Strathprints Institutional Repository

Greenhalgh, David and Rana, Sourav and Samanta, Sudip and Sardar, Tridip and Bhattacharya, Sabyasachi and Chattopadhyay, Joydev (2015) Awareness programs control infectious disease - Multiple delay induced mathematical model. Applied Mathematics and Computation, 251. pp. 539-563. ISSN 0096-3003 , http://dx.doi.org/10.1016/j.amc.2014.11.091

Awareness programs control infectious disease - Multiple delay induced mathematical model.

David Greenhalgh[1], Sourav Rana[2], Sudip Samanta[3], Tridip Sardar[3], Sabyasachi Bhattacharya[3], Joydev Chattopadhyay[3,4]

Abstract

We propose and analyze a mathematical model to study the impact of awareness programs on an infectious disease outbreak. These programs induce behavioral changes in the population, which divide the susceptible class into two subclasses, aware susceptible and unaware susceptible. The system can have a disease-free equilibrium and an endemic equilibrium. The expression of the basic reproduction number and the conditions for the stability of the equilibria are derived. We further improve and study the model by introducing two time-delay factors, one for the time lag in memory fading of aware people and one for the delay between cases of disease occurring and mounting awareness programs. The delayed system has positive bounded solutions. We study various cases for the time delays and show that in general the system develops limit cycle oscillation through a Hopf bifurcation for increasing time delays. We show that under certain conditions on the parameters, the system is permanent. To verify our analytical findings, the numerical simulations on the model, using realistic parameters for Pneumococcus are performed.

Keywords: Epidemic model, Awareness programs, Time delay, Stability analysis, Hopf bifurcation, Numerical simulation.

Mathematics Subject Classification: 34D20, 92B05, 92D20, 92D39.

[1]Department of Mathematics and Statistics, University of Strathclyde, Livingstone Tower, 26 Richmond Street, Glasgow G1 1XH, UK, Email: david.greenhalgh@strath.ac.uk. Tel.: +44-141-548-3653, Fax: +44-141-548-3345

[2]Department of Statistics, Visva-Bharati University, Santiniketan, West Bengal, Pin 731235, India

[3]Agricultural and Ecological Research Unit, Indian Statistical Institute, 203, B. T. Road, Kolkata 700108, India

[4]Corresponding author. E-mail: joydev@isical.ac.in, Fax: +91-33-25773049, Tel: +91-33-25753231.

1. Introduction

In developing countries more than 11 million people died each year due to infectious disease including premature deaths and deaths of young children. Pneumonia, Tuberculosis (TB), Diarrheal diseases (Cholera), Malaria, Measles and more recently HIV/AIDS are the major deadly infectious diseases [1].

The major cause of global childhood mortality is Pneumonia which is caused by a number of infectious agents, including viruses, bacteria and fungi. Approximately 1.4 million children die every year because of Pneumonia [2]. Diarrheal diseases (for example Cholera, Bacillary Dysentery, Typhoid, Giardia and Rotavirus) are the second leading cause of death taking the lives of about 1.5 million children under five every year [3]. In 2010, 8.8 million people were infected with, and 1.4 million died from, TB [4]. Malaria is a life-threatening vector-borne disease caused by the bites of infected mosquitoes. In 2010, Malaria caused an estimated 655,000 deaths, mostly among African children (with an uncertainty range of 537,000 to 907,000) [5]. In 2010, 139,300 people died worldwide due to Measles [6]. Recently, HIV/AIDS has become the major concern in a global pandemic. More than 25 million people died of HIV/AIDS in the last three decades. There were approximately 34.2 million people infected by HIV up to the end of 2011 [7]. Another infectious disease is Influenza which causes serious public health and economic problems. Globally, these annual epidemics result in about three to five million cases of severe illness, and about 250,000 to 500,000 deaths [8]. Other major deadly infectious diseases in humans include Dengue, Yellow Fever, Hepatitis B, Avian Influenza (Bird Flu) and Chagas Disease.

The above description clearly indicates the severity of infectious disease. These diseases are a major threat to developing and underdeveloped countries. Some diseases can be prevented through vaccinations. However this is costly and sometimes the effect is only temporary. On the other hand sometimes disease appropriate awareness in a population can control an infection most effectively. In developing and underdeveloped countries, the mass media plays an important role in changing behavior related to public health. The government and other health organizations should immediately make people aware about the disease and relevant precautions through the media. The media not only make the population acquainted with the disease but also suggest the necessary preventive practices such as social distancing, wearing protective masks or vaccination. In general the people who are aware adopt these practices so that their chances of becoming infected are minimized. Depending on the behavior associated with a given infectious disease, improved levels of awareness may increase the use of mosquito coils, mosquito nets [9], or face masks [10, 11], practice of better hygiene [12, 13], application of preventive medicine or vaccination [14], voluntary quarantine [15], avoidance of places containing large numbers of people

[12], practice of safe sex [16], or other appropriate measures. A comprehensive review of the existing mathematical literature related to the effect of media awareness programs on disease outbreaks is given in Table 1. However, behavioral responses can change the transmission patterns and reduce the prevalence of disease. So there is a need of epidemiological models that explicitly include the effect of awareness programs and behavioral responses. It is to be noted that in general the effect of awareness can strongly depend on local interactions. The individuals in the local spatial or geographical neighbourhood of an outbreak may have a much stronger incentive to adopt preventive practices and this local adoption of suitable preventive practices may cause a local outbreak to die out without the whole population having to adopt them. It would be possible to model this using some sort of spatial model. However in this paper we shall not pursue this line instead we shall study a mean field model and assume that the impact of the awareness program is uniform across the whole population. This is common in the study of disease awareness programs [17, 18, 19, 20] where sometimes we wish to use a relatively simple model to study the effect of awareness programs applied to the whole population to reduce the disease levels in the entire population rather than stop a local outbreak.

A comprehensive review on the impact of media awareness programmes is presented in Section 2. In Section 3 the model without time delays is formulated and analyzed to observe the local stability of the system around the feasible equilibria. The model with multiple time delays is proposed and analyzed in Section 4. The conditions under which the system enters Hopf bifurcation and conditions for permanence of the system are also worked out. In Section 5, numerical simulations are carried out to verify our analytical findings and the paper ends with a brief conclusion.

2. Review of media awareness program in infectious disease outbreak

In this section we review the literature on the effect of media awareness programs on infectious disease outbreaks. These studies are essentially of two different types. In the first type mathematical models are used to investigate the impact of media coverage on the spread and control of infectious disease. The mathematical models are either compartmental models such as susceptible-infected-susceptible (SIS), susceptible-infected-recovered (SIR), susceptible-exposed-infected (SEI), susceptible-infected-recovered-susceptible (SIRS), exposed-infected-hospitalized (EIH), susceptible-exposed-infected-hospitalized-recovered (SEIHR) and similar models, or economic or game-theoretic models. In the second type of study statistical analysis is used to identify the association between media awareness and disease related cases. A comprehensive summary of such studies is given in Table 1.

Table 1: Review on the impact of media awareness programs on infectious disease.

Year	References	Summary of study
2007	[21]	Cui et al. developed and analyzed an SEI model to include media influence on the spreading of a communicable disease in a given area. They concluded that if the basic reproduction number is greater than one and the media effect is high, the model shows several endemic equilibria, which causes a threat to control the disease outbreak.
	[17]	Liu et al. developed an EIH compartmental model to investigate the role of the media and its psychological impact on multiple disease outbreaks. Their model analysis reveals that this impact leads to differences in the transmission pattern.
	[22]	Using the data from the Bangladesh Demography and Health Survey (1999-2000), Rahman and Rahman identified that media and education could play a major role in controlling HIV/AIDS.
	[23]	Tai and Sun investigated media dependency amongst Chinese individuals during the SARS epidemic of 2003. Their study was mainly focused into the situation where the information was highly monitored and not easily available from the mainstream media. In those circumstances, short message service (SMS) and the Internet are the possible substitute resources of information.
2008	[24]	Cui et al. formulated and analyzed an SIS infection model to investigate the role of media coverage during an infectious disease outbreak in a given population. They concluded that increasing media coverage causes a lower infection rate, although it may not absolutely remove the infection.

Table 1 – continued from previous page

Year	References	Summary of study
	[25]	Joshi et al. investigated the effect of an information and education campaign on the HIV epidemic in Uganda. They compare their model with three types of susceptibles to a standard SIR model.
	[26]	Li et al. developed and analyzed an SIS epidemic model, including media coverage in which the susceptible population is subjected to impulsive vaccination. They showed that the disease-free solution is globally asymptotically stable.
	[27]	Liu and Cui developed a compartmental model to study the role of the media in an infectious disease outbreak. They assume a standard epidemiological model but with a reduced transmission term due to the media campaign.
	[28]	Young et al. showed that a high level of media coverage plays a crucial role in making the public aware of many diseases and influencing their perception of risk. Participants in their study often considered diseases that appeared in the media more serious, even when this was not the actual case.
2009	[29]	Chen formulated an economic game-theoretic model of epidemics incorporating self-protection of susceptible populations. He suggests that an individual makes his or her behavioral changes through the information about the disease and expanding the supply of information may decrease the likelihood of eradication.

Table 1 – continued from previous page

Year	References	Summary of study
	[30]	Funk et al. develop and study a mathematical model where the host population is less susceptible due to the spread of awareness. They reveal that change in behavioral response can reduce the size of an outbreak though the epidemic threshold will be unaffected.
	[31]	Li and Cui propose an SIS epidemic model in the presence of media coverage and analyze the model under two distinct types of vaccination strategies namely constant vaccination and pulse vaccination. They compare these two different types of vaccination policies.
2010	[32]	Kiss et al. formulated a mathematical model where the total populations are aware of the disease threat but only a certain proportion of them is responsive. They showed that the infection can be removed when the spreading of information is fast enough, otherwise information transmission can play a major role in controlling the disease.
	[33]	Mummert and Weiss proposed a modified SIR model incorporating the impact of media coverage. They conclude that the severity of the disease outbreak can be lower if the media and the public health agencies work together.
	[34]	Yoo et al. showed using a statistical analysis that there is a connection between Influenza vaccination 1999-2001 and media reporting, specifically headlines on flu-related issues. They studied three media sources: a wire service news agency, a newspaper and four television channels.

Table 1 – continued from previous page

Year	References	Summary of study
2011	[18]	Misra et al. developed and analyzed a nonlinear SIS mathematical model in the presence of a media awareness program. They suggest that an awareness program can control the diffusion of the disease but immigration of susceptibles causes the disease to be endemic.
	[35]	Misra et al. proposed and analyzed a delay induced mathematical model in the presence of an awareness program. They concluded that the awareness program plays a crucial role in controlling the spread of disease, but it cannot remove the infection completely.
	[36]	Sun et al. used the SIS model in a two patch setting with media coverage present in each patch. They analyze their model both analytically and numerically. They find that both epidemic burden and duration of the disease spread are significantly lowered by the media coverage.
	[19]	Tchuenche et al. developed a Susceptible-Infected-Vaccinated-Recovered (SIVR) epidemic model to study the effect of media broadcasting on the spread and control over an Influenza outbreak. Using optimal control theory they obtained the effect of costs due to media coverage.
2012	[37]	Olowukure et al. investigated if there is any connection between volume of newspaper reports and laboratory testing for Influenza A (H1N1) pdm09, (the swine flu Influenza A (H1N1) pandemic of 2009) in one English health region during the early phase of the pandemic. They inferred that there exists a temporal association between volume of media reporting and number of laboratory tests.

Table 1 – continued from previous page

Year	References	Summary of study
	[38]	Tchuenche and Bauch formulated an SIHR model incorporating a signal function which captures the effect of media coverage. They suggest that the disease cannot be eliminated through media coverage, but it can control the spread of the infection.
2013	[39]	Funk and Jansen studied how the interplay between the network of an awareness program and the network of infection determines the dynamics of the disease outbreak.
	[40]	Liu investigated an SIRS epidemic model with media coverage and random perturbation. The disease transmission term was reduced by media coverage as in Liu and Cui [27], Tchuenche et al. [19] and Sun et al. [36] and stochastic white noise perturbation was added. The resulting stochastic differential equation model was studied analytically and numerically.
	[20]	Samanta et al. studied an SIS epidemic model for the effect of media awareness programs on epidemic outbreaks. They concluded that although media awareness programs can have a substantial effect on controlling disease prevalence, above a threshold value of their execution rate, the system shows limit cycle oscillations.
	[41]	Wang et al. studied an SIS network model incorporating the impact of media coverage on disease transmission and suggested effective control strategies to prevent disease through media coverage and education. They find the basic reproduction number, equilibrium and global stability results for their model and explore the results by simulation.

Table 1 – continued from previous page

Year	References	Summary of study
2014	[42]	Kaur et al. proposed and analyzed an SIRS epidemic model incorporating the effects of an awareness program driven by the media. Their model is based on that of Misra et al. [18] with some significant differences in modeling the awareness programs. They conduct an equilibrium and stability analysis and use simulation to verify their results.
	[43]	Samanta and Chattopadhyay proposed and analyzed a slow-fast epidemic model in the presence of the awareness program, where a susceptible individual switches between aware and unaware states very fast, whereas the disease transmission and other biological processes are comparatively slow.
	[44]	Sharma and Misra investigated an SIR model of hepatitis B with varying population size, which couples vaccination and awareness created by the media within a single framework.
	[45]	Wang and Xiao studied an SIR Filippov epidemic model with media coverage by incorporating a piecewise continuous transmission rate to describe that the media coverage exhibits its effects once the number of infected individuals exceeds a certain critical level. The disease transmission coefficient is reduced by an exponential term as a result of a media campaign. They find that a given level of infecteds can be reached if the threshold policy and other parameters are chosen correctly.

Table 1 – continued from previous page

Year	References	Summary of study
	[46]	Zhao et al. proposed and analyzed an SIRS epidemic model incorporating media coverage with time delay. They showed that the time delay in media coverage cannot affect the stability of the disease-free equilibrium when the basic reproduction number is less than unity. However, the time delay affects the stability of the endemic equilibrium and produces limit cycle oscillations while the basic reproduction number is greater than unity.
2015	[47]	Sahu and Dhar studied the complex dynamics of an SEQIHRS epidemic model incorporating media coverage, quarantine and isolation studies in a community with pre-existing immunity. Media coverage does not alter the effective reproduction number but lowers the number of infecteds at the endemic steady state, also lowering the maximum number of infected individuals. The results of isolation and quarantine depend on the amount of transmission from isolated individuals. Higher amounts of pre-existing immunity amongst the population cause the peak infection level to happen earlier and decrease it.

The above descriptions clearly indicate that awareness programmes play a crucial role in controlling the disease during an epidemic outbreak. In the next section we formulate a mathematical model to capture the impact of media awareness programs in an infectious disease outbreak. The model that we shall consider is a deterministic differential equation mean field SIS epidemic model for the spread of an infection in the presence of awareness programs. We model the awareness programs explicitly unlike the models of Cui et al. [24], Li, Ma and Cui [26] and Liu and Cui [27] who model the effect of awareness through a reduction in the disease transmission term. Our work builds on the work of Misra et al. [18, 35] although we allow aware people to become infected and some recovered individuals to become aware. It also builds on Samanta et al. [20] After analysing the basic model we introduce and analyse two types of time delays and then perform simulations based on real parameter values for Pneumococcus to verify our theoretical results.

3. Model with awareness program

3.1. Model Formulation

To formulate the mathematical model we suppose that the whole population is divided into three separate classes, the susceptible aware class, the susceptible unaware class and the infected class. We assume that both susceptible classes can be infected by contact with infectives but the aware class has less chance to be infected compared to the unaware class and the infection rate among aware populations is dependent on the awareness programs. The unaware susceptible population becomes aware through the interaction with the awareness programs [18, 35] which is considered to be a saturating function [27] (Holling type-II) of the awareness programs and a proportion of infected individuals recover from the infection through treatment. After recovery, a fraction of recovered people will join the aware susceptible class and the remaining fraction will remain unaware susceptible. The model does not necessarily assume that the transmission routes of the disease and the information are the same, indeed these may well be different.

We consider that in the region under consideration, the total population is $N(t)$ at time t and the rate of immigration of susceptibles is A, where immigrants are assumed to be unaware. The total population is divided into three classes: the susceptible unaware population $X_-(t)$, the infective population $Y(t)$ and the susceptible aware population $X_+(t)$. Also, let $M(t)$ be the number of campaigns due to the awareness programs driven by the media in that region at time t. μ denotes the implementation rate of awareness programs which is proportional to the number of infective individuals in the population. We assume that unaware susceptible individuals become aware under the influence of the awareness program at the rate λ and the interaction between the unaware susceptible population and the awareness program follows the Holling type-II functional form with half-saturating constant k. It is assumed that the disease spreads only due to direct contact between susceptibles and infectives. Let β be the contact rate of unaware susceptible individuals with infective individuals and it is assumed that the disease transmission follows the mass action law ($\beta X_-(t)Y(t)$). However, our basic assumption is that the interaction between aware susceptibles and infecteds depends on the number of campaigns due to the awareness programs. Large numbers of campaigns causes less interaction between susceptible aware and infected populations, a mathematical form of this assumption can be written as $\frac{\beta X_+(t)Y(t)}{1+\beta_1 M(t)}$, where β_1 is the efficacy of the awareness programs - a monotonic decreasing function of the number of campaigns $M(t)$. It is also a monotonic decreasing function of β_1. We assume that aware susceptible individuals transfer to unaware susceptible individuals due to fading of memory or social factors at a per capita rate λ_0. It is also assumed that a proportion of infected individuals recover through treatment. After recovery, a fraction p of recovered people will become aware and join the aware susceptible class whereas the remaining fraction $(1-p)$ will remain unaware susceptible.

Keeping the above facts in mind, the dynamics of the model is governed by the following systems of nonlinear ordinary differential equations :

$$
\begin{aligned}
\frac{dX_-}{dt} &= A - \beta X_-(t)Y(t) - \lambda X_-(t)\frac{M(t)}{k+M(t)} - dX_-(t) + \lambda_0 X_+(t) + (1-p)\gamma Y(t),\\
\frac{dX_+}{dt} &= \lambda X_-(t)\frac{M(t)}{k+M(t)} + p\gamma Y(t) - dX_+(t) - \lambda_0 X_+(t) - \frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t),\\
\frac{dY}{dt} &= \beta X_-(t)Y(t) + \frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t) - \gamma Y(t) - \alpha Y(t) - dY(t),\\
\frac{dM}{dt} &= \mu Y(t) - \mu_0 M(t),
\end{aligned}
\tag{3.1}
$$

where $X_-(0) > 0$, $X_+ \geq 0$, $Y \geq 0$, $M \geq 0$.

Here the constants γ, α, d represent the recovery rate, disease induced death and natural death rate respectively. The constant μ_0 denotes the depletion rate of awareness programs due to ineffectiveness, social problems in the population, and similar factors. Note that p is a fraction and its value lies between 0 and 1.

Using the fact $N = X_- + X_+ + Y$, the system (3.1) reduces to the following system:

$$
\begin{aligned}
\frac{dY}{dt} &= \beta(N(t) - X_+(t) - Y(t))Y(t) + \frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t) - (\gamma+\alpha+d)Y(t),\\
\frac{dX_+}{dt} &= \lambda(N(t) - X_+(t) - Y(t))\frac{M(t)}{k+M(t)} + p\gamma Y(t) - dX_+(t) - \lambda_0 X_+(t)\\
&\quad - \frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t),\\
\frac{dN}{dt} &= A - dN(t) - \alpha Y(t),\\
\frac{dM}{dt} &= \mu Y(t) - \mu_0 M(t).
\end{aligned}
\tag{3.2}
$$

For the analysis of model (3.2), we need the region of attraction [48] which is given by the set:

$$\Omega = \Big\{(Y, X_+, N, M) \in \Re_+{}^4 : 0 \le X_+ + Y \le N \le \frac{A}{d}, 0 \le M \le \frac{\mu A}{\mu_0 d}\Big\}$$

and attracts all solutions initiating in the interior of the positive orthant, with $N(0) > X_+(0) + Y(0)$.

3.2. Equilibrium analysis

The above model (3.2) has two non-negative equilibria.

(i) The disease free equilibrium (DFE) $E_0(0, 0, A/d, 0)$.

(ii) The endemic equilibrium $E^*(Y^*, X_+^*, N^*, M^*)$.

Here

$X_+^* = \dfrac{\mu_0}{\beta\beta_1\mu Y^*}\Big[\beta\big(\dfrac{A}{d} - \dfrac{\alpha Y^*}{d} - Y^*\big) - \big(\gamma+\alpha+d\big)\Big]\Big[1 + \dfrac{\beta_1\mu Y^*}{\mu_0}\Big]$,

$N^* = \dfrac{A - \alpha Y^*}{d}$,

$M^* = \dfrac{\mu Y^*}{\mu_0}$,

and Y^* satisfies the equation

$$H_1 Y^{*3} + H_2 Y^{*2} + H_3 Y^* + H_4 = 0, \tag{3.3}$$

with

$$\begin{aligned}
H_1 &= \tfrac{\beta\beta_1\mu^2}{\mu_0^2}\big[\big(d+\lambda_0\big)\big(\tfrac{\alpha}{d}+1\big) + p\gamma\big] + \tfrac{\beta^2\mu}{\mu_0}\big(\tfrac{\alpha}{d}+1\big),\\
H_2 &= \beta(\tfrac{\alpha}{d}+1)(\tfrac{\lambda\mu}{\mu_0}+\beta k) - (\tfrac{\beta A}{d} - \gamma - \alpha - d)(\tfrac{\lambda\beta_1\mu^2}{\mu_0^2} + \tfrac{\beta\mu}{\mu_0}) + \tfrac{\lambda\beta\beta_1\mu^2 A}{\mu_0^2 d}\\
&\quad + \tfrac{p\gamma\beta\beta_1\mu k}{\mu_0} - \tfrac{\beta_1\mu^2}{\mu_0^2}(d+\lambda_0)(\tfrac{\beta A}{d} - \gamma - \alpha - d) + \tfrac{\beta\mu}{\mu_0}(d+\lambda_0)(1+\beta_1 k)(\tfrac{\alpha}{d}+1),\\
H_3 &= -\big(\tfrac{\beta A}{d} - \gamma - \alpha - d\big)\big(\tfrac{\lambda\mu}{\mu_0} + \beta k\big)\\
&\quad + k\beta\big(d+\lambda_0\big)\big(\tfrac{\alpha}{d}+1\big) - \tfrac{\mu}{\mu_0}\big(d+\lambda_0\big)\big(1+\beta_1 k\big)\big(\tfrac{\beta A}{d} - \gamma - \alpha - d\big),\\
H_4 &= -k\big(\tfrac{\beta A}{d} - \gamma - \alpha - d\big)\big(d+\lambda_0\big).
\end{aligned} \tag{3.4}$$

An endemic equilibrium exists if

$$\frac{\beta A}{d} - (\gamma + \alpha + d) > 0. \tag{3.5}$$

Let us define $R_0 = \dfrac{\beta A}{d(\gamma+\alpha+d)}$, which is the basic reproduction number for system (3.2).

H_1 is always positive and H_4 is always negative if $R_0 > 1$. Hence the equation (3.3) has at least one positive root. Therefore the sufficient conditions for the existence of the interior equilibrium point of system (3.2) are as follows:

$$R_0 > 1 \text{ and } Y^* < min\Big\{\tfrac{d(\gamma+\alpha+d)(R_0-1)}{\beta(\alpha+d)}, \tfrac{A}{\alpha}\Big\}.$$

However, H_1, H_2, H_3 and H_4 are always positive if $R_0 < 1$. Hence the system (3.2) does not have any positive interior equilibrium (E^*) for $R_0 < 1$.

Remark 1: $\frac{\partial Y^*}{\partial \mu} < 0$ if $\frac{H_{1\mu}Y^{*2}+H_{2\mu}Y^*+H_{3\mu}}{3H_1Y^{*2}+2Y^*H_2+H_3} > 0$ and $\frac{\partial Y^*}{\partial \beta_1} < 0$ if $\frac{H_{1\beta_1}Y^{*2}+H_{2\beta_1}Y^*+H_{3\beta_1}}{3H_1Y^{*2}+2Y^*H_2+H_3} > 0$, which indicates that the equilibrium number of infective individuals decreases with an increase in the value of the the implementation rate of awareness programs and the efficacy of the awareness programs. Here $H_{i\bullet}$, $(i = 1, 2, 3)$ denotes the partial differentiation of H_i with respect to the parameter '$\bullet$'.

Remark 2: We can find the basic reproduction number of the system (3.1) in the absence of awareness program. Therefore the system (3.1) becomes

$$\begin{aligned} \frac{dS}{dt} &= A - \beta SY - dS + \gamma Y, \\ \frac{dY}{dt} &= \beta SY - \gamma Y - \alpha Y - dY, \end{aligned} \tag{3.6}$$

where S and Y are the number of susceptible and infected individuals and the other parameters are the same as defined in system (3.1).

The above model (3.6) has two non-negative equilibria:

(i) The disease free equilibrium (DFE) $E_0(0, A/d)$,

(ii) The endemic equilibrium $E^*(S^*, Y^*)$,

where $S^* = \frac{\gamma+\alpha+d}{\beta}$, $Y^* = \frac{\beta A-d(\gamma+\alpha+d)}{\beta(\alpha+d)}$ the basic reproduction number for the system (3.6) is $R_{01} = \frac{\beta A}{d(\gamma+\alpha+d)}$, which is the same as R_0. So the awareness program cannot eradicate the infection whenever $R_0 > 1$, but it can reduce the equilibrium number of infected individuals (see Figure 2).

3.3. Local stability behavior

The roots of the characteristic equation corresponding to $E_0(0, 0, A/d, 0)$ are $\frac{\beta A}{d} - \gamma - \alpha - d$, $-d$, $-(d + \lambda_0)$, $-\mu_0$.

The DFE E_0 is locally asymptotically stable (LAS) if $\frac{\beta A}{d} - \gamma - \alpha - d < 0$, i.e. $R_0 < 1$.

The variational matrix at an endemic equilibrium $E^*(Y^*, X_+^*, N^*, M^*)$ is

$$J = \begin{pmatrix} -\Pi_1 - \xi & \Pi_2 & \Pi_3 & -\Pi_4 \\ \Pi_5 & -\Pi_6 - \xi & \Pi_7 & \Pi_8 \\ -\Pi_9 & 0 & -\Pi_{10} - \xi & 0 \\ \Pi_{11} & 0 & 0 & -\Pi_{12} - \xi \end{pmatrix}.$$

Here $\Pi_1 = \beta Y^*$, $\Pi_2 = -\beta Y^* + \frac{\beta Y^*}{1+\beta_1 M^*}$, $\Pi_3 = \beta Y^*$, $\Pi_4 = \frac{\beta\beta_1 X_+^* Y^*}{(1+\beta_1 M^*)^2}$, $\Pi_5 = -\frac{\lambda M^*}{k+M^*} + p\gamma - \frac{\beta X_+^*}{1+\beta_1 M^*}$, $\Pi_6 = \frac{\lambda M^*}{k+M^*} + d + \lambda_0 + \frac{\beta Y^*}{1+\beta_1 M^*}$, $\Pi_7 = \frac{\lambda M^*}{k+M^*}$, $\Pi_8 = \frac{\lambda(N^*-X_+^*-Y)k}{(k+M^*)^2} + \frac{\beta\beta_1 X_+^* Y^*}{(1+\beta_1 M^*)^2}$, $\Pi_9 = \alpha$, $\Pi_{10} = d$, $\Pi_{11} = \mu$, $\Pi_{12} = \mu_0$.

The characteristic equation of the system (3.2) around the interior equilibrium (E^*) is

$$\xi^4 + \sigma_1\xi^3 + \sigma_2\xi^2 + \sigma_3\xi + \sigma_4 \quad = \quad 0. \tag{3.7}$$

Therefore, E^* is LAS if and only if

$$\sigma_1 > 0,\ \sigma_2 > 0,\ \sigma_3 > 0,\ \sigma_4 > 0,\ \sigma_1\sigma_2 > \sigma_3 \text{ and } \sigma_1\sigma_2\sigma_3 > \sigma_3^2 + \sigma_1^2\sigma_4. \tag{3.8}$$

Here,

$\sigma_1 = \Pi_1 + \Pi_6 + \Pi_{10} + \Pi_{12}$,

$\sigma_2 = \Pi_1\Pi_{10} + \Pi_1\Pi_{12} + \Pi_{10}\Pi_{12} + \Pi_3\Pi_9 + \Pi_4\Pi_{11} + \Pi_6\Pi_{10} + \Pi_6\Pi_{12} + \Pi_1\Pi_6 - \Pi_2\Pi_5$,

$\sigma_3 = -\Pi_2\Pi_5\Pi_{10} - \Pi_2\Pi_5\Pi_{12} + \Pi_2\Pi_8\Pi_{11} + \Pi_1\Pi_{10}\Pi_{12} + \Pi_3\Pi_9\Pi_{12} + \Pi_4\Pi_{10}\Pi_{11} + \Pi_6\Pi_{10}\Pi_{12}$
$\quad +\Pi_1\Pi_6\Pi_{10} + \Pi_1\Pi_6\Pi_{12} + \Pi_3\Pi_6\Pi_9 + \Pi_4\Pi_6\Pi_{11} + \Pi_2\Pi_7\Pi_9$,

$\sigma_4 = -\Pi_2\Pi_5\Pi_{10}\Pi_{12} + \Pi_2\Pi_7\Pi_9\Pi_{12} - \Pi_2\Pi_8\Pi_{10}\Pi_{11} + \Pi_1\Pi_6\Pi_{10}\Pi_{12} + \Pi_3\Pi_6\Pi_9\Pi_{12} + \Pi_4\Pi_6\Pi_{10}\Pi_{11}$.

4. Model with delay

4.1. Model Formulation

In the previous section we assumed that aware susceptible individuals transfer to unaware susceptible individuals due to fading of memory or certain social factors. However, it is reasonable to consider a time lag in memory fading of aware people. Here we assume that the aware susceptible individual will become unaware susceptible at time t due to forgetting the impact of disease at time $t - \tau_1$ (for some $\tau_1 > 0$).

We need to consider the probability that an aware susceptible individual remains in the aware susceptible class throughout the interval $[t - \tau_1, t]$ which we denote by $P(t, \tau_1)$. An aware susceptible individual leaves the aware susceptible class at time ξ through death at rate d, surviving the time interval $[\xi - \tau_1, \xi]$ and becoming unaware at rate $\lambda_0 P(\xi, \tau_1)$ or becoming infected at rate $\frac{\beta Y(\xi)}{1+\beta_1 M(\xi)}$. Hence

$$P(t, \tau_1) = e^{-\int_{t-\tau_1}^{t}\left[d + \lambda_0 P(\xi,\tau_1) + \frac{\beta Y(\xi)}{1+\beta_1 M(\xi)} d\xi\right]}, \qquad \text{for } t \geq t_1. \tag{4.1}$$

Usually, the number of infective cases known to the policy makers are cases that occurred some time previously and thus the intensity of the awareness program depends on this data. So it is more plausible to consider a time delay in execution of awareness programs. We suppose that at time t the intensity of the awareness programs being executed will be in accordance with the number of infected cases reported at time $t - \tau_2$ (for some $\tau_2 > 0$).

Incorporating these two delays and the survival probability into the system of equations (3.1) and writing $P(t) \equiv P(t, \tau_1)$ as τ_1 is fixed we obtain the system of delay differential equations:

$$
\begin{aligned}
\frac{dX_-}{dt} &= A - \beta X_-(t)Y(t) - \lambda X_-(t)\frac{M(t)}{k+M(t)} - dX_-(t) + \lambda_0 X_+(t-\tau_1)P(t) + (1-p)\gamma Y(t),\\
\frac{dX_+}{dt} &= \lambda X_-(t)\frac{M(t)}{k+M(t)} + p\gamma Y(t) - dX_+(t) - \lambda_0 X_+(t-\tau_1)P(t) - \frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t),\\
\frac{dY}{dt} &= \beta X_-(t)Y(t) + \frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t) - \gamma Y(t) - \alpha Y(t) - dY(t),\\
\frac{dM}{dt} &= \mu Y(t-\tau_2) - \mu_0 M(t),\\
\frac{dP}{dt} &= \Big[-\lambda_0 P(t) + \lambda_0 P(t-\tau_1) - \frac{\beta Y(t)}{1+\beta_1 M(t)} + \frac{\beta Y(t-\tau_1)}{1+\beta_1 M(t-\tau_1)}\Big]P(t).
\end{aligned}
\tag{4.2}
$$

We denote by C the Banach space of continuous functions $\phi : [-\tau, 0] \to \mathbb{R}^5$ with norm

$$\|\phi\| = \sup_{-\tau\le\theta\le 0}\{|\phi_1(\theta)|, |\phi_2(\theta)|, |\phi_3(\theta)|, |\phi_4(\theta)|, |\phi_5(\theta)|\}$$

where $\tau = max\{\tau_1, \tau_2\}$ and $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5)$. As usual, the initial conditions of (4.2) are given as

$$X_-(\theta) = \phi_1(\theta), \quad X_+(\theta) = \phi_2(\theta), \quad Y(\theta) = \phi_3(\theta), \quad M(\theta) = \phi_4(\theta), \quad P(\theta) = \phi_5(\theta), \qquad \theta \in [-\tau, 0], \tag{4.3}$$

where the initial function $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5)$ belongs to the Banach space $C = C([-\tau, 0], \mathbb{R}^5)$ of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^5$. For biological reasons, the initial functions are assumed as

$$\phi_i(\theta) \ge 0, \qquad i = 1, 2, 3, 4 \quad \text{and} \qquad 1 \ge \phi_5(\theta) \ge 0, \qquad \theta \in [-\tau, 0]. \tag{4.4}$$

We also need the consistency condition

$$P(0) = e^{-\int_{-\tau}^{0}\left[d+\lambda_0 P(\xi,\tau_1)+\frac{\beta Y(\xi)}{1+M(\xi)}\right]d\xi}.$$

By the fundamental theory of functional differential equations [49], we know that there is a unique solution $(X_-(t), X_+(t), Y(t), M(t), P(t))$ to system (4.2) with initial conditions (4.3).

4.2. Preliminaries

In this section, we will present some preliminaries, such as positive invariance, boundedness of solutions, existence of equilibria and the characteristic equation.

4.2.1. Positive invariance

Theorem 4.1. *All the solutions of (4.2) with initial conditions (4.3) are positive.*

Proof : The model (4.2) can be written in the following form: $X = col(X_-(t), X_+(t), Y(t), M(t), P(t)) \in \mathbb{R}^5_+$, $(\phi_1(\theta), \phi_2(\theta), \phi_3(\theta), \phi_4(\theta), \phi_5(\theta)) \in \bar{C}_+ = ([-\tau, 0], \mathbb{R}^5_+)$, $\phi_1(0), \phi_2(0), \phi_3(0), \phi_4(0) \geq 0, \phi_5(0) \geq 0$,

$$F(X) = \begin{pmatrix} F_1(X) \\ F_2(X) \\ F_3(X) \\ F_4(X) \\ F_5(X) \end{pmatrix}$$

$$= \begin{pmatrix} A - \beta X_-(t)Y(t) - \lambda X_-(t)\frac{M(t)}{k+M(t)} - dX_-(t) + \lambda_0 X_+(t-\tau_1)P(t) + (1-p)\gamma Y(t) \\ \lambda X_-(t)\frac{M(t)}{k+M(t)} + p\gamma Y(t) - dX_+(t) - \lambda_0 X_+(t-\tau_1)P(t) - \frac{\beta}{1+\beta_1 M(t)} X_+(t)Y(t) \\ \beta X_-(t)Y(t) + \frac{\beta}{1+\beta_1 M(t)} X_+(t)Y(t) - \gamma Y(t) - \alpha Y(t) - dY(t) \\ \mu Y(t-\tau_2) - \mu_0 M(t) \\ \left[-\lambda_0 P(t) + \lambda_0 P(t-\tau_1) - \frac{\beta Y(t)}{1+\beta_1 M(t)} + \frac{\beta Y(t-\tau_1)}{1+\beta_1 M(t-\tau_1)}\right] P(t) \end{pmatrix}.$$

Then the model system (4.2) becomes

$$\dot{X} = F(X) \tag{4.5}$$

with $X(\theta) = (\phi_1(\theta), \phi_2(\theta), \phi_3(\theta), \phi_4(\theta), \phi_5(\theta)) \in C_+$ and $\phi_1(0), \phi_2(0), \phi_3(0), \phi_4(0), \phi_5(0) > 0$. It is easy to check in system (4.5) that whenever choosing $X(\theta) \in \mathbb{R}_+$ such that $X_- = 0, X_+ = 0, Y = 0, M = 0$ or $P = 0$ then

$$F_i(X)|_{x_i=0, X\in\mathbb{R}^5_+} \geq 0, \qquad \text{for} \qquad i = 1, 2, 3, 4, 5,$$

with $x_1(t) = X_-(t),\ x_2(t) = X_+(t),\ x_3(t) = Y(t),\ x_4(t) = M(t),\ x_5(t) = P(t)$. Using the lemma of [50] we claim that any solution of (4.5) with $X(\theta) \in C_+$, say $X(t) = X(t, X(\theta))$, is such that $X(t) \in \mathbb{R}^5_+$ for all $t \geq 0$. From (4.1) we can see that $P(t) \leq 1$ for all t as well.

Next, we will prove the boundedness of solutions. Using the fact $N = X_- + X_+ + Y$, the system (4.2) reduces to the following system:

$$
\begin{aligned}
\frac{dY}{dt} &= \beta(N(t)-X_+(t)-Y(t))Y(t)+\frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t)-(\gamma+\alpha+d)Y(t),\\
\frac{dX_+}{dt} &= \lambda(N(t)-X_+(t)-Y(t))\frac{M(t)}{k+M(t)}+p\gamma Y(t)-dX_+(t)-\lambda_0 X_+(t-\tau_1)P(t)\\
&\quad -\frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t),\\
\frac{dN}{dt} &= A-dN(t)-\alpha Y(t),\\
\frac{dM}{dt} &= \mu Y(t-\tau_2)-\mu_0 M(t),\\
\frac{dP}{dt} &= \Big[-\lambda_0 P(t)+\lambda_0 P(t-\tau_1)-\frac{\beta Y(t)}{1+\beta_1 M(t)}+\frac{\beta Y(t-\tau_1)}{1+\beta_1 M(t-\tau_1)}\Big]P(t).
\end{aligned}
\tag{4.6}
$$

4.2.2. Boundedness

Theorem 3.2. *All the solutions of (4.6) with initial conditions (4.3) are ultimately bounded.*

Proof : Let, $(Y(t), X_+(t), N(t), M(t), P(t))$ be any solution of system (4.6) with initial conditions (4.3). Applying the theorem of differential inequality [51] on the third equation of the system (4.6), we have $N(t) \le e^{-dt}\big(N(0)-\frac{A}{d}\big)+\frac{A}{d}$. Therefore, $\limsup_{t\to\infty} N(t) \le \frac{A}{d}$ as $t \to \infty$. Since $N(t) = Y(t) + X_+(t) + X_-(t)$, we can conclude that for t sufficiently large, $0 \le Y(t), X_+(t) \le \frac{A}{d}$.

Similarly, from the fourth equation of the system (4.6) we have

$$\dot{M}(t) = \mu Y(t-\tau_2)-\mu_0 M(t).$$

This implies that $$\dot{M}(t)+\mu_0 M(t) = \mu Y(t-\tau_2).$$

So $$\dot{M}(t)+\mu_0 M(t) \le \mu\frac{A}{d}, \qquad \text{for } t \ge t_0,\ \text{for some } t_0>0.$$

Hence $$M(t) \le M(t_0)e^{-\mu(t-t_0)}+\frac{\mu A}{\mu_0 d}, \qquad \text{for } t \ge t_0,$$

so $$limsup_{t\to\infty} M(t) \le \frac{\mu A}{\mu_0 d}.$$

It is straightforward to show that if $P(t)$ is part of a solution of (4.6) then $0 \le P(t) \le 1$. Hence, $(Y(t), X_+(t), N(t), M(t), P(t))$ is ultimately bounded above.

4.2.3. Equilibrium Analysis

Now the equilibrium points $(Y^*, X_+^*, N^*, M^*, P^*)$ of the delay model (4.6) satisfy

$$\begin{aligned}
\beta(N^*-X_+^*-Y^*)Y^*+\tfrac{\beta}{1+\beta_1 M^*}X_+^*Y^*-(\gamma+\alpha+d)Y^* &= 0,\\
\lambda(N^*-X_+^*-Y^*)\tfrac{M^*}{k+M^*}+p\gamma Y^*-dX_+^*-\lambda_0 X_+^*P^*-\tfrac{\beta}{1+\beta_1 M^*}X_+^*Y^* &= 0,\\
A-dN^*-\alpha Y^* &= 0,\\
\mu Y^*-\mu_0 M^* &= 0.
\end{aligned}\tag{4.7}$$

Here P^* will depend on $\tau_1(\geq 0)$ through the following equation

$$P^*\ (\equiv\ F_1,\ \text{say})\ =\ e^{-\left[d\tau_1+\lambda_0 P^*\tau_1+\frac{\beta Y^*\tau_1}{1+\beta_1 M^*}\right]}\ \Big(\equiv\ F_2(P^*,\tau_1),\ \text{say}\Big).\tag{4.8}$$

The expression on the righthand side (i.e. $F_2(P^*,\tau_1)$) is a decreasing function of τ_1 such that $F_2(P^*,0)=1$, $F_2(P^*,\infty)=0$. Note that Y^* and M^* depend on τ_1 only through $P^*(\tau_1)$. So there exists at least one positive root (depending on τ_1) of the transcendental equation (4.8) as P^* lies between 0 and 1. A graphical analysis to visualize this scenario is presented in Appendix B.

4.3. Stability analysis and local Hopf bifurcation

Case (a) : $\tau_1=\tau_2=0$

In absence of both delays the system (4.6) reduces to the system (3.2).

Case (b) : $\tau_1=0,\tau_2>0$

Then the system (4.6) reduces to the following system:

$$\begin{aligned}
\frac{dY}{dt} &= \beta(N(t)-X_+(t)-Y(t))Y(t)+\frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t)-(\gamma+\alpha+d)Y(t),\\
\frac{dX_+}{dt} &= \lambda(N(t)-X_+(t)-Y(t))\frac{M(t)}{k+M(t)}+p\gamma Y(t)-dX_+(t)-\lambda_0 X_+(t)\\
&\quad -\frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t),\\
\frac{dN}{dt} &= A-dN(t)-\alpha Y(t),\\
\frac{dM}{dt} &= \mu Y(t-\tau_2)-\mu_0 M(t).
\end{aligned}\tag{4.9}$$

It has the equilibrium point $E^*(Y^*, X_+^*, N^*, M^*)$ the same as the system (3.2). The variational matrix at the endemic equilibrium $E^*(Y^*, X_+^*, N^*, M^*)$ is

$$J = \begin{pmatrix} -M_1 - \xi & M_2 & M_3 & -M_4 \\ M_5 & -M_6 - \lambda_0 - \xi & M_7 & M_8 \\ -M_9 & 0 & -M_{10} - \xi & 0 \\ \mu e^{-\xi\tau_2} & 0 & 0 & -M_{11} - \xi \end{pmatrix}.$$

Here $M_1 = \beta Y^*$, $M_2 = -\beta Y^* + \frac{\beta Y^*}{1+\beta_1 M^*}$, $M_3 = \beta Y^*$, $M_4 = \frac{\beta\beta_1 X_+^* Y^*}{(1+\beta_1 M^*)^2}$, $M_5 = -\frac{\lambda M^*}{k+M^*} + p\gamma - \frac{\beta X_+^*}{1+\beta_1 M^*}$, $M_6 = \frac{\lambda M^*}{k+M^*} + d + \lambda_0 + \frac{\beta Y^*}{1+\beta_1 M^*}$, $M_7 = \frac{\lambda M^*}{k+M^*}$, $M_8 = \frac{\lambda(N^* - X_+^* - Y)k}{(k+M^*)^2} + \frac{\beta\beta_1 X_+^* Y^*}{(1+\beta_1 M^*)^2}$, $M_9 = \alpha$, $M_{10} = d$ and $M_{11} = \mu_0$.

The characteristic equation is

$$\begin{aligned} &\xi^4 + (C_1 + D_1)\xi^3 + (C_2 + D_2)\xi^2 + (C_3 + D_3)\xi + (C_4 + D_4) + \\ &(E_1\xi^2 + (E_2 + F_1)\xi + (E_3 + F_2))e^{-\xi\tau_2} = 0. \end{aligned} \tag{4.10}$$

Here

$C_1 = M_1 + M_6 + M_{10} + M_{11}$,

$C_2 = -M_2M_5 + M_1M_6 + M_6M_{10} + M_1M_{10} + M_3M_9 + M_6M_{11} + M_1M_{11} + M_{10}M_{11}$,

$C_3 = -M_2M_5M_{10} + M_1M_6M_{10} + M_3M_6M_9 + M_2M_7M_9 - M_2M_5M_{11} + M_1M_6M_{11} + M_6M_{10}M_{11}$
$\quad + M_1M_{10}M_{11} + M_3M_9M_{11}$,

$C_4 = -M_2M_5M_{10}M_{11} + M_1M_6M_{10}M_{11} + M_3M_6M_9M_{11} + M_2M_7M_9M_{11}$,

$D_1 = \lambda_0$,

$D_2 = \lambda_0(M_{10} + M_{11} + M_1)$,

$D_3 = \lambda_0(M_1M_{10} + M_3M_9 + M_1M_{11} + M_{10}M_{11})$,

$D_4 = \lambda_0(M_3M_9M_{11} + M_1M_{10}M_{11})$,

$E_1 = \mu M_4$,

$E_2 = -\mu(-M_4M_{10} + M_2M_8 - M_4M_6)$,

$E_3 = -\mu(M_2M_8M_{10} - M_4M_6M_{10})$,

$F_1 = \lambda_0\mu M_4$,

$F_2 = \lambda_0\mu M_4M_{10}$.

Theorem (4.1a) : The equilibrium point E^* is locally asymptotically stable (LAS) for $\tau_2 < \tau_{2_0}$ where τ_{2_0} is the minimum positive value of

$$\overline{\tau}_{2_0} = \frac{1}{\omega_{2_0}} \arccos\left\{ \frac{(E_2+F_1)\omega_{2_0}^2[(C_1+D_1)\omega_{2_0}^2-(C_3+D_3)]+(E_1\omega_{2_0}^2-E_3-F_2)[\omega_{2_0}^4-(C_2+D_4)\omega_{2_0}^2+(C_4+D_4)]}{(E_1\omega_{2_0}^2-E_3-F_2)^2+(E_2+F_1)^2\omega_{2_0}^2} \right\}$$

for ω_{2_0} corresponding to all positive real roots of (4.12). If the coefficients A_{1i} $(i = 1, 2, 3, 4)$ of equation (4.12) do not satisfy the Routh-Hurwitz conditions and $(C_4 + D_4)^2 < (E_3 + F_2)^2$ holds then the delay τ_2 will not affect the stability of the system. If the coefficients A_{1i} $(i = 1, 2, 3, 4)$ of equation (4.12) satisfy the Routh-Hurwitz conditions then the system is LAS for all $\tau_2 \geq 0$, provided that it is stable in the absence of delay.

Proof : Put $\xi = i\omega$ in (4.10) and separating real and imaginary parts we get

$$\begin{aligned} (E_1\omega^2 - E_3 - F_2)\cos\omega\tau_2 - (E_2 + F_1)\omega\sin\omega\tau_2 &= \omega^4 - (C_2 + D_2)\omega^2 + (C_4 + D_4), \\ (E_1\omega^2 - E_3 - F_2)\sin\omega\tau_2 + (E_2 + F_1)\omega\cos\omega\tau_2 &= (C_1 + D_1)\omega^3 - (C_3 + D_3)\omega. \end{aligned} \tag{4.11}$$

Eliminating τ_2 from (4.11) and put $\omega^2 = \omega_1$ we get

$$\omega_1^4 + A_{11}\omega_1^3 + A_{12}\omega_1^2 + A_{13}\omega_1 + A_{14} = 0, \tag{4.12}$$

where

$A_{11} = (C_1 + D_1)^2 - 2(C_2 + D_2)$,

$A_{12} = (C_2 + D_2)^2 + 2(C_4 + D_4) - 2(C_1 + D_1)(C_3 + D_3) - E_1^2$,

$A_{13} = -2(C_2 + D_2)(C_4 + D_4) + (C_3 + D_3)^2 + 2E_1(E_3 + F_2) - (E_2 + F_1)^2$,

$A_{14} = (C_4 + D_4)^2 - (E_3 + F_2)^2$.

Case (b.1) : If the A_{1i} $(i = 1, 2, 3, 4)$ satisfy the Routh-Hurwitz conditions, then (4.12) has no positive real roots. In that case E^* (if it exists) is LAS $\forall \tau_2 > 0$, provided that it is stable in the absence of delay, i.e. τ_2 will not affect the stability of the system, when equation (4.12) has no positive real root.

Case (b.2) : If the A_{1i} $(i = 1, 2, 3, 4)$ do not satisfy the Routh-Hurwitz conditions, in that case $A_{14} < 0$ implies that equation (4.12) has at least one positive real root, i.e. if $(C_4 + D_4)^2 < (E_3 + F_2)^2$ then equation (4.10) has a pair of purely imaginary roots say $\pm i\omega_{2_0}$ and for this value of ω_{2_0} we can get the value of τ_{2_n} from equation (4.11) as

$$\tau_{2_n} = \frac{1}{\omega_{2_0}} \arccos\left\{ \frac{(E_2+F_1)\omega_{2_0}^2[(C_1+D_1)\omega_{2_0}^2-(C_3+D_3)]+(E_1\omega_{2_0}^2-E_3-F_2)[\omega_{2_0}^4-(C_2+D_4)\omega_{2_0}^2+(C_4+D_4)]}{(E_1\omega_{2_0}^2-E_3-F_2)^2+(E_2+F_1)^2\omega_{2_0}^2} \right\} + \frac{2n\pi}{\omega_{2_0}},$$

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

By Butler's lemma, [52] the endemic equilibrium remains stable for $\tau_2 < \overline{\tau}_{2_0}$. Without loss of generality suppose that ω_{2_0} represents the value of ω_{2_0} corresponding to τ_{2_0}.

Theorem (4.1b) : If $\Phi_1(\omega_{2_0}) > 0$, the system (4.6) undergoes a Hopf Bifurcation at the positive equilibrium as τ_2 increases through τ_{2_0}, where the expression of $\Phi_1(\omega_{2_0})$ satisfies (4.13).

Proof : <u>Transversality condition for Hopf-bifurcation</u> :

Differentiating (4.10) with respect to τ_2 we get

$$\frac{d\tau_2}{d\xi} = \frac{4\xi^3+3(C_1+D_1)\xi^2+2(C_2+D_2)\xi+(C_3+D_3)}{E_1\xi^3+(E_2+F_1)\xi^2+(E_3+F_2)\xi}e^{\xi\tau_2} + \frac{2E_1\xi+(E_2+F_1)}{E_1\xi^3+(E_2+F_1)\xi^2+(E_3+F_2)\xi} - \frac{\tau_2}{\xi},$$

$$Sgn\left[\frac{d(Re\xi)}{d\tau_2}\right]_{\tau_2=\tau_{2_0}} = Sgn\left[Re(\frac{d\xi}{d\tau_2})^{-1}\right]_{\xi=i\omega_{2_0}},$$

$$= Sgn\Bigg[Re\frac{[-3(C_1+D_1)\omega_{2_0}{}^2+(C_3+D_3)]\cos\omega_{2_0}\tau_2-[-4\omega_{2_0}{}^3+2(C_2+D_2)\omega_{2_0}]\sin\omega_{2_0}\tau_2}{-(E_2+F_1)\omega_{2_0}{}^2+i\omega_{2_0}[-E_1\omega_{2_0}{}^2+(E_3+F_2)]} +$$

$$Re\frac{[-3(C_1+D_1)\omega_{2_0}{}^2+(C_3+D_3)]\sin\omega_{2_0}\tau_2+[-4\omega_{2_0}{}^3+2(C_2+D_2)\omega_{2_0}]\cos\omega_{2_0}\tau_2}{-(E_2+F_1)\omega_{2_0}{}^2+i\omega_{2_0}[-E_1\omega_{2_0}{}^2+(E_3+F_2)]}i +$$

$$Re\frac{2i\omega_{2_0}E_1+(E_2+F_1)}{-(E_2+F_1)\omega_{2_0}{}^2+i\omega_{2_0}[-E_1\omega_{2_0}{}^2+(E_3+F_2)]}\Bigg],$$

$$= Sgn\Bigg[\frac{-[-3(C_1+D_1)\omega_{2_0}{}^2+(C_3+D_3)]\omega_{2_0}[(E_2+F_1)\omega_{2_0}\cos\omega_{2_0}\tau_2+(E_1\omega_{2_0}{}^2-E_3-F_2)\sin\omega_{2_0}\tau_2]}{(E_2+F_1)^2\omega_{2_0}{}^4+\omega_{2_0}{}^2[-E_1\omega_{2_0}{}^2+(E_3+F_2)]^2}$$

$$+\frac{[-4\omega_{2_0}{}^2+2(C_2+D_2)]\omega_{2_0}{}^2[(E_2+F_1)\omega_{2_0}\sin\omega_{2_0}\tau_2-(E_1\omega_{2_0}{}^2-E_3-F_2)\cos\omega_{2_0}\tau_2]}{(E_2+F_1)^2\omega_{2_0}{}^4+\omega_{2_0}{}^2[-E_1\omega_{2_0}{}^2+(E_3+F_2)]^2}$$

$$+\frac{\omega_{2_0}{}^2[-(E_2+F_1)^2+2E_1(-E_1\omega_{2_0}{}^2+E_3+F_2)]}{(E_2+F_1)^2\omega_{2_0}{}^4+\omega_{2_0}{}^2[-E_1\omega_{2_0}{}^2+(E_3+F_2)]^2}\Bigg].$$

Using relation (4.11) we get the above expression as

$$= Sgn\Bigg[\frac{[3(C_1+D_1)\omega_{2_0}{}^2-(C_3+D_3)][(C_1+D_1)\omega_{2_0}{}^2-(C_3+D_3)]+[4\omega_{2_0}{}^2-2(C_2+D_2)][\omega_{2_0}{}^4-(C_2+D_2)\omega_{2_0}{}^2+C_4+D_4]}{(E_2+F_1)^2\omega_{2_0}{}^2+[-E_1\omega_{2_0}{}^2+(E_3+F_2)]^2}$$

$$+\frac{-(E_2+F_1)^2+2E_1(-E_1\omega_{2_0}{}^2+E_3+F_2)}{(E_2+F_1)^2\omega_{2_0}{}^2+[-E_1\omega_{2_0}{}^2+(E_3+F_2)]^2}\Bigg],$$

$$= Sgn\Bigg[\frac{4\omega_{2_0}{}^6+B_1\omega_{2_0}{}^4+B_2\omega_{2_0}{}^2+B_3}{(E_2+F_1)^2\omega_{2_0}{}^2+[-E_1\omega_{2_0}{}^2+(E_3+F_2)]^2}\Bigg],$$

where

$B_1 = 3(C_1+D_1)^2 - 6(C_2+D_2),$

$B_2 = 2(C_2+D_2)^2 + 4(C_4+D_4) - 4(C_1+D_1)(C_3+D_3) - 2E_1^2,$

$B_3 = (C_3+D_3)^2 - 2(C_2+D_2)(C_4+D_4) - (E_2+F_1)^2 + 2E_1(E_3+F_2).$

Let

$$\Phi_1(\omega_{2_0}) = 4{\omega_{2_0}}^6 + B_1{\omega_{2_0}}^4 + B_2{\omega_{2_0}}^2 + B_3. \tag{4.13}$$

If $\Phi_1(\omega_{2_0}) > 0$ then $Sgn\left[\frac{d(Re\xi)}{d\tau_2}\right]_{\tau_2=\tau_{2_0}} > 0$, i.e. the transversality condition holds and the system undergoes Hopf bifurcation.

Case (c) : $\tau_1 > 0, \tau_2 = 0$

The endemic equilibrium of the model (4.6) is $E^*(Y^*, X_+^*, N^*, M^*, P^*)$ (see section 4.2.3). The variational matrix at endemic equilibrium $E^*(Y^*, X_+^*, N^*, M^*, P^*)$ is

$$J = \begin{pmatrix} -M_1-\xi & M_2 & M_3 & -M_4 & 0 \\ M_5 & -M_6-m_1e^{-\xi\tau_1}-\xi & M_7 & M_8 & -M_9 \\ -M_{10} & 0 & -M_{11}-\xi & 0 & 0 \\ \overline{m} & 0 & 0 & -M_{12}-\xi & 0 \\ M_{13}-m_2e^{-\xi\tau_1} & 0 & 0 & -M_{14}+m_3e^{-\xi\tau_1} & -M_{15}+m_4e^{-\xi\tau_1}-\xi \end{pmatrix}.$$

Here $M_1 = \beta Y^*$, $M_2 = -\beta Y^* + \frac{\beta Y^*}{1+\beta_1 M^*}$, $M_3 = \beta Y^*$, $M_4 = \frac{\beta\beta_1 X_+^* Y^*}{(1+\beta_1 M^*)^2}$, $M_5 = -\frac{\lambda M^*}{k+M^*} + p\gamma - \frac{\beta X_+^*}{1+\beta_1 M^*}$, $M_6 = \frac{\lambda M^*}{k+M^*} + d + \frac{\beta Y^*}{1+\beta_1 M^*}$, $M_7 = \frac{\lambda M^*}{k+M^*}$, $M_8 = \frac{\lambda(N^*-X_+^*-Y)k}{(k+M^*)^2} + \frac{\beta\beta_1 X_+^* Y^*}{(1+\beta_1 M^*)^2}$, $M_9 = \lambda_0 X_+^*$, $M_{10} = \alpha$, $M_{11} = d$, $M_{12} = \mu_0$, $M_{13} = m_2 = \frac{\beta P^*}{1+\beta_1 M^*}$, $M_{14} = m_3 = \frac{\beta\beta_1 Y^* P^*}{(1+\beta_1 M^*)^2}$, $M_{15} = m_1 = m_4 = \lambda_0 P^*$ and $\overline{m} = \mu$.

The characteristic equation is

$$\begin{aligned} &[\xi^5 + A_1\xi^4 + (A_2+F_1)\xi^3 + (A_3+F_2)\xi^2 + (A_4+F_3)\xi + (A_5+F_4)]e^{\xi\tau_1} + \\ &[C_1\xi^3 + C_2\xi^2 + (B_1+C_3)\xi + (B_2+C_4)]e^{-\xi\tau_1} + [D_1\xi^4 + D_2\xi^3 + \\ &(D_3+E_1)\xi^2 + (D_4+E_2)\xi + (D_5+E_3)] = 0. \end{aligned} \tag{4.14}$$

Here $A_1, A_2, A_3, A_5, B_1, B_2, \ldots F_4$ are given in Appendix A.

Theorem (4.2a) : Let $(A_5+B_2+C_4+F_4)^2 < (D_5+E_3)^2$ then the equilibrium E^* is LAS for $\tau_1 \in (0, \tau_{1_0})$ where τ_{1_0} is the minimum positive value of

$$\overline{\tau}_{1_0} = \frac{1}{\omega_{1_0}}\left[\arccos\left(-\frac{\overline{A}_{22}}{\overline{A}_{21}}\frac{\overline{A}_{26}+\overline{A}_{23}}{\overline{A}_{25}+\overline{A}_{22}}\frac{\overline{A}_{25}}{\overline{A}_{24}}\right)\right]$$

for ω_{1_0} corresponding to all positive real roots of (4.16) and the coefficients $\overline{A}_{2i}$ $(i = 1,2,3,4,5,6)$ are described below, provided it is stable in the absence of delay.

Proof : Put $\xi = i\omega$ in (4.14) and separating real and imaginary parts we get

$$\begin{aligned} &A_{21}\cos\omega\tau_1 - A_{22}\sin\omega\tau_1 + A_{23} = 0, \\ &A_{24}\cos\omega\tau_1 + A_{25}\sin\omega\tau_1 + A_{26} = 0, \end{aligned} \tag{4.15}$$

where

$A_{21} = A_1\omega^4 - (A_3 + C_2 + F_2)\omega^2 + (A_5 + B_2 + C_4 + F_4),$

$A_{22} = \omega^5 - (A_2 - C_1 + F_1)\omega^3 + (A_4 - B_1 - C_3 + F_3)\omega,$

$A_{23} = D_1\omega^4 - (D_3 + E_1)\omega^2 + (D_5 + E_3),$

$A_{24} = \omega^5 - (A_2 + C_1 + F_1)\omega^3 + (A_4 + B_1 + C_3 + F_3)\omega,$

$A_{25} = A_1\omega^4 - (A_3 - C_2 + F_2)\omega^2 + (A_5 - B_2 - C_4 + F_4),$

$A_{26} = -D_2\omega^3 + (D_4 + E_2)\omega.$

Eliminating τ_1 from (4.15) we get

$$H_1(\omega) = (A_{21}A_{25} + A_{22}A_{24})^2 - (A_{22}A_{26} + A_{23}A_{25})^2 - (A_{23}A_{24} - A_{21}A_{26})^2 = 0. \tag{4.16}$$

If $(A_5 + B_2 + C_4 + F_4)^2 - (D_5 + E_3)^2 < 0$ then $H_1(0) < 0$ and $H_1(\infty) = +\infty$. So equation (4.16) has at least one positive real root ω_{1_0}.

When $\omega = \omega_{1_0}$, equations (4.15) can be written as

$$\begin{aligned} &\overline{A}_{21}\cos\omega_{1_0}\tau_1 - \overline{A}_{22}\sin\omega_{1_0}\tau_1 + \overline{A}_{23} = 0, \\ &\overline{A}_{24}\cos\omega_{1_0}\tau_1 + \overline{A}_{25}\sin\omega_{1_0}\tau_1 + \overline{A}_{26} = 0. \end{aligned} \tag{4.17}$$

Here $\overline{A}_{21}, \overline{A}_{22}, \overline{A}_{23}, \overline{A}_{24}, \overline{A}_{25}$ and $\overline{A}_{26}$ are obtained by substituting $\omega = \omega_{1_0}$ into $A_{21}, A_{22}, A_{23}, A_{24}, A_{25}$ and A_{26}.

Equations (4.18) are simplified to give

$$\tau'_{1_n} = \frac{1}{\omega_{1_0}}\Big[\arccos\big(-\frac{\overline{A}_{22}}{\overline{A}_{21}}\frac{\overline{A}_{26}+\overline{A}_{23}}{\overline{A}_{25}+\overline{A}_{22}}\frac{\overline{A}_{25}}{\overline{A}_{24}}\big)\Big] + \frac{2n\pi}{\omega_{1_0}}; \qquad n = 0, 1, 2, \ldots ,$$

here $i\omega_{1_0}$ is a purely imaginary root of equation (4.14).

If $(A_5 + B_2 + C_4 + F_4)^2 - (D_5 + E_3)^2 < 0$ then the equilibrium $E^*(Y^*, X_+^*, N^*, M^*, P^*)$ is LAS for $\tau_1 < \tau_{1_0}$. Without loss of generality suppose that ω_{1_0} represents the value of ω_{1_0} corresponding to τ_{1_0}.

Theorem (4.2b) : If $\Phi_2(\omega_{1_0}) > 0$, where $\Phi_2(\omega_{1_0})$ satisfies (4.18) the system (4.6) undergoes a Hopf Bifurcation at the positive equilibrium as τ_1 increases through τ_{1_0}.

Proof : Transversality condition for Hopf-bifurcation :

Differentiating (4.14) with respect to τ_1 , we get $\frac{d\tau_1}{d\xi} =$

$$\frac{[5\xi^4+4A_1\xi^3+3(A_2+F_1)\xi^2+2(A_3+F_2)\xi+(A_4+F_3)]e^{\xi\tau_1}+[4D_1\xi^3+3D_2\xi^2+2(D_3+E_1)\xi+(D_4+E_2)]+[3C_1\xi^2+2C_2\xi+(B_1+C_3)e^{-\xi\tau_1}]}{[D_1\xi^5+D_2\xi^4+(D_3+E_1)\xi^3+(D_4+E_2)\xi^2+(D_5+E_3)\xi]+2[C_1\xi^4+C_2\xi^3+(B_1+C_3)\xi^2+(B_2+C_4)\xi]e^{-\xi\tau_1}} - \frac{\tau_1}{\xi},$$

$$Sgn\Big[\frac{d(Re\xi)}{d\tau_1}\Big]_{\tau_1=\tau_{1_0}} = Sgn\Big[Re(\frac{d\xi}{d\tau_1})^{-1}\Big]_{\xi=i\omega_{1_0}} = Sgn\Big[Re\frac{P_{11}+iP_{12}}{G_{11}+iG_{12}} + Re\frac{i\tau_1}{\omega_{1_0}}\Big] = Sgn\Big[\frac{P_{11}G_{11}+P_{12}G_{12}}{G_{11}^2+G_{12}^2}\Big].$$

P_{11}, P_{12}, G_{11} and G_{12} are given in Appendix A. Let

$$\Phi_2(\omega_{1_0}) = P_{11}G_{11} + P_{12}G_{12}. \tag{4.18}$$

If $\Phi_2(\omega_{1_0}) > 0$ then $Sgn\left[\frac{d(Re\xi)}{d\tau_1}\right]_{\tau_1=\tau_{1_0}} > 0$, i.e. the transversality condition holds and the system undergoes Hopf bifurcation.

Case (d) : $\tau_1 > 0$ and τ_2 fixed in $(0, \tau_{2_0})$

The endemic equilibrium of the model (4.6) is $E^*(Y^*, X_+^*, N^*, M^*, P^*)$ (see section 4.2.3). The variational matrix at the endemic equilibrium $E^*(Y^*, X_+^*, N^*, M^*, P^*)$ is

$$J = \begin{pmatrix} -M_1 - \xi & M_2 & M_3 & -M_4 & 0 \\ M_5 & -M_6 - m_1 e^{-\xi\tau_1} - \xi & M_7 & M_8 & -M_9 \\ -M_{10} & 0 & -M_{11} - \xi & 0 & 0 \\ \overline{m}e^{-\xi\tau_2} & 0 & 0 & -M_{12} - \xi & 0 \\ M_{13} - m_2 e^{-\xi\tau_1} & 0 & 0 & -M_{14} + m_3 e^{-\xi\tau_1} & -M_{15} + m_4 e^{-\xi\tau_1} - \xi \end{pmatrix}.$$

The characteristic equation is

$$\begin{aligned} &[\xi^5 + A_1\xi^4 + A_2\xi^3 + A_3\xi^2 + A_4\xi + A_5]e^{\xi\tau_1} + [B_1\xi + B_2]e^{-\xi(\tau_1+\tau_2)} + \\ &[C_1\xi^3 + C_2\xi^2 + C_3\xi + C_4]e^{-\xi\tau_1} + [D_1\xi^4 + D_2\xi^3 + D_3\xi^2 + D_4\xi + D_5] + \\ &[E_1\xi^2 + E_2\xi + E_3]e^{-\xi\tau_2} + [F_1\xi^3 + F_2\xi^2 + F_3\xi + F_4]e^{\xi(\tau_1-\tau_2)} = 0. \end{aligned} \tag{4.19}$$

Here M_{i_1} $(i_1 = 1{-}15)$, m_{i_2} $(i_2 = 1{-}4)$, $\overline{m}$, A_{i_3} $(i_3 = 1{-}5)$, B_{i_4} $(i_4 = 1{-}2)$, C_{i_5} $(i_3 = 1{-}4)$, D_{i_6} $(i_6 = 1{-}5)$, E_{i_7} $(i_7 = 1-3)$, F_{i_8} $(i_8 = 1-4)$ are the same as described in Case (c).

Theorem (4.3a) : Let $(A_5 + B_2 + C_4 + F_4)^2 < (D_5 + E_3)^2$ and $\tau_2 \in [0, \tau_{2_0})$ then the equilibrium E^* is LAS for $\tau_1 \in (0, \tau_{1_0}^{'})$ where

$$\tau_{1_0}^{'} = \frac{1}{\omega_{3_0}}\left[\arccos\left(-\frac{\overline{A}_{32}\ \overline{A}_{36}+\overline{A}_{33}\ \overline{A}_{35}}{\overline{A}_{31}\ \overline{A}_{35}+\overline{A}_{32}\ \overline{A}_{34}}\right)\right]$$

and the coefficients $\overline{A}_{3i}$ $(i = 1, 2, 3, 4, 5, 6)$ are described below.

Proof : It is assumed that with equation (4.19), τ_2 is in its stable interval and τ_1 is considered as a parameter. Put $\xi = i\omega$ in (4.19) and separating real and imaginary parts we get

$$\begin{aligned} A_{31}\cos\omega\tau_1 - A_{32}\sin\omega\tau_1 + A_{33} = 0, \\ A_{34}\cos\omega\tau_1 + A_{35}\sin\omega\tau_1 + A_{36} = 0. \end{aligned} \tag{4.20}$$

Here

$$A_{31} = [A_1\omega^4 - C_2\omega^3 - A_3\omega^2 + (A_5 + C_4)] + [-F_2\omega^2 + (B_2 + F_4)]\cos\omega\tau_2 + [-F_1\omega^3 + (B_1 + F_3)\omega]\sin\omega\tau_2,$$

$$A_{32} = [\omega^5 - (A_2 - C_1)\omega^3 + (A_4 - C_3)\omega] + [-F_1\omega^3 - (B_1 - F_3)\omega]\cos\omega\tau_2 + [F_2\omega^2 + (B_2 - F_4)]\sin\omega\tau_2,$$

$$A_{33} = [D_1\omega^4 - D_3\omega^2 + D_5] + [-E_1\omega^2 + E_3]\cos\omega\tau_2 + E_2\omega\sin\omega\tau_2,$$

$$A_{34} = [\omega^5 - (A_2 + C_1)\omega^3 + (A_4 + C_3)\omega] + [-F_1\omega^3 + (B_1 + F_3)\omega]\cos\omega\tau_2 + [F_2\omega^2 - (B_2 + F_4)]\sin\omega\tau_2,$$

$$A_{35} = [A_1\omega^4 + C_2\omega^3 - A_3\omega^2 + (A_5 - C_4)] + [-F_2\omega^2 - (B_2 - F_4)]\cos\omega\tau_2 + [-F_1\omega^3 - (B_1 - F_3)\omega]\sin\omega\tau_2,$$

$$A_{36} = [-D_2\omega^3 + D_4\omega] + E_2\omega\cos\omega\tau_2 - [-E_1\omega^2 + E_3]\sin\omega\tau_2.$$

Eliminating τ_1 from (4.20) we get

$$H_2(\omega) = (A_{31}A_{35} + A_{32}A_{34})^2 - (A_{32}A_{36} + A_{33}A_{35})^2 - (A_{33}A_{34} - A_{31}A_{36})^2 = 0. \tag{4.21}$$

Note that if $(A_5 + B_2 + C_4 + F_4)^2 - (D_5 + E_3)^2 < 0$ then $H_2(0) < 0$ and $H_2(\infty) = +\infty$.

Now the above equation (4.21) is a transcendental equation in ω. The form of equation (4.21) is very complicated and it is difficult to predict the nature of its roots. Without going into detailed analysis with (4.21) it is assumed there exists at least one real positive root ω_{3_0}.

When $\omega = \omega_{3_0}$, equation (4.20) can be written as

$$\begin{aligned} \overline{A}_{31}\cos\omega_{3_0}\tau_1 - \overline{A}_{32}\sin\omega_{3_0}\tau_1 + \overline{A}_{33} = 0, \\ \overline{A}_{34}\cos\omega_{3_0}\tau_1 + \overline{A}_{35}\sin\omega_{3_0}\tau_1 + \overline{A}_{36} = 0, \end{aligned} \tag{4.22}$$

where $\overline{A}_{31}, \overline{A}_{32}, \overline{A}_{33}, \overline{A}_{34}, \overline{A}_{35}, \overline{A}_{36}$ are obtained by substituting $\omega = \omega_{3_0}$ into $A_{31}, A_{32}, A_{33}, A_{34}, A_{35}$ and A_{36}.

Equations (4.22) are simplified to give

$$\tau'_{1_n} = \frac{1}{\omega_{3_0}}\Big[\arccos\big(-\frac{\overline{A}_{32}\ \overline{A}_{36}+\overline{A}_{33}\ \overline{A}_{35}}{\overline{A}_{31}\ \overline{A}_{35}+\overline{A}_{32}\ \overline{A}_{34}}\big)\Big] + \frac{2n\pi}{\omega_{3_0}}; \qquad n = 0, 1, 2, ...$$

here $i\omega_{3_0}$ is a purely imaginary root of equation (4.19).

If $(A_5 + B_2 + C_4 + F_4)^2 < (D_5 + E_3)^2$ and $\tau_2 \in [0, \tau_{2_0})$, then the equilibrium $E^*(Y^*, X_+^*, N^*, M^*, P^*)$ is LAS for $\tau_1 \in (0, \tau'_{1_0})$. Without loss of generality suppose that ω_{3_0} represents the value of ω_{3_0} corresponding to τ'_{1_0}.

Theorem (4.3b) : If $\Phi_3(\omega_{3_0}) > 0$, the system (4.6) undergoes a Hopf Bifurcation at the positive equilibrium as τ_1 increases through τ'_{1_0}, where the expression of $\Phi_3(\omega_{3_0})$ satisfies (4.23).

Proof : Transversality condition for Hopf-bifurcation :

Differentiating (4.19) with respect to τ_1 we get

$$Sgn\Big[\frac{d(Re\xi)}{d\tau_1}\Big]_{\tau_1=\tau'_{1_0}} = Sgn\Big[Re(\frac{d\xi}{d\tau_1})^{-1}\Big]_{\xi=i\omega_{3_0}} = Sgn\Big[Re\frac{P_{21}+iP_{22}}{G_{21}+iG_{22}} + Re\frac{i\tau'_{1_0}}{\omega_{3_0}}\Big] = Sgn\Big[\frac{P_{21}G_{21}+P_{22}G_{22}}{G_{21}^2+G_{22}^2}\Big].$$

Here P_{21}, P_{22}, G_{21} and G_{22} are given in the Appendix. Let

$$\Phi_3(\omega_{3_0}) = P_{21}G_{21} + P_{22}G_{22}. \tag{4.23}$$

If $\Phi_3(\omega_{3_0}) > 0$ then $Sgn\Big[\frac{d(Re\xi)}{d\tau_1}\Big]_{\tau_1=\tau_{1_0}^{'}} > 0$, i.e. the transversality condition holds and the system undergoes Hopf bifurcation.

Case (e) : $\tau_2 > 0$ and τ_1 fixed in $(0, \tau_{1_0})$

In a similar way as in Case (d) we can find the characteristic equation as

$$\begin{aligned}&[\xi^5 + A_1\xi^4 + A_2\xi^3 + A_3\xi^2 + A_4\xi + A_5] + [B_1\xi + B_2]e^{-\xi(2\tau_1+\tau_2)} +\\ &[C_1\xi^3 + C_2\xi^2 + C_3\xi + C_4]e^{-2\xi\tau_1} + [D_1\xi^4 + D_2\xi^3 + D_3\xi^2 + D_4\xi + D_5]e^{-\xi\tau_1} +\\ &[E_1\xi^2 + E_2\xi + E_3]e^{-\xi(\tau_1+\tau_2)} + [F_1\xi^3 + F_2\xi^2 + F_3\xi + F_4]e^{-\xi\tau_2} = 0.\end{aligned} \tag{4.24}$$

Theorem (4.4a) : Let $(A_5 + C_4 + D_5)^2 < (B_2 + E_3 + F_4)^2$ and $\tau_1 \in [0, \tau_{1_0})$ then the equilibrium E^* is LAS for $\tau_2 \in (0, \tau_{2_0}^{'})$ where $\tau_{2_0}^{'}$ is the minimum value of

$$\tau_{2_0}^{'} = \frac{1}{\omega_{4_0}}\Big[\arccos\big(-\frac{\overline{A}_{42}\ \overline{A}_{46}+\overline{A}_{43}\ \overline{A}_{45}}{\overline{A}_{41}\ \overline{A}_{45}+\overline{A}_{42}\ \overline{A}_{44}}\big)\Big]$$

over ω_{4_0} corresponding to all positive real roots of (4.26) and the coefficients $\overline{A}_{4i},\ (i = 1, 2, 3, 4, 5, 6)$ are described below.

Proof : It is considered that with equation (4.24), τ_1 is in its stable interval and τ_2 is considered as a parameter. Put $\xi = i\omega$ in (4.24) and separating real and imaginary parts we get

$$\begin{aligned}&A_{41}\cos\omega\tau_2 - A_{42}\sin\omega\tau_2 + A_{43} = 0,\\ &A_{44}\cos\omega\tau_2 + A_{45}\sin\omega\tau_2 + A_{46} = 0.\end{aligned} \tag{4.25}$$

Here

$$A_{41} = [-F_2\omega^2 + F_4] - [E_1\omega^2 - E_3]\cos\omega\tau_1 + E_2\omega\sin\omega\tau_1 + B_2\cos 2\omega\tau_1 + B_1\omega\sin 2\omega\tau_1,$$

$$A_{42} = [F_1\omega^3 - F_3\omega] - E_2\omega\cos\omega\tau_1 - [E_1\omega^2 - E_3]\sin\omega\tau_1 - B_1\omega\cos 2\omega\tau_1 + B_2\sin 2\omega\tau_1,$$

$$\begin{aligned}A_{43} = &[A_1\omega^4 - A_3\omega^2 + A_5] + [D_1\omega^4 - D_3\omega^2 + D_5]\cos\omega\tau_1 - [D_2\omega^3 - D_4\omega]\sin\omega\tau_1\\ &-[C_2\omega^2 - C_4]\cos 2\omega\tau_1 - [C_1\omega^3 - C_3\omega]\sin 2\omega\tau_1,\end{aligned}$$

$$A_{44} = [-F_1\omega^3 + F_3\omega] + E_2\omega\cos\omega\tau_1 + [E_1\omega^2 - E_3]\sin\omega\tau_1 + B_1\omega\cos 2\omega\tau_1 - B_2\sin 2\omega\tau_1,$$

$$A_{45} = [F_2\omega^2 - F_4] + [E_1\omega^2 - E_3]\cos\omega\tau_1 - E_2\omega\sin\omega\tau_1 - B_2\cos 2\omega\tau_1 - B_1\omega\sin 2\omega\tau_1,$$

$$\begin{aligned}A_{46} = &[\omega^5 - A_2\omega^3 + A_4\omega] - [D_2\omega^3 - D_4\omega]\cos\omega\tau_1 - [D_1\omega^4 - D_3\omega^2 + D_5]\sin\omega\tau_1\\ &-[C_1\omega^3 - C_3\omega]\cos 2\omega\tau_1 + [C_2\omega^2 - C_4]\sin 2\omega\tau_1.\end{aligned}$$

Eliminating τ_1 from (4.20) we get

$$H_2(\omega) = (A_{42}A_{46} + A_{43}A_{45})^2 + (A_{43}A_{44} - A_{41}A_{46})^2 - (A_{41}A_{45} + A_{42}A_{44})^2 = 0. \tag{4.26}$$

Note that if $(A_5 + C_4 + D_5)^2 < (B_2 + E_3 + F_4)^2 < 0$ then $H_2(0) < 0$ and $H_2(\infty) = +\infty$.

Again we assume that there exists at least one real positive root ω_{4_0}. When $\omega = \omega_{4_0}$ equation (4.25) can be written as

$$\begin{aligned} \overline{A}_{41} \cos \omega_{4_0} \tau_2 - \overline{A}_{42} \sin \omega_{4_0} \tau_2 + \overline{A}_{43} = 0, \\ \overline{A}_{44} \cos \omega_{4_0} \tau_2 + \overline{A}_{45} \sin \omega_{4_0} \tau_2 + \overline{A}_{46} = 0, \end{aligned} \tag{4.27}$$

where $\overline{A}_{41}, \overline{A}_{42}, \ldots \overline{A}_{46}$ are obtained by substituting $\omega = \omega_{4_0}$ into $A_{41}, A_{42}, \ldots A_{46}$.

Equations (4.27) are simplified to give

$$\tau'_{2_n} = \frac{1}{\omega_{4_0}} \Big[\arccos\big(- \frac{\overline{A_{42}}\,\overline{A_{46}} + \overline{A_{43}}\,\overline{A_{45}}}{\overline{A_{41}}\,\overline{A_{45}} + \overline{A_{42}}\,\overline{A_{44}}} \big) \Big] + \frac{2n\pi}{\omega_{4_0}}; \qquad n = 0, 1, 2, ...$$

here $i\omega_{4_0}$ is a purely imaginary root of equation (4.24).

If $(A_5 + C_4 + D_5)^2 < (B_2 + E_3 + F_4)^2$ and $\tau_1 \in [0, \tau_{1_0})$, then the equilibrium $E^*(Y^*, X^*_+, N^*, M^*, P^*)$ is LAS for $\tau_2 \in (0, \tau'_{2_0})$. Without loss of generality suppose that ω_{4_0} represents the value of ω_{4_0} corresponding to τ'_{2_0}.

Theorem (4.4b) : If $\Phi_4(\omega_{4_0}) > 0$, the system (4.6) undergoes a Hopf Bifurcation at the positive equilibrium as τ_2 increases through τ'_{2_0}, where $\Phi_4(\omega_{4_0})$ satisfies (4.28).

Proof : Transversality condition for Hopf-bifurcation :

Differentiating (4.24) with respect to τ_2 we get

$$Sgn\Big[\frac{d(Re\xi)}{d\tau_2}\Big]_{\tau_2 = \tau'_{2_0}} = Sgn\Big[Re\big(\frac{d\xi}{d\tau_2}\big)^{-1}\Big]_{\xi = i\omega_{4_0}} = Sgn\Big[Re\frac{P_{31} + iP_{32}}{G_{31} + iG_{32}} + Re\frac{i\tau'_{2_0}}{\omega_{4_0}}\Big] = Sgn\Big[\frac{P_{31}G_{31} + P_{32}G_{32}}{G_{31}^2 + G_{32}^2}\Big],$$

where P_{31}, P_{32}, G_{31} and G_{32} are given in the Appendix. Let

$$\Phi_4(\omega_{4_0}) = P_{31}G_{31} + P_{32}G_{32}. \tag{4.28}$$

If $\Phi_4(\omega_{4_0}) > 0$ then $Sgn\Big[\frac{d(Re\xi)}{d\tau_2}\Big]_{\tau_2 = \tau'_{2_0}} > 0$, i.e. the transversality condition holds and the system undergoes Hopf bifurcation.

4.4. Permanence

Biologically, persistence of a system means the survival of all populations of the system in future time. Mathematically, persistence of a system means that strictly positive solutions do not have omega limit points on the boundary of the non-negative cone. Butler, Freedman and Waltman [53], [54] developed the following definition of persistence:

Definition 4.4.1. System (4.6) is said to be permanent if there are positive constants l, L such that each positive solution $(Y(t), X_+(t), N(t), M(t), P(t))$ of system (4.6) with initial conditions corresponding to (4.3) satisfies

$$l \le \lim_{t\to+\infty} \inf Y(t) \le \lim_{t\to+\infty} \sup Y(t) \le L,$$

$$l \le \lim_{t\to+\infty} \inf X_+(t) \le \lim_{t\to+\infty} \sup X_+(t) \le L,$$

$$l \le \lim_{t\to+\infty} \inf N(t) \le \lim_{t\to+\infty} \sup N(t) \le L,$$

$$l \le \lim_{t\to+\infty} \inf M(t) \le \lim_{t\to+\infty} \sup M(t) \le L,$$

$$l \le \lim_{t\to+\infty} \inf P(t) \le \lim_{t\to+\infty} \sup P(t) \le L.$$

In order to prove permanence of system (4.6), we present the theory of permanence of infinite dimensional systems from Theorem 4.1 of Hale and Waltman [55]. Let X be a complete metric space. Suppose that $X^0 \in X$, $X_0 \in X$, $X^0 \bigcap X_0 = \emptyset$. Assume that $T(t)$ is a C_0 semigroup on X satisfying

$$\begin{aligned} T(t): X^0 &\rightarrow X^0, \\ T(t): X_0 &\rightarrow X_0. \end{aligned} \tag{4.29}$$

Let $T_b(t) = T(t)|_{X_0}$ and let A_b be the global attractor for $T_b(t)$.

Lemma 4.4.1 [55]. Suppose that $T(t)$ satisfies (4.29) and we have the following

(i) there is a $t_0 \ge 0$ such that $T(t)$ is compact for $t > t_0$;

(ii) $T(t)$ is point dissipative in X;

(iii) $\bar{A}_b = \bigcup_{x\in A_b} w(x)$ is isolated and has an acyclic covering L, where

$$L = \{L_1, L_2, \ldots, L_n\};$$

(iv) $W^s(L_i) \bigcap X^0 = \emptyset$ for $i = 1, 2, \ldots, n$.

Then X_0 is a uniform repeller with respect to X^0, i.e., there is an $\epsilon_0 > 0$ such that, for any $x \in X^0$, $\lim_{t\to+\infty} \inf \tilde{d}(T(t)x, X_0) \ge \epsilon$, where $\tilde{d}$ is the distance of $T(t)x$ from X_0.

Theorem 4.4.1. *If* $\frac{\beta\epsilon_0}{(\gamma+\alpha+d)} + 1 < R_0 < \frac{\beta\epsilon_0+p\gamma+d+\lambda_0}{(\gamma+\alpha+d)} + 1$, *then the system (4.6) is permanent.*

Proof : We begin by showing that the boundary planes of $\mathbb{R}^5_+$ repel the positive solutions to system (4.2) uniformly. Let us define C_0 to be

$$\{(\psi_1, \psi_2, \psi_3, \psi_4) \in C([-\tau, 0], \mathbb{R}^4_+ \times [0, 1]) : \psi_1(\theta_1) \ne 0, \psi_2(\theta_1) = 0, \psi_3(\theta_1) = 0, \psi_4(\theta_1) = 0 \text{ and } \psi_5(\theta_1) = 0\}.$$

If C^0 =int$C([-\tau,0],\mathbb{R}^4_+\times[0,1])$, it suffices to show that there exists an ϵ_0 such that for all solutions u_t of system (4.2) initiating from C^0, $\lim\limits_{t\to+\infty}\inf \tilde{d}(u_t,C_0)\geq\epsilon_0$. To this end we verify below that the conditions of Lemma 4.4.1 are satisfied. It is easy to see that C_0 and C^0 are positive invariant. Moreover, conditions (i) and (ii) of Lemma 4.4.1 are clearly satisfied. Thus, we only need to verify conditions (iii) and (iv).

There is a constant solution E_0 in C_0. That is $X_-(t)=\frac{A}{d}$, $X_+(t)=0$, $Y(t)=M(t)=P(t)=0$. If $(X_-(t),X_+(t),Y(t),M(t),P(t))$ is a solution of system (4.2) initiating in C_0, then $X_-(t)\to\frac{A}{d}, X_+(t)\to 0, Y(t)\to 0, M(t)\to 0$ and $P(t)\to 0$ as $t\to\infty$. It is obvious that E_0 is isolated invariant.

We now show that $W^s(E_0)\bigcap C^0=\emptyset$. Assuming the contrary, i.e. $W^s(E_0)\bigcap C^0\neq\emptyset$, then there exists a positive solution $(X_-(t),X_+(t),Y(t),M(t),P(t))$ of the system (4.2) such that $(Y(t),X_+(t),N(t),M(t),P(t))\to(0,0,\frac{A}{d},0,0)$ as $t\to+\infty$. Let us choose $\epsilon_0>0$ small enough such that $R_0>1+\epsilon_0$. Let $t_0>0$ be sufficiently large such that $\frac{A}{d}-\epsilon_0<X_-(t)<\frac{A}{d}+\epsilon_0$ for $t>t_0-\tau$. Then we have, for $t>t_0$,

$$\frac{dY}{dt} \;\geq\; \beta\Big(\frac{A}{d}-\epsilon_0-X_+(t)-Y(t)\Big)Y(t)+\frac{\beta}{1+\beta_1 M(t)}X_+(t)Y(t)-(\gamma+\alpha+d)Y(t). \tag{4.30}$$

Hence
$$\frac{dY}{dt} \;\geq\; \beta\Big(\frac{A}{d}-\epsilon_0-X_+(t)-Y(t)\Big)Y(t)-(\gamma+\alpha+d)Y(t), \tag{4.31}$$

or
$$\frac{1}{Y}\frac{dY}{dt} \;\geq\; \beta\Big[\Big(\frac{A}{d}-\epsilon_0\Big)-X_+(t)-Y(t)\Big]Y(t)-(\gamma+\alpha+d). \tag{4.32}$$

For X_+,Y sufficiently small and $R_0>1+\frac{\beta\epsilon_0}{(\gamma+\alpha+d)}$, $\frac{1}{Y}\,\frac{dY}{dt}\geq\epsilon_1>0$ for some $\epsilon_1>0$. Hence $\exists t_1\geq t_0$ such that $\frac{1}{Y}\frac{dY}{dt}\geq\epsilon_1>0$ for $T\geq t_1$. So $Y(t)\geq Y(t_1)e^{\epsilon_1(t-t_1)}$ for $t\geq t_1$ and $Y(t_1)>0$. This contradicts $Y(t)\to 0$ as $t\to\infty$. Therefore $(Y(t),X_+(t),N(t),M(t),P(t))\nrightarrow(0,0,\frac{A}{d},0,0)$, which is a contradiction. Hence $W^s(E_0)\bigcap C^0=\emptyset$. At this time, we are able to conclude from Lemma 4.4.1 that C_0 repels the positive solutions of the system (4.2) uniformly, then the conclusion of Theorem 4.4.1 follows.

5. Numerical simulations

To observe the dynamics of the system, numerical experiments were carried out using Matlab. We base our parameters on the spread of Pneumococcus amongst children under two in Scotland [56]. Pneumococcus is a bacterial disease which has no permanent immunity. Hence an SIS model is suitable. We try to illustrate the analytical results of this paper with realistic parameter values although the objective is more to illustrate the analytical results rather than obtain accurate predictions.

Lamb et al. estimate the size of the population at risk as $N=150,000$ and the per capita death rate as $d=1.3736\times10^{-3}$ day^{-1} giving $A=dN=206.04$ day^{-1}. The infectious period $\frac{1}{\gamma}$ is given by Weir [57] as $\frac{1}{\gamma}=7.1$ weeks giving $\gamma=0.1408$ week$^{-1}=0.02011$ day^{-1}. There is extremely low disease-related mortality from Pneumococcus carriage so we take $\alpha = 0.0$ day^{-1}. A Pneumococcus study by Zhang et al. [58] gives the basic reproduction number R_0 to be in the range 1.8-2.2. We take $R_0 = 2$ which then implies that $\beta = 2.865 \times 10^{-7}$ day^{-1}. The remaining parameters are concerned with the disease awareness program and as we do not have the data on this these are estimated hypothetically as follows: $\lambda = 0.9$ day^{-1}, $\lambda_0 = 0.3$ day^{-1}, $\mu = 1.3736 \times 10^{-3}$ day^{-1}, $\mu_0 = 0.01$ day^{-1}, $k = 500$, $\beta_1 = 1$ and $p = 0.6$.

For the above set of parameter values we obtain $E^* = (1787.4, 73524, 150000, 245.51)$, $\sigma_1 = 0.6097 > 0$, $\sigma_2 = 0.0068 > 0$, $\sigma_3 = 3.1364 \times 10^{-5} > 0$, $\sigma_4 = 3.1425 \times 10^{-8} > 0$, $\sigma_1\sigma_2 - \sigma_3 = 0.0041 > 0$ and $\sigma_1\sigma_2\sigma_3 - \sigma_3^2 - \sigma_1^2\sigma_4 = 1.179 \times 10^{-7} > 0$. Hence this clearly indicates that for the above set of parameter values the system is LAS around the positive interior equilibrium. Figure 1 illustrates that, as expected, simulations carried out for a long time appear to converge to this equilibrium. For the above parameter values and initial conditions we observe that the solutions converge to the steady state in approximately three years. We repeated the simulations with the same parameters and other starting values and found similar behaviour and convergence times. Note that including environmental or demographic stochasticity, and seasonal forcing (or more than one of these together) might change the behaviour of the system.

Next, we find the values of $\partial Y^*/\partial\mu$, Y^* and $\partial Y^*/\partial\beta_1$, Y^* and plot them with respect to μ, β_1 in Figure 2, 3 respectively. It is clear from Figure 2 and Figure 3 that if we increase either μ or β_1 or both, the equilibrium number of infected individuals decreases, which confirms the result given in Remark 1.

To study the impact of delays in system (4.2) we first fix $\tau_1 = 0$ days, and increase the value of τ_2 gradually. We observed that the system is LAS below a critical value τ_{2_0} (≈ 146 days, see Theorem 4.1) of τ_2 and undergoes Hopf bifurcation as τ_2 increases through τ_{2_0} (see Figure 4). For $\tau_2 \leq \tau_{2_0}$ there is a unique LAS endemic equilibrium whose components are plotted on the y-axes in Figure 4. For $\tau > \tau_{2_0}$ a stable limit cycle arises by Hopf bifurcation from this endemic equilibrium and Figure 4 plots the minimum and maximum values of these long-term stable limit cycle oscillations. Then we fixed $\tau_1 = 120$ days and drew the bifurcation diagram of the system (4.2) with respect to τ_2, we observe that the system enters into limit cycle oscillation from a stable equilibrium as we increase the value of τ_2 (see Figure 5). The system undergoes a Hopf bifurcation at $\tau_2 \approx 90$ days (i.e. $\tau'_{2_0} \approx 90$ days, see Theorem 4.4). Similarly, the system (4.2) loses its stability and enters into limit cycle oscillations through Hopf bifurcation at $\tau_{1_0} \approx 128.4$ days, when the second delay is absent ($\tau_2 \approx 0$). In a similar way, keeping τ_2 fixed at 60 days we observe that the system (4.2) undergoes a Hopf bifurcation at $\tau_1 \approx 134.7$ days (i.e. $\tau'_{1_0} \approx 134.7$ days, see Theorem 4.3). In Figure 6 we have drawn the domain of the stability region with respect to τ_1 and τ_2 to visualize the impact of delays in the stability of the system (4.2).

It is worth mentioning here that the interior equilibrium point of the system (4.6) depends on τ_1, which is very different from traditional delay models. In traditional delay models the equilibrium points of the delay model and the non-delay model are the same. However in the present investigation, we have considered the survival probability (P) in the interval of the time lag τ_1 corresponding to aware people forgetting the impact of disease after this time lag. The equilibrium value of P depends on τ_1 explicitly (see Appendix B). Consequently, the value of τ_1 directly influences equilibrium population numbers. In Figure 7 we have plotted the equilibrium number of infected individuals, Y^*, and the value of the survival probability, P^*, against τ_1. We observe that as τ_1 increases the equilibrium number of infected individuals decreases.

Our numerical computation also shows that for $\tau_1 = 0$ days, $P^* = 1$ and $Y^* = 1787.4$ and for $\tau_1 = 180$ days, $P^* = 0.051$ and $Y^* = 81.9$. Therefore, it is clear that if the susceptible individuals become aware and remain aware for a long time then the equilibrium number of infected individuals decreases. However, we have also observed that for $\tau_1 > \tau_{1_0}$ ($\tau_{1_0} \approx 128.4$ days), the system shows limit cycle oscillation, which poses a challenge for controlling the epidemic outbreak.

6. Conclusion

In this paper we have considered the effect of disease awareness programs on disease dynamics where the susceptible population is divided into two different classes, aware susceptible and unaware susceptible. The model was considered first without any time delay and then with two time delays. The first time delay was due to people forgetting the impact of the disease after a time lag τ_1. The second time delay was due to the media mounting a disease awareness campaign because of cases that had previously occurred after a time lag τ_2.

A differential equation model was used to examine the disease spread firstly with no time delay and then with a time delay. For the model with no time delay an expression for the basic reproduction number R_0 was calculated. The DFE is LAS if and only if $R_0 < 1$. For $R_0 > 1$ the DFE becomes unstable and an endemic equilibrium exists.

For the model with no time delay sufficient conditions for the endemic equilibrium to be LAS were derived. For the model with two time delays sufficient conditions for the stability of the endemic equilibrium and the existence of Hopf bifurcations were obtained for four different sets of values of the delay parameters, i.e. when $\tau_1 = 0,\ \tau_2 > 0;\ \tau_2 = 0,\ \tau_1 > 0$ and the two cases when $\tau_1 > 0$ and $\tau_2 > 0$ (see Theorems 4.1, 4.2, 4.3 and 4.4).

Numerical simulations were performed to investigate the behavior of the system. They indicated that the system was LAS with realistic parameter values. We used the numerical simulations to visualize the effect of increasing time delays on the dynamics of the system.

We observed that in our model if we increase the number of campaigns due to the awareness program then the disease transmission rate amongst the susceptible population declines. The numerical simulations also indicate that if the implementation rate of the awareness program increases then the equilibrium number of infected individuals decreases. We have also observed that if the time lag (τ_1) in rejoining the unaware class of aware individuals increases, i.e. the susceptible individuals remain aware for a longer time, then the equilibrium number of infected individuals reduces. However, sustained oscillation may arise if the the time lag increases over a threshold value which could possibly pose a challenge in controlling the epidemic.

However, the restrictions on the rate of immigration could have the ability to control the epidemic. It might be possible to control oscillations by controlling the rate of immigration [20]. Restricting immigration might have a stabilizing effect on disease dynamics.

In the present study we have considered the impact of an awareness campaign that acts on the whole population uniformly. This is a commonly made assumption in the literature on modeling media awareness campaigns. It would be appropriate for control of a disease that is established over a wide area. However it would not be appropriate for controlling a local outbreak of disease where an awareness campaign would have to be much more geographically focussed and act mostly on the local population. In those circumstances we would expect the impact of an awareness campaign to decrease as we move away from the epidemic outbreak or the number of infected individuals reduces. This would require a more sophisticated model and is a possible direction for future research. Note also that although the functional forms of the disease transmission term and the spread of information term have similarities we are not necessarily assuming the same transmission routes. Some other possible information transfer mechanisms could require fundamentally different information transmission terms [32]. This is also another potential direction for future work.

Acknowledgement The authors are thankful to the anonymous reviewers and editor for their useful comments and suggestions. The research works of S. Samanta and T. Sardar are supported by Council of Scientific and Industrial Research (CSIR), Human Resource Development Group, New Delhi, India. The research of J. Chattopadhyay is supported by a DAE project (Ref No. 2/48(4)/2010-R&D II/8870).

References

[1] DCPP. Infectious diseases. Changes in individual behavior could limit the spread of infectious diseases. *Disease Control Priorities Project*, http://www.dcp2.org/file/6/, 2006.

[2] WHO. World Health Organisation. *Media centre, Pneumonia - fact sheet no 331: August 2012*, http://www.who.int/mediacentre/factsheets/fs331/en/index.html, 2012.

[3] WHO. World Health Organisation. *Media centre, Diarrhoeal disease - fact sheet no 330: August 2009*, http://www.who.int/mediacentre/factsheets/fs330/en/index.html, 2009.

[4] WHO. World Health Organisation. *Media centre, Tuberculosis - fact sheet no 104: March 2012*, http://www.who.int/mediacentre/factsheets/fs104/en/index.html, 2012.

[5] WHO. World Health Organisation. *Media centre, Malaria - fact sheet no 094: April 2012*, http://www.who.int/mediacentre/factsheets/fs094/en/index.html, 2012.

[6] WHO. World Health Organisation. *Media centre, Measles - fact sheet no 286: April 2012*, http://www.who.int/mediacentre/factsheets/fs286/en/index.html, 2012.

[7] WHO. World Health Organisation. *Media centre, HIV/AIDS - fact sheet no 360: July 2012*, http://www.who.int/mediacentre/factsheets/fs360/en/index.html, 2012.

[8] WHO. World Health Organisation. *Media centre, Influenza - fact sheet no 211: April 2009*, http://www.who.int/mediacentre/factsheets/fs211/en/index.html, 2009.

[9] N. Pandit, Y. Patel, and B. Bhavsar. Awareness and practice about preventive method against mosquito bite in Gujarat. *Healthline*, 1 (2010) 16-20.

[10] J. T. Lau, X. Yang, H. Y. Tusi, and J. H. Kim. Impacts of SARS on health-seeking behaviors in general population in Hong Kong. *Preventive Medicine*, 41 (2005) 454-462.

[11] I. S. Kristiansen, P. A. Halvorsen, and D. Gyrd-Hansen. Influenza pandemic: perception of risk and individual precautions in a general population. Cross sectional study. *BMC Public Health*, 7 (2007) 48.

[12] J. H. Jones and M. Salathe. Early assessment of anxiety and behavioral response to novel swine-origin influenza A (H1N1). *PLoS ONE*, 4 (2009) e8032.

[13] G. J. Rubin, R. Amlôt, L. Page, and S. Wessely. Public perceptions, anxiety, and behaviour change in relation to the swine flu outbreak: cross sectional telephone survey. *British Medical Journal*, 339 (2009) doi: 10.1136/bmj.b2651.

[14] N. T. Brewer, G. B. Chapman, F. X. Gibbons, M. Gerrard, K. D. McCaul, and N. D. Weinstein. Meta-analysis of the relationship between risk perception and health behavior: the example of vaccination. *Health Psychology*, 26 (2007) 136-145.

[15] C. S. Tracy, E. Rea, and R. Upshur. Public perceptions of quarantine: community-based telephone survey following an infectious disease outbreak. *BMC Public Health*, 9 (2009):doi:10.1186/1471–2458–9–470.

[16] A. Ahituv, V. J. Hotz, and T. Philipson. The responsiveness of the demand for condoms to the local prevalence of AIDS. *The Journal of Human Resources*, 31 (1996) 869-897.

[17] R. Liu, J. Wu, and H. Zhu. Media/psychological impact on multiple outbreaks of emerging infectious diseases. *Computational and Mathematical Methods in Medicine*, 8 (2007) 153-164.

[18] A. K. Misra, A. Sharma, and J. B. Shukla. Modeling and analysis of effects of awareness programs by media on the spread of infectious diseases. *Mathematical and Computer Modelling*, 53 (2011) 1221-1228.

[19] J. M. Tchuenche, N. Dube, C. P. Bhunu, R. J. Smith, and C. T. Bauch. The impact of media coverage on the transmission dynamics of human influenza. *BMC Public Health*, 11 (2011).

[20] S. Samanta, S. Rana, A. Sharma, A. K. Misra, and J. Chattopadhyay. Effect of awareness programs by media on the epidemic outbreaks: a mathematical model. *Applied Mathematics and Computation*, 219 (2013) 6965-6977.

[21] J. Cui, Y. Sun, and H. Zhu. The impact of media on the control of infectious diseases. *Journal of Dynamics and Differential Equations*, 20 (2007) 31-53.

[22] M. S. Rahman and M. L. Rahman. Media and education play a tremendous role in mounting AIDS awareness among married couples in Bangladesh. *AIDS Research and Therapy*, 4 (2007) 10.

[23] Z. Tai and T. Sun. Media dependencies in a changing media environment: the case of the 2003 SARS epidemic in China. *New Media and Society*, 9 (2007) 987-1009.

[24] J. Cui, X. Tao, and H. Zhu. An SIS infection model incorporating media coverage. *Rocky Mountain Journal of Mathematics*, 38 (2008) 1323-1334.

[25] H. Joshi, S. Lenhart, K. Albright, and K. Gipson. Modeling the effect of information campaigns on the HIV epidemic in Uganda. *Mathematical Biosciences and Engineering*, 5 (2008) 557-570.

[26] Y. Li, C. Ma, and J. Cui. The effect of constant and mixed impulsive vaccination on SIS epidemic models incorporating media coverage. *Rocky Mountain Journal of Mathematics*, 38 (2008) 1437-1455.

[27] Y. Liu and J. Cui. The impact of media convergence on the dynamics of infectious diseases. *International Journal of Biomathematics*, 1 (2008) 65-74.

[28] M. E. Young, G. R. Norman, and K. R. Humphreys. Medicine in the popular press: The influence of the media on perceptions of disease. *PLoS ONE*, 3 (2008) e3552.

[29] F. H. Chen. Modeling the effect of information quality on risk behavior change and the transmission of infectious diseases. *Mathematical Biosciences*, 217 (2009) 125-133.

[30] S. Funk, E. Gilad, C. Watkins, and V. A. A. Jansen. The spread of awareness and its impact on epidemic outbreaks. *Proceedings of the National Academy of Sciences USA*, 106 (2009) 6872-6877.

[31] Y. Li and J. Cui. The effect of constant and pulse vaccination on SIS epidemic models incorporating media coverage. *Communications in Nonlinear Science and Numerical Simulation*, 14 (2009) 2353-2365.

[32] I. Z. Kiss, J. Cassell, M. Recker, and P. L. Simon. The impact of information transmission on epidemic outbreaks. *Mathematical Biosciences*, 225 (2010) 1-10.

[33] A. Mummert and H. Weiss. Get the news out loudly and quickly: Modeling the influence of the media on limiting infectious disease outbreaks. *http://arxiv.org/pdf/1006.5028v2.pdf*, 2010.

[34] B. K. Yoo, M. L. Holland, J. Bhattacharya, C. E. Phelps, and P. G. Szilagyi. Effects of mass media coverage on timing and annual receipt of influenza vaccination among medicare elderly. *Health Services Research*, 45 (2010) 1287-1309.

[35] A. K. Misra, A. Sharma, and V. Singh. Effect of awareness programs in controlling the prevalence of an epidemic with time delay. *Journal of Biological Systems*, 19 (2011) 389-402.

[36] C. Sun, W. Yang, J. Arino, and K. Khan. Effect of media-induced social distancing on disease transmission in a two patch setting. *Mathematical Biosciences*, 230 (2011) 87-95.

[37] B. Olowokure, O. Odedere, A.J. Elliot, A. Awofisayo, E. Smit, A. Fleming, and H. Osmanc. Volume of print media coverage and diagnostic testing for influenza A (H1N1) pdm09 virus during the early phase of the 2009 pandemic. *Journal of Clinical Virology*, 55 (2012) 75-78.

[38] J. M. Tchuenche and C. T. Bauch. Dynamics of an infectious disease where media coverage influences transmission. *ISRN Biomathematics*, 2012 (2012).

[39] S. Funk and V.A. Jansen. *The Talk of the Town: Modelling the Spread of Information and Changes in Behaviour. In: Modeling the Interplay Between Human Behavior and the Spread of Infectious Diseases (Editors P. Manfredi and A. D'Onofrio).* Springer, New York, 2013, pp. 93-102.

[40] W. Liu. A SIRS epidemic model incorporating media coverage with random perturbation. *In Abstract and Applied Analysis*, 2013 (2013) Article ID 792308, 9 pages.

[41] Y. Wang, J. Cao, Z. Jin, H. Zhang, and G.Q. Sun. Impact of media coverage on epidemic spreading in complex networks. *Physica A: Statistical Mechanics and its Applications*, 392 (2013) 5824-5835.

[42] N. Kaur, M. Ghosh, and S.S. Bhatia. Modeling and analysis of an SIRS epidemic model with effect of awareness programs by media. *International Journal of Mathematical, Computational, Physical and Quantum Engineering*, 8 (2014) 233-239.

[43] S. Samanta and J. Chattopadhyay. Effect of awareness program in disease outbreak - A slow-fast dynamics. *Applied Mathematics and Computation*, 237 (2014) 98-109.

[44] A. Sharma and A. Misra. Modeling the impact of awareness created by media campaigns on vaccination coverage in a variable population. *Journal of Biological Systems*, 22 (2014) 249-270.

[45] A. Wang and Y. Xiao. A Filippov system describing media effects on the spread of infectious diseases. *Nonlinear Analysis: Hybrid Systems*, 11 (2014) 84-97.

[46] H. Zhao, Y. Lin, and Y. Dai. An SIRS epidemic model incorporating media coverage with time delay. *Computational and Mathematical Methods in Medicine*, 2014 (2014) Article ID 680743, 10 pages.

[47] G.P. Sahu and J. Dhar. Dynamics of an SEQIHRS epidemic model with media coverage, quarantine and isolation in a community with pre-existing immunity. *Journal of Mathematical Analysis and Applications*, 421 (2015) 1651-1672.

[48] H. I. Freedman and J. W. H. So. Global stability and persistence of simple food chains. *Mathematical Biosciences*, 76 (1985) 69-86.

[49] J. K. Hale. *Theory of Functional Differential Equations.* Springer-Verlag, New York, Heidelberg, Berlin, 1977.

[50] X. Yang, L. S. Chen, and J. F. Chen. Permanence and positive periodic solution for the single species nonautonomous delay diffusive model. *Computers and Mathematics with Applications*, 32 (1996) 109-116.

[51] G. Birkhoff and G. C. Rota. *Ordinary Differential Equations.* John Wiley and Sons, Ginn, Boston, 1989.

[52] H.L. Freedman and V. Sree Hari Rao. The trade-off between mutual interference and time lags in predator-prey systems. *Bulletin of Mathematical Biology*, 45 (1983) 991-1004.

[53] G. Butler, H. I. Freedman, and P. Waltman. Uniformly persistent systems. *Proceedings of the American Mathematical Society*, 96 (1986) 425-430.

[54] H. I. Freedman and P. Waltman. Persistence in a model of three competitive populations. *Mathematical Biosciences*, 73 (1985) 89-101.

[55] J. K. Hale and P. Waltman. Persistence in infinite-dimensional systems. *SIAM Journal on Mathematical Analysis*, 20 (1989) 388-395.

[56] K. E. Lamb, D. Greenhalgh, and C. Robertson. A simple mathematical model for genetic effects in pneumococcal carriage and transmission. *Journal of Computational and Applied Mathematics*, 235 (2011) 1812-1818.

[57] A. Weir. *Modelling the Impact of Vaccination and Competition on Pneumococcal Carriage and Disease in Scotland*. Unpublished Ph.D. Thesis, University of Strathclyde, Glasgow, Scotland, 2009.

[58] Q. Zhang, K. Arnaoutakis, C. Murdoch, R. Lakshman, G. Race, R. Burkinshaw, and A. Finn. Mucosal immune responses to capsular pneumococcal polysaccharides in immunized preschool children and controls with similar nasal pneumococcal colonization rates. *Pediatric Infectious Disease Journal*, 23 (2004) 307-313.

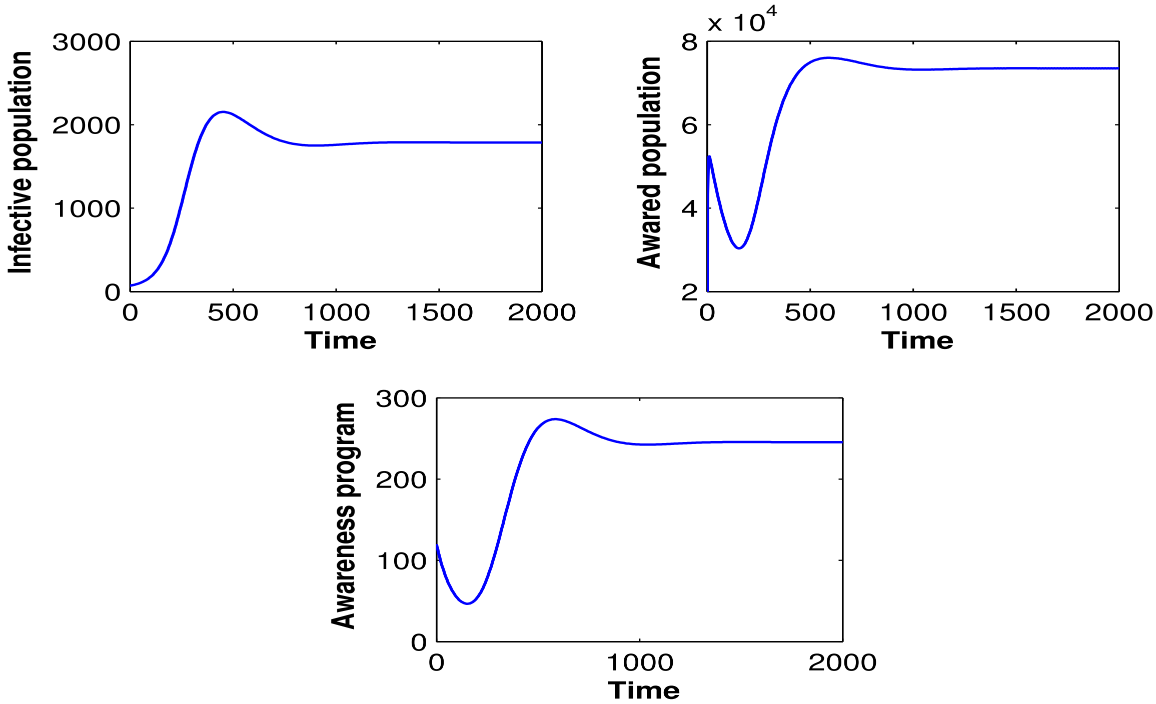

Figure 1: Stable population distribution of (3.2) in absence of both delays ($\tau_1 = \tau_2 = 0$ days). Other parameter values are $\beta = 2.8650 \times 10^{-7}$ day^{-1}, $\lambda = 0.9$ day^{-1}, $\lambda_0 = 0.3$ day^{-1}, $\gamma = 0.02011$ day^{-1}, $d = 1.3736 \times 10^{-3}$ day^{-1}, $\mu = 1.3736 \times 10^{-3}$ day^{-1}, $\mu_0 = 0.01$ day^{-1}, $\alpha = 0, k = 500, \beta_1 = 1, A = 206.04$ day^{-1}, $p = 0.6$.

.

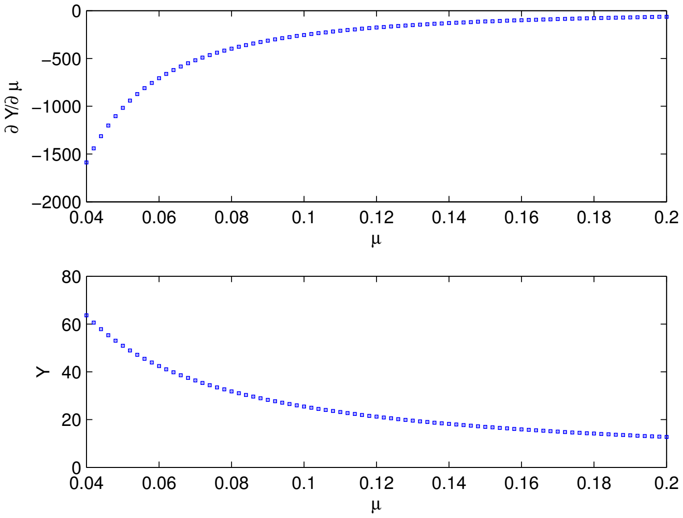

Figure 2: The figure depicts that the equilibrium number of infected individuals reduces with increasing μ (day^{-1}) where other parameter values are kept the same as in Figure 1.

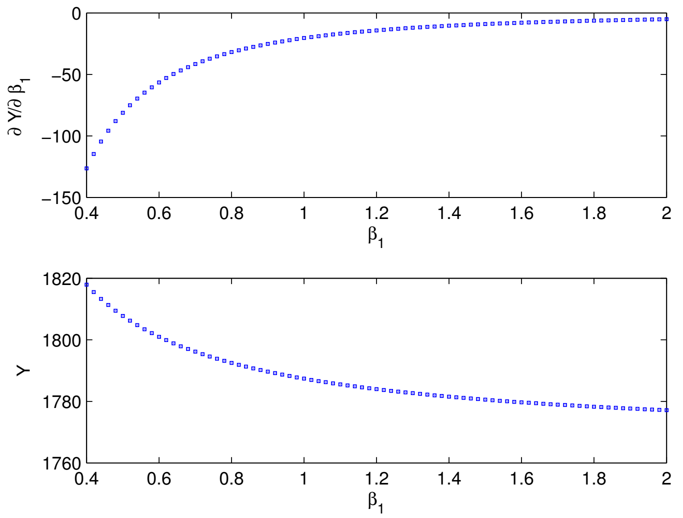

Figure 3: The figure depicts that the equilibrium number of infected individuals reduces with increasing β_1.

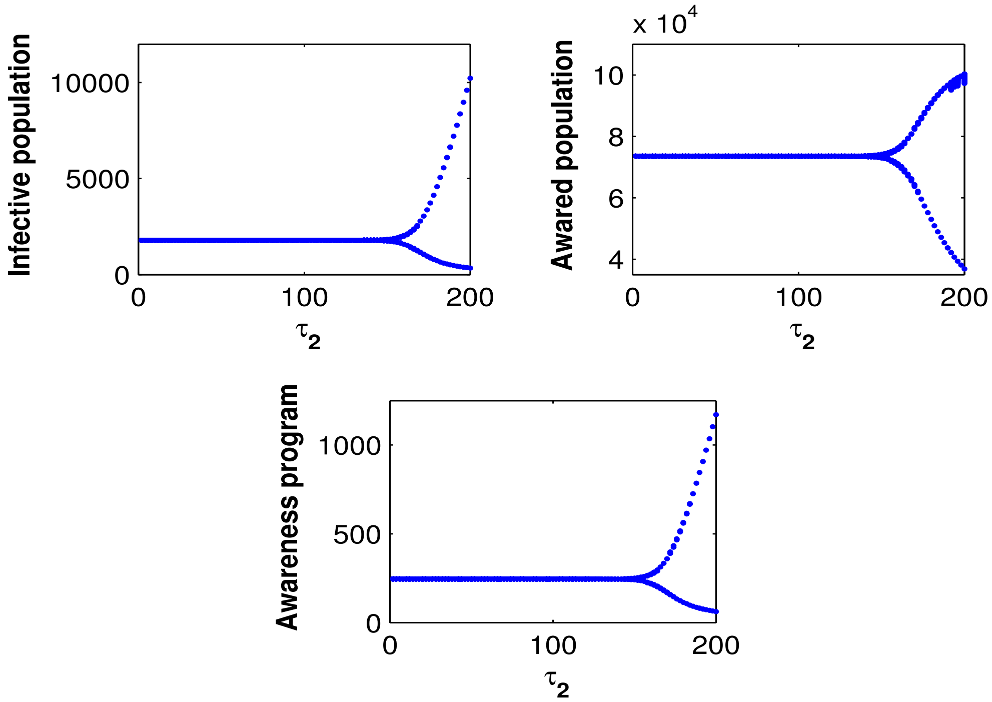

Figure 4: Diagram showing Hopf bifurcation of system (4.2) with respect to τ_2 (days) when $\tau_1 = 0$ days.

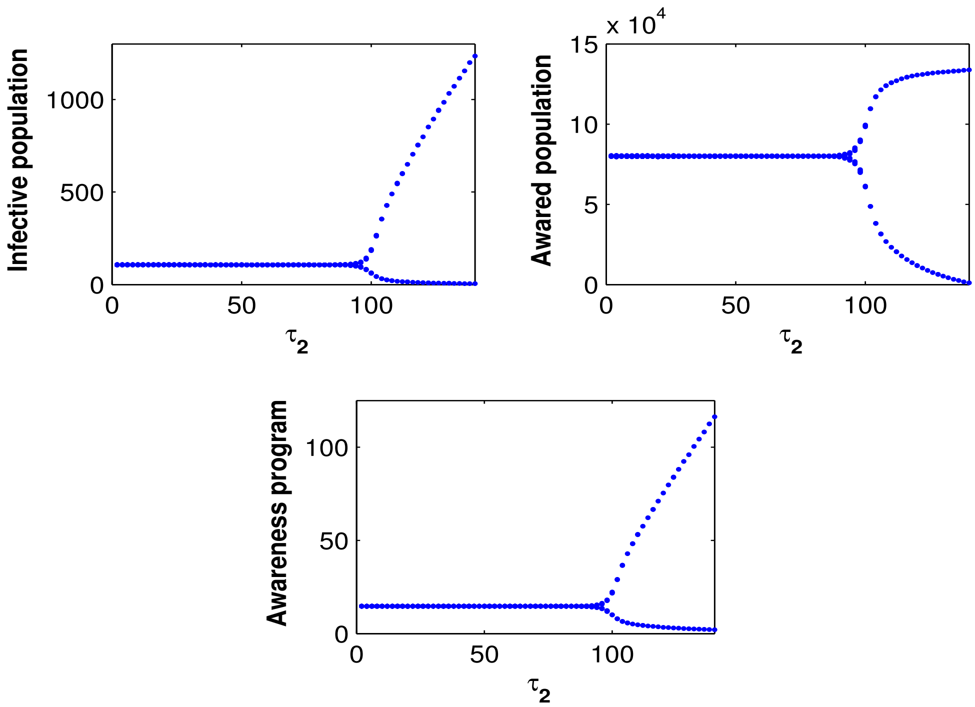

Figure 5: Diagram showing Hopf bifurcation of system (4.2) with respect to τ_2 (days) when $\tau_1 = 120$ days.

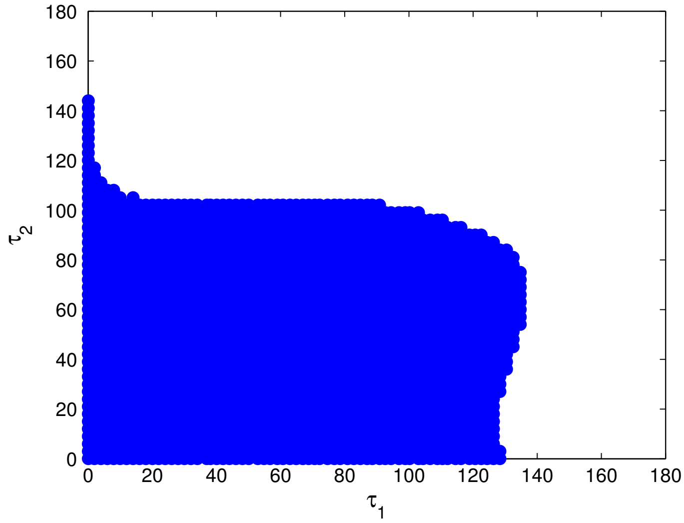

Figure 6: Domain of stability region with respect to τ_1 (days) and τ_2 (days) for the model (4.2). Other parameter values are kept the same as in Figure 1.

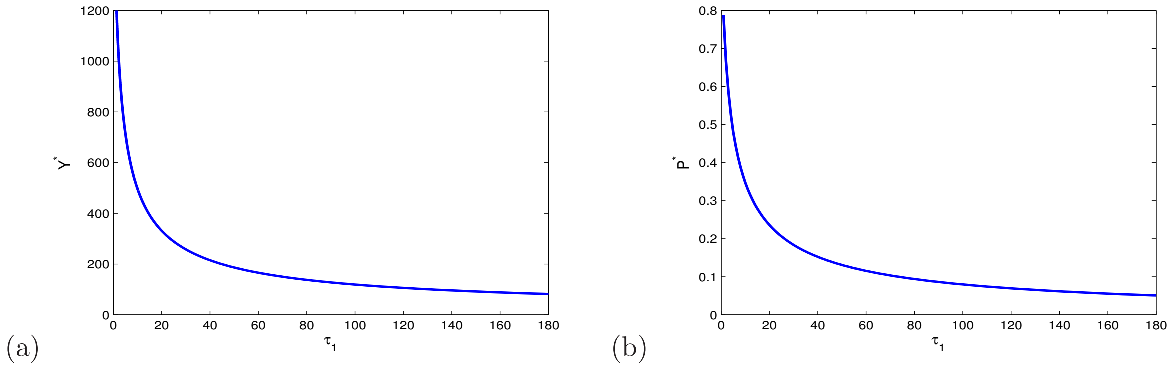

Figure 7: Figures 7(a) and 7(b) show that the equilibrium number of infected individuals (Y^*) and survival probability (P^*) decrease for increase in τ_1.

Appendix A

Detailed mathematical expansions of terms in the paper.

A.1 Terms in characteristic equation (4.14).

$$A_1 = M_1 + M_6 + M_{11} + M_{12} + M_{15},$$

$$A_2 = M_1M_{11} + M_1M_{12} + M_1M_{15} + M_{11}M_{12} + M_{11}M_{15} + M_{12}M_{15} + M_3M_{10} + M_1M_6 - M_2M_5 + M_6M_{11} + M_6M_{12} + M_6M_{15},$$

$$A_3 = M_1M_{11}M_{12} + M_1M_{11}M_{15} + M_1M_{12}M_{15} + M_{11}M_{12}M_{15} + M_3M_{10}M_{12} + M_3M_{10}M_{15} + M_1M_6M_{11} - M_2M_5M_{11} + M_1M_6M_{12} - M_2M_5M_{12} + M_1M_6M_{15} - M_2M_5M_{15} + M_6M_{11}M_{12} + M_6M_{11}M_{15} + M_6M_{12}M_{15} + M_2M_7M_{10} + M_3M_6M_{10} + M_2M_9M_{13},$$

$$A_4 = M_1M_{11}M_{12}M_{15} + M_3M_{10}M_{12}M_{15} + M_1M_6M_{11}M_{12} - M_2M_5M_{11}M_{12} + M_1M_6M_{11}M_{15} - M_2M_5M_{11}M_{15} + M_1M_6M_{12}M_{15} - M_2M_5M_{12}M_{15} + M_6M_{11}M_{12}M_{15} + M_2M_7M_{10}M_{12} + M_3M_6M_{10}M_{12} + M_2M_7M_{10}M_{15} + M_3M_6M_{10}M_{15} + M_2M_9M_{11}M_{13} + M_2M_9M_{12}M_{13},$$

$$A_5 = M_1M_6M_{11}M_{12}M_{15} - M_2M_5M_{11}M_{12}M_{15} + M_2M_7M_{10}M_{12}M_{15} + M_3M_6M_{10}M_{12}M_{15} + M_2M_9M_{11}M_{12}M_{13},$$

$$B_1 = -M_4\ \overline{m}\ m_1m_4,$$

$$B_2 = -M_{11}M_4\ \overline{m}\ m_1m_4,$$

$$C_1 = -m_1m_4,$$

$$C_2 = -(M_1 + M_{11} + M_{12})m_1m_4,$$

$$C_3 = -(M_1M_{11} + M_1M_{12} + M_{11}M_{12} + M_3M_{10})m_1m_4,$$

$$C_4 = -(M_1M_{11}M_{12} + M_3M_{10}M_{12})m_1m_4,$$

$$D_1 = m_1 - m_4,$$

$$D_2 = (M_1 + M_{11} + M_{12} + M_{15})m_1 - (M_1 + M_{11} + M_{12} + M_6)m_4,$$

$$D_3 = (M_1M_{11} + M_1M_{12} + M_1M_{15} + M_{11}M_{12} + M_{11}M_{15} + M_{12}M_{15} + M_3M_{10})m_1 - (M_1M_{11} + M_1M_{12} + M_{11}M_{12} + M_3M_{10} + M_1M_6 - M_2M_5 + M_6M_{11} + M_6M_{12})m_4 - M_2M_9m_2,$$

$$D_4 = (M_1M_{11}M_{12} + M_1M_{11}M_{15} + M_1M_{12}M_{15} + M_{11}M_{12}M_{15} + M_3M_{10}M_{12} + M_3M_{10}M_{15})m_1 - (M_1M_{11}M_{12} + M_3M_{10}M_{12} + M_1M_6M_{11} - M_2M_5M_{11} + M_1M_6M_{12} - M_2M_5M_{12} + M_6M_{11}M_{12} + M_2M_7M_{10} + M_3M_6M_{10})m_4 - (M_2M_9M_{11} + M_2M_9M_{12})m_2,$$

$$D_5 = (M_1M_{11}M_{12}M_{15} + M_3M_{10}M_{12}M_{15})m_1 - (M_1M_6M_{11}M_{12} - M_2M_5M_{11}M_{12} + M_2M_7M_{10}M_{12} + M_3M_6M_{10}M_{12})m_4 - M_2M_9M_{11}M_{12}m_2,$$

$$E_1 = M_4\ \overline{m}\ (m_1 - m_4),$$
$$E_2 = (M_4M_{11}m_1 + M_4M_{15}m_1 - M_4M_{11}m_4 + M_2M_8m_4 + M_2M_9m_3 - M_4M_6m_4)\ \overline{m},$$
$$E_3 = (M_4M_{15}m_1 + M_2M_8m_4 + M_2M_9m_3 - M_4M_6m_4)M_{11}\ \overline{m},$$

$$F_1 = M_4\ \overline{m},$$
$$F_2 = (M_4M_{11} + M_4M_{15} - M_2M_8 + M_4M_6)\ \overline{m},$$
$$F_3 = (M_4M_{11}M_{15} - M_2M_8M_{11} + M_4M_6M_{11} - M_2M_8M_{15} + M_4M_6M_{15} - M_2M_9M_{14})\ \overline{m},$$
$$F_4 = -(M_2M_8M_{15} - M_4M_6M_{15} + M_2M_9M_{14})M_{11}\ \overline{m}.$$

A.2 Terms in the transversality condition of Theorem 4.2b.

$$P_{11} = [5\omega_{1_0}^4 - 3(A_2 + F_1 + C_1)\omega_{1_0}^2 + (A_4 + F_3 + B_1 + C_3)]\cos\omega_{1_0}\tau_{1_0}$$
$$+[4A_1\omega_{1_0}^3 - 2(A_2 + F_2 - C_2)\omega_{1_0}]\sin\omega_{1_0}\tau_{1_0} + [-3D_2\omega_{1_0}^2 + (D_4 + E_2)],$$
$$P_{12} = -[4A_1{\omega_{1_0}}^3 - 2(A_3 + F_2 + C_2)\omega_{1_0}]\cos\omega_{1_0}\tau_{1_0}$$
$$+[5{\omega_{1_0}}^4 - 3(A_2 + F_1 - C_1)\omega_{1_0}^2 + (A_4 + F_3 - B_1 - C_3)]\sin\omega_{1_0}\tau_{1_0} + [-4D_1\omega_{1_0}^3 + 2(D_3 + E_1)\omega_{1_0}],$$
$$G_{11} = 2[C_1\omega_{1_0}^4 - (B_1 + C_3)\omega_{1_0}^2]\cos\omega_{1_0}\tau_{1_0} + 2[-C_2\omega_{1_0}^3 + (B_2 + C_4)\omega_{1_0}]\sin\omega_{1_0}\tau_{1_0}$$
$$+[D_2\omega_{1_0}^4 - (D_4 + E_2)\omega_{1_0}^2],$$
$$G_{12} = 2[-C_2\omega_{1_0}^3 + (B_2 + C_4)\omega_{1_0}]\cos\omega_{1_0}\tau_{1_0} - 2[C_1\omega_{1_0}^4 - (B_1 + C_3)\omega_{1_0}^2]\sin\omega_{1_0}\tau_{1_0}$$
$$+[D_1\omega_{1_0}^5 - (D_4 + E_1)\omega_{1_0}^3 + (D_5 + E_3)\omega_{1_0}].$$

A.3 Terms in the transversality condition of Theorem 4.3b.

$$P_{21} = [5\omega_{3_0}^4 - 3A_2\omega_{3_0}^2 + A_4] + [B_1 - \tau_2B_2]\cos\omega_{3_0}(2\tau_{1_0}' + \tau_2) - \tau_2B_1\omega_{3_0}\sin\omega_{3_0}(2\tau_{1_0}' + \tau_2)$$
$$+[-3C_1\omega_{3_0}^2 + C_3]\cos 2\omega_{3_0}\tau_{1_0}' + 2C_2\sin 2\omega_{3_0}\tau_{1_0}' + [-3D_2\omega_{3_0}^2 + D_4]\cos\omega_{3_0}\tau_{1_0}' + [-4D_1\omega_{3_0}^3 + 2D_3\omega_{3_0}]\sin\omega_{3_0}\tau_{1_0}'$$
$$+[E_2 - \tau_2(-E_1\omega_{3_0}^2 + E_3)]\cos\omega_{3_0}(\tau_{1_0}' + \tau_2) + [2E_1\omega_{3_0} - \tau_2E_2\omega_{3_0}]\sin\omega_{3_0}(\tau_{1_0}' + \tau_2)$$
$$+[(-3F_1\omega_{3_0}^2 + F_3) - \tau_2(-F_2\omega_{3_0}^2 + F_4)]\cos\omega_{3_0}\tau_2 + 2[F_2\omega_{3_0} - \tau_2(-F_1\omega_{3_0}^3 + F_3\omega_{3_0})]\sin\omega_{3_0}\tau_2,$$
$$P_{22} = [-4A_1\omega_{3_0}^3 + 2A_3\omega_{3_0}] - \tau_2B_1\omega_{3_0}\cos\omega_{3_0}(2\tau_{1_0}' + \tau_2) - [B_1 - \tau_2B_2]\sin\omega_{3_0}(2\tau_{1_0}' + \tau_2) + 2C_2\cos 2\omega_{3_0}\tau_{1_0}'$$
$$+[3C_1\omega_{3_0}^2 - C_3]\sin 2\omega_{3_0}\tau_{1_0}' + [-4D_1\omega_{3_0}^3 + 2D_3\omega_{3_0}]\cos\omega_{3_0}\tau_{1_0}' + [3D_2\omega_{3_0}^2 - D_4]\sin\omega_{3_0}\tau_{1_0}'$$
$$+[2E_1\omega_{3_0} - \tau_2E_2\omega_{3_0}]cos\omega_{3_0}(\tau_{1_0}' + \tau_2) - [E_2 + \tau_2(E_1\omega_{3_0}^2 - E_3)]\sin\omega_{3_0}(\tau_{1_0}' + \tau_2)$$
$$+[2F_2\omega_{3_0} + \tau_2(F_1\omega_{3_0}^3 - F_3\omega_{3_0})]\cos\omega_{3_0}\tau_2 + [(3F_1\omega_{3_0}^2 - F_3) + \tau_2(-F_2\omega_{3_0}^2 + F_4)]\sin\omega_{3_0}\tau_2,$$
$$G_{21} = -2B_1\omega_{3_0}^2\cos\omega_{3_0}(2\tau_{1_0}' + \tau_2) + 2B_2\omega_{3_0}\sin\omega_{3_0}(2\tau_{1_0}' + \tau_2) + 2[C_1\omega_{3_0}^4 - C_3\omega_{3_0}^2]\cos 2\omega_{3_0}\tau_{1_0}'$$
$$-2\omega_{3_0}[C_2\omega_{3_0}^2 - C_4]\sin 2\omega_{3_0}\tau_{1_0}' + \omega_{3_0}^2[D_2\omega_{3_0}^2 - D_4]\cos\omega_{3_0}\tau_{1_0}' + \omega_{3_0}[D_1\omega_{3_0}^4 - D_3\omega_{3_0}^2 + D_5]\sin\omega_{3_0}\tau_{1_0}'$$

$$-E_2\omega_{3_0}^2\cos\omega_{3_0}(\tau^{'}_{1_0}+\tau_2)-\omega_{3_0}[E_1\omega_{3_0}^2-E_3]\sin\omega_{3_0}(\tau^{'}_{1_0}+\tau_2),$$

$$G_{22}=2B_2\omega_{3_0}\cos\omega_{3_0}(2\tau^{'}_{1_0}+\tau_2)+2B_1\omega_{3_0}^2\sin\omega_{3_0}(2\tau^{'}_{1_0}+\tau_2)-2\omega_{3_0}[C_2\omega_{3_0}^2-C_4]\cos 2\omega_{3_0}\tau^{'}_{1_0}$$
$$-2\omega_{3_0}^2[C_1\omega_{3_0}^2-C_3]\sin 2\omega_{3_0}\tau^{'}_{1_0}+\omega_{3_0}[D_1\omega_{3_0}^4-D_3\omega_{3_0}^2+D_5]\cos\omega_{3_0}\tau^{'}_{1_0}-\omega_{3_0}^2[D_2\omega_{3_0}^2-D_4]\sin\omega_{3_0}\tau^{'}_{1_0}$$
$$-\omega_{3_0}[E_1\omega_{3_0}^2-E_3]\cos\omega_{3_0}(\tau^{'}_{1_0}+\tau_2)+E_2\omega_{3_0}^2\sin\omega_{3_0}(\tau^{'}_{1_0}+\tau_2).$$

A.4 Terms in the transversality condition of Theorem 4.4b.

$$P_{31}=[5\omega_{4_0}^4-3A_2\omega_{4_0}^2+A_4]+[B_1-2\tau_1B_2]\cos\omega_{4_0}(2\tau_1+\tau^{'}_{2_0})-2\tau_1B_1\omega_{4_0}\sin\omega_{4_0}(2\tau_1+\tau^{'}_{2_0})$$
$$+[-3C_1\omega_{4_0}^2+C_3+2\tau_1(C_2\omega_{4_0}^2-C_4)]\cos 2\omega_{4_0}\tau_1+[2C_2\omega_{4_0}+2\tau_1(C_1\omega_{4_0}^3-C_3\omega_{4_0})]\sin 2\omega_{4_0}\tau_1$$
$$+[-3D_2\omega_{4_0}^2+D_4-\tau_1(D_1\omega_{4_0}^4-D_3\omega_{4_0}^2+D_5)]\cos\omega_{4_0}\tau_1+[-4D_1\omega_{4_0}^3+2D_3\omega_{4_0}+\tau_1(D_2\omega_{4_0}^3-D_4\omega_{4_0})]\sin\omega_{4_0}\tau_1$$
$$+[E_2+\tau_1(E_1\omega_{4_0}^2-E_3)]\cos\omega_{4_0}(\tau_1+\tau^{'}_{2_0})+[2E_1\omega_{4_0}-\tau_1E_2\omega_{4_0}]\sin\omega_{4_0}(\tau_1+\tau^{'}_{2_0})-[3F_1\omega_{4_0}^2-F_3]\cos\omega_{4_0}\tau^{'}_{2_0}$$
$$+2F_2\omega_{4_0}\sin\omega_{4_0}\tau^{'}_{2_0},$$

$$P_{32}=[-4A_1\omega_{4_0}^3+2A_3\omega_{4_0}]-2\tau_1B_1\omega_{4_0}\cos\omega_{4_0}(2\tau_1+\tau^{'}_{2_0})-[B_1-2\tau_1B_2]\sin\omega_{4_0}(2\tau_1+\tau^{'}_{2_0})$$
$$+[2C_2\omega_{4_0}+2\tau_1(C_1\omega_{4_0}^3-C_3\omega_{4_0})]\cos 2\omega_{4_0}\tau_1+[3C_1\omega_{4_0}^2-C_3-2\tau_1(C_2\omega_{4_0}^2-C_4)]\sin 2\omega_{4_0}\tau_1$$
$$+[-4D_1\omega_{4_0}^3+2D_3\omega_{4_0}+\tau_1(D_2\omega_{4_0}^3-D_4\omega_{4_0})]\cos\omega_{4_0}\tau_1+[3D_2\omega_{4_0}^2-D_4+\tau_1(D_1\omega_{4_0}^4-D_3\omega_{4_0}^2+D_5)]\sin\omega_{4_0}\tau_1$$
$$+[2E_1\omega_{4_0}-\tau_1E_2\omega_{4_0}]\cos\omega_{4_0}(\tau_1+\tau^{'}_{2_0})-[E_2+\tau_1(E_1\omega_{4_0}^2-E_3)]\sin\omega_{4_0}(\tau_1+\tau^{'}_{2_0})+2F_2\omega_{4_0}\cos\omega_{4_0}\tau^{'}_{2_0}$$
$$+[3F_1\omega_{4_0}^2-F_3]\sin\omega_{4_0}\tau^{'}_{2_0},$$

$$G_{31}=-B_1\omega_{4_0}^2\cos\omega_{4_0}(2\tau_1+\tau^{'}_{2_0})+B_2\omega_{4_0}\sin\omega_{4_0}(2\tau_1+\tau^{'}_{2_0})-E_2\omega_{4_0}^2\cos\omega_{4_0}(\tau_1+\tau^{'}_{2_0})$$
$$-[E_1\omega_{4_0}^3-E_3\omega_{4_0}]\sin\omega_{4_0}(\tau_1+\tau^{'}_{2_0})+[F_1\omega_{4_0}^4-F_3\omega_{4_0}^2]\cos\omega_{4_0}\tau^{'}_{2_0}-[F_2\omega_{4_0}^3-F_4\omega_{4_0}]\sin\omega_{4_0}\tau^{'}_{2_0},$$

$$G_{32}=B_2\omega_{4_0}\cos\omega_{4_0}(2\tau_1+\tau^{'}_{2_0})+B_1\omega_{4_0}^2\sin\omega_{4_0}(2\tau_1+\tau^{'}_{2_0})-[E_1\omega_{4_0}^3-E_3\omega_{4_0}]\cos\omega_{4_0}(\tau_1+\tau^{'}_{2_0})$$
$$+E_2\omega_{4_0}^2\sin\omega_{4_0}(\tau_1+\tau^{'}_{2_0})-[F_2\omega_{4_0}^3-F_4\omega_{4_0}]\cos\omega_{4_0}\tau^{'}_{2_0}-[F_1\omega_{4_0}^4-F_3\omega_{4_0}^2]\sin\omega_{4_0}\tau^{'}_{2_0}.$$

Appendix B

B.1 Numerical simulations to find the equilibria.

In this appendix we obtain the equilibrium point of the equation (4.6) using the equations (4.7) and (4.8).

First we fix parameters of the system (4.7) to be the same as in Figure 1 and vary P^* in the entire range within 0 and 1 to find (Y^*, X_+^*, N^*, M^*) for each value of P^*. Now we use equation (4.8) which is a transcendental equation in P^* to draw Figure 8. Let us consider the right hand side of the equation (4.8) as $F_2(P^*, \tau_1) = e^{-\left[d\tau_1+\lambda_0 P^*\tau_1+\frac{\beta Y^*\tau_1}{1+\beta_1 M^*}\right]}$. We fix τ_1 and plot $F_2(P^*, \tau_1)$ for P^* lying between 0 and 1. In Figure 8, we have taken some values of τ_1 and plot F_2, here blue, red, black and green solid curves correspond to the value of F_2 at τ_1 equal to $25, 50, 100$ and 150 respectively. Lastly we plot the left hand side of the equation (4.8), i.e. $F_1(P^*) = P^*$ (the dashed blue line). The intersection between F_1 and F_2 is the equilibrium value of P^* for different values of τ_1.

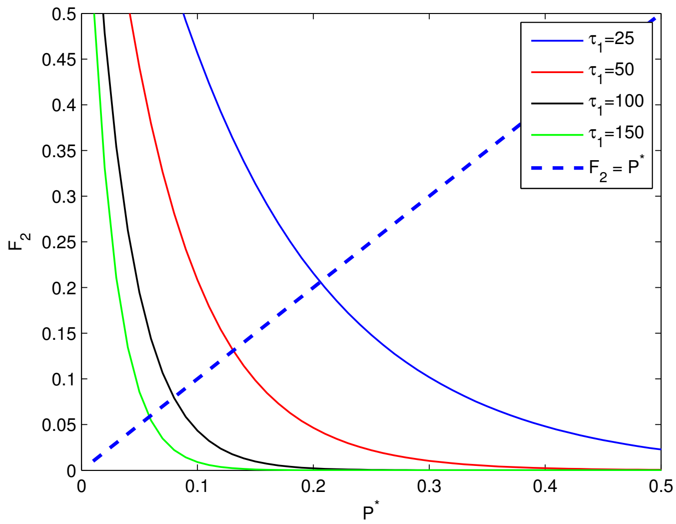

Figure 8: Graphical representation of equation (4.8) to find P^* for different τ_1.