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Mathematical analysis of epidemic model to assess the impact of lockdown on COVID-19

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ABSTRACT: COVID-19 and its variants have been the worst pandemic the entire world has witnessed. Tens of millions of cases have been recorded in over 210 countries and territories as part of the ongoing global pandemic that is still going on today. In this paper, we propose a SEI mathematical model to investigate the impact of lockdown on the control and spreading of infectious disease. COVID-19. The epidemic model incorporates constant recruitment, experiencing infectious force in the latent period and the infected period. The equilibrium states are computed. Under some conditions, results for local asymptotic stability and global stability of disease-free and endemic equilibrium are established by using the stability theory of ordinary differential equations. It is seen that when the basic reproduction number is high, the dynamical system is stable and diseases die out of the system, and when the disease persists in the dynamical system, when transcritical bifurcation appears. The numerical simulations are carried out to validate the analytical results.

KEYWORDS: COVID-19 epidemic; SEI model; lockdown; stability analysis; non-linear incidence

1. Introduction

Coronavirus disease 2019 (COVID-19) is an infectious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The disease was first identified in the city of Wuhan, Hubei Province, China, at the end of 2019 and spread globally, resulting in the ongoing 2019–2020 coronavirus pandemic. As per WHO situation reports on 4 October 4 2020, COVID-19 has claimed 1,030,738 lives, along with 34,804,348 confirmed cases worldwide[1]. On 11 March 2020, the novel COVID-19 outbreak was declared a pandemic by the WHO, and the call for countries to take quick action and scale up the response to treat, detect, and reduce dynamical transmission to save people's lives was reiterated. India stood at an important turning point in its challenge in opposition to COVID-19. To protect the country and control the spread of COVID-19 outbreaks in India, bold and decisive steps were taken on 24 March 2020, by announcing a 21-day nationwide lockdown. Despite control policies such as finding, isolating, testing, treating, and tracing the infected individuals, apart from a complete ban on all ages of people from stepping out of their homes, the closure of commercial and private establishments, the suspension of all research institutions, training, and educational institutions, the closure of all worship places, the suspension of non-essential private and public transport, and the prohibition of all political, social, sports,

entertainment, cultural, academic, and religious activities were imposed in various phases. According to the interim compliance report for the G20 extraordinary virtual summit, which was released on 14 September 2020[2], the spread of COVID-19 in India had been slowed considerably by lockdown measures. The effectiveness of lockdown[3–5] can be judged by the fact that, keeping in view the second wave of coronavirus infections, some countries have reimposed or extended the lockdown[6,7]. This served as the motivation for this paper. Several mathematical models based on integer order and fractional order differential equations for the epidemic COVID-19 have been proposed and analyzed by various mathematicians and researchers since its outbreak in December 2019[8–11]. Over 67,753 research papers and 19,789 preprints have been published so far on coronavirus disease from January 2020 to 2 August 2020. For more recent papers, we refer to the readers[12–16].

Since, interventions such as the role of the media, advertisements, lockdown, etc. have a massive impact on the course of infectious diseases and hence have been discussed via mathematical models for various diseases, for example, a mathematical epidemiological model of COVID-19 cases in Italy[17], modelling the COVID-19 epidemic and implementation of population-wide interventions in Italy[18], the effect of therapy and awareness campaigns on a SIR model[19], a mathematical model via non-linear systems describe the spread of the COVID-19 virus[20], fundamental theory of infectious disease transmission using straightforward compartmental models based on ordinary differential equations, such as the straightforward Kermack-McKendrick epidemic model[21] and a fresh COVID-19 epidemic model with media coverage[22], a nonlinear SEIRS type epidemic model with media impact for transmission dynamics of infectious diseases[23], reproduction numbers of infectious disease models[24], and reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission[25]. In this paper, we demonstrate theoretically how the preventive measure of lockdown impacts the dynamical transmission of coronavirus disease by proposing the SEI mathematical epidemic model, in which illness is infectious not only in the infected period but in the latent period too. Our model is like that proposed by Liu and Cui[26], but there are fundamental differences between the two models. The model proposed by Liu and Cui is the classical SIR model, where the disease is infectious in the infected period only.

The paper is organized as follows: In Section 2, an SEI model incorporating the impact of lockdown on the spreading of COVID-19 is proposed with the consideration that the disease is infectious in the latent period too. In Section 3, the threshold parameter R_0 is obtained, and based on this obtained number, the feasibility of equilibria has been discussed. Section 4 deals with the discussion and analysis of the local and global stability of disease-free equilibrium (DFE) along with the local stability of endemic equilibrium (EE) by the Routh-Hurwitz criterion and the construction of a suitable Lyapunov function. To illustrate the viability of theoretical analysis, the numerical simulations of the proposed model are carried out using MATLAB in Section 5. Section 7 is dedicated to the discussion and conclusion of the paper.

2. An SEI model with lockdown impact

Coronavirus disease continues to race around the world at a worrisome and fierce pace. Governments, organizations, and people are yelling to find out the safeguards to fight back effectively. To get these answers, accurate and comprehensive models are needed that are good enough to depict as many aspects of the disease as possible. Every mathematical epidemic model emphasizes certain components of actual real phenomena and ignores others due to the limitations of relevant mathematical theories and the complexities involved therein. Likewise, it is almost impossible to reflect all aspects of

genuineness for the disease COVID-19 via a single mathematical model[27]. In this section, we propose a mathematical model for infectious coronavirus disease in which an infected person does not become infected for some time. Such a person who is infected but not infective is called exposed. The total population N is partitioned into three compartments: S(t) is the class of susceptible, E(t) is the class of exposed, and I(t) is the class of infectious. In most cases, a host must go through a latent stage after the original infection before becoming contagious. To the best of our knowledge, in the SEI models related to COVID-19 proposed till now, the disease has not been considered infectious in the latent period. But, keeping in view the transmitting feature of COVID-19, here we consider the following SEI model with the feature that the disease is infectious in a latent period as well, and our model is depicting the effect of lockdown on the dynamical transmission of the disease.

$$\begin{cases} \dfrac{dS}{dt} = A - \delta S - \big(\alpha - g(E)\big)\dfrac{S}{N}E - \big(\beta - h(I)\big)\dfrac{S}{N}I \\ \dfrac{dE}{dt} = -\gamma E + \big(\alpha - g(E)\big)\dfrac{S}{N}E + \big(\beta - h(I)\big)\dfrac{S}{N}I - (\delta + \lambda)E \\ \dfrac{dI}{dt} = \gamma E - kI - (\delta + \mu)I \end{cases} \tag{1}$$

$S(0) > 0, E(0) \geq 0, I(0) > 0$ (one infectious person) so that for all $t \geq 0, S(t) + E(t) + I(t) = N$.

The Schematic flow chart diagram of the system (1) is given in **Figure 1**.

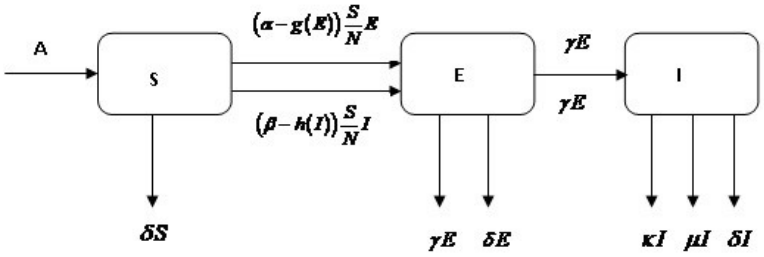

Figure 1. The schematic flow chart of system (1).

The model is grounded on mass action incidence and positive parameters, with the following epidemiological interpretations:

- A is the constant rate at which the susceptible are recruited in the population.
- δ is the average natural death rate across all groups.
- α is the rate of efficient contact in the latent period before lockdown.
- β is the effective contact rate prior to lockdown during the infected time.
- γ is the rate of transmission between infected and exposed people.
- λ is the rate of disease-caused deaths in an exposed population.
- μ is the rate of disease-caused deaths in an infectious population.
- κ is the rate of segregating after disease is constant.

The control measures implemented by public health officials during a disease outbreak, such as lockdowns, restaurant closures, school closures, isolating infected people, postponing conferences, etc., can have an impact on the homogeneous incidence rate. The contact per unit of time t is typically decreased by these required actions. This causes a high number of infected people but a smaller chance of infection per contact, which could cause non-linearity in dynamic transmission rates. In the proposed model (1), $g(E) = \frac{aE}{m+E}$ and $h(I) = \frac{aI}{n+I}$ are the rate of contacts reduced as the impact of lockdown in the

infected and latent period, respectively, where a can be thought of as the rate of implementation of lockdown. Here, m and n reflect the reactive velocity of lockdown and people in infected and latent periods, respectively. Thus $\alpha - g(E)$ and $\beta - h(I)$ are the frequencies of effective contact during the latent and infected periods during lockdown. Further, it is assumed that $a < \delta$. The functions $g(E)$ and $h(I)$ satisfy the following:

$$\text{(i) } g(0) = 0, g'(E) > 0, \lim_{E\to\infty} g(E) = a \text{ (ii) } h(0) = 0, h'(I) > 0, \lim_{I\to\infty} h(I) = a.$$

Since lockdown cannot prevent the disease completely, we take $\alpha,\ \beta > a$. Note that when the reported exposed and infectious numbers arrive at m and n respectively, then in each case, the corresponding reduced value of transmission is equal to $\frac{a}{2}$. Here$S + E + I = N$. Therefore, we have $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} \leq A - \delta N$. Thus, $N(t) \leq N(0)e^{-\delta t} + \frac{A}{\delta}\left(1 - e^{-\delta t}\right)$ so that $\lim_{t\to\infty} Sup(N(t)) = \frac{A}{\delta}$. From a biological point of view, we discuss the system (1) in the following feasible region $\phi = \left\{(S,E,I) \in R^3 : N = S + E + I \leq \frac{A}{\delta}\right\}$.

Thus, the total population remains bounded for all future $t \geq 0$. Here the domain ϕ is non- negative invariant as no solution paths leave through any boundary. The right-hand side (RHS) of each of the equations in the system (1) is smooth and continuously differentiable so that the initial value problems have singe solutions that exist on maximal intervals. Since paths can't leave ϕ, solutions exist $\forall\ t > 0$.

Thus, the model is epidemiologically and mathematically well posed.

3. Steady states and reproduction number (R_0)

The system (1) has a DFE $E_0 = \left(\frac{A}{\delta}, 0, 0\right)$. Since the local stability of the DFE of compartmental models is governed by R_0 of the system, referring to Rafiq et al.[7] and Liu and Cui[26], we compute the R_0 by the next-generation matrix method[28,29]. We have the following two vectors $\mathcal{F}$ and $\mathcal{V}$ to represent the new infection terms and remaining transfer terms respectively.

$$\mathcal{F} = \begin{bmatrix} \left(\alpha - \frac{aE}{m+E}\right)\frac{S}{N}E + \left(\beta - \frac{aI}{+I}\right)\frac{S}{N}I \\ 0 \end{bmatrix}, \mathcal{V} = \begin{bmatrix} \gamma E + (\delta + \lambda)E \\ -\gamma E + \kappa I + (\delta + \mu)I \end{bmatrix}$$

Thus, $F = \frac{\partial(F_1,F_2)}{\partial(E,I)} = \begin{bmatrix} \left(\alpha - \frac{aE}{m+E} - \frac{amE}{(m+E)^2}\right)\frac{S}{N} & \left(\beta - \frac{aI}{n+I} - \frac{anI}{(n+I)^2}\right)\frac{S}{N} \\ 0 & 0 \end{bmatrix}$

$E_0 = \left(\frac{A}{\delta}, 0, 0\right)$, $F = \begin{bmatrix} \alpha & \beta \\ 0 & 0 \end{bmatrix}$ and $V = \frac{\partial(V_1,V_2)}{\partial(E,I)} = \begin{bmatrix} \gamma + \delta + \lambda & 0 \\ -\gamma & \delta + \mu + k \end{bmatrix}$ so that

$|V| = (\gamma + \delta + \lambda)(\delta + \mu + k)$ and $V^{-1} = \frac{1}{(\gamma+\delta+\lambda)(\delta+\mu+k)}\begin{bmatrix} \delta + \mu + k & 0 \\ \gamma & \gamma + \delta + \lambda \end{bmatrix}$

Thus, the reproduction number $R_0 = \rho(FV^{-1}) = \left[\frac{\alpha(\delta+\mu+k)+\beta\gamma}{(\gamma+\delta+\lambda)(\delta+\mu+k)}\right] = \frac{\alpha\omega+\beta\gamma}{\eta\omega}$, where $\omega = \delta + \mu + k$ and $\eta = \gamma + \delta + \lambda$ (say).

Also, let us denote $\theta = \frac{(m\gamma+n\omega)\eta}{\gamma}$.

Theorem 3.1. *For model (1), with R_0 and θ defined as above,*

(i) when $R_0 > 1$ and $A < \theta$, there is a unique EE,

(ii) when $R_0 = 1$, there is no EE.

Proof. For EE, set the RHS of system (1) equal to zero. Note that the EE $E_1(S, E, I)$ satisfies $S > 0, E \geq 0, I > 0$, we have

$$A - \delta S - \left(\alpha - a\frac{E}{m+E}\right)\frac{S}{N}E - \left(\beta - a\frac{I}{n+I}\right)\frac{S}{N}I = 0 \tag{2}$$

$$-\gamma E + \left(\alpha - a\frac{E}{m+E}\right)\frac{S}{N}E + \left(\beta - a\frac{I}{n+I}\right)\frac{S}{N}I - (\delta+\lambda)E = 0 \tag{3}$$

$$\gamma E - kI - (\delta+\mu)I = 0 \tag{4}$$

Adding Equations (2), (3) and (4), we get

$$A - \delta N - \lambda E - (k+\mu)I = 0 \tag{5}$$

Now, $\lambda(4) + \gamma(5)$ gives, $[\lambda(\delta+\mu+k) + \gamma(\mu+k)]I = \gamma A - \gamma\delta N \Rightarrow I = \frac{\gamma(A-\delta N)}{\lambda\omega+\gamma(\omega-\delta)} = \frac{\gamma(A-\delta N)}{(\lambda+\gamma)\omega-\gamma\delta} = \frac{\gamma(A-\delta N)}{(\eta-\delta)\omega-\gamma\delta}$.

Using this, from Equation (5), we get $E = \frac{(\delta+\mu+k)}{\gamma}I = \frac{\omega(A-\delta N)}{(\eta-\delta)\omega-\gamma\delta}$. For convenience, let us say $P = \frac{(A-\delta N)}{(\eta-\delta)\omega-\gamma\delta}$ and hence write $I = \gamma P$and $E = \omega P$. Also $S = N - E - I$ gives

$$S = N - E - I = \frac{A - P\left(\overline{\eta-\delta}\omega - \gamma\delta\right)}{\delta} - \omega P - \gamma P = \frac{A}{\delta} - P\left(\frac{l}{\delta} + \omega + \gamma\right) \tag{6}$$

where for the sake of convenience in expression, say $\overline{\eta-\delta}\omega - \gamma\delta = l$. Now from Equation (3), we have

$$-\eta\omega P(A - Pl)[\omega\gamma P^2 + (m\gamma + n\omega)P + mn] + [\gamma(\alpha-a)\omega P^2 + (\alpha m\gamma + n\overline{\alpha-a})\omega P + \alpha mn][A - P\left(l + \delta\overline{\omega+\gamma}\right)]\omega P + \left[(\beta-a)\gamma\omega P^2 + \left(\beta n\omega + \overline{\beta-a}\right)\gamma mP + \beta mn\right]\left[A - \mathrm{P}\left(l + \delta\overline{\omega+\gamma}\right)\right]\gamma P = 0 \tag{7}$$

Since $P = 0$ corresponds to disease free equilibrium, therefore cancelling P throughout, and using $P = \frac{I}{\gamma}$ the above equation takes the form

$$A'I^3 + B'I^2 + C'I + D' = 0 \tag{8}$$

where

$$\begin{aligned} A' &= -l\gamma\eta\omega^2 + (\alpha-a)\gamma\eta\omega^3 + (\beta-a)\eta\gamma^2\omega^2,\ B' \\ &= [A\omega\gamma - l(m\gamma+n\omega)]\gamma\omega\eta + [(\alpha m\gamma + n\omega\overline{\alpha-a})\eta - (\alpha-a)\gamma A]\gamma\omega^2 \\ &+ \left[\left(\beta n\omega + m\gamma\overline{\beta-a}\right)\eta - (\beta-a)\gamma A\right]\gamma^2\omega, C' \\ &= [A(m\gamma+n\omega) - lmn]\omega\eta\gamma^2 + [\alpha mn\omega\eta - A(\alpha m\gamma + n\omega\overline{\alpha-a})]\omega\gamma^2 \\ &+ \left[\beta mn\omega\eta - A\left(\beta n\omega + \gamma m\overline{\beta-a}\right)\right]\gamma^3, D' = -Amn[\alpha\omega + \beta\gamma - \omega\eta]\gamma^3 \end{aligned}$$

Case (i) When $R_0 > 1$ and $A < \theta$, we have

$$A' = [\eta\omega(R_0 - 1) + (\omega+\gamma)(\delta - a)] > 0$$

$$\begin{aligned} B' = (R_0-1)\omega^2\gamma\eta[-A\gamma + (m\gamma+n\omega)\eta] + A(a\gamma^2\omega^2 + a^2\gamma^3\omega) + n\gamma\omega^3\eta(\delta-a) + m\gamma^3\omega\eta(\delta-a) \\ + m\omega^2\gamma^2\eta\delta + n\omega^2\gamma^2\eta\delta > 0 \end{aligned}$$

and

$$D' = -Amn\omega\eta\gamma^3(R_0 - 1) < 0$$

Therefore, the product of roots of Equation (8) i.e., $-\frac{D'}{A'} > 0$ therfore there are two possibilities: either one root is positive or all the three roots are positive. But the sum of roots i.e., $-\frac{B'}{A'} < 0$, therefore, we cannot have all roots positive. Henece there is only one positive root i.e., unique EE of the system(1) which is given by $E^1(S^*, E^*, I^*)$ where $S^* = \frac{A}{\delta} - P\left(\frac{l}{\delta} + \omega + \gamma\right), E^* = \omega P, I^* = \gamma P, \frac{(A-\delta N)}{(\eta-\delta)\omega-\gamma\delta} = P$.

Case (ii) When $R_0 = 1$, as above we have,

$$A' = (\omega+\gamma)(\delta - a) > 0$$

$$B' = A(a\gamma^2\omega^2 + a^2\gamma^3\omega) + (\delta - a)\gamma\omega\eta(n\omega^2 + m\gamma^2) + m\omega^2\gamma^2\eta\delta + n\omega^2\gamma^2\eta\delta$$

$$C' = A(an\omega^2\gamma^2 + am\gamma^4) + mn(\omega\eta\gamma)^2\delta(\omega + \gamma) > 0$$

$$D' = 0$$

Therefore, Equation (8) becomes $I(A'I^2 + B'I + C') = 0$. Since $I' = 0$ corresponds to disease free equilibrium, therefore for endemic equilibrium, we consider $A'I^2 + B'I + C' = 0$ which is a quadratic equation with sum of roots $-\frac{B'}{A'} < 0$ and the product of roots $\frac{C'}{A'} > 0$. Therefore, no positive root of this equation exists and hence no EE exists for $R_0 = 1$. □

4. Stability analysis

In this Section we will evaluate the local stability of the model (1).

Theorem 4.1. *The DEF of the model (1) is Locally asymptotically stable (LAS) if* $R_0 < 1$*, and unstable if* $R_0 > 1$.

Proof. The Jacobian matrix $J = [j_{ik}]$of the linearization of system (1) at point arbitrary point (S, E, I) is

$$J(S,E,I) = \begin{bmatrix} -\delta - \left(\alpha - \frac{aE}{m+E}\right)\frac{E}{N} - \left(\beta - \frac{aI}{n+I}\right)\frac{I}{N} & -\left[\alpha - \frac{aE}{m+E} - \frac{amE}{(m+E)^2}\right]\frac{S}{N} & -\left[\beta - \frac{aI}{n+I} - \frac{anI}{(n+I)^2}\right]\frac{S}{N} \\ -\left[\beta - \frac{aI}{n+I} - \frac{anI}{(n+I)^2}\right]\frac{S}{N} & -(\gamma+\delta+\lambda) + \left[\alpha - \frac{aE}{m+E} - \frac{amE}{(m+E)^2}\right]\frac{S}{N} & \frac{S}{N}\left[\beta - \frac{aI}{n+I} - \frac{anI}{(n+I)^2}\right]\frac{S}{N} \\ 0 & \gamma & (\delta+\mu+k) \end{bmatrix}$$

Thus, at DFE $E_0 = \left(\frac{A}{\delta}, 0, 0\right)$, we have $J\left(\frac{A}{\delta}, 0, 0\right) = \begin{bmatrix} -\delta & -\alpha & -\beta \\ 0 & -(\gamma+\delta+\lambda) & +\alpha\beta \\ 0 & \gamma & -(\delta+\mu+k) \end{bmatrix}$

Its characteristic equation is $(x + \delta)(x^2 + a_1x + a_2) = 0$ (9)

where, $a_1 = \omega + \eta\left(1 - \frac{\alpha}{\eta}\right) > 0$ and $a_2 = -(\omega\alpha + \beta\gamma - \omega\eta) = \omega\eta[1 - R_0] > 0$. □

Thus clearly, Equation (9), has one real root $x = -\delta$ and other two roots negative or complex conjugate with negative real parts as $a_1 > 0$and $a_2 > 0$. Therefore, all the eigenvalues of $J\left(\frac{A}{\delta}, 0, 0\right)$ i.e., $J(E_0)$ have negative real parts, and by Ruth Hurwitz criteria, E_0 is LAS equilibrium point of Equation (1).

Remark 4.1. From Equation (9), we see that if $R_0 = 1$, then one eigenvalue of the Jacobian matrix $J(E_0)$is zero with multiplicity one, and the other two values are $x = -\delta$ and $x = -\omega - \eta + \alpha = -\frac{1}{\omega}(\omega^2 + \omega\eta - \alpha\omega) = -\frac{1}{\omega}(\omega^2 + \beta\gamma)$, which are real and negative. So, E_0 is marginally locally stable. More precisely, as R_0 increases through 1, there is an exchange of stability between DFE and the EE (which is biologically meaningless if $R_0 < 1$). Hence the equilibrium infective and infectious population sizes depend continuously on R_0 and there is a forward, or transcritical, bifurcation in equilibrium behavior, at $R_0 = 1$.

Theorem 4.2. *The DFE of the model (1) is globally asymptotically stable (GAS) if* $R_0 \leq 1$ *and* $R_0 > 1$.

Proof. Since, $L = \frac{\alpha\omega+\beta}{\eta}E - \beta I$, its derivative along the solutions of the system (1) is

$$L' = (\alpha E + \beta I)\omega\left[R_0\frac{S}{N} - 1\right] - \left(a\frac{E^2}{m+E} + a\frac{I^2}{n+I}\right)\frac{S}{N}.$$

Clearly, as $\frac{S}{N} \leq 1$, for $R_0 \leq 1, L' \leq 0$. Also $L' = 0$ only if $E = I = 0$. Therefore, the maximum invariant set in ϕ is the singleton set $\{E_0\}$ and hence the global stability of $E_0$when $R_0 \leq 1$ follows from LaSalle's invariance principle.

In the next theorem, we will prove the asymptotic stability of the EE $E_1(S^*, E^*, I^*)$ and for that, the following notations have been used.

$$P_1 = -\delta - \left(\alpha - \frac{aE^*}{m+E^*}\right)\frac{E^*}{N^*} - \left(\beta - \frac{aI^*}{n+I^*}\right)\frac{I^*}{N^*}, P_2 = -\left[\alpha - \frac{aE^*}{m+E^*} - \frac{amE^*}{(m+E^*)^2}\right]\frac{S^*}{N^*},$$

$$P_3 = -\left[\beta - \frac{aI^*}{n+I^*} - \frac{anI^*}{(n+I^*)^2}\right]\frac{S^*}{N^*}, P_4 = \left(\alpha - \frac{aE^*}{m+E^*}\right)\frac{E^*}{N^*} + \left(\beta - \frac{aI^*}{n+I^*}\right)\frac{I^*}{N^*},$$

$$P_5 = -\eta + \left[\alpha - \frac{aE^*}{m+E^*} - \frac{amE^*}{(m+E^*)^2}\right]\frac{S^*}{N^*}, P_6 = \left[\beta - \frac{aI^*}{n+I^*} - \frac{anI^*}{(n+I^*)^2}\right]\frac{S^*}{N^*}, P_7 = 0, P_8 = \gamma, P_9 = -\omega.$$

Theorem 4.3. *The EE of the model (1) is LAS if the following conditions hold true:*

k, the rate constant of segregating after disease and hence ω is chosen satisfying:

$$\frac{1}{(\delta + P_1)}\left(-\eta + P_1 + P_2 + \frac{P_3\gamma}{\omega - P_1}\right) = 1 + \frac{P_2}{\omega - P_1} + \frac{\gamma}{P_3} \tag{10}$$

$$P_3(P_1 - \omega - P_2) > \gamma(\omega - P_1) \tag{11}$$

Let us choose η and a_{31} such that

$$\eta + P_2 > 0, \frac{P_3}{\omega - P_1} > \frac{\omega}{P_3}, \left|\frac{P_3}{\omega - P_1}\right|^2 < 2L,$$

where,

$$L = 1 - \frac{1}{2\left(1 + \frac{P_2}{\omega - P_1} + \frac{\gamma}{P_3}\right)} \tag{12}$$

Proof. Let $x = S - S^*, y = E - E^*, z = I - I^*$ be small perturbations about the E_1. Using these new variables, we linearize the model (1) around the E_1 i.e.,

$$\dot{x} = P_1x + P_2y + P_3z, \qquad \dot{y} = P_4x + P_5y + P_6z, \dot{z} = P_7x + P_8y + P_9z \tag{13}$$

$$\dot{V} = (\nabla V)^T[\dot{x}\dot{y}\dot{z}]^T$$

$$= (a_{11}P_1 + a_{21}P_4)x^2 + (a_{11}P_2 + a_{21}P_5 + a_{31}\gamma + a_{12}P_1 + a_{22}P_4)\text{xy}$$
$$+ (a_{11}P_3 + a_{21}P_6 - a_{31}\omega + a_{13}P_1 + a_{23}P_4)\text{zx} + (a_{12}P_2 + a_{22}P_5 + a_{32}\gamma)y^2$$
$$+ (a_{12}P_3 + a_{22}P_6 - a_{32}\omega + a_{13}P_2 + a_{23}P_5 + a_{33}\gamma)yz + (a_{31}P_3 + a_{23}P_6 - a_{33}\omega)z^2$$

Now we will select the $P_{i's}$and $a_{ij's}$ such that coefficients of xy, yz and zx vanish and those of $x^2, y^2$and z^2 are all negative. Also curl conditions $\frac{\partial \nabla V_1}{\partial y} = \frac{\partial \nabla V_2}{\partial x}$, etc. are also satisfied i.e., $a_{ij} = a_{ji}\ \forall 1 \leq i, j \leq 3, i \neq j$ and

$$a_{11}P_1 + a_{21}P_4 < 0 \tag{14a}$$

$$a_{12}P_2 + a_{22}P_5 + a_{32}\gamma < 0 \tag{14b}$$

$$a_{31}P_3 + a_{23}P_6 - a_{33}\omega < 0 \tag{14c}$$

$$a_{11}P_2 + a_{21}P_5 + a_{31}\gamma + a_{12}P_1 + a_{22}P_4 = 0 \tag{14d}$$

$$a_{11}P_3 + a_{21}P_6 - a_{31}\omega + a_{13}P_1 + a_{23}P_4 = 0 \tag{14e}$$

$$a_{12}P_3 + a_{22}P_6 - a_{32}\omega + a_{13}P_2 + a_{23}P_5 + a_{33}\gamma = 0 \tag{14f}$$

Let us choose $a_{11} = 2, a_{21} = a_{12} = 1, a_{23} = a_{32} = 0, a_{33} = 1$

Note that Equations (i) and (ii) follows by the fact that $P_1, P_2, P_3 < 0$ and hypothesis (12). Also, if we select

$$a_{31} = \frac{P_3}{\omega - P_1} (< 0) \tag{14g}$$

it can be seen easily that (iii) and (v) are valid.

From (iv), $2P_2 + P_5 + a_{31}\gamma + P_1 + a_{22}P_4 = 0$, then using the fact that

$$P_2 + P_5 = -\eta \tag{14h}$$

$$a_{22} = \frac{1}{(\delta + P_1)}\left(-\eta + P_1 + P_2 + \frac{P_3\gamma}{\omega - P_1}\right) \tag{14i}$$

From (vi), $P_3 + a_{22}P_6 + a_{13}P_2 + a_{33}\gamma = 0$, then using the fact that $P_3 + P_6 = 0$ and (vii), we get

$$a_{22} = 1 + \frac{P_2}{\omega - P_1} + \frac{\gamma}{P_3} \tag{14j}$$

Clearly assumption in hypothesis (10) validate (viii) & (ix) and hence (iv) and (vi) are valid. Again, by hypothesis (12) and (iv), we observe that

$$a_{31} = \frac{\eta - P_2 - P_1 - a_{22}P_4}{\gamma} > \frac{\omega}{P_3},\ a_{22} > \frac{1}{(P_1+\delta)}\left(\frac{\omega\gamma}{P_3} - \eta + P_1 + P_2\right) > 0 \text{ (since } P_1, P_2, P_3 < 0)$$

Also, from (ix), $a_{22} < 1$ (last two terms are -ve). Here we note that $0 < a_{22} < 1$ and in particular if we select $\frac{1}{2} < a_{22} < 1$.

Thus,

$$\nabla V = \begin{bmatrix} a_{11}x + a_{12}y + a_{13}z \\ a_{21}x + a_{22}y + a_{23}z \\ a_{31}x + a_{32}y + a_{33}z \end{bmatrix} = \begin{bmatrix} 2x + 1y + a_{13}z \\ 1x + a_{22}y + 0z \\ a_{13}x + 0y + 1z \end{bmatrix}$$

Therefore,

$$V = \left(\frac{1}{\sqrt{2a_{22}}}\right)^2 x^2 + xy + \left(\sqrt{\frac{a_{22}}{2}}\right)^2 y^2 + L\left(x^2 + \frac{a_{13}}{L}xz + \frac{z^2}{2L}\right)$$

$$= \left(\frac{1}{\sqrt{2a_{22}}}x + \sqrt{\frac{a_{22}}{2}}y\right)^2 + L\left(x + \frac{a_{13}}{2L}z\right)^2 + \left(\frac{2L - {a_{13}}^2}{4L}\right)z^2$$

where $L = \left(1 - \frac{1}{2a_{22}}\right) > 0$. Here first two terms, being perfect squares and the last term above via assumption in hypothesis (12) are positive. Therefore, $V > 0$ and hence the theorem follows. □

5. Numerical simulations and discussions

In this Section, we present computer simulation results for model Equation (1) by using MATLAB 16.0.

5.1. Existence of DFE for $R_0 \leq 1$ and EE for $R_0 > 1$

The following set of values of parameters satisfying the conditions of theorem 3.1 ensuring the existence of DFE and EE are chosen: For Equation (1), using parameters in **Table 1 and Figure 2** along with a = 0.015, when α=0.15, by computing, we get R_0 = 0.8 < 1 and the Equation (1) has a DFE when α = 0.19, by computing, we get R_0 = 1 and the Equation (1) has a DFE. When α = 0.7 and λ = 0.4 so that η = 0.5 by computing, we get R_0 = 1.42 and θ = 190 satisfying the condition A (=80) < θ and Equation (1) has endemic equilibrium (3476.048, 20.95808, 2.994012).

Table 1. Parameters for the simulation.

S.N.	Param.	m	n	α	β	δ	μ	κ	ω	γ	λ	η	R_0	A	N
1	$R_0 < 1$	100	40	0.15	0.07	0.02	0.3	0.24	0.56	0.08	0.1	0.2	0.8	80	4000
2	$R_0 = 1$	100	40	0.19	0.07	0.02	0.3	0.24	0.56	0.08	0.1	0.2	1	80	4000
3	$R_0 > 1$	100	40	0.7	0.07	0.02	0.3	0.24	0.56	0.08	0.4	0.5	1.42	80	4000

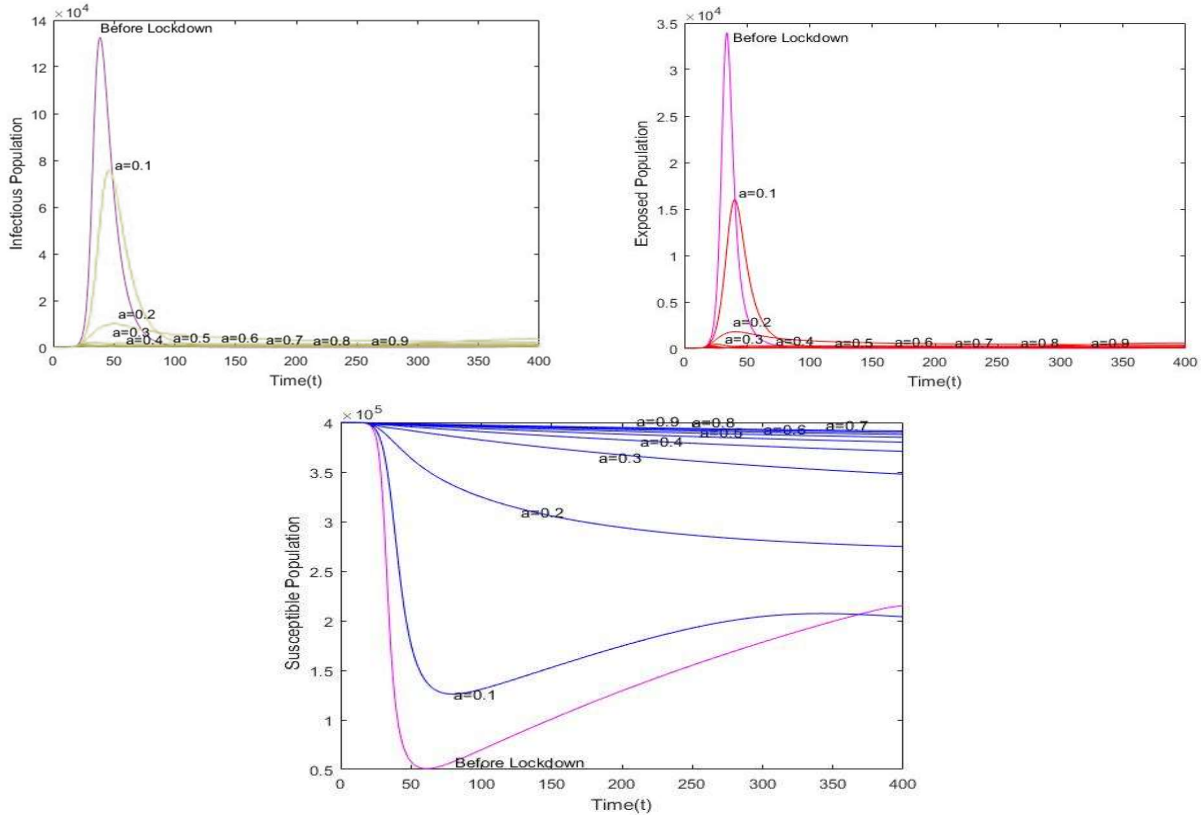

Figure 2. Plots of Infectious, Exposed and Susceptible population v/s time for S. N. 1 of **Table 1**.

5.2. Impact of Implementation of Lockdown in case of EE i.e., $R_0 > 1$

Different values of the parameter are reflecting the rate of implementation of lockdown are taken, as $a = 0, 0.1, 0.2, 0.3, \ldots, 0.9$ with i.c.'s (S, E, I) are (39999, 0, 1) and $N^* = 38000$. The EE point for the values of S. N. 1 of **Table 2** exists as $E_1(S^*, E^*, I^*)$=E_1 (315201.1, 917.758, 5562.17).

For the same values of parameter a, i.c.'s and N^*as used for S.N.1 of **Table 2**, we see that the endemic equilibrium point for the set of values of S. N. 2 of **Table 2** and **Figure 3** exists as $E_1(S^*, E^*, I^*) = E_1$ (36630.24, 82.08925, 1287.675).

Table 2. Parameters for the simulation.

S. N.	m	n	α	β	δ	μ	κ	ω	γ	λ	η	θ	R_0	A	N
1	1000	800	0.9	0.2	0.002	0.03	0.1	0.132	0.8	0.001	0.803	909	2.63	800	400000
2	1000	800	0.2	0.9	0.02	0.03	0.001	0.051	0.8	0.001	0.821	862.87	17.4	800	40000

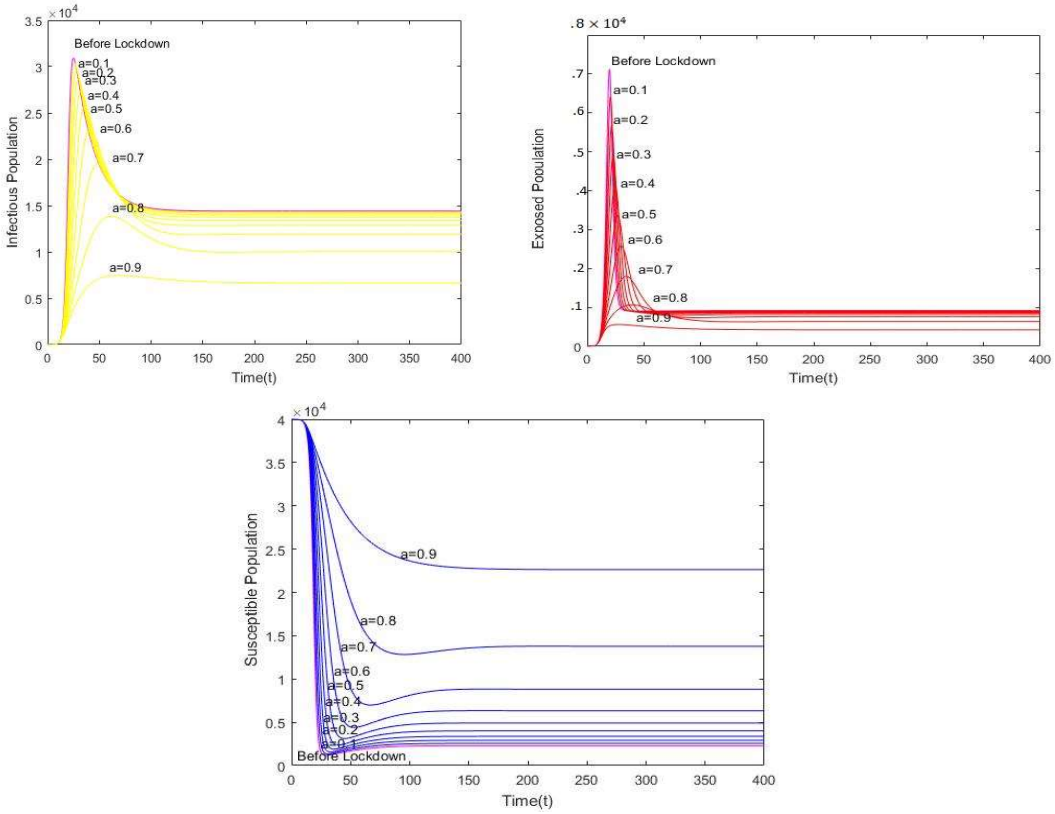

Figure 3. Plots of Infectious, Exposed and Susceptible population v/s time for S. N. 2 of **Table 2**.

5.3. Global asymptotic stability of DEF

Using the parameters in **Table 1**, S. N. 1 along with different i.c's: (1800, 1400, 800), (200, 1500, 500), (2000, 1300, 700), (2000, 1000, 1000), (2000, 1100, 900), (2500, 500, 1000), (2200, 1000, 800), (2500, 1000, 500), (1500, 1400, 1100). Jacobian curves of S, E and I with t in the **Figure 4**, validating theorem 4.2 shows the dynamics of Equation (1) when $R_0 < 1$.

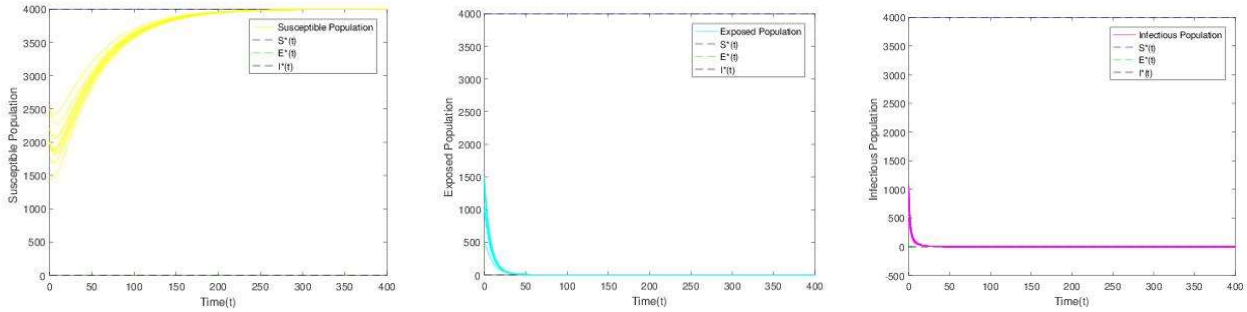

Figure 4. GAS of DFE for $R_0 = 0.8 < 1$, when $\alpha = 0.015$ and $\alpha = 0.15$.

Using the parameters in **Table 1**, S. N. 2 along with the i.c.'s stated above in this subsection, variational curves of S, E and I with t in the **Figure 5** validate the results of theorem 4.2 when $R_0 = 1$.

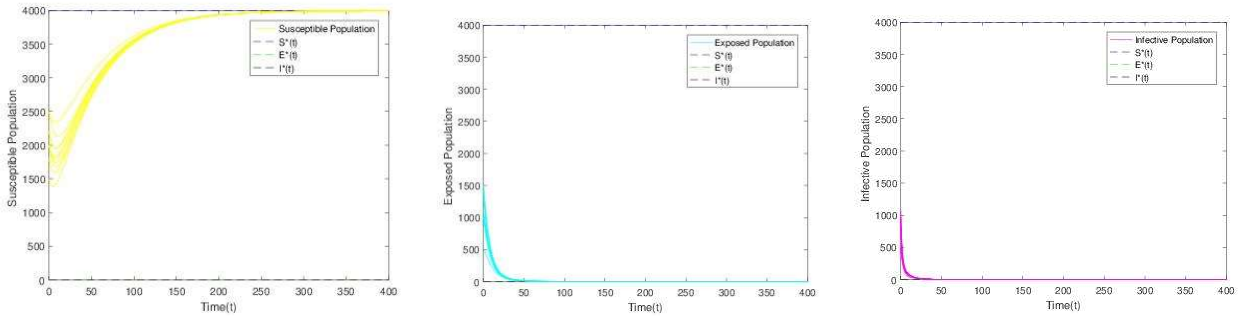

Figure 5. GAS of DFE for $R_0 = 1, when\ a = 0.0015$ and $\alpha = 0.19$.

6. Discussion and conclusion

In this manuscript, we have formulated an SEI mathematical epidemic model combined with the impact of lockdown on COVID-19, taking into consideration the fact that the disease is infectious in a latent period too. For this model, we have found R_0 and shown that if this parameter is less than 1, then DFE is GAS. Under certain restrictions, the existence of an EE for $R_0 > 1$, followed by conditions ensuring the local asymptotic stability of EE is presented. By means of the results of the work and the numerical simulation done in Section 5, it has been verified that the lockdown significantly decreases the transmission rate of the disease. Since R_0 is independent of a, m and n, it can be said that the lockdown does not change the reproduction number.

Author contributions

Conceptualization, PK, KPD, SS, BD and RK; methodology, PK, KPD, SS, BD and RK; software, PK, KPD, SS, BD and RK; validation, PK, KPD, SS, BD and RK; formal analysis, PK, KPD, SS, BD and RK; investigation, PK, KPD, SS, BD and RK; resources, PK, KPD, SS, BD and RK; data curation, PK, KPD, SS, BD and RK; writing—original draft preparation, PK, KPD, SS, BD and RK; writing—review and editing, PK, KPD, SS, BD and RK; visualization, PK, KPD, SS, BD and RK; supervision, PK, KPD, SS, BD and RK; project administration, PK, KPD, SS, BD and RK; funding acquisition, PK, KPD, SS, BD and RK. All authors have read and agreed to the published version of the manuscript.

Conflict of interest

The authors declare no conflict of interest.

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