

RESEARCH

Open Access



A study on COVID-19 transmission dynamics: stability analysis of SEIR model with Hopf bifurcation for effect of time delay

M. Radha¹ and S. Balamuralitharan^{1*}

*Correspondence:

balamurs@srmist.edu.in

¹Department of Mathematics, College of Engineering and Technology, SRM Institute of Science and Technology, SRM Nagar, Kattankulathur, 603203, Kanchipuram, Chennai TN, India

Abstract

This paper deals with a general SEIR model for the coronavirus disease 2019 (COVID-19) with the effect of time delay proposed. We get the stability theorems for the disease-free equilibrium and provide adequate situations of the COVID-19 transmission dynamics equilibrium of present and absent cases. A Hopf bifurcation parameter τ concerns the effects of time delay and we demonstrate that the locally asymptotic stability holds for the present equilibrium. The reproduction number is brief in less than or greater than one, and it effectively is controlling the COVID-19 infection outbreak and subsequently reveals insight into understanding the patterns of the flare-up. We have included eight parameters and the least square method allows us to estimate the initial values for the Indian COVID-19 pandemic from real-life data. It is one of India's current pandemic models that have been studied for the time being. This Covid19 SEIR model can apply with or without delay to all country's current pandemic region, after estimating parameter values from their data. The sensitivity of seven parameters has also been explored. The paper also examines the impact of immune response time delay and the importance of determining essential parameters such as the transmission rate using sensitivity indices analysis. The numerical experiment is calculated to illustrate the theoretical results.

MSC: 34D20; 34E05; 34K50

Keywords: Covid19 Indian pandemic; SEIR; Stability; Hopf bifurcation; Sensitivity parameters

1 Introduction

Globally COVID-19 coronavirus affects 235 countries and territories in which 31,561,520 peoples are affected and 970,688 died and recoverable populations 23,168,689. According to WHO (World Health Organization) data (on September 22, 2020), 5,562,663 have been affected and 88,935 people have died and 44,97,867 have been cured of diseases for the viruses in India. No mass transmission has taken place, and there is a possibility that it will peak in November and December 2020 and will start tapering slowly. Nevertheless, it will continue as an infectious virus, which will threaten the healthcare system by restoring in the future [1, 2]. The progressing COVID-19 episode, developed in Wuhan, China, has

© The Author(s) 2020. This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>.

concerned more than 2600 lives starting on 24 February 2020 and represented a colossal danger to worldwide general wellbeing [3–8]. It has actually required different methods adding current situations and uncommon medical clinics and travel limitations to relieve the infection. Here we have indicated that coronavirus ailment has been engendered in the network of China quickly by revealed information and the Government of China make important strides, for example, occasion augmentation, travel limitation, hospitalization and isolation. These vital limitations have been useful to diminish the infection transmission among the populace and this is legitimized by information results [9–15]. It is earnest to give increasingly logical data to a superior comprehension of the novel coronavirus and promote control of the outbreak [16].

At the beginning of the time episode, it was dispersed, and connected to market places [17]. It has received outrageous measures to relieve flare-up. On 10 March 2020, the neighborhood legislature of Wuhan controlled every open traffic inside the city and shut all inbound and outbound transportation [18–24]. Muhammad Altaf Khan and Abdon Atangana discuss the model that the interaction between the bats and unidentified hosts are the reservoir of infections (seafood market) and the neighborhoods of people. The key cause of the infection is labeled seafood. The buying of goods from people's seafood markets will infect asymptotically or symptomatically. They reduced the model on the premise that the market in seafood has a sufficient source of infection to infect people [25]. Lim et al. discussed the spread of COVID-19 in South Korea through secondary transmission from the people who traveled from China [26]. Hu et al. revealed the potential for asymptomatic transmission by COVID-19 by examining the medical features of 24 asymptomatic patients developing near-contact infection [27]. The open frenzy progressing COVID-19 episode helps us in the records to remember the 1920 flu pandemic in London, United Kingdom. Besides, its attributes mellow side effects as a rule and short sequential interim are like pandemic flu, as opposed to the next two coronaviruses [28]. In 1918, critical extents of the passing were from pneumonia followed by flu contamination [29]. Hence, it may be sensible to return to the demonstrating system of the 1918 flu pandemic, and specifically, to catch the impacts of the individual response and government activity. We assume it will keep going for the following not many days for the occasion and shall refresh. The variables estimation might be evaluated after data have become accessible [30–33]. It contends all avoidance and limiting cases might be ordered up to three huge gatherings, that are portrayed as stage work and reaction work, separately. They likewise consider a COVID-19 transmission time of 14 days and gigantic resettlement from China [34]. A contact is an individual who encountered any of the accompanying exposures during the 2 days prior and the 14 days after the beginning of side effects of a plausible or affirmed case [35].

The median time before the onset of symptoms is 3 days, the shortest is 1 day, and the longest 24 days as recorded. These intervals have a significant role to play in understanding the dynamics of COVID-19 transmission. The mathematical model by Abdon Atangana agrees with its lock-down performance. The harmful effects of inadequate testing should be stated. The asymptomatic person tested may be good and spread the infection or may reach the virus within days after testing, and after the contradictory results the disease may spread further [36].

For COVID-19 among the human population and its stability we have proposed a SEIR pandemic model. Another scientific model in pestilence elements, known as the Ware-

house theory, generally has been discussed for quite a while since it was proposed by Kermack and McKendrick in 1927. It incorporates a few essential improved models, for example, SIR, SIS, and SEIR, among which SEIR is an ordinary model that takes the incubation period into account. Giordano et al’s findings show that the ongoing COVID-19 pandemic involves the combination of restrictive social-discriminatory behavior with widespread testing and contact monitoring. For the Italian COVID-19 epidemic, they estimate model parameters based on data dates from 20 February 2020 (day 1) to 5 April 2020 (day 46) and demonstrate the effect on the spread of the epidemic of progressive restrictions like the latest lock-down, slowly enforced as of 9 March 2020. They also model possible longer-term scenarios that show the impacts of various countermeasures, such as social separation and population-wide SARS-CoV-2 testing. The asymptomatic case is usually not reported to medical authorities as mentioned, and reported cases are typically only a fraction of the total number of symptomatic infectious people. The number of asymptomatic infectious cases and non-confirmed infectious cases and the number of recorded COVID cases in mainland China are discussed in this paper. The disparity between those diagnosed and those not diagnosed is significant. The former are being isolated usually and thus less likely is the infection to spread. This also helps clarify this descent. We mention the fatality rate and propagation of the disease misconception [37].

The SEIR, a widely utilized scourge model, can show the progressions of individuals between four states: Susceptible (S) (population not resistant to illness), Exposed (E) (population as of now in brooding), Infectious (I) (number of contamination effectively circling), and Recovered (R) (population not at this point irresistible because of confinement or in susceptibility or full recuperation). Here the population total size at time t is defined by $N(t)$, with $N(t) = S(t)+E(t)+I(t)+R(t)$. This system is portrayed by accompanying the nonlinear differential equations for the Indian current pandemic COVID19 [38]:

$$\begin{cases} \frac{dS(t)}{dt} = b + \gamma I(t) - (\mu + p)S(t) - \beta S(t)I(t), \\ \frac{dE(t)}{dt} = \beta S(t)I(t) - \mu E(t) - \eta E(t)R(t), \\ \frac{dI(t)}{dt} = (\eta + \sigma \beta)E(t) - (\alpha + \mu + \gamma)I(t), \\ \frac{dR(t)}{dt} = pS(t)E(t)R(t) - (\mu + \sigma \beta)R(t). \end{cases} \tag{1}$$

The parameters $p, b, \gamma, \beta, \mu, \eta, \sigma, \alpha$ are positive constants, p is the proportion of asymptomatic infection, b is the birth rate of people while newborn cells are created, γ is the incubation period of human infection, β is the transmission rate from one compartment to another compartment, μ is the death rate of people, η is the infectious period of symptomatic infection of people, σ is the infectious period of asymptomatic infection of people, α is the multiple of the transmissibility while infected cells are created from the viruses.

Transmission dynamics generating COVID-19 may require a duration of time delay τ , i.e. the delay of the immune system at time (days) t may be governed on the previous time $t - \tau$. We obtain an immune response of length of incubation period, $pS(t)E(t)R(t) = pS(t - \tau)E(t - \tau)R(t - \tau)$ and the duration of the patient being infectious. Tian-Mu Chen et al. [39] investigated the effect of including time delay to acquire the following nonlinear

differential equations:

$$\begin{cases} \frac{dS(t)}{dt} = b + \gamma I(t) + \epsilon R(t) - (\mu + p)S(t) - \beta S(t)I(t), \\ \frac{dE(t)}{dt} = \beta S(t)I(t) - \mu E(t) - \eta E(t)R(t), \\ \frac{dI(t)}{dt} = (\eta + \sigma \beta)E(t) - (\alpha + \mu + \gamma)I(t), \\ \frac{dR(t)}{dt} = pS(t - \tau)E(t - \tau)R(t - \tau) - (\epsilon + \mu + \sigma \beta)R(t), \end{cases} \tag{2}$$

where ϵ is the latent period of human infection in population no longer infectious due to being fully recovered. The aim of the research work is to discuss the SEIR delay model in (2). If $\tau = 0$, Eq. (2) narrates the population inputs between size of population and number of initial infections. The COVID-19 basic reproduction number for the system (2) is defined by

$$R_0 = \frac{b\beta(\eta + \sigma\beta)}{\mu(\mu + p)(\alpha + \mu + \gamma)}.$$

We have likewise determined the basic reproduction number R_0 classical SIR model and we have seen that if $R_0 < 1$ the disease does not proliferate into the population yet on the off chance that $R_0 > 1$ infection will spread among the population. We presented an isolated SIR model and SEIR model portraying disease movement under the presumption that all contaminated individuals are separated after the hatching time frame so that they cannot taint others [40]. Ailment movement in these models is controlled by the basic reproduction number R_0 , which is different from that for the standard SIR model. In the event that $R_0 > 1$ (95%, ranges 1.4 to 3.9), at that point the quantity of inertness contaminated people exponentially develops. Be that as it may, if $R_0 < 1$, at that point the number of contaminated behaves exponentially. This investigation of R_0 catches the course of COVID-19 flare-up and subsequently reveals insight and understanding of the patterns of the flare-up and gives some preventive, measure not to spread the COVID-19 malady (97%, ranges 2.47 to 3.9). This portrays the normal number of recently contaminated cells produced from one tainted cell toward the start of the irresistible procedure. The current scenario will evolve to account for these continuing advancements as new drugs and vaccines are being developed and tested [41].

2 Preliminaries

Let $S(t) = C([-\tau, 0]; \mathbb{R})$ be the continuous norm function of Banach space mappings. The initial conditions for the model (2) are given as follows:

$$\begin{cases} S(t) \geq 0, & E(t) \geq 0, & I(t) \geq 0, & R(t) \geq 0, & t \in [-\tau, 0], \\ S(0) > 0, & E(0) > 0, & I(0) > 0, & R(0) > 0. \end{cases} \tag{3}$$

Let $(S(t), E(t), R(t))$ be three main variables of the system with initial conditions and verify that there is a unique solution. The accompanying lemma is helpful for examining the positivity of the bounded solutions.

Lemma 2.1 *In the system $(S(t), E(t), R(t))$ of (2) with initial conditions (3), we state that*

$$\limsup S(t)_{t \rightarrow +\infty} \leq \frac{b}{\mu + p}.$$

Proof If there is $t_1 > 0$ with the end goal that $S(t_1) > \frac{b}{\mu+p}$ and $\dot{S}(t_1) > 0$, then we have

$$\dot{S}(t_1) = b - (\mu + p)S(t) - \beta S(t)I(t) \leq -\beta S(t)I(t) \leq 0.$$

Hence we have utilized $S(t_1) > \frac{b}{\mu+p}$. This is an inconsistency to $\dot{S}(t_1) > 0$. Along these lines, Lemma 2.1 is verified. \square

Lemma 2.2 *Let $(S(t), E(t), I(t), R(t))$ be the system (2) with initial conditions (3). At that point $(S(t), E(t), I(t))$ and $R(t)$ are certain and there exists a positive constant $\Gamma > 0$, to such an extent that $S(t) < \Gamma, E(t) < \Gamma, I(t) < \Gamma$ and $R(t) < \Gamma$ at an adequately huge time t .*

Proof Considering Eq. (2), we get

$$\begin{aligned} S(t) &= S(0)e^{-\int_0^t ((\mu+p)-\beta I(\epsilon))d\epsilon} + \int_0^t be^{-\int_\eta^t ((\mu+p)-\beta I(\epsilon))d\epsilon} d\eta, \\ E(t) &= E(0)e^{-\int_0^t (\mu+\eta R(\epsilon))d\epsilon} + \int_0^t \beta S(\eta)I(\eta)e^{-\int_0^t (\mu+\eta R(\epsilon))d\epsilon} d\eta, \\ I(t) &= I(0)e^{-(\mu+\alpha+\gamma)t} + \int_0^t (\eta + \beta\sigma)E(\eta)e^{-(\mu+\alpha+\gamma)(t-\eta)} d\eta, \\ R(t) &= R(0)e^{-(\sigma\beta+\epsilon+\mu)t} + \int_0^t pS(\eta - \tau)E(\eta - \tau)R(\eta - \tau)e^{(\sigma\beta+\epsilon+\mu)t} d\eta. \end{aligned}$$

It is anything but difficult to see that $S(t)$ is positive on the existence interval. At that point, we demonstrate that $E(t)$ is positive. Truth be told, let $t_1 > 0$ be the first run through to such an extent that $E(t_1) = 0$. From Eq. (2), we get

$$I(t) = I(0)e^{-(\mu+\alpha+\gamma)t_1} + \int_0^{t_1} (\eta + \sigma\beta)E(\eta)e^{-(\mu+\alpha+\gamma)(t-\eta)} d\eta > 0.$$

Then again, from the second equation of (2), we have $\dot{E}(t_1) = \beta S(t_1)I(t_1) > 0$. This implies $E(t) < 0$ for $t \in (t_1 - \xi, t_1)$, where ξ is a subjectively small positive constant, which prompts an inconsistency. It is follows that $E(t) > 0$ and $I(t) > 0$. By the comparative contention as mentioned above, it is difficult to see that $R(t)$ is positive. Here, we discuss the contentions for an extreme solution of (2).

Here $N(t) = S(t) + E(t) + (\frac{\mu}{2(\eta+\sigma\beta)})I(t) + (\frac{\eta(\mu+p)}{pb})R(t + \tau)$, and we assume $q = \min\{(\mu + p), \frac{\mu}{2}, (\alpha + \mu + \gamma), (\epsilon + \mu + \sigma\beta)\}$.

From (2), we get

$$\begin{aligned} \frac{d}{dt}[N(t)] &= b - (\mu + p)S(t) - \left(\frac{\mu}{2}\right)E(t) - \eta E(t)R(t) - \left(\frac{\mu(\alpha + \mu + \gamma)}{2(\eta + \sigma\beta)}\right)I(t) \\ &\quad + \left(\frac{\eta(\mu + p)}{b}\right)S(t)E(t)R(t) - \left(\frac{\eta(\epsilon + \mu + \sigma\beta)(\mu + p)}{pb}\right)R(t + \tau) \\ &\leq b - (\mu + p)S(t) - \left(\frac{\mu}{2}\right)E(t) - \left(\frac{\mu(\alpha + \mu + \gamma)}{2(\eta + \sigma\beta)}\right)I(t) \\ &\quad - \left(\frac{\eta(\epsilon + \mu + \sigma\beta)(\mu + p)}{pb}\right)R(t + \tau) \end{aligned}$$

$$\begin{aligned}
 &< b - q \left[S(t) + E(t) + \left(\frac{\mu}{2(\eta + \sigma\beta)} \right) I(t) + \left(\frac{\eta(\mu + p)}{pb} \right) R(t + \tau) \right] \\
 &= b - qN.
 \end{aligned}$$

Along these lines, $N(t) < \frac{b}{(\mu+p)\eta}$ for all large t . Subsequently, $S(t), E(t), I(t)$ and $R(t)$ are at last limited by any positive constant Γ . Hence, we finish the verification of Lemma 2.2. \square

3 Theorems for stability analysis

There are three equilibria for system (2):

- (i) COVID-19 infection free equilibrium: $E_0 = (\frac{b}{(\mu+p)}, 0, 0, 0)$.
- (ii) COVID-19 infection absent equilibrium:
 $E_1 = (\frac{\mu(\alpha+\mu+\gamma)}{\beta}(\eta + \sigma\beta), \frac{b\beta(\eta+\sigma\beta)-\mu(\mu+p)(\alpha+\mu+\gamma)}{\mu\beta(\eta+\sigma\beta)}, \frac{b\beta(\eta+\sigma\beta)-\mu(\mu+p)(\alpha+\mu+\gamma)}{\mu\beta(\alpha+\mu+\gamma)}, 0)$.
- (iii) COVID-19 infection present equilibrium: $\bar{E} = (\bar{E}_1, \bar{E}_2, \bar{E}_3, \bar{E}_4)$,

where

$$\begin{aligned}
 \bar{E}_1 &= \frac{p(\alpha+\mu+\gamma)b-\beta(\eta+\sigma\beta)(\epsilon+\mu+\sigma\beta)}{p(\mu+p)(\alpha+\mu+\gamma)}, \bar{E}_2 = \frac{(\epsilon+\mu+\sigma\beta)(\mu+p)(\alpha+\mu+\gamma)}{p(\alpha+\mu+\gamma)b-\beta(\eta+\sigma\beta)(\epsilon+\mu+\sigma\beta)}, \\
 \bar{E}_3 &= \frac{(\eta+\sigma\beta)(\epsilon+\mu+\sigma\beta)(\mu+p)}{p(\alpha+\mu+\gamma)b-\beta(\eta+\sigma\beta)(\epsilon+\mu+\sigma\beta)}, \bar{E}_4 = \frac{1}{\eta} \left[\frac{\beta(\eta+\sigma\beta)(p(\alpha+\mu+\gamma)b-\beta(\eta+\sigma\beta)(\epsilon+\mu+\sigma\beta))}{p(\mu+p)(\alpha+\mu+\gamma)^2} - \mu \right].
 \end{aligned}$$

3.1 Stability of COVID-19 infection free equilibrium

The nonlinear differential equation of (2) at the point E_0 is

$$\begin{cases} \frac{dS(t)}{dt} = -(\mu + p)S(t) - \frac{\beta b}{(\mu+p)}I(t), \\ \frac{dE(t)}{dt} = -\mu E(t) + \frac{\beta b}{(\mu+p)}I(t), \\ \frac{dI(t)}{dt} = (\eta + \sigma\beta)E(t) - (\alpha + \mu + \gamma)I(t), \\ \frac{dR(t)}{dt} = -(\epsilon + \mu + \sigma\beta)R(t). \end{cases} \tag{4}$$

The polynomial equation for (4) is

$$\begin{aligned}
 &[\lambda + (\epsilon + \mu + \sigma\beta)][\lambda + (\mu + p)] \\
 &\left[\lambda^2 + (\mu + (\alpha + \mu + \gamma))\lambda + \mu(\alpha + \mu + \gamma) - \frac{(\eta + \sigma\beta)\beta b}{(\mu + p)} \right] = 0.
 \end{aligned} \tag{5}$$

Two of the roots of the polynomial equation (5) are $\lambda_1 = -(\epsilon + \mu + \sigma\beta)$, $\lambda_2 = -(\mu + p)$. The other roots are calculated by

$$\lambda^2 + (\mu + (\alpha + \mu + \gamma))\lambda + \mu(\alpha + \mu + \gamma) - \frac{(\eta + \sigma\beta)\beta b}{(\mu + p)} = 0. \tag{6}$$

If $R_0 < 1$, then $\mu(\alpha + \mu + \gamma) - \frac{(\eta+\sigma\beta)\beta b}{(\mu+p)} > 0$, and $(\mu + (\alpha + \mu + \gamma))^2 - 4(\mu(\alpha + \mu + \gamma) - \frac{(\eta+\sigma\beta)\beta b}{(\mu+p)}) > 0$. We have

$$\lambda_{3,4} = \frac{-(\mu + (\alpha + \mu + \gamma)) \pm \sqrt{(\mu + (\alpha + \mu + \gamma))^2 - 4(\mu(\alpha + \mu + \gamma) - \frac{(\eta+\sigma\beta)\beta b}{(\mu+p)})}}{2}.$$

Equation (6) has negative real roots. It obeys the accompanying theorem. If $R_0 < 1$, then E_0 is seen to be locally asymptotic stable by developing a Lyapunov functional. If $R_0 > 1$, then E_0 is unstable.

Theorem 3.1 *If $R_0 < 1$, then prove that E_0 is globally asymptotic stable.*

Proof For the Lyapunov functional

$$V = \frac{1}{2} \left[S(t) - \frac{(\epsilon + \mu + \sigma\beta)}{(\mu + \eta)} \right]^2 + \frac{(\epsilon + \mu + \sigma\beta)}{(\mu + \eta)} E(t) + mI(t) + \frac{\eta}{p} R(t) + \eta \int_{t-\tau}^t S(\theta)E(\theta)R(\theta) d\theta,$$

where $m > 0$, we have

$$V'|_{(5)} = \left[S(t) - \frac{b}{(\mu + p)} \right] \left[-(\mu + p) \left(S(t) - \frac{b}{(\mu + p)} \right) - \beta S(t)I(t) \right] + \frac{b}{(\mu + p)} [\beta S(t)I(t) - \mu E(t) - \eta E(t)R(t)] + m [(\eta + \sigma\beta)E(t) - (\alpha + \mu + \gamma)I(t)] - \left(\frac{\eta(\epsilon + \mu + \sigma\beta)}{p} \right) R(t) + \eta S(t)E(t)R(t).$$

Since $\beta S(t)I(t) = \beta I(t) \left[S(t) - \frac{b}{(\mu + p)} \right] + \frac{\beta b}{(\mu + p)} I(t)$, we have

$$V'|_{(5)} = -(\mu + p) \left[S(t) - \frac{b}{(\mu + p)} \right]^2 - \beta I(t) \left[S(t) - \frac{b}{(\mu + p)} \right]^2 + \eta S(t)R(t) \left[S(t) - \frac{b}{(\mu + p)} \right] - \left[\frac{b\mu}{(\mu + p)} - (\eta + \sigma\beta)m \right] E(t) - \left[(\alpha + \mu + \gamma)m - \frac{\beta b^2}{(\mu + p)^2} \right] I(t) - \left[\frac{\eta(\epsilon + \mu + \sigma\beta)}{p} \right] R(t).$$

Hence $R_0 < 1$ decreases to $\frac{b\mu}{(\eta + \sigma\beta)(\mu + p)} - \frac{\beta b^2}{(\alpha + \mu + \gamma)(\mu + p)^2} > 0$, ($m \in [\frac{\beta b^2}{(\alpha + \mu + \gamma)(\mu + p)^2}, \frac{b\mu}{(\eta + \sigma\beta)(\mu + p)}]$), such that $\frac{b\mu}{(\mu + p)} - (\eta + \sigma\beta)m > 0$ and $(\alpha + \mu + \gamma)m - \frac{\beta b^2}{(\mu + p)^2} > 0$.

Letting $S(t), E(t), R(t)$ be positive and $S(t) \leq \frac{b}{(\mu + p)}$ holds, we have $V'|_{(5)} \leq 0$, and $V'|_{(5)} = 0$ iff $(S(t), E(t), I(t), R(t)) = (\frac{b}{(\mu + p)}, 0, 0, 0)$. □

3.2 Stability of COVID-19 infection absent equilibrium

Letting $E_1 = (\tilde{S}, \tilde{E}, \tilde{I}, 0) = (\frac{\mu(\alpha + \mu + \gamma)}{\beta(\eta + \sigma\beta)}, \frac{b\beta - \mu(\mu + p)(\alpha + \mu + \gamma)}{\mu\beta(\alpha + \mu + \gamma)}, \frac{b\beta(\eta + \sigma\beta)}{\mu\beta(\alpha + \mu + \gamma)}, 0)$, the linearized form of equations of system (2) at E_1 is

$$\begin{cases} \frac{dS(t)}{dt} = -((\mu + p) + \beta\tilde{I})S(t) - \beta\tilde{S}I(t), \\ \frac{dE(t)}{dt} = \beta\tilde{I}S(t) - \mu E(t) + \beta\tilde{S}I(t) - \eta\tilde{E}R(t), \\ \frac{dI(t)}{dt} = (\eta + \sigma\beta)E(t) - (\alpha + \mu + \gamma)I(t), \\ \frac{dR(t)}{dt} = p\tilde{S}\tilde{E}R(t - \tau) - (\epsilon + \mu + \sigma\beta)R(t). \end{cases} \tag{7}$$

The characteristic polynomial equation of (7) is

$$(\lambda - p\tilde{S}\tilde{E}e^{-\lambda\tau} + (\epsilon + \mu + \sigma\beta))(\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3) = 0,$$

where

$$\begin{aligned}
 a_1 &= \mu + (\alpha + \mu + \gamma) + (\mu + p) + \frac{b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma)}{\mu(\alpha + \mu + \gamma)}, \\
 a_2 &= (\mu + (\alpha + \mu + \gamma)) \left((\mu + p) + \frac{b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma)}{\mu(\alpha + \mu + \gamma)} \right), \\
 a_3 &= b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma).
 \end{aligned}$$

First we obtain

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0. \tag{8}$$

Clearly, if $R_0 > 1$, we have $a_1 = \mu + (\alpha + \mu + \gamma) + (\mu + p) + \frac{b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma)}{\mu(\alpha + \mu + \gamma)} > 0$ and

$$\begin{aligned}
 a_3 &= b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma), \\
 a_1a_2 - a_3 &= \left(\mu + (\alpha + \mu + \gamma) + (\mu + p) + \frac{b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma)}{\mu(\alpha + \mu + \gamma)} \right) \\
 &\quad \times (\mu + (\alpha + \mu + \gamma)) \\
 &\quad \times \left((\mu + p) + \frac{b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma)}{\mu(\alpha + \mu + \gamma)} \right) \\
 &\quad - (b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma)) \\
 &= \mu^2(\mu + p) + \frac{\mu(b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma))}{(\alpha + \mu + \gamma)} + \mu(\mu + p)(\alpha + \mu + \gamma) \\
 &\quad + \left((\alpha + \mu + \gamma) + (\mu + p) + \frac{b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma)}{\mu(\alpha + \mu + \gamma)} \right) \\
 &\quad \times \left(\mu(\mu + p) + \frac{b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma)}{\mu(\alpha + \mu + \gamma)} \right) \\
 &\quad + (\alpha + \mu + \gamma)(\mu + p) + \frac{b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma)}{\mu(\alpha + \mu + \gamma)} > 0.
 \end{aligned}$$

By the Routh–Hurwitz criteria, (8) has no positive roots. So, we investigate the other polynomial equation

$$\lambda - p\tilde{S}\tilde{E}e^{-\lambda\tau} + (\epsilon + \mu + \sigma\beta) = 0. \tag{9}$$

For $\tau = 0$, $\lambda = \frac{\beta(\eta + \sigma\beta)p(\alpha + \mu + \gamma)b - \beta^2(\eta + \sigma\beta)^2(\epsilon + \mu + \sigma\beta) - \mu p(\mu + p)(\alpha + \mu + \gamma)^2}{\beta^2(\eta + \sigma\beta)^2}$. Obviously, if $R_0 < 1 + \frac{\beta^2(\eta + \sigma\beta)^2(\epsilon + \mu + \sigma\beta)}{\mu p(\mu + p)(\alpha + \mu + \gamma)^2}$, then $\phi < 0$, which illustrates the roots of (9) for some $\phi > 0$ and $\tau > 0$. From (9), we have

$$\begin{cases} \phi = -\frac{(\alpha + \mu + \gamma)p(b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma))}{\beta^2(\eta + \sigma\beta)^2} \sin \phi\tau, \\ (\epsilon + \mu + \sigma\beta) = \frac{(\alpha + \mu + \gamma)p(s\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma))}{\beta^2k^2} \cos \phi\tau, \end{cases} \tag{10}$$

which implies that $\phi^2 = p^2 \left[\frac{(\alpha + \mu + \gamma)p(b\beta(\eta + \sigma\beta) - \mu(\mu + p)(\alpha + \mu + \gamma))}{\beta^2(\eta + \sigma\beta)^2} \right]^2 - (\epsilon + \mu + \sigma\beta)^2$.

Note that if $1 < R_0 < 1 + \frac{\beta^2(\eta+\sigma\beta)^2(\epsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$, then $\phi^2 < 0$. If $1 < R_0 < 1 + \frac{\beta^2(\eta+\sigma\beta)^2(\epsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$, then the COVID-19 infection E_1 is locally asymptotic stable. If $1 < R_0 > 1 + \frac{\beta^2(\eta+\sigma\beta)^2(\epsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$, then the COVID-19 infection E_1 is unstable.

3.3 Stability of COVID-19 infection present equilibrium

In COVID-19 infection, the effects of time delay τ is a bifurcation parameter and it goes through a stationary values. The COVID-19-present equilibrium occurs direct stability and Hopf bifurcation. As a matter of first importance, we interpret the equilibrium $\bar{E} = (\bar{S}, \bar{E}, \bar{I}, \bar{R})$ of system (2) to the source. Let $S_1(t) = S(t) - \bar{S}, E_1(t) = E(t) - \bar{E}, I_1(t) = I(t) - \bar{I}, R_1(t) = R(t) - \bar{R}$. For effortlessness, we likewise use $S(t), E(t), I(t), R(t)$ rather than $S_1(t), E_1(t), I_1(t), R_1(t)$. The system (2) becomes

$$\begin{cases} \frac{dS(t)}{dt} = -((\mu + p) + \beta\bar{I})S(t) - \beta S(t)I(t) - \beta\bar{S}I(t), \\ \frac{dE(t)}{dt} = \beta S(t)I(t) + \beta\bar{I}S(t) - (\mu + \eta\bar{R})E(t) + \beta\bar{S}I(t) - \eta\bar{E}R(t) - \eta E(t)R(t), \\ \frac{dI(t)}{dt} = (\eta + \sigma\beta)E(t) - (\alpha + \mu + \gamma)I(t), \\ \frac{dR(t)}{dt} = pS(t - \tau)E(t - \tau)R(t - \tau) + p\bar{E}S(t - \tau)R(t - \tau) + p\bar{S}E(t - \tau)R(t - \tau) \\ \quad + p\bar{S}\bar{E}R(t - \tau) + p\bar{R}S(t - \tau)E(t - \tau) + p\bar{E}\bar{R}S(t - \tau) + p\bar{S}\bar{R}E(t - \tau) \\ \quad - (\epsilon + \mu + \sigma\beta)R(t). \end{cases} \tag{11}$$

Then the origin $(0, 0, 0, 0)^T$ is a steady state of (11) and the linearized system of equation (11) at the origin is

$$\begin{cases} \frac{dS(t)}{dt} = -((\mu + p) + \beta\bar{I})S(t) - \beta S(t)I(t) - \beta\bar{S}I(t), \\ \frac{dE(t)}{dt} = \beta S(t)I(t) + \beta\bar{I}S(t) - (\mu + \eta\bar{R})E(t) + \beta\bar{S}I(t) - \eta\bar{E}R(t) - \eta E(t)R(t), \\ \frac{dI(t)}{dt} = (\eta + \sigma\beta)E(t) - (\alpha + \mu + \gamma)I(t), \\ \frac{dR(t)}{dt} = pS(t - \tau)E(t - \tau)R(t - \tau) + p\bar{E}S(t - \tau)R(t - \tau) + p\bar{S}E(t - \tau)R(t - \tau) \\ \quad + p\bar{S}\bar{E}R(t - \tau) + p\bar{R}S(t - \tau)E(t - \tau) + p\bar{E}\bar{R}S(t - \tau) + p\bar{S}\bar{R}E(t - \tau) \\ \quad - (\epsilon + \mu + \sigma\beta)R(t). \end{cases} \tag{12}$$

The trivial solution of Eq. (12) is asymptotic stable and Eq. (11) is locally asymptotic stable. The strength of the polynomial equation (12) is given by

$$\Omega(\lambda) = \lambda^4 + x_1\lambda^3 + x_2\lambda^2 + x_3\lambda + x_4 - (x_5\lambda^3 + x_6\lambda^2 + x_7\lambda + x_8)e^{-\lambda\tau}, \tag{13}$$

where

$$\begin{aligned} x_1 &= (\epsilon + \mu + \sigma\beta) + (\mu + p) + \beta\bar{I} + (\alpha + \mu + \gamma) + \mu + \eta\bar{R}, \\ x_2 &= (\epsilon + \mu + \sigma\beta)(\mu + p) + (\epsilon + \mu + \sigma\beta)\beta\bar{I} + (\epsilon + \mu + \sigma\beta)(\alpha + \mu + \gamma) \\ &\quad + (\mu + p)(\alpha + \mu + \gamma) + \beta\bar{I}(\alpha + \mu + \gamma) + (\mu + \eta\bar{R})((\epsilon + \mu + \sigma\beta) + (\mu + p) + \beta\bar{I}), \\ x_3 &= (\epsilon + \mu + \sigma\beta)(\mu + p)(\alpha + \mu + \gamma) + (\epsilon + \mu + \sigma\beta)\beta\bar{I}(\alpha + \mu + \gamma) \\ &\quad + (\mu + \eta\bar{R})((\epsilon + \mu + \sigma\beta)(\mu + p) + (\epsilon + \mu + \sigma\beta)\beta\bar{I}) + (\eta + \sigma\beta)\beta^2\bar{S}\bar{I}, \\ x_4 &= (\epsilon + \mu + \sigma\beta)(\eta + \sigma\beta)\beta^2\bar{S}\bar{I}, \end{aligned}$$

$$\begin{aligned}
 x_5 &= (\epsilon + \mu + \sigma\beta), \\
 x_6 &= (\epsilon + \mu + \sigma\beta)(\mu + p) + (\epsilon + \mu + \sigma\beta)\beta\bar{I} + (\epsilon + \mu + \sigma\beta)(\alpha + \mu + \gamma) + \mu(\epsilon + \mu + \sigma\beta), \\
 x_7 &= (\epsilon + \mu + \sigma\beta)(\mu + p)(\alpha + \mu + \gamma) + (\epsilon + \mu + \sigma\beta)\beta\bar{I}(\alpha + \mu + \gamma) \\
 &\quad + (\mu + \eta\bar{R})((\epsilon + \mu + \sigma\beta)(\mu + p) + (\epsilon + \mu + \sigma\beta)\beta\bar{I}) \\
 &\quad - (\epsilon + \mu + \sigma\beta)\eta\bar{R}((\mu + p) + \beta\bar{I} + (\alpha + \mu + \gamma)), \\
 x_8 &= (\epsilon + \mu + \sigma\beta)(\eta + \sigma\beta)\beta^2\bar{S}\bar{I} - (\epsilon + \mu + \sigma\beta)(\mu + p)(\alpha + \mu + \gamma)\eta\bar{R}.
 \end{aligned}$$

Theorem 3.2 *If the solution of (12) is locally asymptotic stable, then $\tau = 0$ and $R_0 > 1 + \frac{\beta^2(\eta + \sigma\beta)^2(\epsilon + \mu + \sigma\beta)}{\mu p(\mu + p)(\alpha + \mu + \gamma)^2}$.*

Proof Let $\tau = 0$. From (13),

$$\lambda^4 + (x_1 + x_5)\lambda^3 + (x_2 - x_6)\lambda^2 + (x_3 - x_7)\lambda + x_4 - x_8 = 0. \tag{14}$$

Since $R_0 > 1 + \frac{\beta^2(\eta + \sigma\beta)^2(\epsilon + \mu + \sigma\beta)}{\mu p(\mu + p)(\alpha + \mu + \gamma)^2}$, $\bar{S} > 0$, $\bar{E} > 0$, $\bar{I} > 0$, $\bar{R} > 0$.

By the method of the Routh–Hurwitz criteria, we get

$$\begin{aligned}
 x_9 &= x_1 - x_5 = \beta\bar{I} + \eta\bar{R} + 3\mu + p + \alpha + \gamma > 0, \\
 x_{10} &= (x_1 - x_5)(x_2 - x_6) - (x_3 - x_7) \\
 &= ((\mu + p) + \beta\bar{I} + (\alpha + \mu + \gamma) + \mu + \eta\bar{R})[(\mu + p)(\alpha + \mu + \gamma) + \beta\bar{I}(\alpha + \mu + \gamma) \\
 &\quad + (\mu + \eta\bar{R})((\epsilon + \mu + \sigma\beta) + (\mu + p) + \beta\bar{I}) - \mu(\epsilon + \mu + \sigma\beta)] \\
 &\quad - (\epsilon + \mu + \sigma\beta)(\eta + \sigma\beta)\beta^2\bar{S}\bar{I} - (\epsilon + \mu + \sigma\beta)\eta\bar{R}((\mu + p) + \beta\bar{I} + (\alpha + \mu + \gamma)) \\
 &\quad ((\mu + p) + \beta\bar{I} + (\alpha + \mu + \gamma))[(\mu + p)(\alpha + \mu + \gamma) + (\alpha + \mu + \gamma)\beta\bar{I} \\
 &\quad + (\mu + \eta\bar{R})((\mu + p) + \eta\bar{R})((\mu + p) + \beta\bar{I})] + [(\mu + p)(\alpha + \mu + \gamma) \\
 &\quad + (\mu + \eta\bar{R})((\mu + \eta) + \beta\bar{I}) + (\epsilon + \mu + \sigma\beta)\eta\bar{R}]x_{11} \\
 &= \begin{vmatrix} x_1 - x_5 & x_3 - x_7 & 0 \\ 1 & x_2 - x_6 & x_4 - x_8 \\ 0 & x_1 - x_5 & x_3 - x_7 \end{vmatrix} \\
 &= (x_1 - x_5)[(x_2 - x_6)(x_3 - x_7) - (x_1 - x_5)(x_4 - x_8)](x_3 - x_7)^2.
 \end{aligned}$$

Let $m = \mu + \eta\bar{R}$, $n = (\mu + p) + \beta\bar{I}$. Thus,

$$\begin{aligned}
 x_{11} &= [(\alpha + \mu + \gamma)m(n - (\mu + p)) + \eta(\epsilon + \mu + \sigma\beta)\bar{R}n + \eta(\epsilon + \mu + \sigma\beta)\bar{R}(\alpha + \mu + \gamma)] \\
 &\quad ((\alpha + \mu + \gamma)n^2 + mn^2 + (\alpha + \mu + \gamma)^2n + (\alpha + \mu + \gamma)mn \\
 &\quad + (\mu + p)(\alpha + \mu + \gamma)m + m^2n + (\epsilon + \mu + \sigma\beta)m\eta\bar{R}) \\
 &\quad - (\epsilon + \mu + \sigma\beta)(\mu + p)(\alpha + \mu + \gamma)\eta\bar{R}(n^2 + (\alpha + \mu + \gamma)^2 + 2(\alpha + \mu + \gamma)n \\
 &\quad + m^2 + 2mn + 2m(\alpha + \mu + \gamma)) \\
 &= (\alpha + \mu + \gamma)m(n - (\mu + p))((\alpha + \mu + \gamma)n^2 + mn^2 + (\alpha + \mu + \gamma)^2n
 \end{aligned}$$

$$\begin{aligned}
 &+ (\alpha + \mu + \gamma)mn + (\mu + p)(\alpha + \mu + \gamma)m + m^2n + (\epsilon + \mu + \sigma\beta)m\eta\bar{R}) \\
 &+ (\epsilon + \mu + \sigma\beta)\eta\bar{R}(\alpha + \mu + \gamma)^2m(n - (\mu + p)) \\
 &+ (\epsilon + \mu + \sigma\beta)(\alpha + \mu + \gamma)^3\eta\bar{R}(n - d) \\
 &+ 2(\epsilon + \mu + \sigma\beta)\eta\bar{R}(\alpha + \mu + \gamma)^2n(n - (\mu + p)) \\
 &+ (\epsilon + \mu + \sigma\beta)\eta\bar{R}(\alpha + \mu + \gamma)m^2(n - (\mu + p)) \\
 &+ 2(\epsilon + \mu + \sigma\beta)\eta\bar{R}(\alpha + \mu + \gamma)mn(n - (\mu + p)) \\
 &+ (\epsilon + \mu + \sigma\beta)\eta\bar{R}(\alpha + \mu + \gamma)^2m(n - (\mu + p)) \\
 &+ (\epsilon + \mu + \sigma\beta)\eta\bar{R}mn^3 + (\epsilon + \mu + \sigma\beta)\eta\bar{R}(\mu + p)(\alpha + \mu + \gamma)mn \\
 &+ (\epsilon + \mu + \sigma\beta)\eta\bar{R}m^2n^2 + (\epsilon + \mu + \sigma\beta)^2m\eta^2\bar{R}^2n \\
 &+ (\epsilon + \mu + \sigma\beta)^2\eta^2\bar{R}^2(\alpha + \mu + \gamma)m.
 \end{aligned}$$

We have $x_{11} > 0$, since $n - (\mu + p) > 0$.

$$x_{12} = \begin{vmatrix} x_1 - x_5 & x_3 - x_7 & 0 & 0 \\ 1 & x_2 - x_6 & x_4 - x_8 & 0 \\ 0 & x_1 - x_5 & x_3 - x_7 & 0 \\ 0 & 1 & x_2 - x_6 & x_4 - x_8 \end{vmatrix} = a_4x_{11}.$$

Taking note of $a_4 = (\epsilon + \mu + \sigma\beta)(\mu + p)(\alpha + \mu + \gamma)\eta\bar{x}$, it is anything but difficult to acquire that $x_{12} > 0$. Subsequently, the real parts are negative in (14). This completes the verification of Theorem 3.2. Here the roots of $\Omega(\lambda) = 0$ have negative real roots. Hence, there exists a $\tau_0 > 0$ to such that $\tau \in [0, \tau_0)$ in (13), and we have

$$\Omega(\lambda) = 0, \quad \text{Re}(\lambda) < 0 \quad \text{for } \tau \in [0, \tau_0), \tag{15}$$

moreover, when $\tau = \tau_0$, $\text{Re}(\lambda) < 0$. To decide on this τ_0 and the related simply $\phi_0 i (\phi_0 > 0)$ imaginary roots, we understand (13) with $\lambda = \phi_0 i$. For straightforwardness, we use τ, ϕ rather than τ_0, ϕ_0 . From (13), we have

$$\begin{aligned}
 &\phi^4 - x_1\phi^3i - x_2\phi^2 + x_3\phi i + x_4 - (-x_5\phi^3i - x_6\phi^2 + x_7\phi + x_8) \\
 &(\cos \phi\tau - i \sin \phi\tau) = 0.
 \end{aligned} \tag{16}$$

Comparing the coefficients of real and imaginary parts, we get

$$\begin{cases} (x_8 - x_6\phi^2) \cos \phi\tau + (x_7\phi - x_5\phi^3) \sin \phi\tau = \phi^4 - x_2\phi^2 + x_4, \\ (x_5\phi^3 - x_7\phi) \cos \phi\tau + (x_8 - x_6\phi^2) \sin \phi\tau = x_1\phi^3 - x_3\phi, \end{cases} \tag{17}$$

$$\begin{aligned}
 \cos \phi\tau &= \frac{1}{\Delta} \begin{vmatrix} \phi^4 - x_2\phi^2 + x_4 & x_7\phi - x_5\phi^3 \\ x_1\phi^3 - x_3\phi & x_8 - x_6\phi^2 \end{vmatrix} \\
 &= \frac{1}{\Delta} [(x_1x_5 - x_6)\phi^6 + (x_8 + x_2x_6 - x_1x_7 - x_3x_5)\phi^4 \\
 &\quad + (x_3x_7 - x_2x_8 - x_4x_6)\phi^2 + x_4x_8]
 \end{aligned}$$

$$\begin{aligned}
 &= \frac{1}{\Delta} (c_1\phi^6 + c_2\phi^4 + c_3\phi^2 + c_4), \\
 \sin \phi \tau &= \frac{1}{\Delta} \begin{vmatrix} x_8 - x_6\phi^2 & \phi^4 - x_2\phi^2 + x_4 \\ x_5\phi^3 - x_7\phi & x_1\phi^3 - x_3\phi \end{vmatrix} \\
 &= \left(-\frac{\phi}{\Delta}\right) (x_5\phi^6 + (x_1x_6 - x_7 - x_2x_5)\phi^4 + (x_2x_7 + x_4x_5 - x_3x_6 - x_1x_8)\phi^2 \\
 &\quad + (x_3x_8 - x_4x_7)) \\
 &= -\frac{\phi}{\Delta} (x_9\phi^6 + x_{10}\phi^4 + x_{11}\phi^2 + x_{12}),
 \end{aligned}$$

where

$$\begin{aligned}
 \Delta &= \begin{vmatrix} x_8 - x_6\phi^2 & x_7\phi - x_5\phi^3 \\ x_4\phi^3 - x_7\phi & x_8 - x_6\phi^2 \end{vmatrix} \\
 &= (x_8 - x_6\phi^2)^2 + (x_7 - x_5\phi^3)^2 = x_5\phi^6 + (x_6 - 2x_5x_7)\phi^4 + (x_7^2 - 2x_6x_8)\phi^2 \\
 &\quad + x_8^2 = (e_1\phi^6 + e_2\phi^4 + e_3\phi^2 + e_4) > 0.
 \end{aligned}$$

Here $\sin^2 \phi \tau + \cos^2 \phi \tau = 1$, it follows that

$$\phi^{14} + x_{13}\phi^{12} + x_{14}\phi^{10} + x_{15}\phi^8 + x_{16}\phi^6 + x_{17}\phi^4 + x_{18}\phi^2 + x_{19} = 0, \tag{18}$$

where

$$\begin{aligned}
 x_{13} &= \frac{1}{x_9^2} (c_1^2 + 2x_9x_{10} - e_1^2), \\
 x_{14} &= \frac{1}{x_9^2} (2c_1c_2 + x_{10}^2 + 2x_9x_{11} - 2e_1e_3), \\
 x_{15} &= \frac{1}{x_9^2} (c_2^2 + 2c_1c_3 + 2x_9x_{12} + 2x_{10}x_{11} - e_2^2 - 2e_1e_3), \\
 x_{16} &= \frac{1}{x_9^2} (2c_1c_4 + 2c_2c_3 + x_{11}^2 + 2x_{10}x_{12} - 2e_1e_4 - 2e_2e_3), \\
 x_{17} &= \frac{1}{x_9^2} (c_3^2 + 2c_2c_4 + 2x_{11}x_{12} - e_3^2 - 2e_2e_4), \\
 x_{18} &= \frac{1}{x_9^2} (2c_3c_4 + x_{12}^2 - 2e_3e_4), \\
 x_{19} &= \frac{1}{x_9^2} (c_4^2 - e_4^2).
 \end{aligned}$$

Denoting: $x = \phi^2$, (18) becomes

$$x^7 + x_{13}x^6 + x_{14}x^5 + x_{15}x^4 + x_{16}x^3 + x_{17}x^2 + x_{18}x + x_{19} = 0. \tag{19}$$

First, $x = 0$ is not a root of (19) if $x_8 \neq 0$. There is no positive real root in (19). Therefore $\phi = \sqrt{x}$ does not get the solution. Hence the bifurcation parameter τ does not occur and a Hopf bifurcation is not evaluated. Equation (19) always has positive real roots. Let the hypothesis be as follows:

(Ω_1): Equation (19) has possibly one positive real root;

(Ω_2): $\Lambda \triangleq [4\phi^6 + 3(x_1^2 - 2x_2 - x_5^2)\phi^4 + 2(x_2^2 - x_6^2 + 2x_4 + x_5x_7 - 2x_1x_3)\phi^2 + x_3^2 - x_7^2 + 2x_6x_8 - 2x_2x_4] > 0$ for any $\phi > 0$.

Let x_0 be the positive roots of (19), denoting $\phi_0 = \sqrt{x_0}$. From the above, we get

$$\tau_j = \frac{1}{\phi_0} \left(\cos^{-1} \left(\frac{c_1\phi_0^6 + c_2\phi_0^4 + c_3\phi_0^2 + c_4}{e_1\phi_0^6 + e_2\phi_0^4 + e_3\phi_0^2 + e_4} \right) + 2j\pi \right), \quad j = 0, 1, 2, 3, \dots,$$

and

$$\tau_0 = \frac{1}{\phi_0} \cos^{-1} \left(\frac{c_1\phi_0^6 + c_2\phi_0^4 + c_3\phi_0^2 + c_4}{e_1\phi_0^6 + e_2\phi_0^4 + e_3\phi_0^2 + e_4} \right), \quad j = 0.$$

The set of ordered pair is (ϕ_0, τ_0) to find the polynomial roots of (13) in a neighborhood of τ_0 and differentiating with respect to τ , we get

$$\left[\frac{d\lambda}{d\tau} \right]^{-1} = \frac{-(4\lambda^3 + 3x_1\lambda^2 + 2x_2\lambda + x_3)e^{\lambda\tau}}{\lambda(x_5\lambda^3 + x_6\lambda^2 + x_7\lambda + x_8)} + \frac{3x_5\lambda^2 + 2x_6\lambda + x_7}{\lambda(x_5\lambda^3 + x_7\lambda^2 + x_7\lambda + x_8)} - \frac{\tau}{\lambda}. \tag{20}$$

Letting (20), we have

$$\begin{aligned} \operatorname{Re} \left[\frac{d\lambda}{d\tau} \right]^{-1} &= \frac{1}{\phi\Delta} \{ (3x_1\phi^2 - x_3) [(x_5\phi^3 - x_7\phi) \cos \phi\tau + (x_8 - x_6\phi^2) \sin \phi\tau] \\ &\quad + (4\phi^3 - 2x_2\phi) [(x_8 - x_6\phi^2) \cos \phi\tau - (x_5\phi^3 - x_7\phi) \sin \phi\tau] \\ &\quad + (x_7 - 3x_5\phi^2)(x_5\phi^3 - x_7\phi) + 2x_6\phi(x_8 - x_6\phi^2) \} \\ &= \frac{1}{\Delta} [4\phi^6 + 3(x_2^2 - 2x_2 - x_5^2)\phi^4 + 2(x_2^2 - x_6^2 + 2x_4 + 2x_5x_7 - 2x_1x_3)\phi^2 \\ &\quad + x_3^2 - x_7^2 + 2x_6x_8 - 2x_2x_4], \end{aligned}$$

where $\Delta = (x_5\phi^3 - x_7\phi)^2 + (x_8 - x_6\phi^2)^2 > 0$. If (Ω_2) is satisfied, then (20) > 0 will hold for any $\phi > 0$.

So, $\operatorname{sign}\{\operatorname{Re}[\frac{d\lambda}{d\tau}]\mid\tau = \tau_0\} = \operatorname{sign}\{\operatorname{Re}[\frac{d\lambda}{d\tau}]\mid\tau = \tau_0\} \triangleq \operatorname{sign}(\cdot) = 1$.

Therefore, the roots of (14) have negative real parts. If $\tau = \tau_0$, then other negative real roots have $\Omega(\lambda) = 0$. In (14), if $\tau \in [0, \tau_0)$ and (Ω_1, Ω_2) assumption, then the COVID-19 infection is stable. Similarly, if $\tau > \tau_0$, then the COVID-19 infection is unstable and Eq. (2) shows bifurcation at $\tau = \tau_0$. □

4 Analysis of sensitivity parameters

The effects of changing parameter values on the functional value of the reproduction number R_0 are obtainable in this section. The essential parameter must be found, which could be an important threshold for disease management. The algebraic representations of the sensitivity index of R_0 to the parameters $\eta, \beta, \sigma, \gamma, \alpha, b, p$ are as follows:

$$\begin{aligned} \frac{\partial R_0}{\partial \beta} &= \frac{2\beta\sigma b}{\mu(\mu + p)(\mu + \gamma + \alpha)}, & \frac{\partial R_0}{\partial b} &= \frac{\beta(\eta + \sigma\beta)}{\mu(\mu + p)(\mu + \gamma + \alpha)}, \\ \frac{\partial R_0}{\partial \eta} &= \frac{\beta b}{\mu(\mu + p)(\mu + \gamma + \alpha)}, & \frac{\partial R_0}{\partial p} &= \frac{-\beta b(\eta + \sigma\beta)}{\mu(\mu + p)^2(\mu + \gamma + \alpha)}, \end{aligned}$$

$$\begin{aligned} \frac{\partial R_0}{\partial \sigma} &= \frac{\beta^2 \sigma}{\mu(\mu + p)(\mu + \gamma + \alpha)}, & \frac{\partial R_0}{\partial \alpha} &= \frac{-\beta b(\eta + \sigma \beta)}{\mu(\mu + p)(\mu + \gamma + \alpha)^2}, \\ \frac{\partial R_0}{\partial \gamma} &= \frac{-\beta b(\eta + \sigma \beta)}{\mu(\mu + p)(\mu + \gamma + \alpha)^2}. \end{aligned}$$

It is concluded that some partial derivatives are positive, with the increase of any of the above positive value parameters η, β, σ the basic reproductive number R_0 increases. The elasticity is estimated with the proportional response to the proportional perturbation. We have

$$\begin{aligned} E_\beta &= \frac{\beta}{R_0} \frac{\partial R_0}{\partial \beta} = \frac{2\beta\sigma}{(\eta + \sigma\beta)} = 0.9804, & E_\eta &= \frac{\eta}{R_0} \frac{\partial R_0}{\partial \eta} = \frac{\eta}{(\eta + \sigma\beta)} = 0.5098, \\ E_\sigma &= \frac{\sigma}{R_0} \frac{\partial R_0}{\partial \sigma} = \frac{\beta\sigma}{(\eta + \sigma\beta)} = 0.4901. \end{aligned}$$

From the above expressions, it is observed that E_η, E_β and E_σ are positive. This implies an increase in the parameters η, β and σ leads to an increase in the value of the basic reproduction number R_0 . The smallest change in these parameters can cause a high variation in the basic reproduction number. A very sensitive parameter should be carefully calculated since a slight variation can lead to major quantitative changes in the system.

5 Numerical experiment

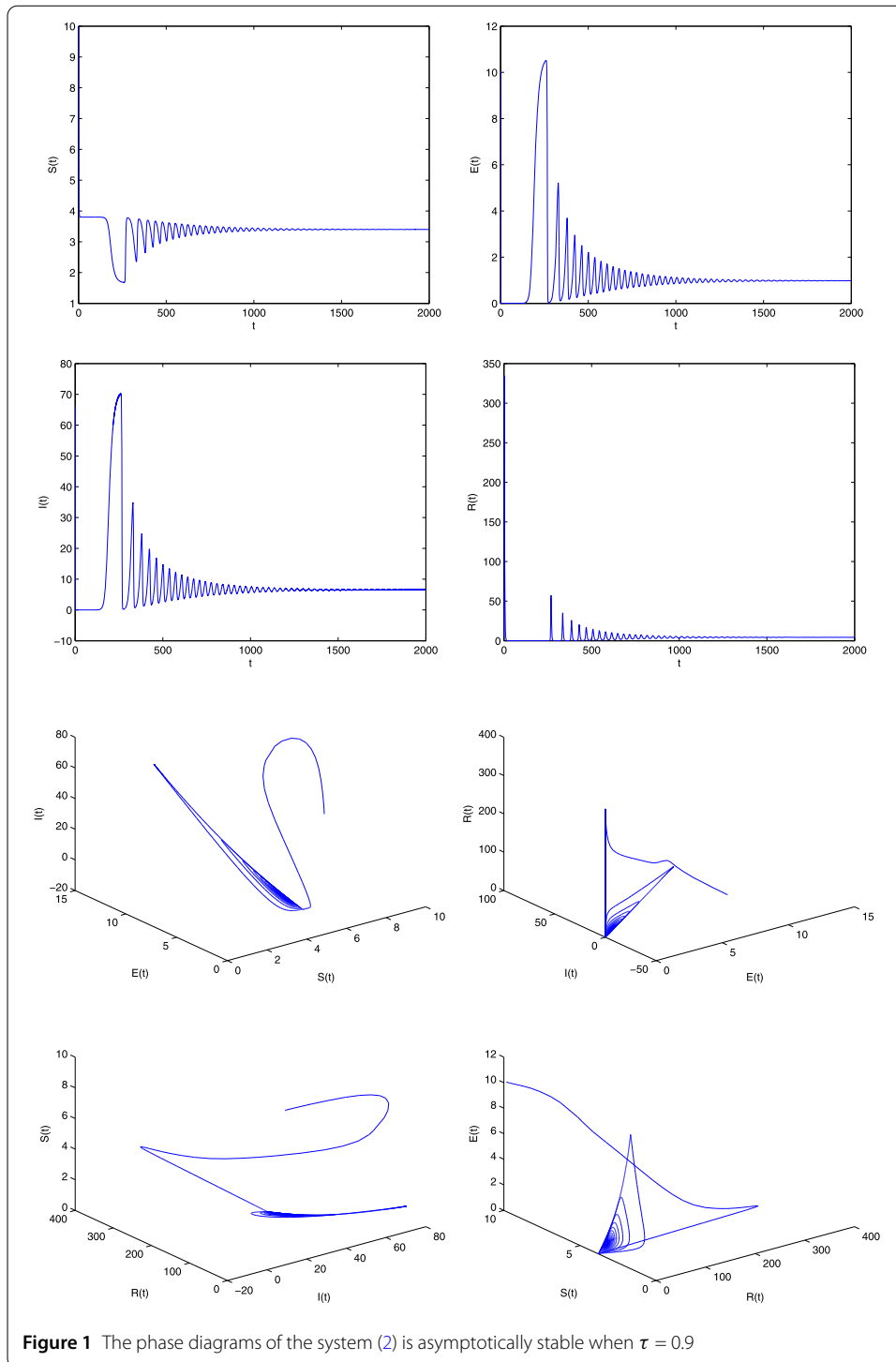
The COVID 19 model has unspecified parameters of SEIR model. The model's identities are investigated by the iterative algorithm and the parameter values (Age-standardized SEIR model) of the real-life data for the COVID 19 pandemic in India should be determined from these model values. COVID 19 data are therefore important in developing and validating the nonlinear ODE. Let us consider the parameters $b = 0.5, \gamma = 0.008, \epsilon = 0.1, \mu = 0.0018, p = 0.5, \beta = 0.1923, \eta = 0.1, \sigma = 0.5, \alpha = 0.5$ with $(S(0), E(0), I(0), R(0))$ [34]. In the event that (19) has no positive roots, at that point the COVID-19 infection present equilibrium is locally asymptotic stable. On the off chance that $R_0 = 2.47$, at that point the COVID-19 disease presents equilibrium $E = (3.1, 1.4, 10.01, 2.01)$. From (19), we have

$$x^7 + 500.01x^6 + 10,025x^5 + 23,423x^4 + 4099x^3 + 28.1x^2 + 0.1x + 6.110^{-5} = 0,$$

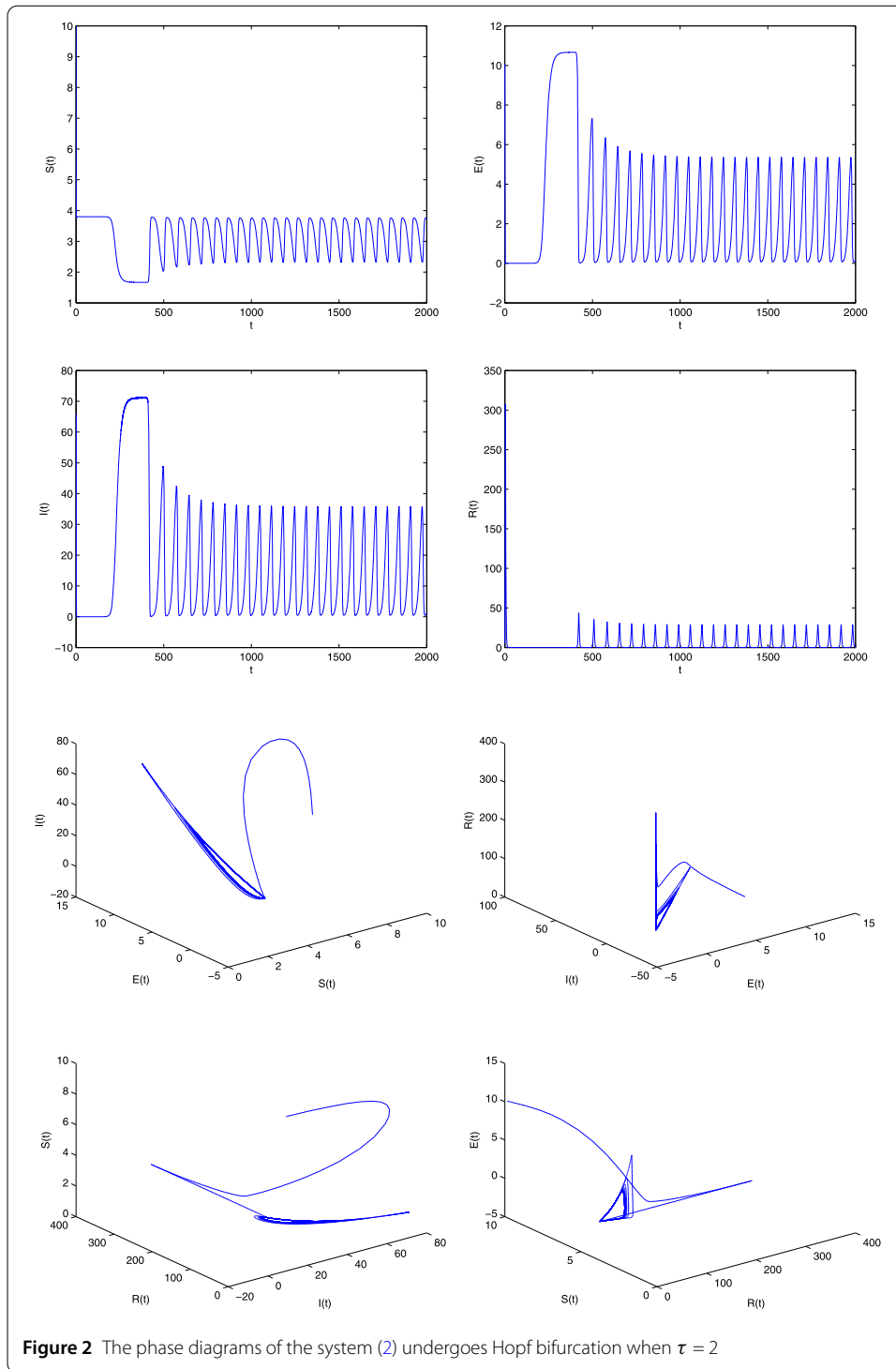
having real negative roots. Therefore the equilibrium is locally asymptotic stable and it represents a Hopf bifurcation. Obviously, $R_0 = 1.92$, and the COVID-19 infection presenting an equilibrium has $E = (3.01, 1.3, 8.1, 3.3)$. From (19), we have

$$x^7 + 477.2304x^6 + 47,123x^5 + 33,257x^4 + 5008x^3 + 27.1x^2 - 1.3x - 0.001 = 0,$$

having positive real roots and others having negative real roots. Accordingly, $\phi_0 = \sqrt{x} = 0.1$ It is not hard to evaluate the bifurcation stationary value to be $\tau_0 = 1.96$. Also, it is anything but difficult to prove that $\Lambda = 2.8 > 0$, i.e., (Ω_2) is fulfilled. The phase diagram of the system (2) is asymptotic stable when $\tau = 0.9 < \tau_0$ (see Fig. 1). Also, the phase diagrams of the system (2) undergoes a Hopf bifurcation when $\tau = 2 > \tau_0$ (see Fig. 2). We utilize the serious cases and deaths in the individual response work, rather than deaths as it were. We additionally increment the power of the legislative activity to such an extent that the



model results to a great extent in a coordinate of the watched, with a revealing proportion. In Fig. 3 shows the numerical simulations and calculated the different R_0 values such as 2.0317, 1.2922, 1.4809, 1.5972 and 0.9844 from the real-life data already published (WHO). The range of the R_0 values lies between 0.9844 and 2.0317. The time plots of SEIR COVID-19 model for different recruitment rate at $\tau = 2$ (see Fig. 4). To be specific just the extent of the model's created cases will be accounted for as a general rule. Consequently it would



concern testing given a generally brief time frame arrangement, and a few other obscure parameters are to be assessed. Figure 4 depicts how decreasing the transmission rate can change the system dynamics from the limit cycle to stable focus. It implies that without the Hopf bifurcation, the system is stable and controllable. Based on the sensitivity analysis value, decreasing the transmission rate can change the dynamics of the system from a limit cycle to a stable focus.

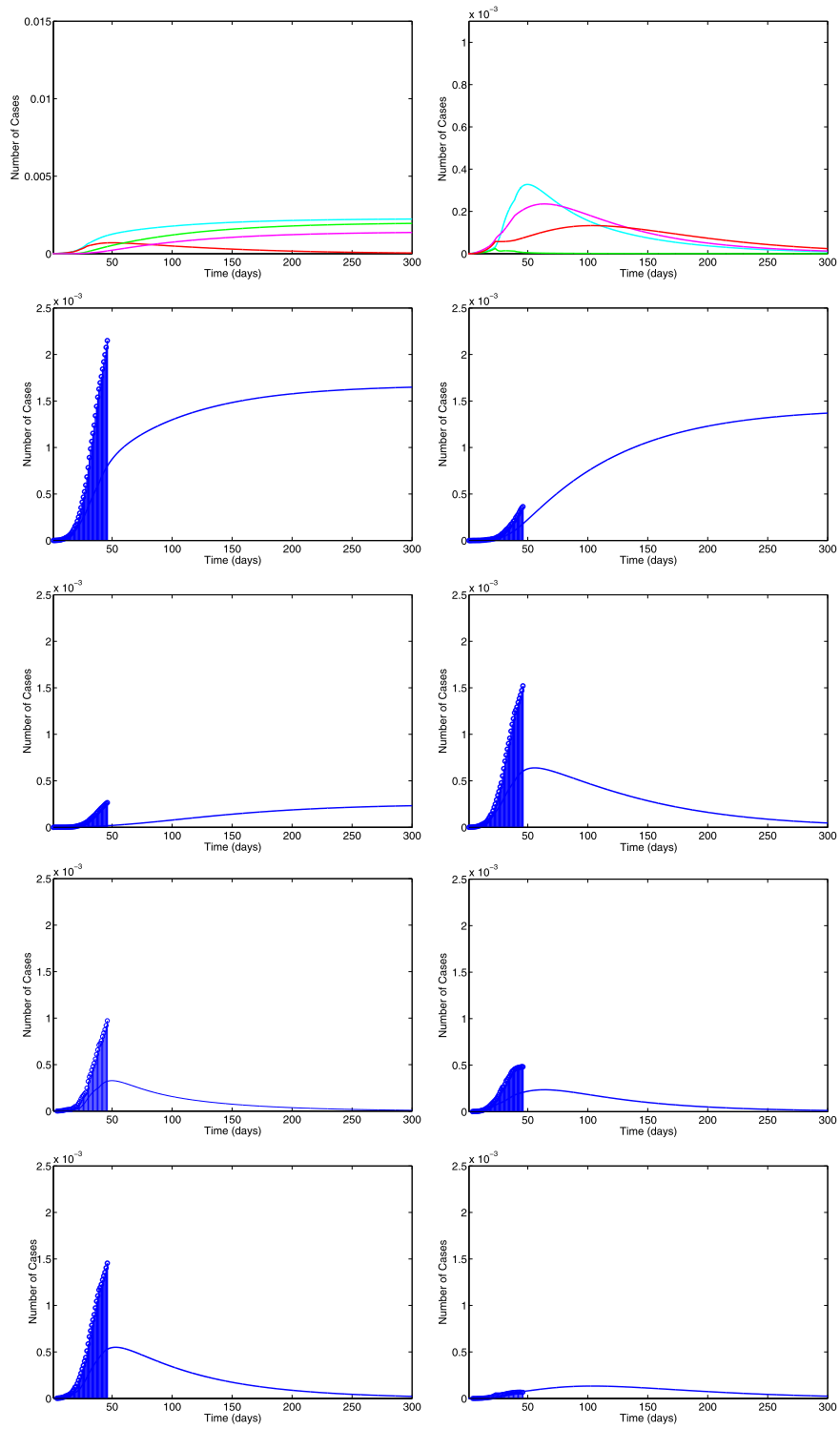
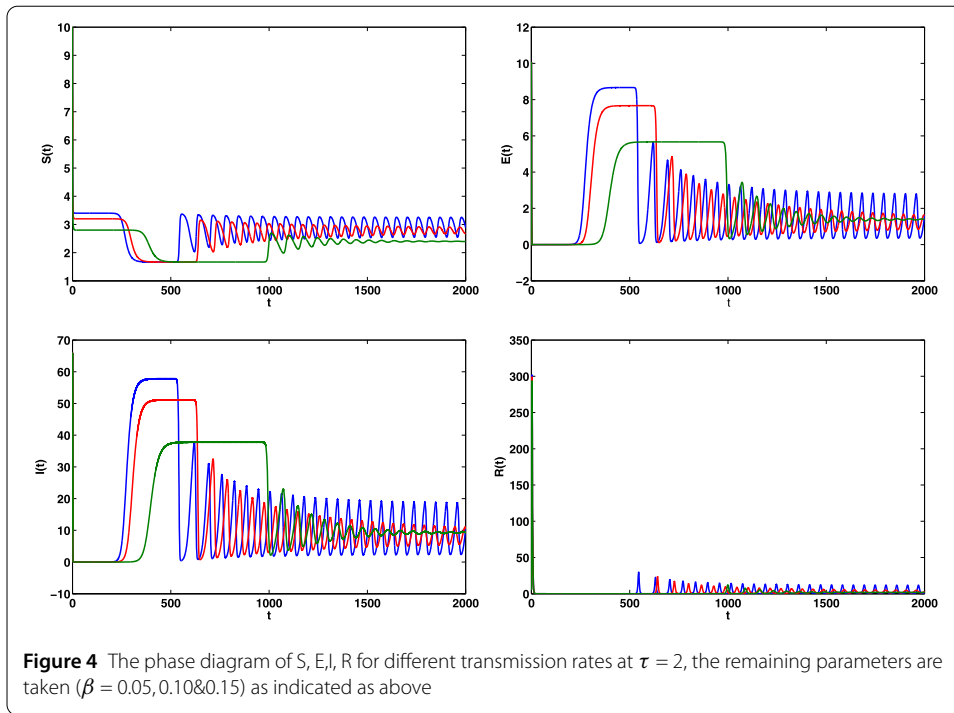


Figure 3 The epidemic numerical simulations of (2) from real-life data



6 Conclusion

There is a shortage of epidemiological information about the rising coronavirus, which would be of essential significance to structure and executing auspicious, specially appointed viable general well being intercessions, isolation and travel limitations. We have contemplated a general SEIR model of COVID-19 infection with delay. If $R_0 < 1$, then stability of the disease-free equilibrium is derived by Lyapunov techniques. Furthermore as regards the effects of time delay $\tau = 0$, the COVID-19 infection is either absent or presents an equilibrium when $R_0 > 1$. Here $1 < R_0 < 1 + \frac{\beta^2(\eta+\sigma\beta)^2(\epsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$, then E_1 is stable. If $R_0 > 1 + \frac{\beta^2(\eta+\sigma\beta)^2(\epsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$, then E_1 is unstable. Hence, $\tau > 0$, $1 < R_0 < 1 + \frac{\beta^2(\eta+\sigma\beta)^2(\epsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$, E_1 is stable. The basic reproductive ratio $R_0 > 1 + \frac{\beta^2(\eta+\sigma\beta)^2(\epsilon+\mu+\sigma\beta)}{\mu p(\mu+p)(\alpha+\mu+\gamma)^2}$, if the susceptible cells birth rate is high. Therefore the linearized system of (2) has no real positive roots and we have stability. The polynomial equation (19) has a single real positive root when $\tau < \tau_0$. The COVID-19 infection presenting equilibrium is stable. If $\tau > \tau_0$, the equilibrium solutions are unstable and a Hopf bifurcation occurs. Supposing Eq. (19) has more than one positive root, it does not exit. In the future, further investigation is needed to this system. The controlling of the reproduction number ratios proposes that the outbreak might be more genuine than what has been accounted for up until now, given the specific period of expanding social contacts, justifying powerful, severe general well being measures planned to relieve the weight produced by the spreading of the new infection. Finally, as regards the transmission rate it can be concluded that the system dynamics can be modified by decreasing its value from a limit cycle to a stable focus. Important measures to reduce the proportion of people susceptible to infection can be taken through increasing their immunity, quarantining infectious people, and decreasing their interaction with susceptible people. By using the least square approach, we used the descendant gradient model and identified the nearly approximate value of the parameters. The most important factor in

preventing the spread of the virus locally is to empower the citizens with the right information and taking precautions as per the advisories being issued by the Ministry of Health & Family Welfare.

Acknowledgements

The authors wish to thank the chief editor, the associate editor and the anonymous reviewers for their helpful feedback to improve the quality of this manuscript. The authors also wish to thank Dr. V. Kumaran, Department of Mathematics, National Institute of Technology, Trichy, India, and Dr. V. Vetrivel, Department of Mathematics, Indian Institute of Technology Madras, Chennai, India, for their valuable guidance.

Funding

Not applicable.

Availability of data and materials

To this article no data sets were generated or analyzed in the current study data.

Competing interests

The authors declare there are no competing interests.

Authors' contributions

For the writing of this paper all authors equally contributed and also read and agreed on the final copy of the manuscript.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Received: 20 April 2020 Accepted: 9 September 2020 Published online: 24 September 2020

References

1. World Health Organization. Novel Coronavirus—Japan (ex-China). World Health Organization. Cited April 16, 2020. Available at <https://www.who.int/csr/don/16-April-2020-novel-coronavirus-japan-ex-china/en/>
2. Zhou, P., Yang, X., Wang, X., et al.: A pneumonia outbreak associated with a new coronavirus of probable bat origin. *Nature* **579**, 270–273 (2020). <https://doi.org/10.1038/s41586-020-2012-7>
3. Li, Q., Guan, X., Wu, P., et al.: Early transmission dynamics in Wuhan, China, of novel coronavirus-infected pneumonia. *N. Engl. J. Med.* **382**(13), 1199–1207 (2020). <https://doi.org/10.1056/NEJMoa2001316>
4. Huang, C., Wang, Y., Li, X., et al.: Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *Lancet* **395**(10223), 497–506 (2020). [https://doi.org/10.1016/S0140-6736\(20\)30183-5](https://doi.org/10.1016/S0140-6736(20)30183-5)
5. Zhu, N., Zhang, D., Wang, W., et al.: A novel coronavirus from patients with pneumonia in China, 2019. *N. Engl. J. Med.* **382**(8), 727–733 (2020). <https://doi.org/10.1056/NEJMoa2001017>
6. Zhao, S., Musa, S.S., Lin, Q., Ran, J., Yang, G., Wang, W., Lou, Y., Yang, L., Gao, D., He, D., Wang, M.H.: Estimating the unreported number of novel coronavirus (2019-nCoV) cases in China in the first half of January 2020: a data-driven modelling analysis of the early outbreak. *J. Clin. Med.* **9**(2), 388 (2020). <https://doi.org/10.3390/jcm9020388>
7. Dye, C., Gay, N.: Epidemiology. Modeling the SARS epidemic. *Science* **300**(5627), 1884–1885 (2003). <https://doi.org/10.1126/science.1086925>
8. Zhou, G., Yan, G.: Severe acute respiratory syndrome epidemic in Asia. *Emerg. Infect. Dis.* **9**, 1608–1610 (2003). <https://doi.org/10.3201/eid0912.030382>
9. Wu, Z., McGoogan, J.M.: Characteristics of and important lessons from the Coronavirus disease 2019 (COVID-19) outbreak in China: summary of a report of 72 314 cases from the Chinese center for disease control and prevention. *JAMA* **323**(13), 1239–1242 (2020). <https://doi.org/10.1001/jama.2020.2648>
10. Huang, C., Wang, Y., Li, X., et al.: Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China [published correction appears in *Lancet* 2020 Jan 30]. *Lancet* **395**(10223), 497–506 (2020). [https://doi.org/10.1016/S0140-6736\(20\)30183-5](https://doi.org/10.1016/S0140-6736(20)30183-5)
11. Guan, W.J., Ni, Z.Y., Hu, Y., et al.: Clinical characteristics of coronavirus disease 2019 in China. *N. Engl. J. Med.* **382**(18), 1708–1720 (2020). <https://doi.org/10.1056/NEJMoa2002032>
12. Nishiura, H., Kobayashi, T., Yang, Y., Hayashi, K., Miyama, T., Kinoshita, R., et al.: The rate of under ascertainment of novel Coronavirus (2019-nCoV) infection: estimation using Japanese passengers data on evacuation flights. *J. Clin. Med.* **9**(2), 419 (2020). Available at <https://www.mdpi.com/2077-0383/9/2/419>
13. Bogoch, I.I., Watts, A., Thomas-Bachli, A., Huber, C., Kraemer, M.U.G., Khan, K.: Pneumonia of unknown etiology in Wuhan, China: potential for international spread via commercial air travel. *J. Travel. Med.* **27**(2), taaa008 (2020). <https://doi.org/10.1056/NEJMoa2002032>
14. Wu, J.T., Leung, K., Leung, G.M.: Now casting and forecasting the potential domestic and international spread of the 2019-nCoV outbreak originating in Wuhan, China: a modelling study [published correction appears in *Lancet* 2020 Feb 4]. *Lancet* **395**(10225), 689–697 (2020). [https://doi.org/10.1016/S0140-6736\(20\)30260-9](https://doi.org/10.1016/S0140-6736(20)30260-9)
15. Lina, Q., Zhaob, S., Gaod, D., Loue, Y., Yangf, S., Musae, S.S., Wangb, M.H., Caig, Y., Wangg, W., Yangh, L., Hee, D.: A conceptual model for the coronavirus disease 2019 (COVID-19) outbreak in Wuhan, China with individual reaction and governmental action. *Int. J. Infect. Dis.* **93**, 211–216 (2020)
16. Bauch, C.T., et al.: Dynamically modeling SARS and other newly emerging respiratory illnesses: past, present, and future. *Epidemiology* **16**(6), 791–801 (2005). <https://doi.org/10.1097/01.ede.0000181633.80269.4c>
17. Cauchemez, S., et al.: Unraveling the drivers of MERS-CoV transmission. *Proc. Natl. Acad. Sci. USA* **113**(32), 9081–9086 (2016). <https://doi.org/10.1073/pnas.1519235113>

18. Chan, J.F.-W., et al.: A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster. *Lancet* (2020). [https://doi.org/10.1016/s0140-6736\(20\)30154-9](https://doi.org/10.1016/s0140-6736(20)30154-9)
19. Chan, J.F.-W., et al.: A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster. *Lancet* (2020). [https://doi.org/10.1016/s0140-6736\(20\)30154-9](https://doi.org/10.1016/s0140-6736(20)30154-9)
20. Chowell, G., et al.: Model parameters and outbreak control for SARS. *Emerg. Infect. Dis.* **10**(7), 1258–1263 (2004). <https://doi.org/10.3201/eid1007.030647>
21. Lessler, J., et al.: Incubation periods of acute respiratory viral infections: a systematic review. *Lancet Infect. Dis.* **9**(5), 291–300 (2009). [https://doi.org/10.1016/S1473-3099\(09\)70069-6](https://doi.org/10.1016/S1473-3099(09)70069-6)
22. Lipsitch, M., et al.: Transmission dynamics and control of severe acute respiratory syndrome. *Science* **300**(5627), 1966–1970 (2003). <https://doi.org/10.1126/science.1086616>
23. Tan, W., et al.: A novel coronavirus genome identified in a cluster of pneumonia cases Wuhan, China 2019–2020, China CDC weekly. *China CDC Weekly* **2**(4), 61–62 (2020). <https://doi.org/10.46234/ccdcw2020.017>
24. Chen, Y., Liu, Q., Guo, D.: Emerging coronaviruses: genome structure, replication, and pathogenesis [published correction appears in *J. Med. Virol.* 2020 Aug 2]. *J. Med. Virol.* **92**(4), 418–423 (2020). <https://doi.org/10.1002/jmv.25681>
25. Khan, M.A., Atangana, A.: Modeling the dynamics of novel coronavirus (2019-nCov) with fractional derivative [published online ahead of print, 2020 Mar 14]. *Alex. Eng. J.* (2020). <https://doi.org/10.1016/j.aej.2020.02.033>
26. Lim, J., Jeon, S., Shin, H.Y., Kim, M.J., Seong, Y.M., Lee, W.J., Choe, K.W., Kang, Y.M., Lee, B., Park, S.J.: Case of the index patient who caused tertiary transmission of coronavirus disease 2019 in Korea: the application of lopinavir/ritonavir for the treatment of COVID-19 pneumonia monitored by quantitative RT-PCR. *J. Korean Med. Sci.* **35**(7), 1–6 (2020). <https://doi.org/10.3346/jkms.2020.35.e88>
27. Hu, Z., Song, C., Xu, C., et al.: Clinical characteristics of 24 asymptomatic infections with COVID-19 screened among close contacts in Nanjing, China. *Sci. China Life Sci.* **63**, 706–711 (2020). <https://doi.org/10.1007/s11427-020-1661-4>
28. Hui, D.S.C., Zumla, A.: Severe acute respiratory syndrome: historical, epidemiologic, and clinical features. *Infect. Dis. Clin. North Am.* **33**, 869–889 (2019). <https://doi.org/10.1016/j.idc.2019.07.001>
29. Killerby, M.E., Biggs, H.M., Midgley, C.M., Gerber, S.I., Watson, J.T.: Middle East respiratory syndrome coronavirus transmission. *Emerg. Infect. Dis.* **26**, 191–198 (2020). <https://doi.org/10.3201/eid2602.190697>
30. Willman, M., Kobasa, D., Kindrachuk, J.: A comparative analysis of factors influencing two outbreaks of middle eastern respiratory syndrome (MERS) in Saudi Arabia and South Korea. *Viruses* **11**(12), 1119 (2019). <https://doi.org/10.3390/v11121119>
31. Cohen, J., Normile, D.: New SARS-like virus in China triggers alarm. *Science* **367**(6475), 234–235 (2020). <https://doi.org/10.1126/science.367.6475.234>
32. Lu, H., Stratton, C.W., Tang, Y.W.: Outbreak of pneumonia of unknown etiology in Wuhan, China: the mystery and the miracle. *J. Med. Virol.* **92**(4), 401–402 (2020). <https://doi.org/10.1002/jmv.25678>
33. Rothe, C., Schunk, M., Sothmann, P., et al.: Transmission of 2019-nCoV infection from an asymptomatic contact in Germany. *N. Engl. J. Med.* **382**(10), 970–971 (2020). <https://doi.org/10.1056/NEJMc2001468>
34. Hui, D.S., Azhar, E.E.I., Madani, T.A., Ntoumi, F., Kock, R., Dar, O., Ippolito, G., Mchugh, T.D., Memish, Z.A., Drosten, C., et al.: The continuing 2019-nCoV epidemic threat of novel coronaviruses to global health—the latest 2019 novel coronavirus outbreak in Wuhan, China. *Int. J. Infect. Dis.* **91**, 264–266 (2020). <https://doi.org/10.1016/j.ijid.2020.01.009>
35. Cheng, V.C.C., Wong, S.C., To, K.K.W., Ho, P.L., Yuen, K.Y.: Preparedness and proactive infection control measures against the emerging Wuhan coronavirus pneumonia in China. *J. Hosp. Infect.* **104**(3), 254–255 (2020). <https://doi.org/10.1016/j.jhin.2020.01.010>
36. Atangana, A.: Modelling the spread of COVID-19 with new fractal-fractional operators: can the lockdown save mankind before vaccination? *Chaos Solitons Fractals* **136**, 109860 (2020). <https://doi.org/10.1016/j.chaos.2020.109860>
37. Giordano, G., Blanchini, F., Bruno, R., et al.: Modelling the COVID-19 epidemic and implementation of population-wide interventions in Italy. *Nat. Med.* **26**, 855–860 (2020). <https://doi.org/10.1038/s41591-020-0883-7>
38. Zhao, S., Lin, Q., Ran, J., et al.: Preliminary estimation of the basic reproduction number of novel coronavirus (2019-nCoV) in China, from 2019 to 2020: a data-driven analysis in the early phase of the outbreak. *Int. J. Infect. Dis.* **92**, 214–217 (2020). <https://doi.org/10.1016/j.ijid.2020.01.050>
39. Chen, T.-M., Rui, J., Wang, Q.-P., Zhao, Z.-Y., Cui, J.-A., Yin, L.: A mathematical model for simulating the phase-based transmissibility of a novel coronavirus. *Infect. Dis. Poverty* **9**, 24 (2020). <https://doi.org/10.1186/s40249-020-00640-3>
40. Chen, T., Rui, J., Wang, Q., et al.: A mathematical model for simulating the phase-based transmissibility of a novel coronavirus. *Infect. Dis. Poverty* **9**, 24 (2020). <https://doi.org/10.1186/s40249-020-00640-3>
41. Liu, Z., Magal, P., Seydi, O., Webb, G.: Understanding unreported cases in the COVID-19 epidemic outbreak in Wuhan, China, and the importance of major public health interventions. *Biology* **9**, 50 (2020). <https://doi.org/10.3390/biology9030050>