y}_7 - \tilde{y}_8^2) - \tilde{y}_7^2 + \tilde{y}_9^2 \\
&\quad = \Big[g_1^2 + \lambda_o^2 + (\delta+b)^2 + v_s^2 + 2\,v_s\,(\lambda + \delta\,p + b)\Big]\Big[(\lambda_o\,g_1 + \tilde{a}_6\,a_7)(\lambda_o\,g_1 - \tilde{a}_6\,a_7) + \\
&\qquad \lambda_o^2\,\big[v_s^2 + (\delta+b)^2 + 2\,v_s\,(\lambda + \delta\,p + b)\big] + \big[v_s\,g_4 + g_1\,(\delta+b)\big]^2\Big] - \\
&\qquad \lambda_o^2\,\big[v_s\,g_4 + g_1\,(\delta+b)\big]^2 + (\tilde{a}_6\,a_7)^2 \\
&\quad = \Big[g_1^2 + (\delta+b)^2 + v_s^2 + 2\,v_s\,(\lambda + \delta\,p + b)\Big]\Big[(\lambda_o\,g_1 + \tilde{a}_6\,a_7)(\lambda_o\,g_1 - \tilde{a}_6\,a_7) + \\
&\qquad \lambda_o^2\,\big[v_s^2 + (\delta+b)^2 + 2\,v_s\,(\lambda + \delta\,p + b)\big] + \big[v_s\,g_4 + g_1\,(\delta+b)\big]^2\Big] + (\tilde{a}_6\,a_7)^2 + \\
&\qquad \lambda_o^2\,(\lambda_o\,g_1 + \tilde{a}_6\,a_7)(\lambda_o\,g_1 - \tilde{a}_6\,a_7) + \lambda_o^4\,\big[v_s^2 + (\delta+b)^2 + 2\,v_s\,(\lambda + \delta\,p + b)\big] > 0,
\end{aligned}
$$

which shows that $\tilde{x}_1\,\tilde{x}_2 > \tilde{x}_3$, based on (4.23).

Therefore, all the roots μ_p of the cubic equation (4.25) have a negative real part. Thus, there are no purely imaginary roots $k = i\,\mu$ of the characteristic equation (4.20), and the disease-free steady state E_0 is stable if $R_v^d < 1$ for any $\tau \geq 0$. ■

Stability of the endemic state

Next, we turn our attention to the endemic steady state E^*. The characteristic equation for linearisation near this steady state has the form

$$
k^4 + k^3\,P_5 + k^2\,(P_6 - a_\tau\,\tilde{y}_8) + k\,(P_7 + a_\tau\,\tilde{P}_7) + P_8 + a_\tau\,\tilde{P}_8 = 0, \tag{4.26}
$$

where

$$
\begin{aligned}
P_5 &= a_5 + \tilde{a}_5 + g_1 + g_2 + v_s, \\
P_6 &= g_2\,(a_5 + \tilde{a}_5 + g_1) + v_s\,(a_5 + g_4) + \lambda_o\,g_3 + a_5\,(\lambda + a_2 + \tilde{a}_5) + \tilde{a}_5\,(\tilde{a}_0 + a_6), \\
P_7 &= \lambda_o[v_s\,(a_5 + g_4) + (\delta+b)(a_5 + \tilde{a}_5 + g_1)] + a_5[v_s\,(\delta\,q + a_2) + r\,(\lambda + \tilde{a}_5 - \delta\,q)] + \\
&\qquad \tilde{a}_5\,r\,(a_6 - \delta\,p) + g_2\,[a_5\,(\lambda + a_2 + \tilde{a}_5) + \tilde{a}_5\,(\tilde{a}_0 + a_6)], \\
\tilde{P}_7 &= \alpha_o\,\tilde{a}_6\,(a_5 - \tilde{a}_5) - \tilde{y}_8\,(\delta + b + a_5), \\
P_8 &= \lambda_o\big[a_5\,[v_s\,(\delta\,q + a_2) + \lambda\,(\delta+b) + r\,(\lambda + \tilde{a}_5 - \delta\,q)] + (\delta+b)\,[\tilde{a}_5\,(b + a_5 + a_6) + \\
&\qquad a_2\,(a_5 - \tilde{a}_5)] + \tilde{a}_5\,r\,g_4\big], \\
\tilde{P}_8 &= \alpha_o\,\tilde{a}_6\,[v_s\,a_5 + (\delta+b)\,(a_5 - \tilde{a}_5)] - \tilde{y}_8\,a_5\,(r + \delta + b),
\end{aligned}
$$

and

$$\begin{aligned} &a_\tau = e^{-k\,\tau},\ \ \tilde{a}_0 = \frac{\sigma_s\,\beta\,S_a^*}{N},\ \ a_2 = \frac{\beta\,S_n^*}{N},\ \ a_5 = \frac{\beta\,I^*}{N},\ \ \tilde{a}_5 = \frac{\sigma_s\,\beta\,I^*}{N},\\ &a_6 = \eta\,M^*,\ \ \tilde{a}_6 = \eta\,S_n^*,\ \ a_7 = \frac{\alpha}{N},\ \ g_1 = \lambda + b + a_6,\ \ g_2 = \lambda_o + \delta + b,\\ &g_3 = \delta + b + v_s,\ \ g_4 = \delta\,q + b + a_6,\ \ \tilde{y}_8 = \tilde{a}_6\,a_7. \end{aligned} \tag{4.27}$$

For $\tau = 0$, characteristic equation (4.26) turns into a quartic

$$k^4 + k^3\,P_5 + k^2\,(P_6 - \tilde{y}_8) + k\,(P_7 + \tilde{P}_7) + P_8 + \tilde{P}_8 = 0, \tag{4.28}$$

whose roots all have a negative real part if and only if the following Routh-Hurwitz conditions are satisfied

$$P_5 > 0, \qquad P_6 - \tilde{y}_8 > 0, \qquad P_7 + \tilde{P}_7 > 0, \qquad P_8 + \tilde{P}_8 > 0,$$

$$P_5\,[(P_6 - \tilde{y}_8)\,(P_7 + \tilde{P}_7) - P_5\,(P_8 + \tilde{P}_8)] > (P_7 + \tilde{P}_7)^2.$$

Since $P_5\,[(P_6 - \tilde{y}_8)\,(P_7 + \tilde{P}_7) - P_5\,(P_8 + \tilde{P}_8)] > (P_7 + \tilde{P}_7)^2$ can be rewritten as

$$P_5\,(P_6 - \tilde{y}_8)\,(P_7 + \tilde{P}_7) > P_5^2\,(P_8 + \tilde{P}_8) + (P_7 + \tilde{P}_7)^2,$$

it follows that the condition $P_7 + \tilde{P}_7 > 0$ is always satisfied provided

$$\begin{aligned} &P_5 > 0, \quad P_6 - \tilde{y}_8 > 0, \quad P_8 + \tilde{P}_8 > 0,\\ &P_5\,[(P_6 - \tilde{y}_8)\,(P_7 + \tilde{P}_7) - P_5\,(P_8 + \tilde{P}_8)] > (P_7 + \tilde{P}_7)^2. \end{aligned} \tag{4.29}$$

Note that equation (4.13) can be expressed in the form

$$\begin{aligned} (N\,\eta\,\omega_o + \eta\,\alpha\,S_a^*)\,S_n^* + N^2\,\lambda_o\,\delta\,p = N\,\lambda_o\,[(\lambda + \delta\,p + v_s + b)\,S_a^* + \delta\,p\,S_n^*] +\\ [N\,\lambda_o\,\delta\,p + \lambda_o\,\sigma_s\,\beta\,S_a^* - N\,\eta\,\alpha_o\,S_n^*]\,I^*, \end{aligned}$$

$$\begin{aligned} \Rightarrow \quad (N\,\eta\,\omega_o + \eta\,\alpha\,S_a^*)\,S_n^* + N^2\,\lambda_o\,\delta\,p = N\,\lambda_o\,[(\lambda + \delta\,p + v_s + b)\,S_a^* + \delta\,p\,S_n^*] +\\ [N\,\lambda_o\,(r + \delta\,p + b) - (N\,\eta\,\alpha_o + \beta\,\lambda_o)\,S_n^*]\,I^*, \end{aligned} \tag{4.30}$$

since $(N\,\eta\,\omega_o + \eta\,\alpha\,S_a^*)\,S_n^* + N^2\,\lambda_o\,\delta\,p > 0$ it follows that

$$N\,\lambda_o\,[(\lambda + \delta\,p + v_s + b)\,S_a^* + \delta\,p\,S_n^*] + [N\,\lambda_o\,(r + \delta\,p + b) - (N\,\eta\,\alpha_o + \beta\,\lambda_o)\,S_n^*]\,I^* > 0,$$

$$\Rightarrow \qquad I^* < \frac{N\,\lambda_o\,[(\lambda + \delta\,p + v_s + b)\,S_a^* + \delta\,p\,S_n^*]}{(N\,\eta\,\alpha_o + \beta\,\lambda_o)\,S_n^* - N\,\lambda_o\,(r + \delta\,p + b)},$$

the fact that $I^* > 0$, gives

$$(N\,\eta\,\alpha_o + \beta\,\lambda_o)\,S_n^* > N\,\lambda_o\,(r + \delta\,p + b) \quad \Rightarrow \quad \eta\,\alpha_o\,S_n^* + \frac{\beta\,\lambda_o\,S_n^*}{N} > \lambda_o\,(r + \delta\,p + b),$$

which implies

$$\alpha_o\,\tilde{a}_6 + \lambda_o\,a_2 > \lambda_o\,(r + \delta\,p + b). \tag{4.31}$$

By expressing (4.30) in the form

$$[(N\,\eta\,\alpha_o + \beta\,\lambda_o)\,S_n^* - N\,\lambda_o\,(r + \delta\,p + b)]\,I^* = N\,\lambda_o\,[(\lambda + \delta\,p + v_s + b)\,S_a^* + \delta\,p\,S_n^*] - (N\,\eta\,\omega_o + \eta\,\alpha\,S_a^*)\,S_n^* - N^2\,\lambda_o\,\delta\,p,$$

and using the relation $(N\,\eta\,\alpha_o + \beta\,\lambda_o)\,S_n^* > N\,\lambda_o\,(r + \delta\,p + b)$, it implies that

$$N\,\lambda_o\,[(\lambda + \delta\,p + v_s + b)\,S_a^* + \delta\,p\,S_n^*] - (N\,\eta\,\omega_o + \eta\,\alpha\,S_a^*)\,S_n^* - N^2\,\lambda_o\,\delta\,p > 0,$$

which can be recast as

$$N\,\lambda_o\,(\lambda + v_s + b)\,S_a^* > (N\,\eta\,\omega_o + \eta\,\alpha\,S_a^*)\,S_n^* + N\,\lambda_o\,\delta\,p\,(N - S_n^* - S_a^*),$$

$$N\,\lambda_o\,(\lambda + v_s + b)\,S_a^* > \eta\,\alpha\,S_a^*\,S_n^* \quad \Rightarrow \quad \lambda_o\,(\lambda + v_s + b) > \frac{\eta\,\alpha\,S_n^*}{N} = \tilde{a}_6\,a_7,$$

hence,

$$\lambda_o\,(\lambda + v_s + b) > \tilde{y}_8. \tag{4.32}$$

Also note that

$$a_5 - \tilde{a}_5 = \frac{\beta\,I^*}{N} - \frac{\sigma_s\,\beta\,I^*}{N} = \beta\,h_{i_*}\,(1 - \sigma_s) > 0 \quad \Rightarrow \quad a_5 > \tilde{a}_5. \tag{4.33}$$

Consequently, proving the above stability conditions in (4.29) we have

$\rightarrow \quad P_5 = a_5 + \tilde{a}_5 + g_1 + g_2 + v_s > 0,$

this first condition for stability is always satisfied.

$$\begin{aligned}
\rightarrow \quad P_6 - \tilde{y}_8 &= g_2\,(a_5 + \tilde{a}_5 + g_1) + v_s\,(a_5 + g_4) + \lambda_o\,g_3 + a_5\,(\lambda + a_2 + \tilde{a}_5) + \\
&\quad \tilde{a}_5\,(\tilde{a}_0 + a_6) - \tilde{y}_8 > g_2\,g_1 + \lambda_o\,g_3 - \tilde{y}_8 \\
&= (\lambda_o + \delta + b)\,(a_5 + \lambda + b + a_6) + \lambda_o\,(\delta + b + v_s) - \tilde{y}_8 \\
&> \lambda_o\,(\lambda + v_s + b) - \tilde{y}_8 > 0,
\end{aligned}$$

the second condition also holds due to (4.32).

$$\begin{aligned}
\rightarrow \quad P_8 + \tilde{P}_8 &= \lambda_o\big[a_5\,[v_s\,(\delta\,q + a_2) + \lambda\,(\delta + b) + r\,(\lambda + \tilde{a}_5 - \delta\,q)] + \tilde{a}_5\,r\,g_4 + \\
&\quad (\delta + b)\,[\tilde{a}_5\,(b + a_5 + a_6) + a_2\,(a_5 - \tilde{a}_5)]\big] + \alpha_o\,\tilde{a}_6\,[v_s\,a_5 + (\delta + b)\,(a_5 - \tilde{a}_5)] - \\
&\quad \tilde{y}_8\,a_5\,(r + \delta + b) \\
&= a_5\,v_s\,(\alpha_o\,\tilde{a}_6 + \lambda_o\,a_2 + \lambda_o\,\delta\,q) + a_5\,(r + \delta + b)\,[\lambda_o\,(\lambda + v_s + b) - \tilde{y}_8] - \\
&\quad \lambda_o\,a_5\,v_s\,(r + \delta + b) - \lambda_o\,a_5\,b\,(r + \delta + b) - \lambda_o\,a_5\,r\,\delta\,q + \lambda_o\,\tilde{a}_5\,[r\,(\delta\,q + a_5) + \\
&\quad (\delta + b)\,(r + b + a_5 + a_6)] + (a_5 - \tilde{a}_5)(\delta + b)(\alpha_o\,\tilde{a}_6 + \lambda_o\,a_2) \\
&= a_5\,v_s\,[\alpha_o\,\tilde{a}_6 + \lambda_o\,a_2 - \lambda_o\,(r + \delta\,p + b)] + a_5\,(r + \delta + b)\,[\lambda_o\,(\lambda + v_s + b) - \tilde{y}_8] + \\
&\quad \lambda_o\,a_5\,r\,\delta\,p + (a_5 - \tilde{a}_5)(\delta + b)[\alpha_o\,\tilde{a}_6 + \lambda_o\,a_2 - \lambda_o\,(r + b)] + \lambda_o\,\tilde{a}_5\,[r\,(\delta\,q + a_5) + \\
&\quad (\delta + b)(a_5 + a_6)] > 0,
\end{aligned}$$

this condition is satisfied for any parameter values since all the brackets are positive due to the relations in (4.31), (4.32) and (4.33) shown above.

$$\rightarrow \quad P_5\,\big[(P_6 - \tilde{y}_8)\,(P_7 + \tilde{P}_7) - P_5\,(P_8 + \tilde{P}_8)\big] > (P_7 + \tilde{P}_7)^2,$$

this expression can be rewritten as

$$(P_7 + \tilde{P}_7)\,\big[P_5\,(P_6 - \tilde{y}_8) - (P_7 + \tilde{P}_7)\big] - P_5^2\,(P_8 + \tilde{P}_8) > 0,$$

but

$$\begin{aligned}
P_5\,(P_6 - \tilde{y}_8) - (P_7 + \tilde{P}_7) &= (a_5 + \tilde{a}_5 + g_1 + g_2 + v_s)\big[g_2\,(a_5 + \tilde{a}_5 + g_1) + v_s\,(a_5 + g_4) + \\
&\quad \lambda_o\,g_3 + a_5\,(\lambda + a_2 + \tilde{a}_5) + \tilde{a}_5\,(\tilde{a}_0 + a_6) - \tilde{y}_8\big] - \big[\lambda_o[v_s\,(a_5 + g_4) + \\
&\quad (\delta + b)(a_5 + \tilde{a}_5 + g_1)] + a_5[v_s\,(\delta\,q + a_2) + r\,(\lambda + \tilde{a}_5 - \delta\,q)] + \tilde{a}_5\,r\,(a_6 - \delta\,p) + \\
&\quad g_2\,[a_5\,(\lambda + a_2 + \tilde{a}_5) + \tilde{a}_5\,(\tilde{a}_0 + a_6)] + \alpha_o\,\tilde{a}_6\,(a_5 - \tilde{a}_5) - \tilde{y}_8\,(\delta + b + a_5)\big] \\
&= (\lambda_o + v_s + \tilde{a}_5 + g_1)\,[\lambda_o\,(\lambda + v_s + b) - \tilde{y}_8] + \lambda_o\,[P_5\,(\delta + b + a_5 + \tilde{a}_5 + a_6) + \\
&\quad (\delta + b + a_5)(\lambda + v_s + b)] + (a_5 + \tilde{a}_5 + g_1)[g_3\,(\delta + b + a_5) + (\delta + b)(\tilde{a}_5 + g_1)] + \\
&\quad \tilde{a}_5\,[(\tilde{a}_0 + a_6)(v_s + \tilde{a}_5 + g_1) + r\,\delta\,p + \alpha_o\,\tilde{a}_6] + v_s\,(a_5 + g_4)(\tilde{a}_5 + g_1 + g_3) + \\
&\quad a_6\,r\,(a_5 - \tilde{a}_5) + a_5\,[\tilde{a}_5\,(\tilde{a}_5 + g_1) + r\,(\delta\,q + b) + g_1\,(\lambda + b + \tilde{a}_5 - \tilde{a}_0) - \alpha_o\,\tilde{a}_6],
\end{aligned}$$

and $P_8 + \tilde{P}_8$ can also be express as

$$\begin{aligned}
P_8 + \tilde{P}_8 = \lambda_o\,(P_7 + \tilde{P}_7) + \alpha_o\,a_5\,\tilde{a}_6\,g_3 - \big[\lambda_o\,(\delta + b + a_5)[\lambda_o\,(\lambda + v_s + b) - \tilde{y}_8] + \\
a_5\,[\lambda_o^2(\delta + a_2) + \tilde{y}_8\,(r + \delta + b)] + \tilde{a}_5\,[(\delta + b)(\alpha_o\,\tilde{a}_6 + \lambda_o^2) + \\
\lambda_o^2(a_5 + \tilde{a}_0 + a_6)] + \lambda_o^2(a_6\,g_3 - v_s\,\delta\,p) + \alpha_o\,\tilde{a}_6\,\lambda_o\,(a_5 - \tilde{a}_5)\big].
\end{aligned}$$

Therefore,

$$
\begin{aligned}
&(P_7+\tilde{P}_7)\,[P_5\,(P_6-\tilde{y}_8)-(P_7+\tilde{P}_7)]-P_5^2\,(P_8+\tilde{P}_8)\\
&\quad=(P_7+\tilde{P}_7)\Big[(\lambda_o+v_s+\tilde{a}_5+g_1)\,\big[\lambda_o\,(\lambda+v_s+b)-\tilde{y}_8\big]-\lambda_o\,\big[\lambda_o\,P_5+\\
&\qquad(\lambda_o+v_s+\tilde{a}_5+g_1)(\lambda+v_s+b)\big]+(a_5+\tilde{a}_5+g_1)\big[g_3\,(\delta+b+a_5)+\\
&\qquad(\delta+b)(\tilde{a}_5+g_1)\big]+\tilde{a}_5\,\big[(\tilde{a}_0+a_6)(v_s+\tilde{a}_5+g_1)+r\,\delta\,p+\alpha_o\,\tilde{a}_6\big]+\\
&\qquad v_s\,(a_5+g_4)(\tilde{a}_5+g_1+g_3)+a_6\,r\,(a_5-\tilde{a}_5)+a_5\,\big[\tilde{a}_5\,(\tilde{a}_5+g_1)+r\,(\delta\,q+b)+\\
&\qquad g_1\,(\lambda+b+\tilde{a}_5-\tilde{a}_0)-\alpha_o\,\tilde{a}_6\big]\Big]+P_5^2\Big[\lambda_o\,(\delta+b+a_5)\big[\lambda_o\,(\lambda+v_s+b)-\tilde{y}_8\big]+\\
&\qquad a_5\big[\lambda_o^2(\delta+a_2)+\tilde{y}_8\,(r+\delta+b)\big]+\tilde{a}_5\big[(\delta+b)(\alpha_o\,\tilde{a}_6+\lambda_o^2)+\lambda_o^2(a_5+\tilde{a}_0+a_6)\big]+\\
&\qquad \lambda_o^2(a_6\,g_3-v_s\,\delta\,p)+\alpha_o\,\tilde{a}_6\,\lambda_o\,(a_5-\tilde{a}_5)-\alpha_o\,a_5\,\tilde{a}_6\,g_3\Big]>0.
\end{aligned}
$$

Consequently, we obtain the result.

Lemma 2. *Let the condition*

$$
(P_7+\tilde{P}_7)\,[P_5\,(P_6-\tilde{y}_8)-(P_7+\tilde{P}_7)]-P_5^2\,(P_8+\tilde{P}_8)>0 \tag{4.34}
$$

hold, then the endemic steady state E^* is linearly asymptotically stable for $\tau=0$.

Remark 2. *Although it does not appear possible to prove that the condition (4.34) is automatically satisfied, extensive numerical simulations show that it does indeed hold for any parameter values, for which the endemic steady state E^* is biologically feasible.*

Having established stability of the endemic state E^* for $\tau=0$, the next step in the analysis is to investigate whether this steady state can lose stability for $\tau>0$, in which case the characteristic equation (4.26) has the explicit form

$$
k^4+P_5\,k^3+P_6\,k^2+P_7\,k+P_8=(\tilde{y}_8\,k^2-\tilde{P}_7\,k-\tilde{P}_8)\,e^{-k\,\tau}, \tag{4.35}
$$

In order for the steady state E^* to lose its stability, some of the eigenvalues as determined by this equation must cross the imaginary axis. Looking for solutions in the form $k=i\mu$ gives

$$
\begin{aligned}
\mu^4-P_5\,i\,\mu^3-P_6\,\mu^2+P_7\,i\,\mu+P_8&=(-\tilde{y}_8\,\mu^2-\tilde{P}_7\,i\,\mu-\tilde{P}_8)\,e^{-i\,\mu\,\tau}\\
&=(-\tilde{y}_8\,\mu^2-\tilde{P}_7\,i\,\mu-\tilde{P}_8)\,[\cos(\mu\,\tau)-i\,\sin(\mu\,\tau)].
\end{aligned}
$$

Separating real and imaginary parts, we obtain

$$\begin{aligned} \mu^4 - P_6\,\mu^2 + P_8 &= -[(\tilde{y}_8\,\mu^2 + \tilde{P}_8)\,\cos(\mu\,\tau) + \tilde{P}_7\,\mu\,\sin(\mu\,\tau)], \\ -P_5\,\mu^3 + P_7\,\mu &= (\tilde{y}_8\,\mu^2 + \tilde{P}_8)\,\sin(\mu\,\tau) - \tilde{P}_7\,\mu\,\cos(\mu\,\tau). \end{aligned} \tag{4.36}$$

Squaring and adding these two equations gives

$$\mu^8 + \tilde{x}_4\,\mu^6 + \tilde{x}_5\,\mu^4 + \tilde{x}_6\,\mu^2 + \tilde{x}_7 = 0, \tag{4.37}$$

where $\tilde{x}_4 = P_5^2 - 2\,P_6$, $\tilde{x}_5 = 2\,P_8 + P_6^2 - 2\,P_5\,P_7 - \tilde{y}_8^2$, $\tilde{x}_6 = P_7^2 - 2\,P_6\,P_8 - \tilde{P}_7^2 - 2\,\tilde{y}_8\,\tilde{P}_8$, $\tilde{x}_7 = P_8^2 - \tilde{P}_8^2$.

Considering equation (4.37) gives the following equation for the Hopf frequency

$$f(\mu) = \mu^8 + \tilde{x}_4\,\mu^6 + \tilde{x}_5\,\mu^4 + \tilde{x}_6\,\mu^2 + \tilde{x}_7 = 0. \tag{4.38}$$

The derivative of the function, $f(\mu)$ can be found as

$$f'(\mu) = 2\,\mu\,(4\,\mu^6 + 3\,\tilde{x}_4\,\mu^4 + 2\,\tilde{x}_5\,\mu^2 + \tilde{x}_6).$$

Solving the system (4.36) for τ, we obtain

$$(\mu^4 - P_6\,\mu^2 + P_8)(\tilde{y}_8\,\mu^2 + \tilde{P}_8) = -(\tilde{y}_8\,\mu^2 + \tilde{P}_8)^2\,\cos(\mu\,\tau) - \tilde{P}_7\,\mu\,(\tilde{y}_8\,\mu^2 + \tilde{P}_8)\sin(\mu\,\tau),$$

$$\tilde{P}_7\,\mu\,(-P_5\,\mu^3 + P_7\,\mu) = \tilde{P}_7\,\mu\,(\tilde{y}_8\,\mu^2 + \tilde{P}_8)\,\sin(\mu\,\tau) - (\tilde{P}_7\,\mu)^2\,\cos(\mu\,\tau),$$

and adding these two equations yields

$$\cos(\mu\,\tau) = \frac{(P_6\,\mu^2 - \mu^4 - P_8)(\tilde{y}_8\,\mu^2 + \tilde{P}_8) + \tilde{P}_7\,\mu^2\,(P_5\,\mu^2 - P_7)}{(\tilde{y}_8\,\mu^2 + \tilde{P}_8)^2 + \tilde{P}_7^2\,\mu^2}.$$

Similarly, solving for $\sin(\mu\,\tau)$ using (4.36) gives

$$\sin(\mu\,\tau) = \frac{\mu\left[(P_6\,\tilde{P}_7 + \tilde{y}_8\,P_7 - P_5\,\tilde{P}_8)\,\mu^2 - (\tilde{y}_8\,P_5 + \tilde{P}_7)\,\mu^4 + P_7\,\tilde{P}_8 - P_8\,\tilde{P}_7\right]}{(\tilde{y}_8\,\mu^2 + \tilde{P}_8)^2 + \tilde{P}_7^2\,\mu^2}.$$

Hence, expressing τ in terms of cosine, we obtain

$$\tau_n = \frac{1}{\mu}\left[\cos^{-1}\left(\frac{(P_6\,\mu^2 - \mu^4 - P_8)(\tilde{y}_8\,\mu^2 + \tilde{P}_8) + \tilde{P}_7\,\mu^2\,(P_5\,\mu^2 - P_7)}{(\tilde{y}_8\,\mu^2 + \tilde{P}_8)^2 + \tilde{P}_7^2\,\mu^2}\right) + 2\pi n\right],$$

$$n = 0, 1, 2, \ldots$$

Without loss of generality, one can assume that $f(\mu) = 0$ has eight different positive roots μ_j, $j = 1, \ldots, 8$. For each of those roots, we can find the corresponding value of the time delay τ as

$$\tau_{j,n} = \frac{1}{\mu_j}\left[\cos^{-1}\left(\frac{(P_6\,\mu_j^2 - \mu_j^4 - P_8)(\tilde{y}_8\,\mu_j^2 + \tilde{P}_8) + \tilde{P}_7\,\mu_j^2\,(P_5\,\mu_j^2 - P_7)}{(\tilde{y}_8\,\mu_j^2 + \tilde{P}_8)^2 + \tilde{P}_7^2\,\mu_j^2}\right) + 2\pi\,(n-1)\right],$$

$$j = 1, \ldots, 8,\ n \in \mathbb{N},$$

and define

$$\tau_0 = \tau_{j_0,n_0} = \min_{1\le j\le 8, n\ge 1}\{\tau_{j,n}\}, \quad \mu_0 = \mu_{j_0}. \tag{4.39}$$

In order to establish whether the Hopf bifurcation actually occurs at $\tau = \tau_0$, one has to determine the sign of $d[\mathrm{Re}(k)]/d\tau$. Differentiating the characteristic equation (4.35) with respect to τ gives

$$\Big[4\,k^3+3\,P_5\,k^2+2\,P_6\,k+P_7\Big]\frac{dk}{d\tau} = \Big[(2\,\tilde{y}_8\,k-\tilde{P}_7)\,e^{-k\,\tau}-\tau\,(\tilde{y}_8\,k^2-\tilde{P}_7\,k-\tilde{P}_8)\,e^{-k\,\tau}\Big]\frac{dk}{d\tau} - k\,(\tilde{y}_8\,k^2-\tilde{P}_7\,k-\tilde{P}_8)\,e^{-k\,\tau}$$

$$\Rightarrow \quad 4\,k^3+3\,P_5\,k^2+2\,P_6\,k+P_7-\Big[2\,\tilde{y}_8\,k-\tilde{P}_7-\tau\,(\tilde{y}_8\,k^2-\tilde{P}_7\,k-\tilde{P}_8)\Big]\,e^{-k\,\tau} = -\,k\,e^{-k\,\tau}\Big[\tilde{y}_8\,k^2-\tilde{P}_7\,k-\tilde{P}_8\Big]\frac{d\tau}{dk}.$$

Therefore,

$$\left(\frac{dk}{d\tau}\right)^{-1} = \frac{\big[2\,\tilde{y}_8\,k-\tilde{P}_7-\tau\,(\tilde{y}_8\,k^2-\tilde{P}_7\,k-\tilde{P}_8)\big]\,e^{-k\,\tau}-(4\,k^3+3\,P_5\,k^2+2\,P_6\,k+P_7)}{k\,(\tilde{y}_8\,k^2-\tilde{P}_7\,k-\tilde{P}_8)\,e^{-k\,\tau}}$$
$$= \frac{(2\,\tilde{y}_8\,k-\tilde{P}_7)\,e^{-k\,\tau}-(4\,k^3+3\,P_5\,k^2+2\,P_6\,k+P_7)}{(\tilde{y}_8\,k^3-\tilde{P}_7\,k^2-\tilde{P}_8\,k)\,e^{-k\,\tau}}-\frac{\tau}{k}.$$

Evaluating at $\tau=\tau_0$ with $k=i\mu_0$ yields

$$\left(\frac{dk}{d\tau}\right)^{-1}\Bigg|_{\tau=\tau_0} = \frac{(2\tilde{y}_8 i\mu_0-\tilde{P}_7)[\cos(\mu_0\tau_0)-i\sin(\mu_0\tau_0)]+(4i\mu_0^3+3P_5\mu_0^2-2P_6 i\mu_0-P_7)}{(-\tilde{y}_8\,i\,\mu_0^3+\tilde{P}_7\,\mu_0^2-\tilde{P}_8\,i\,\mu_0)\,[\cos(\mu_0\tau_0)-i\,\sin(\mu_0\tau_0)]} - \frac{\tau_0}{i\mu_0}.$$

Collecting the real terms and substituting the values of $\cos(\mu_0\tau_0)$ and $\sin(\mu_0\tau_0)$ give the following after simplification,

$$\mathrm{Re}\left(\frac{dk}{d\tau}\right)^{-1}\Bigg|_{\tau=\tau_0} = \left[\frac{1}{(\tilde{y}_8\,\mu_0^2+\tilde{P}_8)^2+\tilde{P}_7^2\,\mu_0^2}\right]\Big[4\,\mu_0^6+3\,(P_5^2-2P_6)\mu_0^4+ 2\,(2\,P_8+P_6^2-2\,P_5\,P_7-\tilde{y}_8^2)\mu_0^2+P_7^2-2\,P_6\,P_8-2\,\tilde{y}_8\,\tilde{P}_8-\tilde{P}_7^2\Big]$$
$$= \frac{4\,\mu_0^6+3\,\tilde{x}_4\,\mu_0^4+2\,\tilde{x}_5\,\mu_0^2+\tilde{x}_6}{(\tilde{y}_8\,\mu_0^2+\tilde{P}_8)^2+\tilde{P}_7^2\,\mu_0^2}$$
$$= \frac{2\,\mu_0\left[4\,\mu_0^6+3\,\tilde{x}_4\,\mu_0^4+2\,\tilde{x}_5\,\mu_0^2+\tilde{x}_6\right]}{2\,\mu_0\left[(\tilde{y}_8\,\mu_0^2+\tilde{P}_8)^2+\tilde{P}_7^2\,\mu_0^2\right]} = z_v\,f'(\mu_0),$$

where $z_v = \Big[2\,\mu_0\Big((\tilde{y}_8\,\mu_0^2+\tilde{P}_8)^2+\tilde{P}_7^2\,\mu_0^2\Big)\Big]^{-1}$ and since $z_v>0$, it means that

$$\mathrm{sign}\left\{\frac{d\,[\mathrm{Re}(k)]}{d\tau}\right\}_{\tau=\tau_0} = \mathrm{sign}\left\{\mathrm{Re}\left(\frac{dk(\tau_0)}{d\tau}\right)^{-1}\right\} = \mathrm{sign}\big\{z_v\,f'(\mu_0)\big\} = \mathrm{sign}\big\{f'(\mu_0)\big\}.$$

This analysis can be summarised as follows.

Theorem 6. *Let τ_0 and μ_0 be defined as in (4.39) and $f'(\mu_0) > 0$. Then the endemic steady state E^* of the system (4.2) is linearly asymptotically stable for $\tau < \tau_0$, unstable for $\tau > \tau_0$ and undergoes Hopf bifurcation at $\tau = \tau_0$ provided the condition of Lemma 2 holds.*

4.4 Numerical bifurcation analysis and simulations

In order to better understand how different parameters affect the stability of the disease-free and endemic equilibria, we use a pseudospectral method [7] implemented in a traceDDE suite in MATLAB to numerically compute characteristic eigenvalues. Figure 4.2 illustrates how stability of the endemic steady state depends on the disease transmission rate β, local awareness rate α, the value of global awareness campaigns ω_o, the rate of reporting infection cases α_o, and the time delay τ of individuals' response to available information. This figure shows that the endemic equilibrium only exists for a limited range of disease transmission rates, and it is stable for higher rates and unstable for smaller β. Increasing the awareness rate α_o leads to a destabilisation of the endemic steady state, but surprisingly, increasing the value of global awareness campaigns ω_o or a local awareness rate α actually results in stabilising an endemic steady state, whilst increasing these rates above certain values makes the endemic steady state unfeasible, in which case the disease-free steady state is stable.

In terms of two types of vaccination, naturally, vaccination of aware individuals does not have any noticeable effect on stability of the endemic steady state, whereas increasing the vaccination rate of unaware individuals stabilises the endemic steady state, until it makes E^* unfeasible and stabilises the disease-free steady state. Increasing the time delay τ, in accordance with Theorem 6, leads to destabilisation of the endemic steady state and the emergence of periodic solutions. Figure 4.3 further illustrates the stability boundary of the steady state E^*, showing that for higher infant vaccination rates, a lower value of global awareness campaign is required to stabilise the endemic steady state.

Figures 4.4 - 4.7 demonstrates the results of numerical continuation of the Hopf bifurcation of the endemic steady state, as performed using DDE-BIFTOOL con-

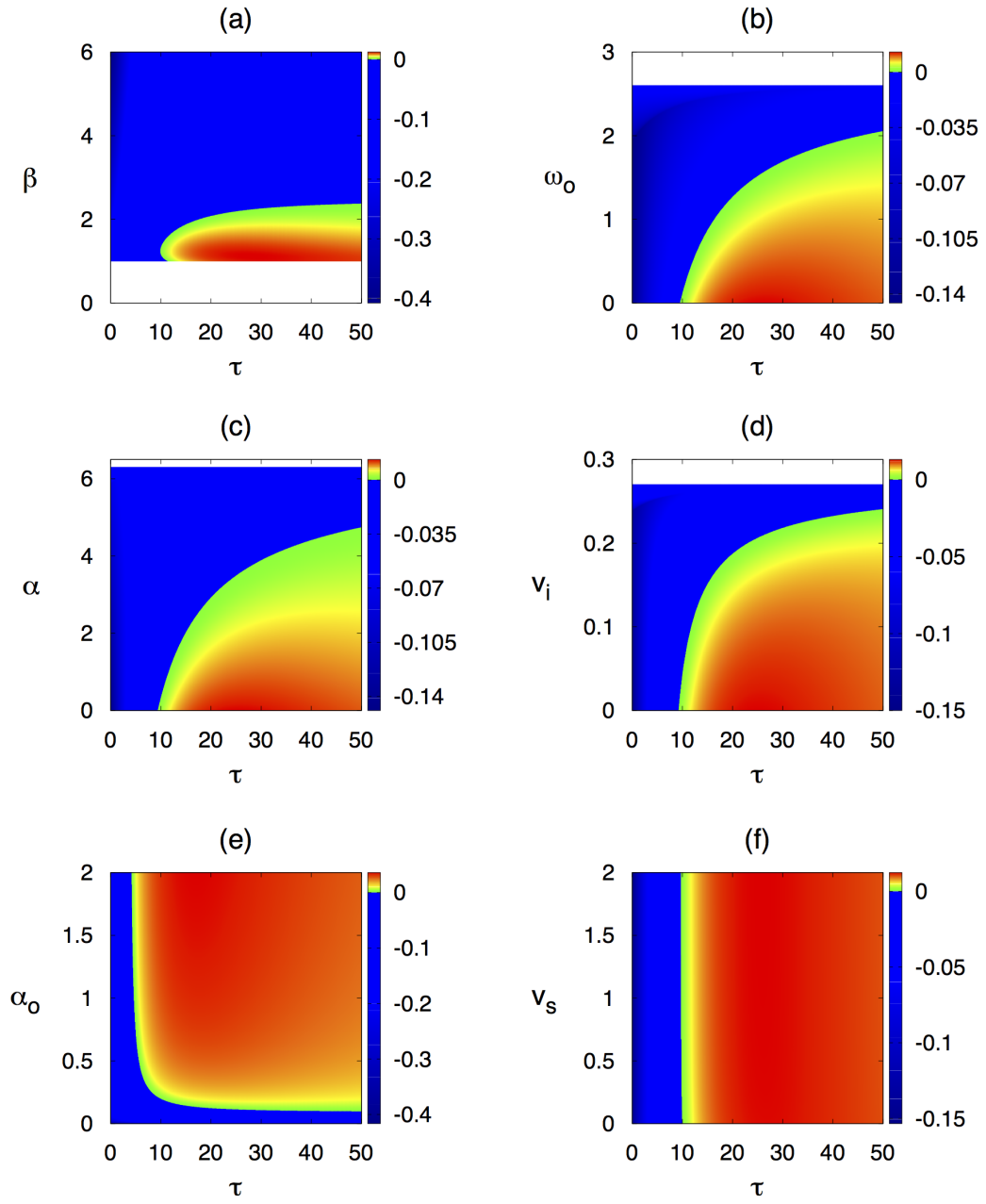

Figure 4.2: Stability of the endemic steady state E^*. Colour code denotes max[Re(k)], and in white regions the endemic steady state is not feasible. Parameter values are as follows, $\beta = 1.2$ but varied in (a), $\omega_o = 0.1$ but varied in (b), $\alpha = 0.3$ but varied in (c), $v_i = 0.04$ but varied in (d), $\alpha_o = 0.2$ but varied in (e), $v_s = 0.06$ but varied in (f), $\lambda = 0.1, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \lambda_o = 0.3, \eta = 0.05, \delta = 0.3, b = 0.4, N = 100$.

tinuation software. Figure 4.4(a) and (c) captured the results for the plot in complex plane of the eigenvalues of the steady state as extracted using p_splot during runtime for $\tau = 8$ and $\tau = 16$ respectively showing a stable state and an unstable state, (b) represents the Hopf point generated also from the p_splot during runtime and (d) shows the outcome of comparing the result obtained using traceDDE for varied ω_o

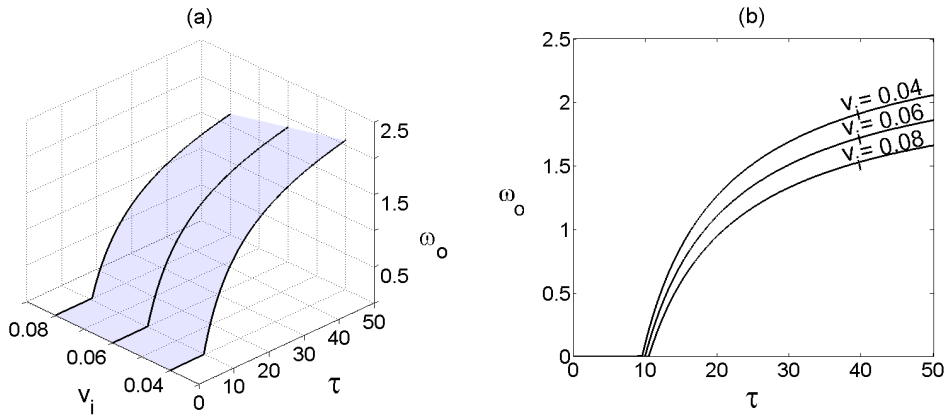

Figure 4.3: Stability boundaries of the endemic steady state E^*. The steady state is stable to the left of the surface in (a), and to the left of the lines in (b). Parameter values used are: $\alpha = 0.3, \lambda = 0.1, \beta = 1.2, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.05, \delta = 0.3, v_s = 0.06, b = 0.4, N = 100$.

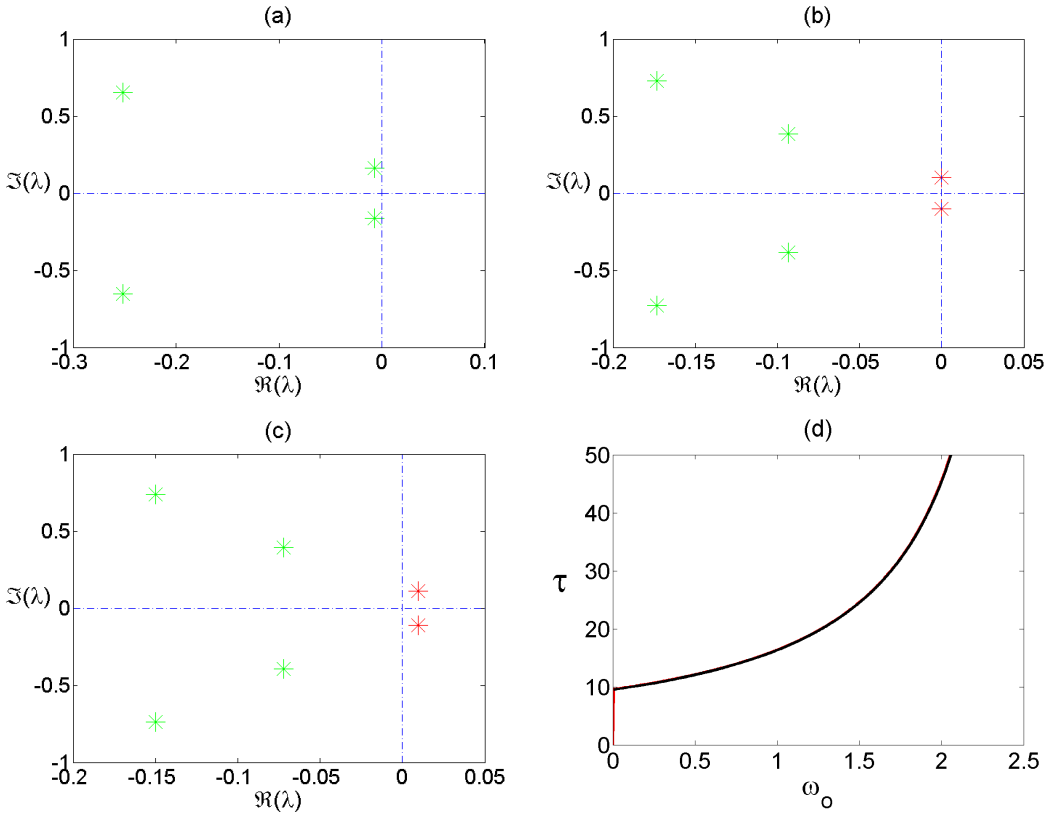

Figure 4.4: Bifurcation analysis of the endemic state (a) distribution of eigenvalues for $\tau = 8$, (b) distribution of eigenvalues at the Hopf point for $\tau = 16$, (c) distribution of eigenvalues for $\tau = 16$, (d) comparison of the Hopf boundary computed using DDE-BIFTOOL (black) and traceDDE (red). The parameter values are $\alpha = 0.3, \lambda = 0.1, \beta = 1.2, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.1, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.05, \delta = 0.3, v_s = 0.06, v_i = 0.04, b = 0.4, N = 100$.

against τ and the result from DDE-BIFTOOL extracted by br_contn for the continuation output of Hopf branch points. The results for the amplitude and period

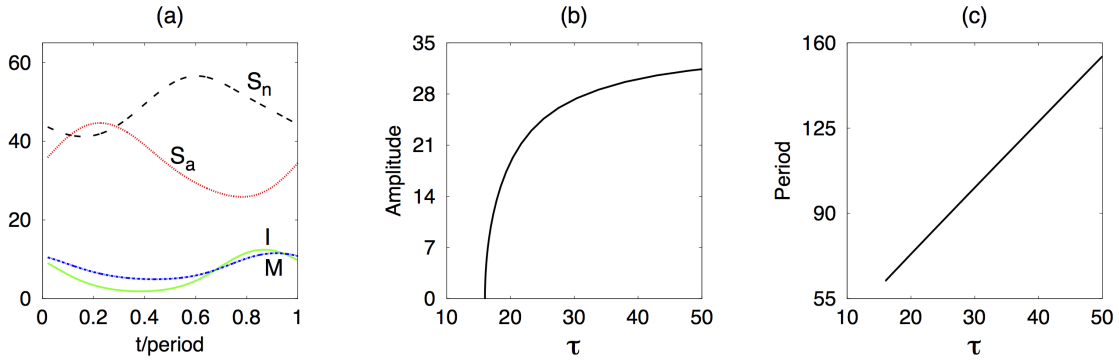

Figure 4.5: Bifurcation analysis of the endemic state for $v_i = 0.04, \tau = 16$ (a) periodic solutions showing the dynamics of variables (b) amplitude of periodic solutions depending on the time delay τ, (c) period depending on time delay. Parameter values are $\alpha = 0.3, \lambda = 0.1, \beta = 1.2, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.1, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.05, \delta = 0.3, v_s = 0.06, b = 0.4, N = 100$.

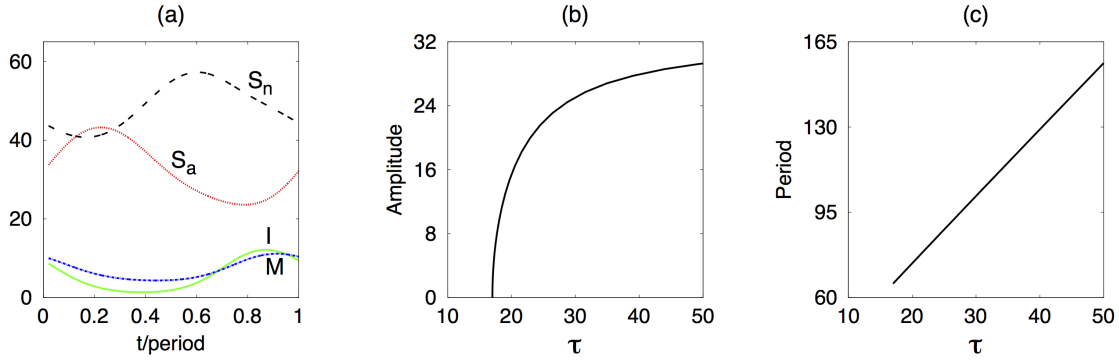

Figure 4.6: Bifurcation analysis of the endemic state for $v_i = 0.06, \tau = 17$ (a) periodic solutions showing the dynamics of variables (b) amplitude of periodic solutions depending on the time delay τ, (c) period depending on time delay. Parameters used are $\alpha = 0.3, \lambda = 0.1, \beta = 1.2, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.1, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.05, \delta = 0.3, v_s = 0.06, b = 0.4, N = 100$.

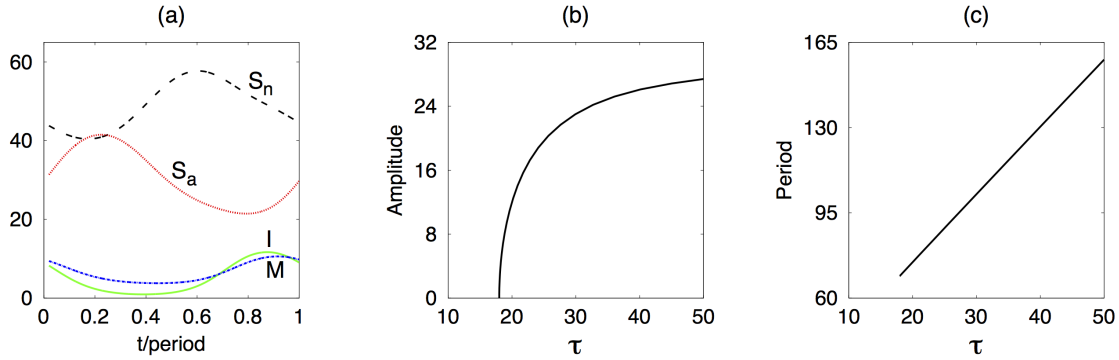

Figure 4.7: Bifurcation analysis of the endemic state for $v_i = 0.08, \tau = 18$, (a) periodic solutions showing the dynamics of variables (b) amplitude of periodic solutions depending on the time delay τ, (c) period depending on time delay. Other parameters are $\alpha = 0.3, \lambda = 0.1, \beta = 1.2, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.1, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.05, \delta = 0.3, v_s = 0.06, b = 0.4, N = 100$.

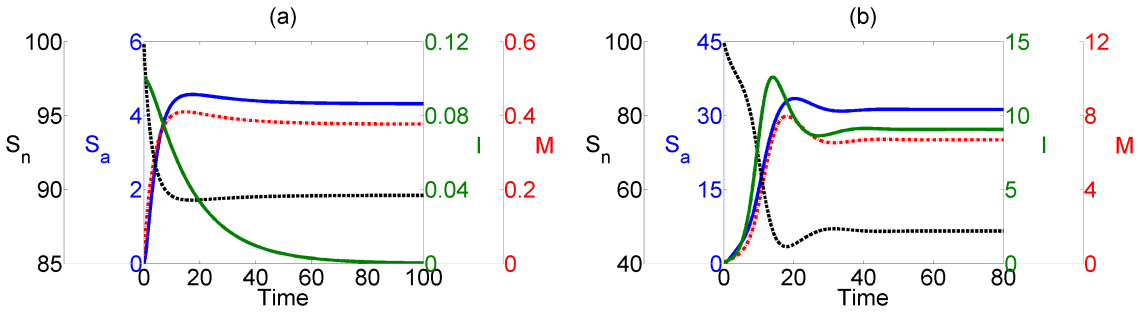

Figure 4.8: Numerical solution of the system (4.5) with $\tau = 0$. (a) Stable disease-free steady state for $\beta = 0.6, R_v^d = 0.8977$. (b) Stable endemic equilibrium for $\beta = 1.2, R_v^d = 1.7955$. Other parameter values are $\alpha = 0.3, \lambda = 0.1, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.1, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.05, \delta = 0.3, v_s = 0.06, v_i = 0.04, b = 0.4, N = 100$.

of periodic solutions of the model equation as against time delay, τ using $v_i = 0.04$ are represented in Figure 4.5. Further evaluations of the model with $v_i = 0.06$ and $v_i = 0.08$ produced the outcome in Figures 4.6 and 4.7 respectively. It shows that both the amplitude, and the period of periodic solutions increase with the time delay τ, and for higher vaccination rates v_i the amplitude of the periodic solution is smaller, while the period is higher.

In Figure 4.8 we illustrate how actual dynamics of the system (4.2) changes depending on system parameters. Figures 4.8(a) and (b) show that for $\tau = 0$, the system approaches the stable disease-free or endemic steady states for $R_v^d < 1$ or $R_v^d > 1$, respectively. One should note that according to Theorem 5, the stability of the disease-fee steady state does not depend on the value of the time delay τ, but rather on the basic reproduction number R_v^d only, so if one keeps the value of $R_v^d < 1$, the same kind of behaviour would be observed for any $\tau > 0$. Figure 4.9(a)-(c) revealed that choosing parameters in the range where $R_v^d > 1$ and increasing the time delays τ results in the system approaching endemic steady state in an oscillatory manner, with the amplitude of oscillations increasing with the time delay. Once the time delay τ exceeds the critical value determined by Theorem 6, the endemic steady state becomes unstable, and the system exhibits stable periodic solutions illustrated in Figure 4.9(d). The amplitude and period of such solutions themselves depend on the time delay, as has been shown earlier in plots (b) and (c) of Figures 4.5, 4.6 and 4.7.

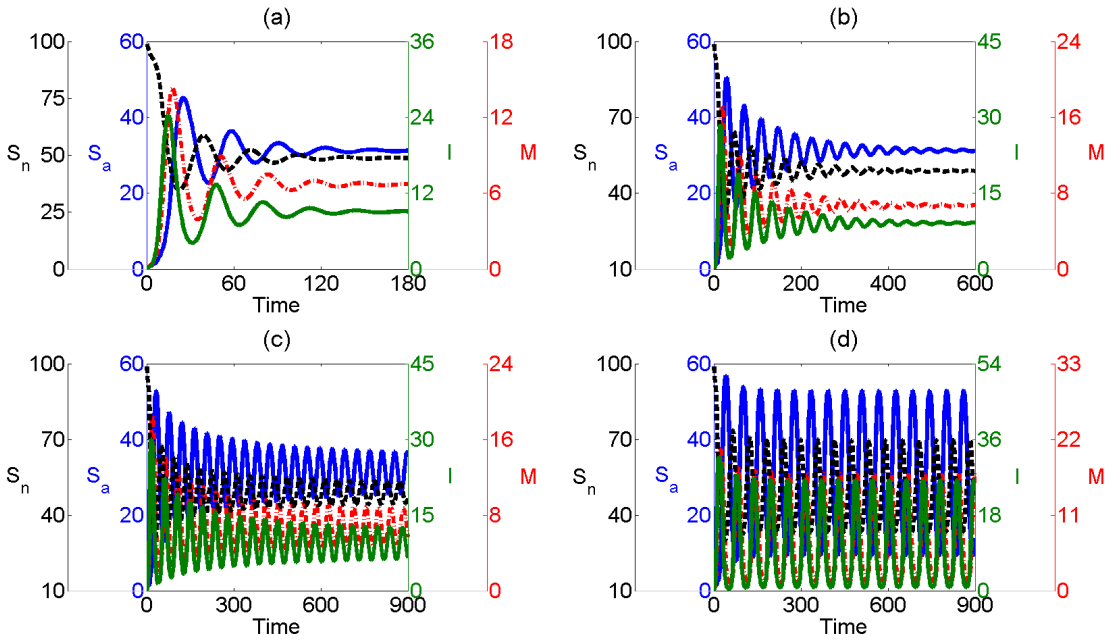

Figure 4.9: Stability evaluation of the endemic state with $R_v^d = 1.7955$ (a) $\tau = 4$ (b) $\tau = 8$ (c) $\tau = 10$ (d) $\tau = 16$. Parameter values are $\alpha = 0.3, \lambda = 0.1, \beta = 1.2, r = 0.2, \sigma_s = 0.04, p = 0.4, q = 0.6, \omega_o = 0.1, \alpha_o = 0.2, \lambda_o = 0.3, \eta = 0.05, \delta = 0.3, v_s = 0.06, v_i = 0.04, b = 0.4, N = 100$.

4.5 Discussion

This chapter has analysed the effects of vaccination and different types of disease awareness on the dynamics of epidemic spread. We have studied analytically and numerically the conditions on system parameters which ensure feasibility and stability of the disease-free and endemic equilibria. These results suggest that stability of the disease-free steady state is independent of the time delay associated with the response of unaware individuals to various types of awareness campaign, but it is rather determined by the basic reproduction number R_v^d that depends on other epidemiological parameters, as well as the values of awareness dissemination and infant vaccination rate. On the contrary, stability of the endemic equilibrium does depend on the response time delay in such a way that while the endemic steady state is stable for $\tau = 0$ (whenever it is biologically feasible), increasing the time delay can destabilise this endemic steady state and lead to the onset of stable periodic oscillations.

The analysis has provided a number of insights into the relative roles of different parameters, some of which are natural, while others were surprising. Vaccination of aware individuals appears to not have a profound effect on the disease dynamics,

while increasing the vaccination rates of unaware individuals (newborn), can make the endemic steady state unfeasible, so that the disease would be eradicated, and the system would settle on a stable disease-free equilibrium. For large values of the time delay, reducing the rate of disease transmission destabilise the endemic equilibrium, which should be expected.

Contrary to intuition, the same behaviour occurs when one reduces the values of global awareness campaign or local awareness, whereas one would expect that reduced awareness would support the maintenance of disease in the population, as is the case for the awareness stemming from the reported cases of disease. Moreover, increasing the values of local awareness and/or global awareness campaign increases the time delay needed to destabilise the endemic steady state. Interestingly, all these different types of disease awareness only affect the stability of the endemic equilibrium for sufficiently large time delay, while for zero and small delays, the endemic steady state is always stable whenever it is feasible, regardless of the rates of awareness.

The results suggest that no matter how efficiently the cases of infection are reported, by itself this is not sufficient to create enough awareness to eradicate an epidemic, whereas global awareness campaign, and increasing the overall awareness level through contacts with other aware individuals are able to achieve this. Furthermore, the analysis shows an important role played by the vaccination of newborns, which can prevent epidemic outbreaks by providing a required level of herd immunity.

Chapter 5

Discussion and future work

5.1 Summary and conclusions

Earlier models of infectious disease dynamics have provided some insights into how the circulation of information about an invading pathogen could assist in curtailing the spread of the disease within a given population. Information in these models can originate from different sources, such as direct contacts between the individuals, also known as a local awareness, and the global sources comprising of the media, mass awareness campaign programmes, social networking via the internet etc. Availability and dissemination of information to individuals from these sources play a vital role in the control of infectious diseases.

This thesis has focused on the spread of infectious diseases within human population that can become aware of the pathogen through both local and global sources of awareness. In real life scenarios, one can observe that the unaware population can access global information regardless of whether it is susceptible, infected or recovered, with all individuals having equal access to global sources of awareness. We have considered three realistic scenarios concerning the circulation of awareness from the media and other global sources, as well as from direct interactions of individuals. The first model focused on analysing the possibility of the unaware population becoming aware, irrespective of its status, of an invading pathogen through direct contact and global sources of awareness. The analysis provided analytical expressions of various steady states together with conditions for their stability. The results show that the presence of awareness tends to generate corresponding behavioural

changes in humans which in turn allows to control the spread of infectious diseases.

The second model focused on the effects of time delay in the response of unaware individuals to information provided during the dissemination of awareness. The results of the analysis show that the disease-free state will remain stable irrespective of the value of the time delay, suggesting that stability of this steady state does not depend on the duration of delay associated with individuals' responses to the awareness but is rather determined by the rates at which the awareness is required. Interestingly, the results suggest that, disease eradication becomes possible when the rate of the spread of local awareness or the level of global awareness campaign are sufficiently high, unlike the case of increasing the level of awareness stemming from the higher number of reported disease cases. The results also show that increasing global awareness campaign or the rate of local awareness tends to stabilise the endemic steady state in the case of delay in the response of individuals to available information, and when these values are sufficiently high, the disease is eradicated. This demonstrates the importance of awareness circulation in reducing the spread of infectious diseases.

The third model considered the optimisation of the impact of awareness when it is operating in parallel with the administration of a vaccine. This was done by analysing the impact of disseminating awareness of the infectious disease using various awareness routes and the administration of vaccine to the unaware susceptible (newborn) and the aware population. Besides the analytical investigation of the model, the numerical simulations were carried out to illustrate how different parameters affect system dynamics. Further to conclusions obtained for earlier models, the results for this model show that vaccination of aware susceptible appears to not have a profound effect on the disease dynamics, while increasing newborn vaccination is much more effective at curtailing or eradicating the spread of infectious diseases.

The overall outcome lies in the significance of awareness for the purpose of controlling the spread of infectious diseases. In light of the results, one can conclude that the dissemination of information to the general public regarding an infectious disease contributes to the positive behavioural reactions of the population in fighting and controlling the spread of the disease, and, as a result, the outbreaks of the disease are minimised. The impact of awareness circulation was found to be enhanced

with the administration of vaccines to newborns, which means that the combined effects of awareness and vaccination on the spread of an invading infectious disease in a given human population tend to be more effective in eradicating the disease from the population. Consequently, educating people serves as a control strategy against the spread of infectious diseases, and this can be further supported by the administration of vaccines.

5.2 Further research

One possible important extension of the work presented in this thesis is the consideration of the geographic spread of infections and disease awareness. Spatial movement of people within and between different regions can result in different rates of the spread of disease and awareness. According to [46], during the 1994 outbreak of the plague in India, as the infected migrated due to a general panic, this resulted in the spread of disease to other states of the country. Similar situations occurred during the recent outbreak of Ebola and Zika virus which spread across countries as a result of the infected population migrating from the disease-endemic region to other countries. Zuo *et al.* [67] considered the influence of media coverage on the spread of infectious disease within a particular region from other regions. In the same light, future research should focus on the issue of migration of the population and the dissemination of awareness, evaluating the impact these have on the spread of infectious disease.

Within spatial models of epidemic spread, one can investigate optimal vaccination aimed at eradicating diseases with constrained resources. This would result in targeted vaccination strategies focusing on specific geographic regions. Another issue that can have a profound effect on developing techniques for disease control is the fact that infectious diseases are often characterized by a non-exponential distribution of infectious periods. More realistic models can include this feature through distributed time delays, and data from actual epidemic outbreaks can then be fed into these models to investigate how this affects the disease spread, and how epidemics can be controlled.

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