Time-delayed modelling of the COVID-19 dynamics with a convex incidence rate ☆

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ABSTRACT

COVID-19 pandemic represents an unprecedented global health crisis which has an enormous impact on the world population and economy. Many scientists and researchers have combined efforts to develop an approach to tackle this crisis and as a result, researchers have developed several approaches for understanding the COVID-19 transmission dynamics and the way of mitigating its effect. The implementation of a mathematical model has proven helpful in further understanding the behaviour which has helped the policymaker in adopting the best policy necessary for reducing the spread. Most models are based on a system of equations which assume an instantaneous change in the transmission dynamics. However, it is believed that SARS-COV-2 have an incubation period before the tendency of transmission. Therefore, to capture the dynamics adequately, there would be a need for the inclusion of delay parameters which will account for the delay before an exposed individual could become infected. Hence, in this paper, we investigate the SEIR epidemic model with a convex incidence rate incorporated with a time delay. We first discussed the epidemic model as a form of a classical ordinary differential equation and then the inclusion of a delay to represent the period in which the susceptible and exposed individuals became infectious. Secondly, we identify the disease-free together with the endemic equilibrium state and examine their stability by adopting the delay differential equation stability theory. Thereafter, we carried out numerical simulations with suitable parameters choice to illustrate the theoretical result of the system and for a better understanding of the model dynamics. We also vary the length of the delay to illustrate the changes in the model as the delay parameters change which enables us to further gain an insight into the effect of the included delay in a dynamical system. The result confirms that the inclusion of delay destabilises the system and it forces the system to exhibit an oscillatory behaviour which leads to a periodic solution and it further helps us to gain more insight into the transmission dynamics of the disease and strategy to reduce the risk of infection.

1. Introduction

The Coronavirus Disease (COVID-19) formerly known as a novel coronavirus (2019-nCoV) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is highly transmissible [1] and has posed a great threat to the global public health, of which was reported by the World Health Organization (WHO) to emerged in Wuhan City, Hubei province of China at the end of the year 2019. Mathematical modelling has proven to be essential in understanding the dynamics of infectious diseases. A direct application of mathematical models to data has been of enormous help in having more knowledge about the infection and control of diseases [2]. In recent times, there has been much concern about airborne transmission such as coronavirus which spread rapidly with high cases of infection still recorded across nations [3]. As a result of the massive rate of daily new cases and high infection rate,

several literature have been dedicated to the study of the disease and multiple models have been developed to foster our understanding of the dynamics of the virus [4]. Many researchers believe that it is of utmost importance to have a schematic that could help to represent the behaviour of the transmission and a model is believed to be highly desirable. The emergence of the mathematical model, therefore, enables researchers to gain useful insight into the nature, evolution and process of modelling infectious diseases [5]. Although many mathematical models have been in existence, the most adopted model is that of Kermack and McKendrick [6] proposed in 1927 which is popularly known as the SIR (Susceptible–Infected–Recovered) epidemic model. Since the formulation of the famous SIR model, there have also been several models that have been developed for the studying of the spread of infectious diseases [7]. For instance [8–12] modifies the popularly accepted epidemic model to investigate the transmission dynamics and control of swine influenza. Also, [13–27] uses a mathematical model for understanding the COVID-19 transmission dynamics with a case study of India and other region using the available dataset where their model predicts the transmission of the virus. Similarly, [28–35] adopted the epidemic to study the spread of HIV infection and many more. Recently, the SIR model has been extensively extended and applied to investigate the transmission of COVID-19 under different incidence rates [2,4,7]. It is worth noting that the incidence rate is considered to play a significant role in infectious disease modelling as it provides a reasonable qualitative description of the disease dynamics. In much literature, the bilinear incidence rate is often adopted with the assumption that the populations are homogeneous mixing with the environment [2]. However, in reality, this assumption seems to be inaccurate as the population mixing could be heterogeneous. Therefore, this led to the modification of the bilinearity of the incidence rate to the adoption of nonlinearity [36]. Many literatures such as [37–41] also backed up this claim and confirmed that the process involving the transmission of the disease is indeed better captured with the use of the nonlinear incidence rate. Although the use of the nonlinear incidence rate in an epidemic model has provided a rich dynamical behaviour, [42] pointed out that the modification to the convex incidence rate could better capture a complex scenario where the disease incubator is not negligence. This is highly relevant in this study as it is believed that the COVID-19 infection does have an incubation period. Therefore, in this work, we shall consider the epidemic model with a convex incidence rate and also incorporate a time delay. Lately, time delays have been included in models to provide a better understanding of a complex system. In fact, in many physical phenomena, the state of the system does not solely depend on the current occurrence, the past information is also crucial for an in-depth understanding of the system. So, incorporating time delay helps to provide a more realistic model [41]. So, the classical ODE models are now being modified to include the delay in order to capture the complexity of the system. For example, the delay differential equation model is used by [43] to analyse the HIV infection where the delay is incorporated to account for the time between viral entry into the cell and the production of new virus particles. Similarly, [44] implemented a time-delayed model for drug therapy and it was also used by [45] for immune response and many more. This shows that to have a more realistic representation of a dynamical system, the inclusion of delay parameters is inevitable. The time-delayed modelling has now been receiving much attention and it adequately enables modellers to account for the lags in responses. Hence, the inclusion of time delay in an epidemic model makes the system to have a representation close to reality and it could also change the behaviour of such a system such as destabilising the steady state. As a result, this paper aims to conduct a qualitative analysis of the time-delayed model for COVID-19 infection using the SEIR (Susceptible–Exposed–Infected–Recovered) epidemic model with a convex incidence rate. Then, we shall investigate the effect of the time delay on the dynamic behaviour of the model. In practice, this delay enable us to adequately account for the pauses between when the exposed population became infected as it

Table 1
Parameter description.

Parameters	Physical description
$S(t)$	Susceptible
$E(t)$	Exposed
$I(t)$	Infected
$R(t)$	Removed
A	Recruitment
μ	Death rate
α, β	Constant rate
σ	Latent period of disease exposure
γ	Recovery rate

is believed that there is not instantaneous change between the exposed and infected. This delay in change of class compartment has already been established through the World Health Organisation (WHO) report where it is confirmed that exposed class only start to show symptoms of infection after few days of contact. In addition, the introduced mechanism of testing also confirmed that there could be false negative if testing is performed immediately after contact. Therefore, this needs to be adequately incorporate into the SEIR model. The organisation of the paper is as follows: In Section 2, we discussed the model both with and without delay and then provides the preliminary result of the model. In Section 3, we discussed the equilibria and the stability of both the disease-free and endemic equilibrium. The numerical simulations and discussion of the results are presented in Section 4 while the conclusion is provided in Section 5.

2. Model formulation

In this section, we present the epidemic model without delay and then discuss the idea of the epidemic model with a time delay.

2.1. SEIR without time delay

We provide some preliminary results of the SEIR model with a convex incidence rate which does not include the time delay. The quantitative analysis of the result shall be discussed which will be used to analyse the stability of the delayed SEIR model. To begin with, suppose an individual in a population who is susceptible to disease is represented by $S(t)$, we regard this group as the individuals who are yet to be infected. The number of an individual that is exposed to the disease is represented by $E(t)$ while the infected population who have the tendency to spread the disease are denoted by $I(t)$. Finally, we denoted the removed class by $R(t)$. As provided in [46,7,42], the recruitment into the susceptible population is through migration or birth at a constant rate A, where σ represents the latent period of the disease exposure, natural death rate occur in all the classes at the rate μ, we have not considered the disease-induced death rate in this model. We considered a convex incidence rate of the form $\beta IS(1 + \alpha I)$ where α, β are positive constants. Therefore, the SEIR model is given as:

$$\begin{aligned}
\dot{S} &= A - \mu S(t) - \beta S(t) I(t)(1 + \alpha I(t)), \\
\dot{E} &= \beta S(t) I(t)(1 + \alpha I(t)) - (\mu + \sigma) E(t), \\
\dot{I} &= \sigma E(t) - (\mu + \gamma) I(t), \\
\dot{R} &= \gamma I(t) - \mu R(t).
\end{aligned} \tag{2.1}$$

The model presented in Eq. (2.1) can be shown in form of a flow chat given in Fig. 1 while the description of the model parameters is given in Table 1. Further information on model description and their biological motivation can be find in [3].

We note that since the model in (2.1) monitors the population of all associated parameters and the state variables are non-negative, then we could observe that the first three equation of the system is independent
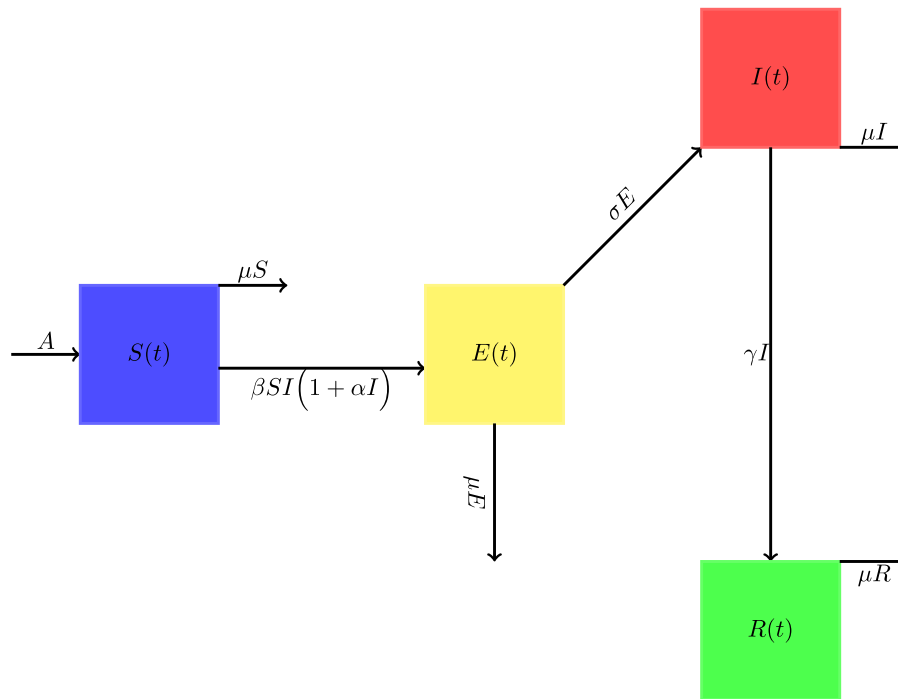
Fig. 1. Flowchart of an SEIR epidemic model with convex incidence rate without delay.

of the fourth. Therefore, without loss of generality, the system in (2.1) can be analysed by considering the subsystem:

$$\dot{S} = A - \mu S(t) - \beta S(t)I(t)(1 + \alpha I(t)),$$
$$\dot{E} = \beta S(t)I(t)(1 + \alpha I(t)) - (\mu + \sigma)E(t), \qquad (2.2)$$
$$\dot{I} = \sigma E(t) - (\mu + \gamma)I(t).$$

The type of system in (2.2) has been extensively studied in literatures where the analysis, stability and existence of hopf bifurcation are discussed. The most recent literature that presents the analysis of such a model can be found in [2].

2.2. SEIR with time delay

In the epidemic model, there has been an assumption that an individual reacts to the disease exposure almost immediately which is practically incorrect [2]. In several physical phenomena, there is often a time delay before a system responds to forces and this is no difference in disease transmission. For infectious diseases, a class of infectious individuals takes a time period before they could transmit a disease to the susceptible class after exposure. Also, it takes a certain interval for an infected individual in a population to recover from the disease and this can be accounted for with the inclusion of a delay term in the model. Therefore, to reflect the behaviour of a dynamic system more adequately, then it is highly desirable to incorporate a time delay into the system. As a result, much literature has adopted the inclusion of time delay into an existing model for a better representation of the system behaviour. For example, [43] extended the HIV model by incorporating a time delay which represents the time of initial infection until the production of new virions. In general, a delay is often introduced when there are some hidden processes within a system that could cause a time lag or whenever there is a pause before a response to stimuli [47]. Further literature where time delayed is extensively used in model formulation can be found in [48]. So, in terms of the epidemic model, there will be a more realistic description of disease dynamics by incorporating delays within the system which could also account for the effect of disease latency or immunity as identified in [49,50]. Therefore, we shall assume that the migration of the individual from the susceptible class to the infectious category is subject to delay. Hence, the model (2.1) is extended by incorporating a time delay represented by τ in the incidence rate which follows a similar argument in [2]. The flowchart is presented in Fig. 2 and the model is given in (2.3).

$$\dot{S} = A - \mu S(t) - \beta S(t - \tau)I(t - \tau)(1 + \alpha I(t - \tau)),$$
$$\dot{E} = \beta S(t - \tau)I(t - \tau)(1 + \alpha I(t - \tau)) - \mu E(t) - \sigma E(t - \tau), \qquad (2.3)$$
$$\dot{I} = \sigma E(t - \tau) - (\mu + \gamma)I(t),$$
$$\dot{R} = \gamma I(t) - \mu R(t),$$

where $\tau > 0$ is representing the latent period of the disease. The initial conditions for the system (2.3) are as follows:

$$\begin{cases} S(0) &= \phi_1(\theta) > 0, \\ E(0) &= \phi_2(\theta) \geq 0, \\ I(0) &= \phi_3(\theta) \geq 0, \\ R(0) &= \phi_4(\theta) \geq 0, \quad \theta \in [-\tau, 0]. \end{cases} \qquad (2.4)$$

Similarly, the first three equation of (2.3) does not depend on the fourth equation which allows us to analyse the system by considering the subsystem,

$$\dot{S} = A - \mu S(t) - \beta S(t - \tau)I(t - \tau)(1 + \alpha I(t - \tau)),$$
$$\dot{E} = \beta S(t - \tau)I(t - \tau)(1 + \alpha I(t - \tau)) - \mu E(t) - \sigma E(t - \tau), \qquad (2.5)$$
$$\dot{I} = \sigma E(t - \tau) - (\mu + \gamma)I(t).$$

Suppose $N = S + E + I$, then

$$\frac{d}{dt}(S + E + I) = A - \mu(S + E + I),$$
$$= A - \gamma I - \mu N,$$
$$\leq A - \mu N.$$

Hence, $\limsup_{t \to \infty} N \leq \frac{A}{\mu}$. Therefore, the dynamic of system (2.5) can be studied in the region,

$$\Omega = \{(S \ E \ I) \mid S > 0, E \geq 0, I \geq 0, 0 \leq N = S + E + I \leq A/\mu\}. \qquad (2.6)$$

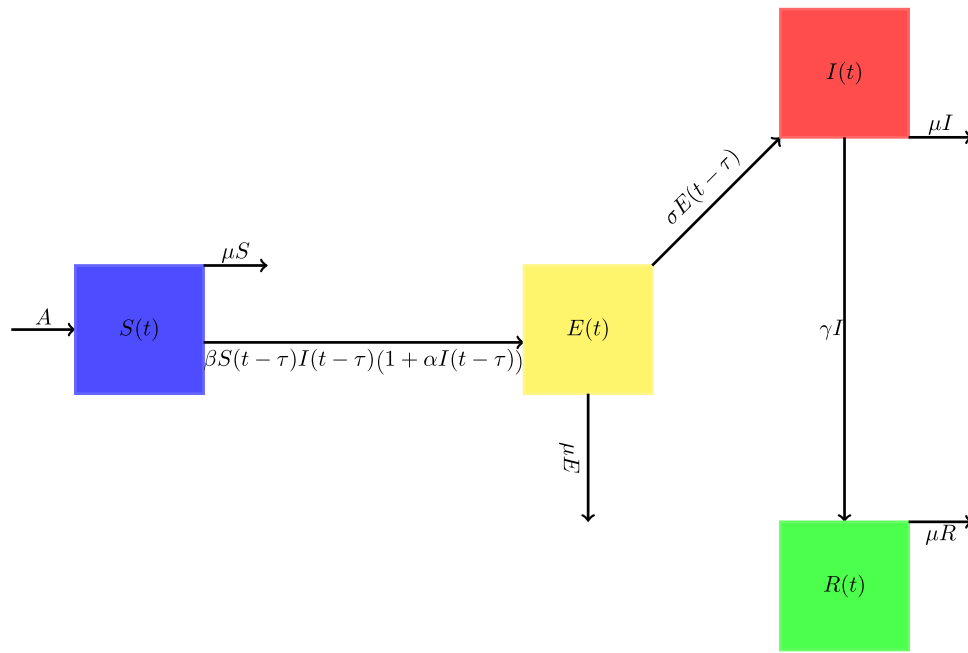which is positive invariant with respect to (2.5).

Fig. 2. Delayed SEIR epidemic model with convex incidence rate flow chart.

3. Equilibrium and stability analysis

In this section, we shall categorised the equilibrium points are the disease free equilibrium and the endemic equilibrium which are denoted by E^0 and E^1 respectively. It is well known that a system have a stable equilibrium if its neighbourhood trajectory approaches the point asymptotically at $t \to \infty$ and same is applicable to a system with a time delay. Thus, we obtain the equilibrium for the system (2.5) by setting $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = 0$ Therefore, we obtain the disease free equilibrium as $E^0 = (A/\mu, 0, 0)$ and for the endemic equilibrium, we have $E^1 = (S^*, E^*, I^*)$ where,

$$S^* = \frac{(\mu + \sigma)(\mu + \gamma)}{\beta\sigma(1 + \alpha I^*)},$$

$$E^* = \frac{(\mu + \gamma)I^*}{\sigma},$$

$$I^* = \frac{1}{2a}\left(\sqrt{b^2 - 4ac} - b\right),$$

and

$$\begin{cases} a &= \alpha\beta\sigma(\mu + \sigma)(\mu + \gamma) \\ b &= \beta\sigma(\mu + \sigma)(\mu + \gamma) - A\alpha\beta\sigma^2 \\ c &= \sigma\mu(\mu + \sigma)(\mu + \gamma) - A\sigma^2\beta \end{cases}$$

3.1. Expression of the reproductive number

We provide the expression of the basic reproductive number R_0 in this section. In epidemiology, the R_0 is considered as the most important parameter which provides an insight into how the disease spread within a population. This also enables us to understand how the disease can be controlled. There are multiple approaches for determining the R_0 such as by using a generation matrix as adopted by [42] or by direct substitution of parameters into the model. By using this approach, we obtain the reproductive number for our model to be

$$R_0 = \frac{A\sigma\beta}{\mu(\mu + \sigma)(\mu + \gamma)}, \tag{3.1}$$

which is consistent with the reproductive number obtained in [2]. Hence, by representing the endemic equilibrium in term of the reproductive number, we have

$$S^* = \frac{A}{\mu R_0(1 + \alpha I)},$$

$$E^* = \frac{(\mu + \gamma)I}{\sigma},$$

$$I^* = \frac{1}{2a}\left(\sqrt{b^2 - 4ac} - b\right),$$

with

$$\begin{cases} a &= \frac{A\alpha\beta^2\sigma^2}{\mu R_0} \\ b &= A\beta\sigma^2\left(\frac{\beta}{\mu R_0} - \alpha\right) \\ c &= A\beta\sigma^2\left(\frac{1}{R_0} - 1\right) \end{cases}$$

3.2. Stabilities of equilibria

We study the stability of the equilibrium through the linearisation of the system. We shall do this by defining $\hat{X} = (\hat{S}, \hat{E}, \hat{I})$ as the equilibrium of our delayed system (2.5). Then by setting $S(t) = x_1(t) + \hat{S}$, $E(t) = x_2(t) + \hat{E}$ and $I(t) = x_3(t) + \hat{I}$, then the linearised system of (2.5) is given as:

$$\frac{dx_1}{dt} = -\mu x_1(t) - \beta\hat{I}\left(1 + \alpha\hat{I}\right)x_1(t) - \beta\hat{S}\left(1 + \alpha\hat{I}\right)x_3(t) - \alpha\beta\hat{S}\hat{I}x_3(t - \tau),$$

$$\frac{dx_2}{dt} = \beta\hat{I}\left(1 + \alpha\hat{I}\right)x_1(t) + \beta\hat{S}\left(1 + \alpha\hat{I}\right)x_3(t) + \alpha\beta\hat{S}\hat{I}x_3(t - \tau)$$
$$\quad - \mu x_2(t) - \sigma x_2(t - \tau), \tag{3.2}$$

$$\frac{dx_3}{dt} = \sigma x_2(t - \tau) - (\mu + \gamma)x_3(t).$$

The Jacobian matrix corresponding to the linearised system (3.2) is obtained as:

$$\begin{pmatrix} -\mu - \beta\hat{I}\left(1 + \alpha\hat{I}\right) & 0 & -\beta\hat{S}\left(1 + \alpha\hat{I}(1 + e^{-\lambda\tau})\right) \\ \\ \beta\hat{I}(1 + \alpha\hat{I}) & -(\mu + \sigma e^{-\lambda\tau}) & \beta\hat{S}\left(1 + \alpha\hat{I}(1 + e^{-\lambda\tau})\right) \\ \\ 0 & \sigma e^{-\lambda\tau} & -(\mu + \gamma), \end{pmatrix} \tag{3.3}$$

with the characteristics equation given as

$$det \begin{pmatrix} -\mu - \beta\hat{I}(1+\alpha\hat{I}) - \lambda & 0 & -\beta\hat{S}\left(1+\alpha\hat{I}(1+e^{-\lambda\tau})\right) \\ \\ \beta\hat{I}(1+\alpha\hat{I}) & -(\mu+\sigma e^{-\lambda\tau})-\lambda & \beta\hat{S}\left(1+\alpha\hat{I}(1+e^{-\lambda\tau})\right) \\ \\ 0 & \sigma e^{-\lambda\tau} & -(\mu+\gamma)-\lambda \end{pmatrix}$$

$$= 0, \tag{3.4}$$

which leads to

$$\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3 + \left(q_1\lambda^2 + q_2\lambda + q_3\right)e^{-\lambda\tau} + \left(r_2\lambda + r_3\right)e^{-2\lambda\tau} = 0 \tag{3.5}$$

where

$$\begin{cases} p_1 &= 3\mu + \gamma + \beta\hat{I}\left(1+\alpha\hat{I}\right), \\ p_2 &= \mu(3\mu+2\gamma) + \beta\hat{I}(2\mu+\gamma)\left(1+\alpha\hat{I}\right), \\ p_2 &= \mu(\mu+\gamma)\left[\mu+\beta\hat{I}(1+\alpha\hat{I})\right], \\ q_1 &= \sigma, \\ q_2 &= \sigma(2\mu+\gamma), \\ q_3 &= \sigma\left[\mu(\mu+\gamma) + \beta\hat{I}\left(1+\alpha\hat{I}\right) - \mu\beta\hat{S}\left(1+\alpha\hat{I}\right)\right], \\ r_2 &= -\sigma\alpha\beta\hat{S}\hat{I}, \\ r_2 &= -\sigma\alpha\mu\hat{S}\hat{I}. \end{cases} \tag{3.6}$$

For the stability, it is well known that an equilibrium point is asymptotically stable if all the roots of its characteristics equation have a negative real parts. Therefore, for our system, we shall adopt the following lemma.

Lemma 3.1 ([2]). *The equilibrium point* (S^*, E^*, I^*) *of the delayed model is absolutely stable iff all root associated with the characteristics equation of the delayed model have negative real part and no purely imaginary root.*

Lemma 3.2 ([2]). *The equilibrium point* (S^*, E^*, I^*) *of the delayed model is conditionally stable iff all root associated with the characteristics equation of the delayed model have negative real part and there exists some positive value* τ *such that the associated characteristics equation of the delayed model has purely imaginary root.*

Hence, we shall these lemmas to study the stability of the disease free equilibrium E^0 and the endemic equilibrium E^1.

3.2.1. Stability of disease free equilibrium

Theorem 3.3. *The disease free equilibrium of the delayed model* (3.2) *is asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

Proof. Recall that $E^0 = (A/\mu, 0, 0)$ and by evaluating the Jacobian matrix of the linearised system at the E^0, we have

$$J(E^0) = \begin{pmatrix} -\mu & 0 & \frac{A\beta}{\mu} \\ \\ 0 & -\left(\mu+\sigma e^{-\lambda\tau}\right) & \frac{A\beta}{\mu} \\ \\ 0 & \sigma e^{-\lambda\tau} & -(\mu+\gamma) \end{pmatrix}$$

we then have the characteristics equation as:

$$(\lambda + \mu)f(\lambda) = 0 \tag{3.7}$$

where $\lambda = -\mu$ is one of the solution and $f(\lambda) = \lambda^2 + (2\mu + \gamma + \sigma e^{-\lambda\tau})\lambda + (\mu + \sigma e^{-\lambda\tau})(\mu+\gamma) - \frac{A\beta\sigma}{\mu}e^{-\lambda\tau}$.

If $\tau = 0$, then $f(\lambda) = \lambda^2 + (2\mu + \gamma + \sigma)\lambda + (\mu+\sigma)(\mu+\gamma)(1 - R_0)$. Since the model parameters are all positives, then for $R_0 < 1$, the roots $F(\lambda)$ have negative real parts which implies that it is asymptotically stable. Conversely, if $R_0 > 1$ then the all the real roots of $f(\lambda)$ will not be strictly negative. Therefore, it is unstable.

Similarly, when $\tau > 0$, then we consider the characteristics equation provided in (3.5) and by setting $\lambda = i\omega$, we have

$$p_3 - p_1\omega^2 + i(p_2\omega - \omega^3) - (q_1\omega^2 - q_3 - iq_2\omega)e^{-i\omega\tau} + (r_3 + ir_2\omega)e^{-2i\omega\tau} = 0 \tag{3.8}$$

by separating the real and the complex part of (3.8), we have

$$p_3 - p_1\omega^2 = (q_1\omega^2 - q_3)\cos(\omega\tau) - q_2\omega\sin(\omega\tau)$$
$$+ r_2\omega\sin(2\omega\tau) - r_3\cos(2\omega\tau)$$
$$p_2\omega - \omega^3 = (-q_1\omega^2 + q_3)\sin(\omega\tau) - q_2\omega\cos(\omega\tau)$$
$$- r_2\omega\cos(2\omega\tau) + r_3\sin(2\omega\tau)$$

which leads to

$$\omega^6 + (p_1 - 2p_2)\omega^4 + (p_2^2 - 2p_1p_3 + q_1 - q_2^2 - r^2)\omega^2 + p_3^2 - q_3 - r_3^2 = 0 \tag{3.9}$$

If $\xi = \omega^2$, then (3.9) becomes

$$\xi^3 + \alpha_1\xi^2 + \alpha_2\xi + \alpha_3 = 0 \tag{3.10}$$

where

$$\begin{cases} \alpha_1 &= p_1 - 2p_2, \\ \alpha_2 &= p_2^2 - 2p_1p_3 + q_1 - q_2^2 - r^2, \\ \alpha_3 &= p_3^2 - q_3 - r_3^2. \end{cases} \tag{3.11}$$

Thus, by substituting (3.6) into (3.11), we have $\alpha_1, \alpha_2, \alpha_3$ all positive for $R_0 < 1$. Therefore, it also follows that the roots of (3.10) have negative real parts which implies that it is asymptotically stable when $R_0 < 1$ and unstable whenever $R_0 > 1$. $\square$

3.2.2. Stability of endemic equilibrium

Theorem 3.4. *The endemic equilibrium of the delayed model* (3.2) *is asymptotically stable for* $R_0 > 1$.

Proof. Here, we shall consider the characteristics equation provided in (3.5) evaluated at E^1 which gives

$$\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3 + \left(q_1\lambda^2 + q_2\lambda + q_3\right)e^{-\lambda\tau} + \left(r_2\lambda + r_3\right)e^{-2\lambda\tau} = 0 \tag{3.12}$$

Then for $\tau = 0$, (3.12) reduces to

$$\lambda^3 + p_1\lambda^2 + p_2\lambda + p_3,$$

where p_1, p_2, p_3 are all positive and by Routh–Hurwitz criterion [51] for stability, all the roots of (3.12) have negative real part. Similarly, when $\tau > 0$, we let $\lambda = i\omega$, where $\omega > 0$ is taken as the root of (3.12) and by following the same approach adopted for the disease free equilibrium, we have that the roots of the characteristics equation has no positive real part and therefore, we can conclude that E^1 is asymptotically stable for $R_0 > 1$. $\square$

4. Numerical simulations and discussion

We calculate the numerical simulation for the model in (2.3) using the following set of parameters available in [2,4].

$$A = 10.7, \mu = 0.0062, \sigma = 0.6, \gamma = 0.9, \alpha = 0.0009$$

with these parameter values, the model becomes

$$\frac{dS}{dt} = 10.7 - 0.062S(t) - \beta S(t-\tau)I(t-\tau)(1+0.0009I(t-\tau)),$$
$$\frac{dE}{dt} = \beta S(t-\tau)I(t-\tau)(1+0.0009I(t)) - 0.062E(t) - 0.6E(t-\tau), \tag{4.1}$$
$$\frac{dI}{dt} = 0.6E(t-\tau) - 0.962I(t),$$
$$\frac{dR}{dt} = 0.9I(t) - 0.062R(t).$$

With the above parameters, we display the time series for the susceptible, exposed and infectious classes. From Fig. 3, we observed that there is an initial sharp decrease in the susceptible class which concise with
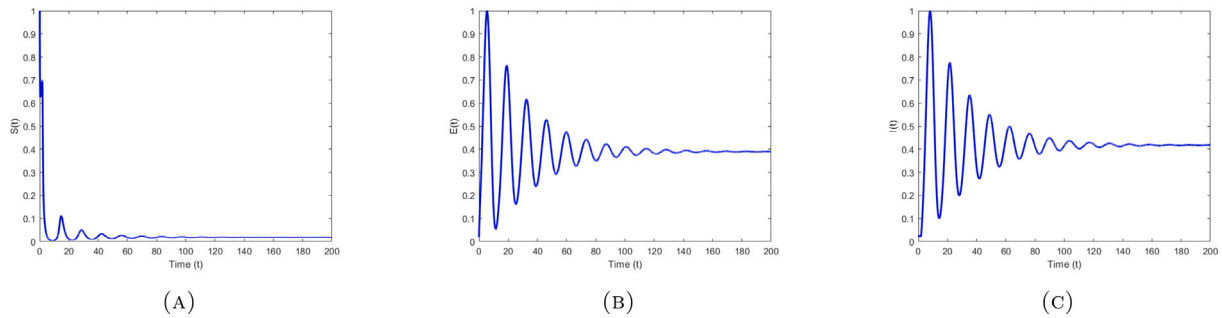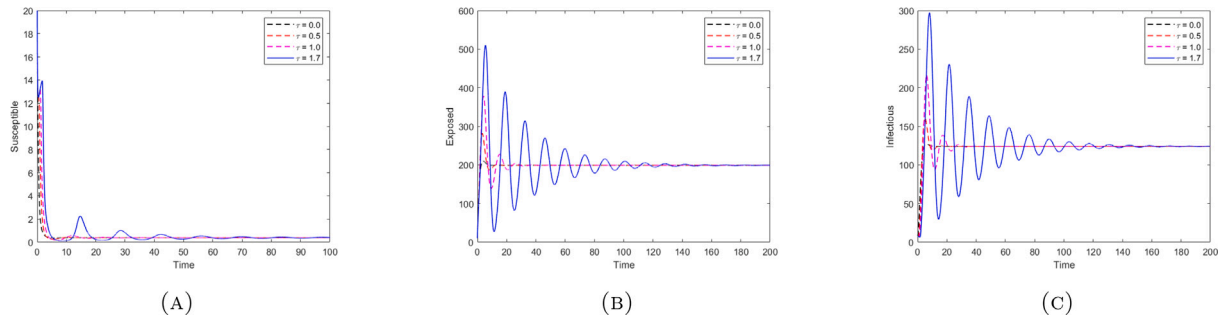
Fig. 3. Time series plot for $\tau = 1.70$.

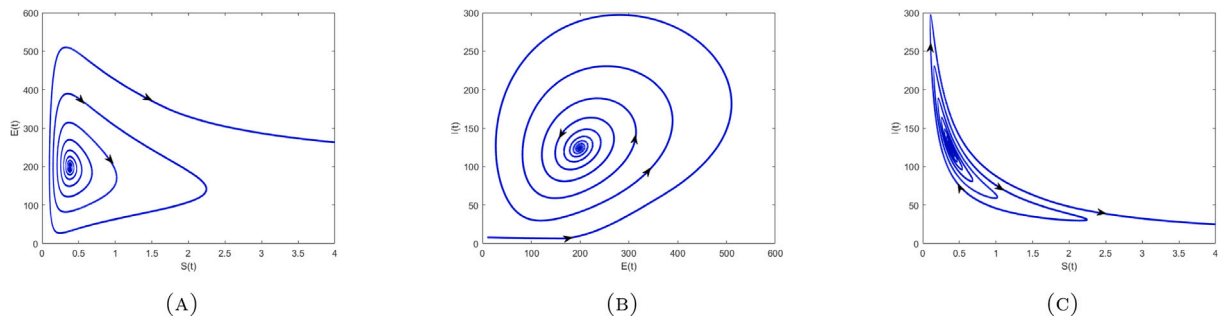**Fig. 4.** Effect of time delay τ on the model dynamics.

Fig. 5. Model phase plane.

the increase in the exposed and infectious group. The subsequent trend, therefore, shows that whenever there is an increase (or decrease) in the class of susceptible individuals, there is a decrease (or increase) in the exposed and infectious population.

Similarly, Fig. 3 also shows that the susceptible, exposed and infectious individuals exhibit an oscillatory behaviour which indicates that the population are randomly and well mixed with high interaction between the classes which leads to a rapid change in the behaviours. However, the system becomes stable over a long time which implies that at a future time, the population becomes less likely to be infected which could be attributed to the immunity upon the recovery from the diseases. Next, we shall examine the effect of the delay parameters on the model.

Fig. 4 shows the population behaviour for a large time delay and a small time delay. By examining the two figures, we could observe a more oscillatory behaviour and lesser stability observed for a large delay which implies that the bigger the delay within the system, the higher the complexity of the model behaviour which results in instability. Finally, the phase plane is displayed in Fig. 5 and a spiral behaviour could be observed most notably between the exposed and infected classes which indicated the level of interaction between the classes.

5. Conclusion

In this work, a delay SEIR model was investigated with a convex incidence rate incorporated with delays. The work shows the oscillatory behaviour of the model compartment when the population are assumed to be well mixed. By incorporating the delay into the system, we are able to provide a more realistic scenario that an individual does get the disease but not instantly which implies that a time period is required which was represented with the parameter τ. The model was analysed to understand the behaviour of the equilibrium state together with their dynamics. Thus, the result shows that when the delay parameter $\tau \leq 1.7$, the endemic equilibrium has locally asymptotic stability which indicates that the disease could be mitigated and will lead to a lower infectious class over a period. However, when τ passes through the critical value, then the endemic equilibrium losses its stability and a hopf bifurcation arise which indicates that the disease would be out of control. In this sense, it is necessary to judge the spread and model the dynamics with various parameters for proper supervision. It is therefore recommended that the level of exposure should be minimised to reduce the rate of infection.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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