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The effect of incidence functions on the dynamics of a quarantine/isolation model with time delay

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ABSTRACT

The problem of the asymptotic dynamics of a guarantine/isolation model with time delay is considered, subject to two incidence functions, namely standard incidence and the Holling type II (saturated) incidence function. Rigorous qualitative analysis of the model shows that it exhibits essentially the same (equilibrium) dynamics regardless of which of the two incidence functions is used. In particular, for each of the two incidence functions, the model has a globally asymptotically stable disease-free equilibrium whenever the associated reproduction threshold quantity is less than unity. Further, it has a unique endemic equilibrium when the threshold quantity exceeds unity. For the case with the Holling type II incidence function, it is shown that the unique endemic equilibrium of the model is globally asymptotically stable for a special case. The permanence of the disease is also established for the model with the Holling type II incidence function. Furthermore, it is shown that adding time delay to and/or replacing the standard incidence function with the Holling type II incidence function in the corresponding autonomous quarantine/isolation model with standard incidence (considered in Safi and Gumel (2010) [10]) does not alter the qualitative dynamics of the autonomous system (with respect to the elimination or persistence of the disease). Finally, numerical simulations of the model with standard incidence show that the disease burden decreases with increasing time delay (incubation period). Furthermore, models with time delay seem to be more suitable for modeling the 2003 SARS outbreaks than those without time delay.

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1. Introduction

The aim of this study is to assess the roles of time delay (to model the incubation period of a disease) and the choice of incidence function in the transmission dynamics of a communicable disease in the presence of quarantine (of exposed individuals) and isolation (of individuals with disease symptoms). Quarantine and isolation measures have been widely used, over the decades, to control the spread of diseases such as yellow fever, smallpox, measles, ebola, pandemic influenza, diphtheria, plague, cholera, and, more recently, severe acute respiratory syndrome (SARS) [1–9]. To achieve the main objective of this study, the autonomous quarantine/isolation presented in [10] will be extended to incorporate time delay and two different incidence functions. The functional forms of the incidence functions to be considered are derived on the basis of the framework described below.

Let S(t), I(t) and N(t) denote the number of susceptible individuals, the number of infectious individuals and the total size of the population at time t, respectively. Further, let $\beta(N)$ be the average number of contacts sufficient for transmitting infection (effective contact rate). Then, the force of infection, given by $\beta(N)I/N$, represents the average number of contacts that a susceptible individual makes with infectious individuals *per* unit time. If $\beta(N) = \beta N$ (i.e., the contact rate depends

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on the total population, N), then the incidence function $g_1(I) = \beta I$ is called *mass action incidence*. If $\beta(N) = \beta$ (a constant), then the incidence function $g_2(I) = \beta I/N$ is called *standard incidence* [11,12]. These two functions are widely used in the modeling the transmission dynamics of the human diseases [13,14]. Another widely used incidence function is the Holling type II incidence function, given by $g_3(I) = \frac{\beta I}{1+\omega I}$, with $\omega > 0$, [15–18]. The non-linear incidence function of type $g_3(I)$ was first introduced by Capasso and Serio [15], in their study of the cholera epidemic in Bari, Italy. The main justification for using such a functional form of the incidence function stems from the fact that the number of effective contacts between infective individuals and susceptible individuals may saturate at high infective levels due to crowding of infective individuals, or due to the preventive measures taken by (and behavioral changes of) the susceptible individuals in response to the severity of the disease [16-18].

The paper is organized as follows. The model with standard incidence is formulated in Section 2. The existence and global asymptotic stability of its disease-free equilibrium (DFE), as well as the existence of its endemic equilibrium point (EEP), are established in Section 3. The model with the Holling type II incidence function is formulated and analyzed in Section 4. The permanence of the disease is also established for this model.

2. Model formulation: standard incidence

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The model to be considered in this study is that for the transmission dynamics of an infectious disease, in the presence of quarantine of exposed individuals and isolation of infected individuals with disease symptoms, and is given by the following delayed system of integro-differential equations:

$$\frac{dS}{dt} = \Pi - \frac{\beta S(t)I(t)}{N(t)} - \mu S(t),
E = \int_{t-\tau}^{t} \frac{\beta S(x)I(x)e^{-(\mu+\sigma)(t-x)}}{N(x)} dx,
\frac{dI}{dt} = \frac{e^{-\tau(\mu+\sigma)}\beta S(t-\tau)I(t-\tau)}{N(t-\tau)} - (\gamma_{1} + \phi + \mu + \delta_{1})I(t),
\frac{dQ}{dt} = \sigma E(t) - (\alpha + \mu)Q(t),
\frac{dH}{dt} = \alpha Q(t) + \phi I(t) - (\gamma_{2} + \mu + \delta_{2})H(t),
\frac{dR}{dt} = \gamma_{1}I(t) + \gamma_{2}H(t) - \mu R(t),$$
(1)

where S, E, I, Q, H, R denote the populations of susceptible, exposed, infectious, guarantined, hospitalized and recovered individuals at time t, respectively.

Thus, the total human population at time t, denoted by N(t), is given by

$$N(t) = S(t) + E(t) + I(t) + Q(t) + H(t) + R(t).$$

The initial data for the model (1) is given by

$$S(\theta) = \phi_1(\theta), \quad E(\theta) = \phi_2(\theta), \quad I(\theta) = \phi_3(\theta), \\ Q(\theta) = \phi_4(\theta), \quad H(\theta) = \phi_5(\theta), \quad R(\theta) = \phi_6(\theta), \quad \theta \in [-\tau, 0],$$
(2)

where $\phi = [\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6] \in \mathcal{C}$ such that $\phi_i(\theta) = \phi_i(0) \ge 0$ for $(\theta \in [-\tau, 0], i = 1, 3, 4, 5, 6), \phi_2(\theta) \ge 0$ $(\theta \in [-\tau, 0])$, and \mathcal{C} denotes the Banach space $\mathcal{C}([-\tau, 0], \mathbb{R}^6)$ of continuous functions mapping the interval $[-\tau, 0]$ into \mathbb{R}^6 , equipped with the uniform norm defined by $\|\phi\| = \sup_{\theta \in [-\tau, 0]} |\phi(\theta)|$. Furthermore, it is assumed that $\phi_i(0) > 0$ (for i = 1, ..., 6).

In (1), the parameter Π represents the rate of recruitment into the population, and β is the effective contact rate. The delay parameter $\tau > 0$ represents the associated incubation period [19] (see Table 1 for a list of some communicable diseases and their respective incubation periods). Exposed individuals are quarantined at a rate α . Quarantined and infectious individuals are hospitalized at the rates α and ϕ , respectively. The parameters γ_1 and γ_2 represent the recovery rates of infectious and hospitalized individuals, respectively, while μ is the natural death rate. Finally, δ_1 and δ_2 are the diseaseinduced death rates for infectious and hospitalized individuals, respectively. A flow diagram of the model (1) is given in Fig. 1, and the associated variables and parameters are described and estimated in Tables 2 and 3. It should be stated that the parameter values in Table 3 are relevant to the transmission dynamics of SARS [1,20–22].

The delayed model (1) is an extension of the autonomous quarantine/isolation model presented in [10] by incorporating a time delay ($\tau > 0$), but with the assumption of loss of infection-acquired immunity relaxed (i.e., that recovered individuals do not become susceptible again) and the assumption that hospitalized individuals do not transmit infection. One of the main aims of this study is to determine whether or not incorporating time delay (for the incubation period) alters the qualitative dynamics of the autonomous quarantine/isolation model considered in [10]. Another major objective is to

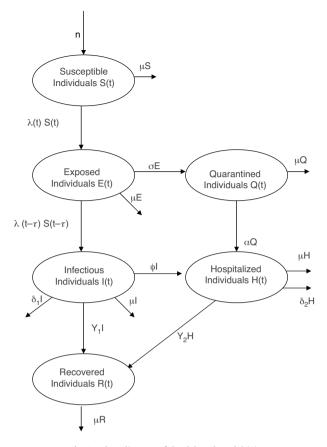


Fig. 1. Flow diagram of the delayed model (3).

determine whether replacing the standard incidence function in the model (1) with the Holling type II incidence function $(g_3(I) = \frac{I}{1+\omega I})$ will introduce new (or different) dynamical features for the delayed model (1).

2.1. Basic properties

Using the generalized Leibnitz rule of differentiation [25], the model (1) can be rewritten as a system of delayed differential difference equation given by

$$\frac{dS}{dt} = \Pi - \frac{\beta S(t)I(t)}{N(t)} - \mu S(t),
\frac{dE}{dt} = \frac{\beta S(t)I(t)}{N(t)} - \frac{e^{-\tau(\mu+\sigma)}\beta S(t-\tau)I(t-\tau)}{N(t-\tau)} - (\sigma+\mu)E,
\frac{dI}{dt} = \frac{e^{-\tau(\mu+\sigma)}\beta S(t-\tau)I(t-\tau)}{N(t-\tau)} - (\gamma_1 + \phi + \mu + \delta_1)I(t),
\frac{dQ}{dt} = \sigma E(t) - (\alpha + \mu)Q(t),
\frac{dH}{dt} = \alpha Q(t) + \phi I(t) - (\gamma_2 + \mu + \delta_2)H(t),
\frac{dR}{dt} = \gamma_1 I(t) + \gamma_2 H(t) - \mu R(t).$$
(3)

The basic qualitative properties of the model (3) will now be investigated.

Lemma 1. The solution (S(t), E(t), I(t), Q(t), H(t), R(t)) of the system (3), with the initial data (2), exists for all $t \ge 0$ and is unique. Furthermore, S(t) > 0, E(t) > 0, I(t) > 0, Q(t) > 0, H(t) > 0, and R(t) > 0 for all $t \ge 0$.

Table 1

Incubation period for some of communicable diseases [14,23].

Disease	Incubation period (days)
Chicken pox	14-16
Ebola	2–21
Influenza	1–3
Measles	9–12
SARS	Up to 10
Smallpox	7–17

Table 2	2
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Description of variables and parameters of the model (3).	on of variables and parameters of the model (3).
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Variable	Description
	1
S(t)	Population of susceptible individuals
E(t)	Population of exposed individuals
I(t)	Population of infectious individuals
Q(t)	Population of quarantined individuals
H(t)	Population of hospitalized individuals
R(t)	Population of recovered individuals
Parameter	Description
П	Recruitment rate into the community
μ	Natural death rate
β	Effective contact rate
τ	Incubation period
ω	Parameter for measuring psychological or inhibitory effect
σ	Quarantine rate for exposed individuals
α	Hospitalization rate for guarantined individuals
ϕ	Hospitalization rate for infectious individuals
$\dot{\psi}$	Rate of loss of infection-acquired immunity
Υ ₁	Recovery rate for infectious individuals
γ_2	Recovery rate for hospitalized individuals
δ_1	Disease-induced death rate for infectious individuals
δ_2	Disease-induced death rate for hospitalized individuals

Estimated values of the parameters of the model (3).				
Parameter	Value (per day)	Source		
П	136	[21]		
β	(0, 0.5)	[21]		
μ	0.0000351	[21]		
γ_1	0.03521	[24]		
Υ2	0.042553	[24]		
δ_1	0.04227	[22]		
δ_2	0.027855	[24]		
κ	0.156986	[20]		
α	0.156986	[20]		
ϕ	0.20619	[24]		
σ	0.1	[21]		
ω	0.1	Assumed		

Proof. System (3) can be written as (where a dot represents differentiation with respect to t)

Table 3

$$\dot{X} = f(t, X_{\tau}),$$

where $X = (S(t), E(t), I(t), Q(t), H(t), R(t)) \in C$. Since f(t, X) is continuous and Lipschitz in X, it follows, by the Fundamental Theory of Functional Differential Equations [26], that the system (3) has a unique solution (S(t), E(t), I(t), Q(t), H(t), R(t)) satisfying the initial data (2).

It is clear from the first equation of the model (3) that

$$\frac{\mathrm{d}S}{\mathrm{d}t} \geq -\left[\frac{\beta S(t)I(t)}{N(t)} + \mu\right]S(t),$$

and so

$$S(t) \ge S(0) \exp\left\{-\int_0^t \left[\frac{\beta S(u)I(u)}{N(u)} + \mu\right] du\right\} > 0, \quad \text{for all } t > 0.$$

Similarly, it follows from the third equation of the system (3) that I(t) > 0 for all t > 0. Since the second equation of (3) is equivalent to the second equation of (1), it follows (by using the fact that S(t) > 0 and I(t) > 0 for all t > 0, together with the fact that all the parameters of the model are positive) that

$$E(t) = \int_{t-\tau}^{t} \frac{\beta S(x)I(x)e^{-(\mu+\sigma)(t-x)}}{N(x)} dx > 0.$$

Furthermore, using the same approach as for S(t) above, it can be shown that Q(t) > 0, H(t) > 0 and R(t) > 0 for all t > 0. \Box

Lemma 2. The closed set

$$\mathcal{D} = \left\{ (S, E, I, Q, H, R) \in \mathbb{R}^6_+ : S + E + I + Q + H + R \le \frac{\Pi}{\mu} \right\}$$

is positively invariant.

Proof. Adding all the equations of the model (3) gives

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \Pi - \mu N - (\delta_1 I + \delta_2 H). \tag{4}$$

Since $dN/dt \le \Pi - \mu N$, it follows that $dN/dt \le 0$ if $N \ge \Pi/\mu$. Thus,

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Pi}{\mu} (1 - e^{-\mu t}).$$

In particular, $N(t) \leq \Pi/\mu$ if $N(0) \leq \Pi/\mu$. Hence, the region \mathcal{D} is positively invariant. Further, if $N(0) > \Pi/\mu$, then either the solution enters \mathcal{D} in finite time, or N(t) approaches Π/μ asymptotically. Hence, the region \mathcal{D} attracts all solutions in \mathbb{R}^6_+ . \Box

3. Global stability of the DFE

.

The DFE of the system (3), obtained by setting the derivatives in the model (3) to zero, is given by

$$\mathcal{E}_{0} = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0, 0\right).$$
(5)

The global asymptotic stability property of \mathcal{E}_0 will be explored using the methodology given in [25,27]. It is convenient to define

$$\mathcal{R}_0^{\mathsf{S}}(\tau) = \mathcal{R}_0^{\mathsf{S}} = \frac{\beta \mathsf{e}^{-\tau(\mu+\sigma)}}{\gamma_1 + \phi + \mu + \delta_1}.$$

The quantity \mathcal{R}_0^S is known as the *basic reproduction number* of the delayed model (3). It measures the average number of new infections generated by a single infectious individual in a completely susceptible population. It is worth noting that $\mathcal{R}_0^S(\tau)$ is a decreasing function of τ (and $\mathcal{R}_0^S(0) > \mathcal{R}_0^S(\tau)$ for all $\tau > 0$).

Theorem 1. The DFE of the model (3), given by (5), is GAS in \mathcal{D} whenever $\mathcal{R}_0^S < 1$.

Proof. Let $\mathcal{R}_0^S < 1$. Furthermore, let (S(t), E(t), I(t), Q(t), H(t), R(t)) be any positive solution of the system (3) with the initial data (2). The third equation of the system (3) can be rewritten as

$$I(t) = \int_{-\infty}^{t} \frac{\beta e^{-\tau(\sigma+\mu)} S(x-\tau) I(x-\tau)}{N(x-\tau)} e^{-(\gamma_1+\phi+\mu+\delta_1)(t-x)} dx,$$

$$\leq \int_{-\infty}^{t} \beta e^{-\tau(\sigma+\mu)} I(x-\tau) e^{-(\gamma_1+\phi+\mu+\delta_1)(t-x)} dx, \quad \text{since } S(t) \leq N(t) \text{ in } \mathcal{D}.$$
(6)

It follows, by using the substitution s = t - x in (6), that

$$I(t) \leq \int_0^\infty \beta e^{-\tau(\sigma+\mu)} I(t-s-\tau) e^{-(\gamma_1+\phi+\mu+\delta_1)(s)} \mathrm{d}s.$$
(7)

Taking the lim sup of both sides of (7), and noting that $\limsup \int f \leq \int \limsup f$ [25], gives

$$\lim_{t \to \infty} \sup I(t) \leq \int_0^\infty \beta e^{-\tau(\sigma+\mu)} e^{-(\gamma_1 + \phi + \mu + \delta_1)(s)} ds \limsup_{t \to \infty} I(t),$$
$$= \frac{\beta e^{-\tau(\mu+\sigma)}}{\gamma_1 + \phi + \mu + \delta_1} \limsup_{t \to \infty} I(t) = \mathcal{R}_0^{\mathsf{S}} \limsup_{t \to \infty} I(t). \tag{8}$$

Since $\mathcal{R}_0^S < 1$, it follows that $\limsup_{t\to\infty} I(t) < \limsup_{t\to\infty} I(t)$. This is a contradiction, unless $\limsup_{t\to\infty} I(t) = 0$. Thus, for any $\epsilon > 0$ sufficiently small, there exists a T > 0 such that if t > T, then $I(t) < \epsilon$.

Using $S(t)/N(t) \le 1$ and $I(t) < \epsilon$, for t > T, in the second equation of (3) gives

$$\dot{E} \leq \beta \epsilon - (\sigma + \mu) E$$

Furthermore, by the comparison theorem [28],

$$\limsup_{t\to+\infty} E(t) \leq \frac{\beta\epsilon}{\sigma+\mu}.$$

Since ϵ is arbitrary, it follows (by setting $\epsilon \rightarrow 0$) that

 $\limsup_{t\to+\infty} E(t) = 0.$

Hence, for $\epsilon_1 > 0$ small, there exists a $T_1 > T$ such that if $t > T_1$, then $E(t) < \epsilon_1$. Using $E(t) < \epsilon_1$, for $t > T_1$, in the fourth equation of (3) gives

$$\dot{Q} \leq \epsilon_1 \sigma - (\alpha + \mu) Q,$$

and so

1

$$\limsup_{t \to +\infty} Q(t) \le \frac{\epsilon_1 \sigma}{\alpha + \mu}$$

Hence,

 $\limsup_{t\to+\infty} Q(t) = 0.$

In a similar way, it can be shown that

 $\limsup_{t \to +\infty} H(t) = 0 \text{ and } \limsup_{t \to +\infty} R(t) = 0.$

Finally, it follows from the first equation of (3), for t > T, that

$$\dot{S} \ge \Pi - \beta \epsilon - \mu S$$
,

and so

$$\liminf_{t \to +\infty} S(t) \ge \frac{\Pi - \beta \epsilon}{\mu}$$

Hence, by letting $\epsilon \rightarrow 0$ in (9),

 $\liminf_{t\to+\infty} S(t) \geq \frac{\Pi}{\mu}.$

Additionally, since $\limsup_{t \to +\infty} S(t) \leq \frac{\Pi}{\mu}$, it follows that

$$\lim_{t\to+\infty}S(t)=\frac{\Pi}{\mu}.$$

Thus,

$$\lim_{t \to +\infty} (S(t), E(t), I(t), Q(t), H(t), R(t)) = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0, 0\right) = \mathcal{E}_0. \quad \Box$$

This result (Theorem 1) is consistent with that given for the non-delayed quarantine/isolation model in [10] (for the case where recovered individuals do not lose their infection-acquired immunity and hospitalized individuals do not transmit infection for the DFE of the model considered in [10]). That is, this result shows that adding time delay to the non-delayed (autonomous) quarantine/isolation model in [10] does not alter the global asymptomatic stability property of the DFE (\mathcal{E}_0) of

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(9)

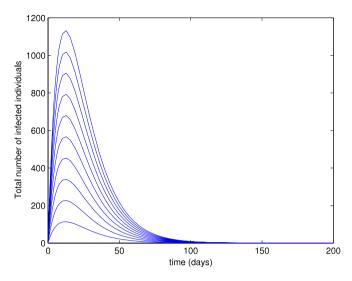


Fig. 2. Simulations of the delayed model (3), showing the total number of infected individuals as a function of time. Parameter values used are as given in Table 3, with $\tau = 20$ and $\beta = 0.15$ (so $\Re_0^S = 0.7150 < 1$ and $\tau > \tau_c^S = 16.6470$).

the corresponding non-delayed model given in [10]. The epidemiological implication of Theorem 1 is that the combined use of quarantine and isolation can lead to disease elimination from the community if the two interventions can bring (and keep) the threshold quantity, \mathcal{R}_0^S , to a value less than unity (i.e., for the delayed model (3), the condition $\mathcal{R}_0^S < 1$ is necessary and sufficient for disease elimination).

By solving for the delay parameter (τ) from the equation $\Re_0^S = 1$ (and noting Theorem 1), the following result can be obtained.

Lemma 3. The DFE of the model (3), given by (5), is GAS in \mathcal{D} whenever

(.)

$$\tau > \ln\left(\frac{\beta}{\mu + \phi + \gamma_1 + \delta_1}\right)^{\left(\frac{1}{\sigma + \mu}\right)} = \tau_c^{S}.$$

In other words, Lemma 3 shows that the disease will be eliminated from the community if and only if $\tau > \tau_c^S$. Furthermore, it follows from Lemma 3 that the longer infected individuals remain in the exposed class (*E*), the higher the likelihood of disease elimination from the community. Fig. 2 depicts the numerical results obtained by simulating the model (3) using the parameter values in Table 3, and various initial conditions, for the case when $\tau > \tau_c^S$ ($\mathcal{R}_0^S < 1$). It is evident from this figure that all solutions converged to the DFE, \mathcal{E}_0 (in line with Theorem 1 and Lemma 3).

3.1. The existence of an EEP

In this section, the possible existence and stability of endemic (positive) equilibria of the model (3) will be explored. Let $\mathcal{E}_1^S = (S^{**}, E^{**}, I^{**}, Q^{**}, H^{**}, R^{**})$ represent any arbitrary endemic equilibrium point of the model (3), so $N^{**} = S^{**} + E^{**} + I^{**} + Q^{**} + H^{**} + R^{**}$. Solving the equations of the model (3) at the steady state gives

$$S^{**} = \frac{\Pi}{\lambda^{**} + \mu}, \qquad E^{**} = \frac{\lambda^{**}S^{**}(1 - e^{-\tau(\sigma + \mu)})}{\sigma + \mu}, \qquad I^{**} = \frac{e^{-\tau(\sigma + \mu)}\lambda^{**}S^{**}}{\gamma_1 + \phi + \mu + \delta_1},$$

$$Q^{**} = \frac{\sigma E^{**}}{\alpha + \mu}, \qquad H^{**} = \frac{\alpha Q^{**} + \phi I^{**}}{\gamma_2 + \mu + \delta_2}, \qquad R^{**} = \frac{\gamma_1 I^{**} + \gamma_2 H^{**}}{\mu},$$
(10)

where

$$\lambda^{**} = \frac{\beta I^{**}}{N^{**}}.$$
(11)

For computational convenience, the expressions in (10) are rewritten in terms of $\lambda^{**}S^{**}$ as below:

$$E^{**} = \frac{\lambda^{**}S^{**}(1 - e^{-\tau(\sigma+\mu)})}{\sigma + \mu}, \qquad I^{**} = \frac{e^{-\tau(\sigma+\mu)}\lambda^{**}S^{**}}{\gamma_1 + \phi + \mu + \delta_1},$$

$$Q^{**} = P_1\lambda^{**}S^{**}, \qquad H^{**} = P_2\lambda^{**}S^{**}, \qquad R^{**} = P_3\lambda^{**}S^{**},$$
(12)

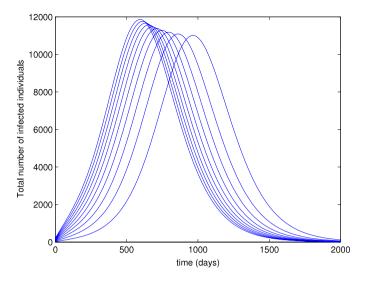


Fig. 3. Simulations of the delayed model (3), showing the total number of infected individuals as a function of time. Parameter values used are as given in Table 3, with $\tau = 18$ and $\beta = 0.1$ (so $\Re_0^5 = 1.0298 > 1$ and $\tau < \tau_c^S = 20.9263$).

where

$$P_{1} = \frac{\sigma(1 - e^{-\tau(\sigma + \mu)})}{(\sigma + \mu)(\alpha + \mu)}, \qquad P_{2} = \frac{\alpha P_{1}}{\gamma_{2} + \mu + \delta_{2}} + \frac{\phi e^{-\tau(\sigma + \mu)}}{(\gamma_{2} + \mu + \delta_{2})(\gamma_{1} + \phi + \mu + \delta_{1})}$$
$$P_{3} = \frac{\gamma_{1} e^{-\tau(\sigma + \mu)}}{\mu(\gamma_{1} + \phi + \mu + \delta_{1})} + \frac{\gamma_{2} P_{2}}{\mu}.$$

Substituting the expressions in (12) into (11) gives

$$\lambda^{**}S^{**} + \frac{\lambda^{**}S^{**}(1 - e^{-\tau(\sigma+\mu)})\lambda^{**}}{\sigma + \mu} + \frac{\lambda^{**}e^{-\tau(\sigma+\mu)}\lambda^{**}S^{**}}{\gamma_1 + \phi + \mu + \delta_1} + \lambda^{**}P_1\lambda^{**}S^{**} + \lambda^{**}P_2\lambda^{**}S^{**} + \lambda^{**}P_3\lambda^{**}S^{**} = \frac{\beta e^{-\tau(\sigma+\mu)}\lambda^{**}S^{**}}{\gamma_1 + \phi + \mu + \delta_1}.$$
(13)

Dividing each term in (13) by $\lambda^{**}S^{**}$ (and noting that, at the endemic steady state, $\lambda^{**}S^{**} \neq 0$) gives

$$1 + P_4 \lambda^{**} = \frac{\beta e^{-\tau(\sigma+\mu)}}{\gamma_1 + \phi + \mu + \delta_1} = \mathcal{R}_0^S.$$
(14)

Since

$$P_4 = \frac{1 - e^{-\tau(\sigma + \mu)}}{\sigma + \mu} + \frac{e^{-\tau(\sigma + \mu)}}{\gamma_1 + \phi + \mu + \delta_1} + P_1 + P_2 + P_3 \ge 0,$$

it follows from (14) that

$$\lambda^{**} = \frac{\mathcal{R}_0^S - 1}{P_4} > 0, \text{ whenever } \mathcal{R}_0^S > 1.$$
 (15)

The components of the endemic equilibrium, \mathcal{E}_1^S , can then be obtained by substituting the unique value of λ^{**} , given in (15), into the expressions in (10). Thus, the following result is established.

Lemma 4. The model (3) has a unique endemic (positive) equilibrium, given by \mathscr{E}_1^S , whenever $\mathscr{R}_0^S > 1$.

Although not proven here, numerical simulations of the model (3) suggest that the EEP (\mathcal{E}_1^S) of the model (3) is asymptotically stable for $\mathcal{R}_0^S > 1$ (Fig. 3). It should be mentioned, however, that the solutions depicted in Fig. 3 did not converge to zero, as they appear to (see Fig. 4 for a blow up of the tail end of Fig. 3). In other words, Figs. 3 and 4 show convergence of the solutions to the unique EEP, \mathcal{E}_1^S , of the model (3) for the case $\mathcal{R}_0^S > 1$. The following conjecture is suggested:

Conjecture 1. The unique EEP, \mathcal{E}_1^S , of the model (3) is LAS whenever $\mathcal{R}_0^S > 1$.

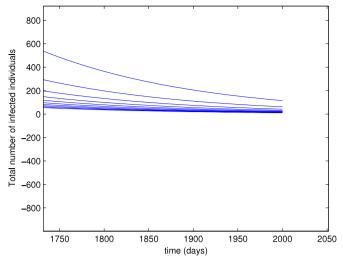


Fig. 4. Blow up of the tail end of Fig. 3.

In summary, the model (3) has a globally asymptotic stable disease-free equilibrium whenever $\Re_0^S < 1$, and it has a unique endemic equilibrium whenever $\Re_0^S > 1$. These results are consistent with those reported for the corresponding autonomous (non-delayed) quarantine/isolation model in [10]. In other words, adding time delay to the non-delayed quarantine/isolation model in [10] does not alter its qualitative (equilibrium) dynamics. The next task is to determine whether or not the dynamics of the non-delayed quarantine/isolation model in [10] is affected by the combined use of time delay and the substitution of the standard incidence function with the Holling type II incidence function. This is considered below.

4. The model with Holling type II incidence

In this section, the delayed model (3) will be analyzed subject to the use of the Holling type II incidence function, given by $g_3(I) = \frac{I}{1+\omega I}$ (with $\omega > 0$), in place of the standard incidence function. The delayed model (3), with the standard incidence function replaced by $g_3(I)$, is given by

$$\frac{dS}{dt} = \Pi - \frac{\beta S(t)I(t)}{1 + \omega I(t)} - \mu S(t),$$

$$\frac{dE}{dt} = \frac{\beta S(t)I(t)}{1 + \omega I(t)} - \frac{e^{-\tau(\mu+\sigma)}\beta S(t-\tau)I(t-\tau)}{1 + \omega I(t-\tau)} - (\sigma+\mu)E,$$

$$\frac{dI}{dt} = \frac{\beta e^{-\tau(\mu+\sigma)}S(t-\tau)I(t-\tau)}{1 + \omega I(t-\tau)} - (\gamma_1 + \phi + \mu + \delta_1)I(t),$$

$$\frac{dQ}{dt} = \sigma E(t) - (\alpha + \mu)Q(t),$$

$$\frac{dH}{dt} = \alpha Q(t) + \phi I(t) - (\gamma_2 + \mu + \delta_2)H(t),$$

$$\frac{dR}{dt} = \gamma_1 I(t) + \gamma_2 H(t) - \mu R.$$
(16)

4.1. Global stability of the DFE

The delayed system (16) has the same DFE, \mathcal{E}_0 , as the system (3). Further, the invariant region, \mathcal{D} , holds for system (16) as well. The GAS property of the DFE of the system (16) will be explored using the methodology given in [29]. Define

$$\mathcal{R}_0^H = \frac{\beta \Pi e^{-\tau(\mu+\sigma)}}{\mu(\gamma_1 + \phi + \mu + \delta_1)}.$$

The proof is based on using the following result.

Lemma 5 ([29]). Consider the following delay differential equation:

$$\dot{u} = \frac{au(t-\tau)}{1+\omega u(t-\tau)} - bu(t), \qquad u(\theta) = \phi(\theta) \ge 0, \quad \theta \in [-\tau, 0), \ \phi(0) > 0$$
(17)

where a, b and ω are positive constants, $\tau > 0$; then:

``

(i) Eq. (17) has a trivial equilibrium u = 0 and it is globally asymptotically stable if a < b. (ii) If a > b, Eq. (17) has a unique positive equilibrium $u^* = \frac{a-b}{\omega b}$ which is globally asymptotically stable.

Theorem 2. The DFE of the model (16), given by (5), is GAS in \mathcal{D} whenever $\mathcal{R}_0^H < 1$.

Proof. Let $\mathcal{R}_0^H < 1$. Furthermore, let (S(t), E(t), I(t), Q(t), H(t), R(t)) be any positive solution of the system (16) with the initial data (2). Since $\mathcal{R}_0^H < 1$, it is clear that

$$\beta e^{-\tau(\mu+\sigma)} \Pi/\mu < \gamma_1 + \phi + \mu + \delta_1.$$
⁽¹⁸⁾

Since $S(t) \leq \Pi/\mu$ in \mathcal{D} for all t > 0, it follows from the second equation of (16) that

$$\dot{I} \le \frac{\beta \Pi e^{-\tau(\mu+\sigma)} I(t-\tau)}{\mu [1+\omega I(t-\tau)]} - (\gamma_1 + \phi + \mu + \delta_1) I(t).$$
(19)

Consider, next, the auxiliary (with equality) equation associated with the inequality (19) (where u is a dummy variable)

$$\dot{u} = \frac{\beta \Pi e^{-\tau(\mu+\sigma)} u(t-\tau)}{\mu [1+\omega u(t-\tau)]} - (\gamma_1 + \phi + \mu + \delta_1) u(t).$$
(20)

Using Item (i) of Lemma 5, together with Eq. (18), in (20) gives

 $\lim_{t \to +\infty} u(t) = 0.$

Thus, it follows from (19), using the comparison theorem [28], that

 $\limsup I(t) = 0.$ $t \rightarrow +\infty$

Thus, for any $\epsilon > 0$ sufficiently small, there exists a T > 0 such that if t > T, then $I(t) < \epsilon$. Using $S \le \Pi/\mu$ in \mathcal{D} and $I < \epsilon$, for t > T, in the second equation of (16) (note that g(I) is monotone increasing) gives

$$\dot{E} \leq \frac{\beta \Pi \epsilon}{\mu (1 + \omega \epsilon)} - (\sigma + \mu) E.$$

Furthermore, by the comparison theorem,

$$\limsup_{t \to +\infty} E(t) \le \frac{\beta \Pi \epsilon}{\mu(\sigma + \mu)(1 + \omega \epsilon)}.$$

Since ϵ is arbitrary, it follows (by setting $\epsilon \rightarrow 0$) that

$$\limsup_{t\to+\infty} E(t)=0.$$

Hence, for $\epsilon_1 > 0$ small, there exists a $T_1 > T$ such that if $t > T_1$, then $E(t) < \epsilon_1$. Using $E(t) < \epsilon_1$, for $t > T_1$, in the fourth equation of (16) gives

$$\dot{Q} \leq \epsilon_1 \sigma - (\alpha + \mu) Q,$$

and so

 $\limsup_{t\to+\infty} Q(t) \leq \frac{\epsilon_1 \sigma}{\alpha+\mu}.$

Hence.

 $\limsup Q(t) = 0.$ $t \rightarrow +\infty$

In a similar way, it can be shown that

 $\limsup H(t) = 0$ and $\limsup R(t) = 0$. $t \rightarrow +\infty$ $t \rightarrow +\infty$

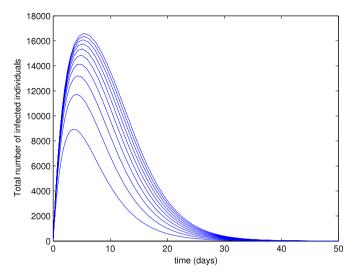


Fig. 5. Simulations of the delayed model (16), showing the total number of infected individuals as a function of time. Parameter values used are as given in Table 3, with $\tau = 20$ and $\beta = 0.0025809$ (so $\mathcal{R}_0^H = 0.1599 < 1$ and $\tau > \tau_c^H = 16.3340$).

Finally, it follows from the first equation of (16), for t > T, that

$$\dot{S} \ge \Pi - \frac{\beta S \epsilon}{1 + \omega \epsilon} - \mu S,$$

so, using the comparison theorem,

$$\liminf_{t \to +\infty} S(t) \ge \frac{\Pi(1 + \omega\epsilon)}{\mu + \epsilon(\beta + \omega\mu)}$$

Hence (by letting $\epsilon \rightarrow 0$)

$$\liminf_{t\to+\infty} S(t) \ge \frac{11}{\mu}.$$

Additionally, since $\limsup_{t\to+\infty} S(t) \leq \frac{\pi}{\mu}$ in \mathcal{D} it follows that

$$\lim_{t\to+\infty} S(t) = \frac{\Pi}{\mu}.$$

Thus,

$$\lim_{t \to +\infty} (S(t), E(t), I(t), Q(t), H(t), R(t)) = \left(\frac{\Pi}{\mu}, 0, 0, 0, 0, 0\right) = \mathcal{E}_0. \quad \Box$$

The epidemiological implication of the above result (Theorem 2) is that the combined use of quarantine and isolation can lead to disease elimination if the two interventions can bring (and keep) the threshold quantity, \mathcal{R}_0^H , to a value less than unity (i.e., the condition $\mathcal{R}_0^H < 1$ is necessary and sufficient for disease elimination). By solving for τ from the equation $\mathcal{R}_0^H = 1$ (and noting Theorem 2), the following result can be obtained.

Lemma 6. The DFE of the model (16), given by (5), is GAS in \mathcal{D} whenever $\tau > \ln\left[\frac{\beta\Pi}{\mu(\mu+\phi+\gamma_1+\delta_1)}\right]^{\left(\frac{1}{\sigma+\mu}\right)} = \tau_c^H$.

In other words, like in the case of system (3), the disease will be eliminated from the community if and only if $\tau > \tau_c^H$. Fig. 5 depicts the numerical results obtained by simulating the model (16) using the parameter values in Table 3 and various initial conditions for the case $\tau > \tau_c^H (\mathcal{R}_0^H < 1)$. It is evident from this figure that all solutions converged to the DFE, \mathcal{E}_0 (in line with Theorem 2 and Lemma 6).

4.2. The existence of an EEP and disease permanence

In this section, the possible existence of endemic (positive) equilibria of the model (16), and the permanence of the disease, will be explored.

4.2.1. The existence of an EEP

Let $\mathcal{E}_1^H = (S^{**}; E^{**}; I^{**}; Q^{**}; H^{**}; R^{**})$ represent any arbitrary endemic equilibrium of the model (16). Solving the equations of the model (16) at the steady state gives

$$S^{**} = \frac{\Pi(1+\omega I^{**})}{\mu(1+\omega I^{**})+\beta I^{**}}, \qquad E^{**} = \frac{\beta(1-e^{-(\sigma+\mu)\tau})S^{**}I^{**}}{\sigma+\mu},$$

$$S^{**} = \frac{(1+\omega I^{**})(\gamma_1+\phi+\mu+\delta_1)}{\beta e^{-(\sigma+\mu)\tau}}, \qquad Q^{**} = \frac{\sigma E^{**}}{\alpha+\mu},$$

$$H^{**} = \frac{\phi I^{**}+\alpha Q^{**}}{\gamma_2+\mu+\delta_2}, \qquad R^{**} = \frac{\gamma_1 I^{**}+\gamma^{**}H^{**}}{\mu}.$$
(21)

Equating the first and third equations of (21), and solving for I^{**} in terms \mathcal{R}_0^H , gives

$$I^{**} = \frac{\mathcal{R}_0^H - 1}{\mu(\beta + \omega\mu)(\gamma_1 + \phi + \mu + \delta_1)^2} > 0, \quad \text{whenever } \mathcal{R}_0^H > 1.$$

$$(22)$$

Substituting for I^{**} from (22) into the first equation of (21) gives

$$S^{**} = \frac{\omega \Pi e^{-(\sigma+\mu)\tau} + (\gamma_1 + \phi + \mu + \delta_1)}{e^{-(\sigma+\mu)\tau} (\beta + \omega\mu)}.$$
(23)

It follows from (21) (noting from (22) and (23) that both I^{**} and S^{**} are positive if $\mathcal{R}_0^H > 1$) that $\mathcal{E}_1^H \in \mathbb{R}_+^6$ whenever $\mathcal{R}_0^H > 1$. Thus, the following result is established.

Lemma 7. The model (16) has a unique endemic (positive) equilibrium, given by \mathcal{E}_1^H , whenever $\mathcal{R}_0^H > 1$.

4.2.2. Permanence of the disease

The permanence of the disease will now be explored in the context of the model (16). That is, the objective is to determine whether or not the number of infectious cases in the population will persist above a certain positive number for a long time period (for the case when $\mathcal{R}_0^H > 1$).

Theorem 3. If $\mathcal{R}_0^H > 1$, then for any solution of (16) with the initial data (2), there exists a positive number $\nu = e^{-\tau(\gamma_1 + \phi + \mu + \delta_1)}I^{**}$, such that $\liminf_{t\to\infty} I(t) \ge \nu$.

Proof. The proof of Theorem 3 is based on using the approach given in [30–33]. It should be noted, first of all, that the second equation of (16) can be rewritten as

$$\dot{I} = \frac{\beta e^{-\tau(\sigma+\mu)} S(t) I(t)}{1 + \omega I(t)} - (\gamma_1 + \phi + \mu + \delta_1) I(t) - \frac{d}{dt} \int_{t-\tau}^t \frac{\beta e^{-\tau(\sigma+\mu)} S(x) I(x)}{1 + \omega I(x)} dx.$$
(24)

Consider the following function:

$$V(t) = I(t) + \int_{t-\tau}^{t} \frac{\beta e^{-\tau(\sigma+\mu)} S(x)I(x)}{1 + \omega I(x)} dx$$

Clearly, V(t) is bounded (since the variables I(t) and S(t) are bounded). Furthermore, it follows, using (24), that

$$\dot{V} = \frac{\beta e^{-\tau(\sigma+\mu)} S(t) I(t)}{1 + \omega I(t)} - (\gamma_1 + \phi + \mu + \delta_1) I(t).$$
(25)

Since, at the endemic steady state, S(t) is given by $S^{**} = \frac{\pi}{\mu + \frac{\beta I^{**}}{1 + \omega I^{**}}} > 0$ whenever $\mathcal{R}_0^H > 1$, it is clear that for any 0 < q < 1, $S^{**} < K$, where $K = \frac{\pi}{\mu + \frac{\beta q I^{**}}{1 + \omega q I^{**}}}$. Hence, there exists a number $m \ge 1$ such that $S^{**} < K(1 - e^{-m\pi\tau/K})$.

The next task is to show that $I(t) \ge qI^{**}$ for all $t \ge (m + 1)\tau$. Suppose, by contradiction, that $I(t) < qI^{**}$ for all $t \ge (m + 1)\tau$. It then follows, from the first equation of (16), for $t \ge (m + 1)\tau$, that

$$\dot{S}(t) > \Pi - \left(\mu + \frac{\beta q I^{**}}{1 + \omega q I^{**}}\right) S(t) = \Pi - \frac{\Pi}{K} S(t).$$

Hence,

$$\begin{split} S(t) \, &> \, K \, - \, \mathrm{e}^{-\Pi/K[t-(m+1)\tau]} \left\{ K - S[(m+1)\tau] \right\}, \\ &> \, K \left\{ 1 - \, \mathrm{e}^{-\Pi/K[t-(m+1)\tau]} \right\}, \end{split}$$

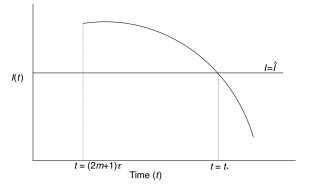


Fig. 6. Plot I(t) for $t \ge (2m + 1)\tau$.

and so, for $t \ge (2m+1)\tau$,

$$S(t) > K(1 - e^{-m\Pi\tau/K}) = \hat{S} > S^{**}.$$
 (26)

Since $I(t) < qI^{**} < I^{**}$, it follows from (25), for $t \ge (2m + 1)\tau$, that

$$\dot{V} > \frac{\beta e^{-\tau(\mu+\sigma)} S(t) I(t)}{1+\omega I^{**}} - (\gamma_1 + \phi + \mu + \delta_1) I(t), > \frac{\beta e^{-\tau(\mu+\sigma)} \hat{S} I(t)}{1+\omega I^{**}} - (\gamma_1 + \phi + \mu + \delta_1) I(t), = \left[\frac{\beta e^{-\tau(\mu+\sigma)} \hat{S}}{1+\omega I^{**}} - (\gamma_1 + \phi + \mu + \delta_1) \right] I(t).$$
(27)

Let $\hat{I} = \min_{\theta \in [-\tau,0]} I(\theta + 2\tau (m+1))$. It can be claimed that $I(t) \ge \hat{I}$ for all $t \ge (2m+1)\tau$. Suppose the claim does not hold. Then there exists a constant $d_1 > 0$ such that $I(t) \ge \hat{I}$ for $t \in ([2m+1]\tau, 2[m+1]\tau + d_1 = t_*)$, $I(t) < \hat{I}$ for $t > t_*$ with $I(t_*) = \hat{I}$ and $\hat{I}(t_*) \le 0$ (see Fig. 6). However, it follows from the third equation of (16), when $t = t_*$, that

$$\begin{split} \dot{I}(t_{*}) &= \frac{e^{-\tau(\sigma+\mu)}\beta S(t_{*}-\tau)I(t_{*}-\tau)}{1+\omega I(t_{*}-\tau)} - (\phi+\gamma_{1}+\mu+\delta_{1})I(t_{*}), \\ &= \frac{e^{-\tau(\sigma+\mu)}\beta S(t_{*}-\tau)I(t_{*}-\tau)}{1+\omega I(t_{*}-\tau)} - (\phi+\gamma_{1}+\mu+\delta_{1})\hat{I}, \quad \text{since } I(t_{*}) = \hat{I}, \\ &\geq \frac{e^{-\tau(\sigma+\mu)}\beta S(t_{*}-\tau)\hat{I}}{1+\omega I(t_{*}-\tau)} - (\phi+\gamma_{1}+\mu+\delta_{1})\hat{I}, \quad \text{since } I(t) \geq \hat{I} \text{ for } t \in ([2m+1]\tau, t_{*}), \\ &> \left[\frac{e^{-\tau(\sigma+\mu)}\beta S(t_{*}-\tau)}{1+\omega I^{**}} - (\phi+\gamma_{1}+\mu+\delta_{1})\right]\hat{I}, \quad \text{since } I(t) < I^{**} \text{ for } t \geq (2m+1)\tau, \\ &> \left[\frac{e^{-\tau(\sigma+\mu)}\beta S^{**}}{1+\omega I^{**}} - (\phi+\gamma_{1}+\mu+\delta_{1})\right]\hat{I} = 0. \end{split}$$

This contradicts the fact that $\dot{I}(t_*) \leq 0$. Hence, $I(t) \geq \hat{I}$ for $t \geq (2m + 1)\tau$. Thus, it follows from (27) that

$$\dot{V} > \left[\frac{\beta e^{-\tau(\mu+\sigma)}\hat{S}}{1+\omega I^{**}} - (\gamma_1 + \phi + \mu + \delta_1)\right]\hat{I} \quad \text{for all } t \ge 2(m+1)\tau.$$

Hence, $\lim_{t\to\infty} V(t) = \infty$, which contradicts the fact that V(t) is bounded. Finally, to complete the proof, we need to show that $I(t) \ge v$ for sufficiently large t.

Let t_1 be sufficiently large and $I(t_1) = qI^{**}$. Consider the following interval, $[t_1, t_2]$. It follows, from the second equation of (16), that

$$\dot{I} \ge -(\phi + \gamma_1 + \mu + \delta_1)I.$$

Hence,

$$I(t) > I(t_1)e^{-(\phi+\gamma_1+\mu+\delta_1)(t-t_1)} = qI^{**}e^{-(\phi+\gamma_1+\mu+\delta_1)(t-t_1)}, \quad \text{for } t \in [t_1, t_2].$$
(28)

It is clear from (28) that if $t_2 - t_1 \le \tau$, then $I(t) \ge qI^{**}e^{-\tau(\phi+\gamma_1+\mu+\delta_1)} = q\nu$. For the other case (where $t_2 - t_1 > \tau$), it is easy to see that the inequity $I(t) \ge qI^{**}e^{-\tau(\phi+\gamma_1+\mu+\delta_1)} = q\nu$ also holds for $t \in [t_1, t_1 + \tau]$. We claim that (28) also holds for $t \in (t_1 + \tau, t_2]$. If not, then there exists a constant d > 0 such that $I(t) \ge q\nu$ for $t \in (t_1 + \tau, t_1 + \tau + d = t_0)$, with $I(t_0) = q\nu$, $I(t) < q\nu$ for $t \in (t_0, t_2)$ and $\dot{I}(t_0) \le 0$. Here, too, it follows from the third equation of (16), when $t = t_0$, that

$$\begin{split} \dot{I}(t_0) &= \frac{e^{-\tau(\sigma+\mu)}\beta S(t_0-\tau)I(t_0-\tau)}{1+\omega I(t_0-\tau)} - (\phi+\gamma_1+\mu+\delta_1)I(t_0), \\ &= \frac{e^{-\tau(\sigma+\mu)}\beta S(t_0-\tau)I(t_0-\tau)}{1+\omega I(t_0-\tau)} - (\phi+\gamma_1+\mu+\delta_1)q\nu, \quad \text{since } I(t_0) = q\nu, \\ &\geq \frac{e^{-\tau(\sigma+\mu)}\beta S(t_0-\tau)q\nu}{1+\omega q\nu} - (\phi+\gamma_1+\mu+\delta_1)q\nu, \quad \text{since } I(t) \ge q\nu, \\ &\geq \left[\frac{e^{-\tau(\sigma+\mu)}\beta S(t_0-\tau)}{1+\omega I^{**}} - (\phi+\gamma_1+\mu+\delta_1)\right]q\nu, \quad \text{since } q\nu \le I^{**}, \\ &> \left[\frac{e^{-\tau(\sigma+\mu)}\beta S^{**}}{1+\omega I^{**}} - (\phi+\gamma_1+\mu+\delta_1)\right]q\nu = 0. \end{split}$$

This contradicts the fact that $\dot{I}(t_0) \leq 0$. Hence, $I(t) \geq q\nu$ for $t \in [t_1, t_1]$. Since this interval and the parameter $q \in (0, 1)$ are chosen arbitrarily, it is concluded that $I(t) \geq \nu$. Thus, $\liminf_{t \to \infty} I(t) \geq \nu$. \Box

The epidemiological implication of Theorem 3 is that the number of infectious cases will persist in the population (as $t \to \infty$) above a certain positive number (ν) whenever $\mathcal{R}_0^H > 1$.

4.3. Global stability of the EEP

Here, the global stability of the EEP, \mathcal{E}_1^H , of the model (16) will be explored. It is convenient to define

$$\mathcal{D}_0 = \{ (S, E, I, Q, H, R) \in \mathcal{D} : E = I = Q = H = R = 0 \}.$$

Theorem 4. The unique endemic equilibrium of the model (16), given by (21), is GAS in $\mathcal{D} \setminus \mathcal{D}_0$ if $\mathcal{R}_0^H > 1$ and $\omega \Pi e^{-\tau(\sigma+\mu)} > \phi + \gamma_1 + \mu + \delta_1$.

Proof. The proof of Theorem 4 is based on using a comparison argument and an iteration technique, as given in [29,34]. Let (S(t), E(t), I(t), Q(t), H(t), R(t)) be any solution of (16) with initial conditions given by (2). Further, let

$$S_{\infty} = \liminf_{t \to \infty} S(t), \qquad S^{\infty} = \limsup_{t \to \infty} S(t), \qquad E_{\infty} = \liminf_{t \to \infty} E(t), \qquad E^{\infty} = \limsup_{t \to \infty} E(t)$$

$$I_{\infty} = \liminf_{t \to \infty} I(t), \qquad I^{\infty} = \limsup_{t \to \infty} I(t), \qquad Q_{\infty} = \liminf_{t \to \infty} Q(t), \qquad Q^{\infty} = \limsup_{t \to \infty} Q(t)$$

$$H_{\infty} = \liminf_{t \to \infty} H(t), \qquad H^{\infty} = \limsup_{t \to \infty} H(t), \qquad R_{\infty} = \liminf_{t \to \infty} R(t), \qquad R^{\infty} = \limsup_{t \to \infty} R(t).$$

The goal is to show that

$$S_{\infty} = S^{\infty} = S^{**}, \qquad E_{\infty} = E^{\infty} = E^{**}, \qquad I_{\infty} = I^{\infty} = I^{**}, \qquad Q_{\infty} = Q^{\infty} = Q^{**}, \\ H_{\infty} = H^{\infty} = H^{**}, \quad \text{and} \quad R_{\infty} = R^{\infty} = R^{**}.$$

It follows from the first equation of (16) that

$$\hat{S}(t) \leq \Pi - \mu S$$

and so, by the comparison theorem,

 $\limsup_{t\to\infty} S(t) \le \Pi/\mu.$

Let $U_1^S = \Pi/\mu$. Thus, for sufficiently small $\epsilon > 0$, there exists a $T_1 > 0$ such that $S(t) \le U_1^S + \epsilon$ for $t > T_1$. It follows from the third equation of (16) that, for $t > T_1 + \tau$,

$$\dot{I}(t) \le \frac{\beta e^{-\tau(\sigma+\mu)} (U_1^S + \epsilon) I(t-\tau)}{1 + \omega I(t-\tau)} - (\phi + \gamma_1 + \mu + \delta_1) I(t).$$
(29)

Consider the auxiliary equation of (29):

$$\dot{u}(t) = \frac{\beta e^{-\tau(\sigma+\mu)} (U_1^S + \epsilon) u(t-\tau)}{1 + \omega u(t-\tau)} - (\phi + \gamma_1 + \mu + \delta_1) u(t).$$
(30)

Since $\mathcal{R}_0^H > 1$, it follows that, for sufficiently small $\epsilon > 0$, $\beta e^{-\tau(\sigma+\mu)}(U_1^S + \epsilon) > (\phi + \gamma_1 + \mu + \delta_1)$. Hence, by Item (ii) of Lemma 5 and (30),

$$\lim_{t \to \infty} u(t) = \frac{\beta e^{-\tau(\sigma+\mu)} (U_1^S + \epsilon) - (\phi + \gamma_1 + \mu + \delta_1)}{\omega(\phi + \gamma_1 + \mu + \delta_1)}$$

Thus, by the comparison theorem,

$$I^{\infty} = \limsup_{t \to \infty} I(t) \le \frac{\beta e^{-\tau(\sigma+\mu)} (U_1^{S} + \epsilon) - (\phi + \gamma_1 + \mu + \delta_1)}{\omega(\phi + \gamma_1 + \mu + \delta_1)},$$

and so

$$I^{\infty} \leq \frac{\beta e^{-\tau(\sigma+\mu)} U_1^{S} - (\phi + \gamma_1 + \mu + \delta_1)}{\omega(\phi + \gamma_1 + \mu + \delta_1)}$$

Similarly, let

$$U_1^I = \frac{\beta e^{-\tau(\sigma+\mu)} U_1^S - (\phi+\gamma_1+\mu+\delta_1)}{\omega(\phi+\gamma_1+\mu+\delta_1)}.$$

Then, for sufficiently small $\epsilon > 0$, there exists a $T_2 > T_1 + \tau$ such that $I(t) \le U_1^l + \epsilon$ for $t > T_2$. It follows from the first equation of (16), for $t > T_2$, that

$$\dot{S}(t) \ge \Pi - \mu S - \frac{\beta(U_1^l + \epsilon)}{1 + \omega(U_1^l + \epsilon)},$$

and so, by the comparison theorem,

$$S_{\infty} = \liminf_{t \to \infty} S(t) \ge \frac{\Pi[1 + \omega(U_1^I + \epsilon)]}{\mu + (\beta + \mu\omega)(U_1^I + \epsilon)}.$$

Hence, $S_{\infty} \ge L_1^S$, where $L_1^S = \frac{\Pi[1+\omega U_1^I]}{\mu+(\beta+\mu\omega)U_1^I}$. In other words, for sufficiently small $\epsilon > 0$, there exists a $T_3 > T_2 + \tau$ such that $S(t) \ge L_1^S - \epsilon$ for $t > T_3$. It follows from the third equation of (16), for $t > T_3 + \tau$, that

$$\dot{I}(t) \geq \frac{\beta e^{-\tau(\sigma+\mu)} (L_1^{\mathrm{S}} - \epsilon) I(t-\tau)}{1 + \omega I(t-\tau)} - (\phi + \gamma_1 + \mu + \delta_1) I(t),$$

and so (by considering the auxiliary equation)

$$\dot{u}(t) = \frac{\beta e^{-\tau(\sigma+\mu)} (L_1^{\mathcal{S}} - \epsilon) u(t-\tau)}{1 + \omega u(t-\tau)} - (\phi + \gamma_1 + \mu + \delta_1) u(t).$$

Hence, it follows from Item (ii) of Lemma 5 (since $\mathcal{R}_0^H > 1$) that

$$\lim_{t \to \infty} u(t) = \frac{\beta e^{-\tau(\sigma+\mu)} (L_1^{\varsigma} - \epsilon) - (\phi + \gamma_1 + \mu + \delta_1)}{\omega(\phi + \gamma_1 + \mu + \delta_1)}$$

and the comparison theorem gives

$$I_{\infty} = \liminf_{t \to \infty} I(t) \ge \frac{\beta e^{-\tau(\sigma+\mu)} (L_1^{\mathsf{S}} - \epsilon) - (\phi + \gamma_1 + \mu + \delta_1)}{\omega(\phi + \gamma_1 + \mu + \delta_1)}.$$

Hence, for sufficiently small $\epsilon > 0$, there exists a $T_4 > T_3 + \tau$ such that $I(t) \ge L_1^l - \epsilon$ for $t > T_4$, where

$$L_1^l = \frac{\beta e^{-\tau(\sigma+\mu)} L_1^{\varsigma} - (\phi + \gamma_1 + \mu + \delta_1)}{\omega(\phi + \gamma_1 + \mu + \delta_1)}$$

Using $S(t) \le U_1^S + \epsilon$, $I(t) \le U_1^I + \epsilon$, $S(t) \ge L_1^S - \epsilon$ and $I(t) \ge L_1^I - \epsilon$ in the second equation of (16), for $t > T_4 + \tau$, gives

$$\dot{E} \leq \frac{\beta(U_1^3 + \epsilon)(U_1^l + \epsilon)}{1 + \omega(U_1^l + \epsilon)} - \frac{\beta e^{-\tau(\sigma + \mu)}(L_1^2 - \epsilon)(L_1^l - \epsilon)}{1 + \omega(L_1^l - \epsilon)} - (\sigma + \mu)E$$

Hence, by the comparison theorem,

$$E^{\infty} = \limsup_{t \to \infty} E(t) \le \frac{\beta(U_1^{\mathsf{S}} + \epsilon)(U_1^{\mathsf{I}} + \epsilon)}{[1 + \omega(U_1^{\mathsf{I}} + \epsilon)](\sigma + \mu)} - \frac{\beta e^{-\tau(\sigma + \mu)}(L_1^{\mathsf{S}} - \epsilon)(L_1^{\mathsf{I}} - \epsilon)}{[1 + \omega(L_1^{\mathsf{I}} - \epsilon)](\sigma + \mu)}.$$

Therefore, for sufficiently small $\epsilon > 0$, there exists a $T_5 > T_4 + \tau$ such that $E(t) \le U_1^E + \epsilon$ for $t > T_5$, where

$$U_{1}^{E} = \frac{\beta U_{1}^{S} U_{1}^{I}}{(1 + \omega U_{1}^{I})(\sigma + \mu)} - \frac{\beta e^{-\tau(\sigma + \mu)} L_{1}^{S} L_{1}^{I}}{(1 + \omega L_{1}^{I})(\sigma + \mu)}.$$

Similarly, by using $S(t) \le U_1^S + \epsilon$, $I(t) \le U_1^I + \epsilon$, $S(t) \ge L_1^S - \epsilon$ and $I(t) \ge L_1^I - \epsilon$ in the second equation of (16), for $t > T_4 + \tau$, we have

$$\dot{E} \leq \frac{\beta(L_1^S - \epsilon)(L_1^l - \epsilon)}{1 + \omega(L_1^l - \epsilon)} - \frac{\beta e^{-\tau(\sigma + \mu)}(U_1^S + \epsilon)(U_1^l + \epsilon)}{1 + \omega(U_1^l + \epsilon)} - (\sigma + \mu)E,$$

and so

$$E_{\infty} = \liminf_{t \to \infty} E(t) \ge \frac{\beta(L_1^S - \epsilon)(L_1^l - \epsilon)}{[1 + \omega(L_1^l - \epsilon)](\sigma + \mu)} - \frac{\beta e^{-\tau(\sigma + \mu)}(U_1^S + \epsilon)(U_1^l + \epsilon)}{[1 + \omega(U_1^l + \epsilon)](\sigma + \mu)}$$

Hence, for sufficiently small $\epsilon > 0$, there exists a $T_6 > T_5 + \tau$ such that $E(t) \ge L_1^E - \epsilon$ for $t > T_6$, where

$$L_{1}^{E} = \frac{\beta L_{1}^{S} L_{1}^{l}}{(1 + \omega L_{1}^{l})(\sigma + \mu)} - \frac{\beta e^{-\tau(\sigma + \mu)} U_{1}^{S} U_{1}^{l}}{(1 + \omega U_{1}^{l})(\sigma + \mu)}$$

Using $E(t) \leq U_1^E + \epsilon$ in the fourth equation of (16), for $t > T_5$, gives

$$\dot{Q}(t) \leq \sigma(U_1^E + \epsilon) - (\alpha + \mu)Q,$$

and so

$$Q^{\infty} = \limsup_{t \to \infty} Q(t) \le \frac{\sigma(U_1^E + \epsilon)}{\alpha + \mu}$$

Thus, for sufficiently small $\epsilon > 0$, there exists a $T_7 > T_6 + \tau$ such that $Q(t) \le U_1^Q + \epsilon$ for $t > T_7$, where $U_1^Q = \frac{\sigma U_1^E}{\alpha + \mu}$. Similarly, by using $E(t) \ge L_1^E - \epsilon$ in the fourth equation of (16), for $t > T_6$, we have

$$\dot{Q}(t) \ge \sigma (L_1^E - \epsilon) - (\alpha + \mu)Q,$$

and

$$Q_{\infty} = \liminf_{t \to \infty} Q(t) \ge \frac{\sigma(L_1^t - \epsilon)}{\alpha + \mu}.$$

Thus, for sufficiently small $\epsilon > 0$, there exists a $T_8 > T_7 + \tau$ such that $Q(t) \ge L_1^Q - \epsilon$ for $t > T_8$, where $L_1^Q = \frac{\sigma L_1^L}{\alpha + \mu}$. Using $I(t) \le U_1^I + \epsilon$ and $Q(t) \le U_1^Q + \epsilon$ in the fifth equation of (16), for $t > T_7$, gives

$$\dot{H}(t) \leq \alpha (U_1^Q + \epsilon) + \phi (U_1^I + \epsilon) - (\gamma_2 + \mu + \delta_2)H,$$

and

$$H^{\infty} = \limsup_{t \to \infty} H \le \frac{\alpha(U_1^{\mathbb{Q}} + \epsilon) + \phi(U_1^{\mathbb{I}} + \epsilon)}{\gamma_2 + \mu + \delta_2}$$

Thus, for sufficiently small $\epsilon > 0$, there exists a $T_9 > T_8 + \tau$ such that $H(t) \le U_1^H + \epsilon$, for $t > T_9$, where $U_1^H = \frac{\alpha U_1^Q + \phi U_1^I}{(\gamma_2 + \mu + \delta_2)}$. Similarly, it follows by using $I(t) \ge L_1^I - \epsilon$ and $Q(t) \ge L_1^Q - \epsilon$ in the fifth equation of (16), for $t > T_8$, that

$$\dot{H}(t) \ge \alpha (L_1^Q - \epsilon) + \phi (L_1^I - \epsilon) - (\gamma_2 + \mu + \delta_2) H,$$

and so

$$H_{\infty} = \liminf_{t \to \infty} H \le \frac{\alpha(L_1^Q - \epsilon) + \phi(L_1^l - \epsilon)}{\gamma_2 + \mu + \delta_2}$$

Hence, for sufficiently small $\epsilon > 0$, there exists a $T_{10} > T_9 + \tau$ such that $H(t) \ge L_1^H - \epsilon$ for $t > T_{10}$, where $L_1^H = \frac{\alpha L_1^Q + \phi L_1^I}{\gamma_2 + \mu + \delta_2}$.

Using $I(t) \le U_1^l + \epsilon$ and $H(t) \le U_1^H + \epsilon$ in the last equation of (16), for $t > T_9$, gives

$$\dot{\mathbf{R}} \le \gamma_1(U_1^I + \epsilon) + \gamma_2(U_1^H + \epsilon) - \mu \mathbf{R}$$

Hence,

$$R^{\infty} = \limsup_{t \to \infty} R(t) \le \frac{\gamma_1(U_1^l + \epsilon) + \gamma_2(U_1^H + \epsilon)}{\mu}.$$

Thus, $R^{\infty} \leq U_1^R$, where $U_1^R = \frac{\gamma_1 U_1^l + \gamma_2 U_1^H}{\mu}$. Using $I(t) \geq L_1^l - \epsilon$ and $H(t) \geq L_1^H - \epsilon$ in the last equation of (16), for $t > T_{10}$, gives

$$R \ge \gamma_1(L_1^I - \epsilon) + \gamma_2(L_1^H - \epsilon) - \mu R,$$

and so (by the comparison theorem)

$$R_{\infty} = \liminf_{t \to \infty} R(t) \ge \frac{\gamma_1(L_1^l - \epsilon) + \gamma_2(L_1^H - \epsilon)}{\mu}$$

Hence, $R_{\infty} \ge L_1^R$, where $L_1^R = \frac{\gamma_1 L_1^I + \gamma_2 L_1^H}{\mu}$. Continuing in this manner leads to the following sequences:

$$\begin{split} U_{n}^{S} &= \frac{\Pi[1 + \omega L_{n-1}^{l}]}{\mu + (\beta + \mu \omega)L_{n-1}^{l}}, \qquad L_{n}^{S} = \frac{\Pi[1 + \omega U_{n}^{l}]}{\mu + (\beta + \mu \omega)U_{n}^{l}}, \\ U_{n}^{I} &= \frac{\beta e^{-\tau(\sigma + \mu)}U_{n}^{S} - (\phi + \gamma_{1} + \mu + \delta_{1})}{\omega(\phi + \gamma_{1} + \mu + \delta_{1})}, \qquad L_{n}^{I} = \frac{\beta e^{-\tau(\sigma + \mu)}L_{n}^{S} - (\phi + \gamma_{1} + \mu + \delta_{1})}{\omega(\phi + \gamma_{1} + \mu + \delta_{1})}, \\ U_{n}^{E} &= \frac{\beta U_{n}^{l}U_{n}^{S}}{(1 + \omega U_{n}^{l})(\sigma + \mu)} - \frac{\beta e^{-\tau(\sigma + \mu)}L_{n}^{l}L_{n}^{S}}{(1 + \omega L_{n}^{l})(\sigma + \mu)}, \\ L_{n}^{E} &= \frac{\beta L_{n}^{l}L_{n}^{S}}{(1 + \omega L_{n}^{l})(\sigma + \mu)} - \frac{\beta e^{-\tau(\sigma + \mu)}U_{n}^{l}U_{n}^{S}}{(1 + \omega U_{n}^{l})(\sigma + \mu)}, \\ U_{n}^{Q} &= \frac{\sigma U_{n}^{E}}{(\alpha + \mu)}, \qquad L_{n}^{Q} &= \frac{\sigma L_{n}^{E}}{(\alpha + \mu)}, \\ U_{n}^{H} &= \frac{\alpha U_{n}^{Q} + \phi U_{n}^{l}}{(\gamma_{2} + \mu + \delta_{2})}, \qquad L_{n}^{H} &= \frac{\alpha L_{n}^{Q} + \phi L_{n}^{l}}{(\gamma_{2} + \mu + \delta_{2})}, \\ U_{n}^{R} &= \frac{\gamma_{1}U_{n}^{l} + \gamma_{2}U_{n}^{H}}{\mu}, \qquad L_{n}^{R} &= \frac{\gamma_{1}L_{n}^{l} + \gamma_{2}L_{n}^{H}}{\mu}. \end{split}$$

Finally, since $L_n^S \leq S_\infty \leq S^\infty \leq U_n^S$, $L_n^E \leq E_\infty \leq U_n^E$, $L_n^I \leq I_\infty \leq I^\infty \leq U_n^I$, $L_n^Q \leq Q_\infty \leq Q^\infty \leq U_n^Q$, $L_n^H \leq H_\infty \leq H^\infty \leq U_n^H$ and $L_n^R \leq R_\infty \leq R^\infty \leq U_n^R$, the proof is concluded by showing that

$$\begin{split} &\lim_{n\to\infty}U_n^S=S^{**}=\lim_{n\to\infty}L_n^S,\qquad \lim_{n\to\infty}U_n^I=I^{**}=\lim_{n\to\infty}L_n^I,\\ &\lim_{n\to\infty}U_n^E=E^{**}=\lim_{n\to\infty}L_n^E,\qquad \lim_{n\to\infty}U_n^Q=Q^{**}=\lim_{n\to\infty}L_n^Q\\ &\lim_{n\to\infty}U_n^H=H^{**}=\lim_{n\to\infty}L_n^H,\qquad \lim_{n\to\infty}U_n^R=R^{**}=\lim_{n\to\infty}L_n^R \end{split}$$

Using the first four sequences of (31), it is easy to see that the sequence U_{n+1}^S can be written in terms of U_n^S as

$$U_{n+1}^{S} = \frac{\omega^{2} \Pi^{2} e^{-2\tau(\sigma+\mu)} U_{n}^{S}}{k^{2} + e^{-\tau(\sigma+\mu)} (\beta + \omega\mu) [\omega \Pi e^{-\tau(\sigma+\mu)} - k] U_{n}^{S}},$$
(32)

where $k = \phi + \gamma_1 + \mu + \delta_1$. Furthermore, it can be shown that whenever $\omega \Pi e^{-\tau(\sigma+\mu)} > k$, the sequence U_n^S is monotone as follows:

$$U_{n+1}^{S} - U_{n}^{S} = \frac{[\omega \Pi e^{-\tau(\sigma+\mu)} - k][\omega \Pi e^{-\tau(\sigma+\mu)} + k - (\beta + \omega\mu)e^{-\tau(\sigma+\mu)}U_{n}^{S}]U_{n}^{S}}{k^{2} + e^{-\tau(\sigma+\mu)}(\beta + \omega\mu)[\omega \Pi e^{-\tau(\sigma+\mu)} - k]U_{n}^{S}}.$$

Since $S^{**} \leq U_n^S$, it follows that

$$U_{n+1}^{S} - U_{n}^{S} \leq \frac{[\omega \Pi e^{-\tau(\sigma+\mu)} - k][\omega \Pi e^{-\tau(\sigma+\mu)} + k - (\beta + \omega\mu)e^{-\tau(\sigma+\mu)}S^{**}]U_{n}^{S}}{k^{2} + e^{-\tau(\sigma+\mu)}(\beta + \omega\mu)[\omega \Pi e^{-\tau(\sigma+\mu)} - k]U_{n}^{S}}$$
$$= 0 \quad \left(\operatorname{since} S^{**} = \frac{\omega \Pi e^{-\tau(\sigma+\mu)} + k}{e^{-\tau(\sigma+\mu)}(\beta + \omega\mu)}\right).$$

Thus, $\lim_{n\to\infty} U_n^S$ exists.

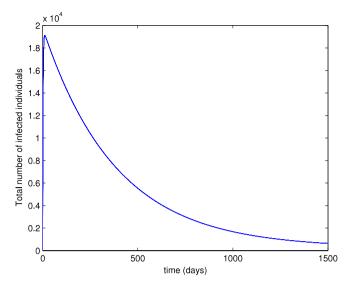


Fig. 7. Simulations of the delayed model (16), showing the total number of infected individuals as a function of time. Parameter values used are as given in Table 3, with $\tau = 10$ and $\beta = 0.0025809$ (so $\mathcal{R}_0^H = 2.3741 > 1$ and $\tau < \tau_c^H = 16.3340$).

Let $M = \lim_{n \to \infty} U_n^S$. Then, it follows from (32) that

$$M = \frac{\omega^2 \Pi^2 e^{-2\tau(\sigma+\mu)} M}{k^2 + e^{-\tau(\sigma+\mu)} (\beta + \omega\mu) [\omega \Pi e^{-\tau(\sigma+\mu)} - k] M}$$

and so

$$M = \lim_{t \to \infty} U_n^S = \frac{\omega \Pi e^{-\tau(\sigma+\mu)} + k}{e^{-\tau(\sigma+\mu)}(\beta + \omega\mu)} = S^{**}.$$

Taking the limit as $n \to \infty$ of both sides of the third sequence of (31) gives

$$\lim_{n \to \infty} U_n^I = \frac{\beta e^{-\tau(\sigma+\mu)} S^{**} - (\phi+\gamma_1+\mu+\delta_1)}{\omega(\phi+\gamma_1+\mu+\delta_1)} = I^{**}$$

Similarly, taking the limits of both sides of the remaining sequences in (31), and using the previous results, gives

$$\lim_{n \to \infty} L_n^S = S^{**}, \qquad \lim_{n \to \infty} L_n^I = I^{**}, \qquad \lim_{n \to \infty} U_n^E = \lim_{n \to \infty} L_n^E = I$$
$$\lim_{n \to \infty} U_n^Q = \lim_{n \to \infty} L_n^Q = Q^{**}, \qquad \lim_{n \to \infty} U_n^H = \lim_{n \to \infty} L_n^H = H^{**},$$
$$\lim_{n \to \infty} U_n^R = \lim_{n \to \infty} L_n^R = R^{**}.$$

Hence, $\lim_{t\to\infty} (S(t), E(t), I(t), Q(t), H(t), R(t)) = \mathcal{E}_1^H$. \Box

Theorem 4 shows that the disease will persist in the population whenever $\mathcal{R}_0^H > 1$. Here, too, by solving for τ from $\mathcal{R}_0^H > 1$, the following result can be shown.

Lemma 8. The unique endemic equilibrium of the model (16), given by (21), is GAS in $\mathcal{D} \setminus \mathcal{D}_0$ if $\tau < \ln \left[\frac{\beta \Pi}{\mu(\mu + \phi + \gamma_1 + \delta_1)} \right]^{\left(\frac{1}{\sigma + \mu} \right)} = \tau_c$ and $\omega \Pi e^{-\tau(\sigma + \mu)} > \phi + \gamma_1 + \mu + \delta_1$.

Theorem 4 shows that the disease will persist in the population provided that $\Re_0^H > 1$ ($\tau < \tau_c$) and $\omega \Pi e^{-\tau(\sigma+\mu)} > \phi + \gamma_1 + \mu + \delta_1$. Thus, Lemmas 6 and 8 suggest that $\tau = \tau_c$ is a sharp epidemiological threshold that governs the persistence ($\tau < \tau_c$) and elimination ($\tau > \tau_c$) of the disease in the population. Fig. 7 shows a time series plot of the total number of infected individuals for various initial conditions. This figure clearly shows convergence of the solutions to the EEP for the case $\tau < \tau_c$ ($\Re_0^H > 1$) (in line with Theorem 4 and Lemma 8). Fig. 8 depicts of the total number of cases as a function of time for various values of τ . This figure shows a decreasing number of cases with increasing values of delay parameter τ . That is, the longer individuals stay in the exposed class, the lower the disease burden.

To assess the impact of using time delay to model the incubation period on the suitability of the model (3) for realistically capturing the observed SARS data (cumulative probable cases) for the Greater Toronto Area (GTA) of Canada, the model (3) is simulated in the presence and absence of time delay. It should be stated that the GTA recorded about 250 probable cases of

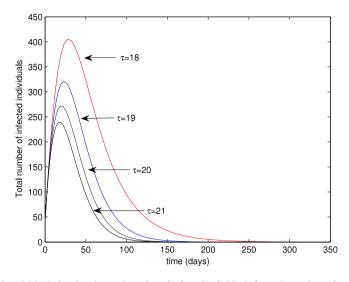


Fig. 8. Simulations of the delayed model (16), showing the total number of infected individuals for various values of τ . Parameter values used are as given in Table 3, with $\beta = 0.15$.

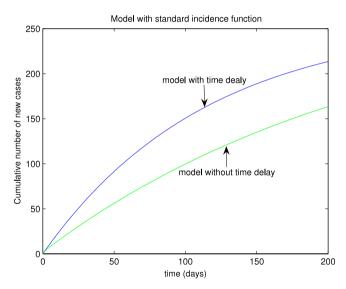


Fig. 9. Numerical simulations of the standard incidence delayed model (3), showing the cumulative number of probable SARS cases for the GTA, in the presence and absence of time delay.

SARS during the 2003 outbreaks [21]. The simulation results obtained, depicted in Fig. 9, show that while the model without time delay (considered in [10]) underestimated the observed cumulative number of probable cases (about 170 cases), the model with time delay (i.e., model (3)) gave a good estimate of the observed data (about 220 cases). Thus, this study suggests that the model (3), with time delay, is more appropriate for modeling the SARS outbreaks in the GTA than the corresponding model without time delay (given in [10]). Similar simulation results were obtained for the case of the model with Holling type II function (16) (see Fig. 10).

5. Conclusions

A deterministic quarantine/isolation model with time delay is considered, subject to two incidence functions, namely standard incidence and the Holling type II incidence function. The main findings of this study are summarized below:

- (i) The model with standard incidence function, given by (3), has a globally asymptotically stable disease-free solution whenever a certain epidemiological threshold quantity (\mathcal{R}_0^S) is less than unity (Theorem 1). Furthermore, this model has a unique positive endemic equilibrium whenever the threshold quantity (\mathcal{R}_0^S) exceeds unity (Lemma 4).
- (ii) The model with Holling type II incidence function, given by (16), has a globally asymptotically stable disease-free solution whenever its associated epidemiological threshold quantity (\mathcal{R}_0^H) is less than unity (Theorem 2). This model has

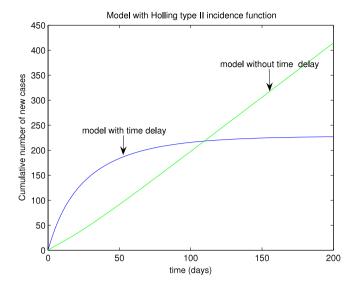


Fig. 10. Numerical simulations of the Holling type II delayed model (16), showing the cumulative number of probable SARS cases for the GTA, in the presence and absence of time delay.

a unique positive endemic equilibrium whenever the threshold quantity (\mathcal{R}_0^H) exceeds unity (Lemma 7). Furthermore, the model system is permanent whenever $\mathcal{R}_0^H > 1$ (Theorem 3). The unique endemic equilibrium of the model (16) is globally asymptotically stable under certain conditions (Theorem 4).

In summary, the theoretical analyses in this study show that adding time delay to and/or replacing the standard incidence function by a Holling type II incidence function in the autonomous (non-delayed) quarantine/isolation model in [10] does not alter the qualitative dynamics (as regards the elimination or persistence of the disease) of the non-delayed model considered in [10]. In other words, the theoretical results in this study show that the quarantine/isolation model with time delay ($\tau > 0$) and standard or non-linear incidence function of Holling type II has essentially the same qualitative (equilibrium) dynamics as the corresponding autonomous quarantine/isolation model ($\tau = 0$) with the standard incidence function considered in [10].

Numerical simulations of the delayed model with the standard incidence function show that the associated disease burden decreases with increasing time delay (τ). Furthermore, models with time delay seem to be more appropriate for modeling the SARS epidemic than those without time delay (regardless of which of the incidence functions is used).

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