Mathematical Modelling of COVID-19 Pandemic with Demographic Effects

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Abstract

In this paper, an asymptomatic infection transmission Susceptible-Exposed-Infectious-Recovered (SEIR) model with demographic effects is used to understand the dynamics of the COVID-19 pandemics. We calculate the basic reproduction number ($R_0$) and prove the global stability of the model by solving the differential equations of the model using the disease-free equilibrium (DFE) and endemic equilibrium (EE) equations, respectively. We showed that when $R_0 < 1$ or $R_0 \leq 1$ and $R_0 > 1$ or $R_0 \geq 1$ the DFE and EE asymptotic stability exist theoretically and numerically respectively. We also demonstrate the detrimental impact of the direct and asymptomatic infections for the COVID-19 pandemic.

Keywords: Mathematical modelling, COVID-19, demographic effects, asymptotic stability

1. Introduction

The COVID-19 is a novel flu infection belong to the Coronaviruses family that causes illness ranging from a common cold to severe illness in humans like the Middle East Respiratory Syndrome (MERS) and Severe Acute Respiratory Syndrome (SARS) in adults and children [1,2]. The COVID-19 started in the city of Wuhan, Hubei Province, China, in 2019 and has spread to all parts of the world, affecting 213 countries and territories [3]. It is the third coronavirus species to infect human populations in the past two decades [4–6]. As of 10 June 2020, there have been global confirmed cases of 7,145,539, and 408,025 resulted in deaths[3]. Symptoms of the virus are fever, cough, shortness of breath, fatigue, body aches, headache, the loss of taste or smell, sore throat, congestion or runny nose, nausea or vomiting, and diarrhea [7]. Close contact and respiratory droplets within 6 feet (1.8 m) approximately are the most common primary ways of transmission[8].

However, people who contracted the virus can take between 2 to 14 days before signs and symptoms manifested [7], and within this periods they can infect others[9,10]. That is an asymptomatic individuals can infect an uninfected person. Although most of those infected get cured, there is currently no vaccine or specific antiviral therapy to prevent contacting the virus. In a mild case, usual flu treatments like antibiotic drugs are used, and in severe cases, supportive treatment like a breathing machine is given to protect vital functions of the organs. The virus infected all ages of humans, but the higher risk is more on adult individuals with severe illness relating to respiratory diseases, organ diseases, and blood diseases [7].

The basic reproduction number ($R_0$) is a critical threshold quantity associated with viral transmissibility, and it has been used to understand the transmission of the COVID-19.
Epidemiological $R_0$ is an equation used to describe the contagiousness of the pathogen. It determined the number of people on average that would be infected from a case introduced into a population. The initial COVID-19 pandemic $R_0$, according to the World Health Organization (WHO) was estimated to be between ranges of 1.4 to 2.5[6]. That is, one infected person will infect an average of 2 persons in his/her lifetime. Also, in other studies of the epidemic, Zhao et al.[11] estimated the average $R_0$ for COVID-19, from 3.3 to 5.5, and Read et al.[12], estimated to range between 3.6 and 4.0.

Stability analysis which has a direct relationship with $R_0$ is also another way to understand infectious disease. It is believed that when $R_0$ is above unity, the disease will persist, and the stability is endemic, and when $R_0$ less than unity, the disease will die out, and the stability is disease-free. The analysis is done by partition the state of individuals in the population into different compartments. For instance, since COVID-19 have an incubation period, the population can be divided into those who are susceptible (S) to the virus, those who are exposed (E) to the virus, those who are infected (I) with the virus and those recovered (R) from the illness. The SEIR is interpreted using differential equations, where calculus and simple algebraic methods are used to study the dynamic of the disease. However, during these approaches the $R_0$ is calculated directly from the differential equations model at the state when the disease is free from the population.

In this study, a deterministic four compartments SEIR model with an asymptomatic infection transmission is used to inspect the stability of the COVID-19 pandemic using differential equations techniques. This is done by formulating four nonlinear differential equations and provides a theoretical and numerical analysis of the model. Our results show that, theoretically, the disease-free and endemic equilibria of the model locally and globally asymptotically stable and the direct and symptomatic infection transmissions are detrimental for the COVID-19 pandemic.

2. Model framework

In this section, we describe an epidemic transmission SEIR model with demographic changes. The model is used in epidemiology to compute the amount of susceptible, exposed (infected), infectious, recovered people in a population ($N$). This model is used under the following assumptions:

✓ The population is constant but large.
✓ The only way a person can leave the susceptible state \((S)\) is to become infected either from the exposed \((E)\) or infectious \((I)\) state.

✓ The only way a person can leave the \(E\) state is to show signs and symptoms of the illness or die of natural death.

✓ The only way a person can leave the \(I\) state is to recover from the disease or die from natural death or die as a result of the disease.

✓ A person who recovered \((R)\) from the illness received permanent immunity.

✓ Age, sex, social status, and race do not affect the probability of being infected.

✓ The member of the population has the same contacts with one another equally.

✓ All births are into the susceptible state, and it is assumed that the birth and natural death rates are equal.

The transmission is measured at \(S\beta(I + \kappa E)/N\), where \(\beta\) is the direct transmission rate, and \(\kappa\) is the probability of getting infected when an uninfected individual comes into contact with an individual from state \(E\). We assume natural birth and death rate to be measured at an equal rate \(\mu\) and the disease induced death rate measure at \(\delta\). The rate for an individual to move from state \(E\) to state \(I\) is measured at rate \(\sigma\), and the rate of recovery is measured at \(\gamma\). Figure 1 represents the SEIR model with an asymptomatic infection flow diagram.

\[\begin{align*}
\frac{dS(t)}{dt} &= \mu N - \beta \frac{S(I + \kappa E)}{N} - \mu S, \\
\frac{dE(t)}{dt} &= \frac{S(I + \kappa E)}{N} - (\mu + \sigma)E, \\
\frac{dI(t)}{dt} &= \sigma E - (\mu + \gamma + \delta)I, \\
\frac{dR(t)}{dt} &= \gamma I - \mu R,
\end{align*}\]  

(1)

**Figure 1.** The SEIR model with an asymptomatic infection flow diagram.
Where $S(t) = S, E(t) = E, I(t) = I$ and $R(t) = R$, denote the number of susceptible, exposed, infectious, and remove individuals at time $t$, respectively, and $N = S + E + I + R$. System (1) is subjected to the initial condition

$$S(0) = S_0, \ E(0) = E_0, I(0) = I_0, R(0) = R_0.$$  \hfill (2)

For simplicity system (1) is reduced to a proportional framework given as

$$\frac{ds(t)}{dt} = \mu - \beta s(i + \kappa e) - \mu s,$$
$$\frac{de(t)}{dt} = \beta s(i + \kappa e) - (\mu + \sigma)e,$$
$$\frac{di(t)}{dt} = \sigma e - (\mu + \gamma + \delta)i,$$
$$\frac{dr(t)}{dt} = \gamma i - \mu r,$$  \hfill (3)

where $s = S/N, e = E/N, i = I/N$, and $r = r/N$. By considering the total population

$$s + e + i + r = 1 \Rightarrow r = 1 - s - e - i,$$

system (3) can be reduced to

$$\frac{ds(t)}{dt} = \mu - \beta s(i + \kappa e) - \mu s,$$
$$\frac{de(t)}{dt} = \beta s(i + \kappa e) - (\mu + \sigma)e,$$
$$\frac{di(t)}{dt} = \sigma e - (\mu + \gamma + \delta)i.$$  \hfill (4)

3. **Positivity of the solution**

Assume that system (4) has a global solution corresponding to non-negative initial conditions. Then the solution is non-negative at all times. The statement is confirmed by the following Lemma.

**Lemma 1.** If $s(0) \geq 0, e(0) \geq 0$ and $i(0) \geq 0$ then the solution $s(t), e(t)$ and $i(t)$ are all positive for all $t \geq 0$.

**Proof.** We use the contradiction concept by assuming there exists positive real $t_1, t_2$ and $t_3$ for which the following conditions hold:

I. $s(t_1) = 0, ds(t_1)/dt < 0$, and for all $0 \leq t \leq t_1$ one has that $e(t) \geq 0$ and $i(t) \geq 0$;

II. $e(t_2) = 0, de(t_2)/dt < 0$, and for all $0 \leq t \leq t_2$ one has that $s(t) \geq 0$ and $i(t) \geq 0$;
III. \( i(t_3) = 0, di(t_3)/dt < 0, \) and for all \( 0 \leq t \leq t_3 \) one has that \( s(t) \geq 0 \) and \( e(t) \geq 0. \)

Condition (I) contradict if \( s(t) \geq 0, ds(t_1)/dt = \mu > 0. \) Also, condition (II) contradicts because 
\( e(t) \geq 0, de(t_2)/dt = \beta si \geq 0. \) Finally, condition (III) contradict since for \( i(t) \geq 0, di(t_3)/dt = \sigma E \geq 0. \) Thus, the solutions of \( s(t), e(t) \) and \( i(t) \) remains positive for all \( t > 0. \)

Hence the positively invariant for the system (4) is
\[ \Omega = \{ s(t), e(t), i(t) \in R^3, s(t), +e(t) + i(t) \leq 1 \}. \] (5)

3.1 The equilibria of the model

There are two equilibrium points for the system (4), i.e., the disease-free equilibrium (DFE), the state when the disease is absence, and the endemic equilibrium (EE), which is the state when the disease continues to persist in the population. To understand the stability of the model we need an expression to estimate the basic reproduction number \( (R_0). \)

Let the DFE points of the model denoted as \( E^0 = (s^0, e^0, i^0) \) and represent system (4) at \( E^0 \) as
\[
\mu - \beta s^0(i^0 + \kappa e^0) - \mu e^0 = 0,
\beta s^0(i^0 + \kappa e^0) - (\mu + \sigma)e^0 = 0,
\sigma e^0 - (\mu + \gamma + \delta)i^0 = 0.
\] (6)

In terms of \( i^0 \), from the last equation of (6), we get
\[ e^0 = \frac{(\mu + \gamma + \delta)i^0}{\sigma}. \]

Adding the first two equations of (6) and substitute for \( e^0 \) we get
\[ s^0 = 1 - \frac{(\mu + \sigma)(\mu + \gamma + \delta)i^0}{\mu\sigma}. \]

Because at the disease-free state no one have the infection then, \( i^0 = 0. \) We can see that \( E^0 = (s^0, e^0, i^0) = (1,0,0). \)

Substitute \( s^0 \) and \( e^0 \) into the second equation of (6), we get
\[
\beta i^0 \left( 1 + \frac{\kappa(\mu + \gamma + \delta)}{\sigma} \right) \left( 1 - \frac{(\mu + \sigma)(\mu + \gamma + \delta)i^0}{\mu\sigma} \right) - \frac{(\mu + \sigma)(\mu + \gamma + \delta)i^0}{\sigma} = 0. \] (7)

By expanding the bracket of (7) and simplify we get
\[
\beta \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) i^0 - \beta \left( \frac{\mu + \sigma}{\mu \sigma} \right) \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) (i^0)^2 - \frac{\mu + \sigma}{\sigma} (\mu + \gamma + \delta) i^0 = 0 ,
\]

Divide (8) by \( i^0 \) we get
\[
\beta \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) - \beta \left( \frac{\mu + \sigma}{\mu \sigma} \right) \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) i^0 - \frac{\mu + \sigma}{\sigma} (\mu + \gamma + \delta) = 0 ,
\]

Substitute \( i^0 = 0 \), from \( E^0 = (s^0, e^0, i^0) = (1,0,0) \) we get
\[
\frac{\sigma \beta}{(\mu + \sigma)(\mu + \gamma + \delta)} \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) = 1.
\]

Since the threshold for \( R_0 \) is unity (1), we then assume
\[
R_0 = \frac{\sigma \beta}{(\mu + \sigma)(\mu + \gamma + \delta)} \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) .
\]

Also, the EE points are denoted as \( E^* = (s^*, e^*, i^*) \), where \( s^*, e^* \) and \( i^* \) are calculated by letting \( s^0 = s^* \), \( e^0 = e^* \), \( i^0 = i^* \) from (6) and we get
\[
\mu - \beta s^*(i^* + \kappa e^*) - \mu s^* = 0 ,
\]
\[
\beta s^*(i^* + \kappa e^*) - (\mu + \sigma) e^* = 0 ,
\]
\[
\sigma e^* - (\mu + \gamma + \delta) i^* = 0 .
\]

Following (6), then (9) becomes
\[
\beta \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) - \beta \left( \frac{\mu + \sigma}{\mu \sigma} \right) \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) i^* - \frac{\mu + \sigma}{\sigma} (\mu + \gamma + \delta) = 0 .
\]

Divide both sides by \( \sigma/(\mu + \sigma)(\mu + \gamma + \delta) \) we get
\[
\frac{\sigma \beta}{(\mu + \sigma)(\mu + \gamma + \delta)} \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) - \frac{\beta}{\mu} \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) i^* - 1 = 0 ,
\]

Substituting \( R_0 \) we get
\[
\frac{\beta}{\mu} \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) i^* = R_0 - 1 ,
\]
Hence

\[ i^* = \frac{\mu}{\beta} \left( \frac{\sigma}{\sigma + \kappa(\mu + \gamma + \delta)} \right) (R_0 - 1), \]

also

\[ e^* = \left( \frac{(\mu + \gamma + \delta)}{\sigma} \right) i^*, \]

\[ s^* = 1 - \frac{\mu(\mu + \sigma)(\mu + \gamma + \delta)i^*}{\sigma}. \]

### 3.2 Stability analysis of the Disease-free equilibrium points

**Theorem 1.** If \( R_0 < 1 \) and \( \kappa \beta < (\mu + \sigma) + (2\mu + \gamma + \delta + \sigma) \) the DFE is locally asymptotically stable in \( \Omega \).

**Proof.** The Jacobian matrix of system (3) associated with DFE is given as

\[
 J_{(1,0,0)} = \begin{pmatrix} -\mu & -\kappa \beta & -\beta \\ 0 & \kappa \beta - (\mu + \sigma) & \beta \\ 0 & \sigma & -(\mu + \gamma + \delta) \end{pmatrix}, \tag{13}
\]

with characteristic polynomial

\[
 P(\omega) = (\mu + \omega)[\omega^2 + ((\mu + \sigma) - \kappa \beta + (\mu + \gamma + \delta))\omega + ((\mu + \sigma) - \kappa \beta)(\mu + \gamma + \delta) - \sigma \beta],
\]

where \( \omega \) is the eigenvalues and Theorem 1 can satisfy if and only if

\[
 (\mu + \sigma) - \kappa \beta + (\mu + \gamma + \delta) > 0 \implies \kappa \beta < (\mu + \sigma) + (2\mu + \gamma + \delta + \sigma),
\]

and

\[
 ((\mu + \sigma) - \kappa \beta)(\mu + \gamma + \delta) - \sigma \beta > 0 \implies R_0 = \frac{\beta}{(\mu + \sigma)(\mu + \gamma + \delta + \kappa)} < 1.
\]

The proof of Theorem 1 is complete.

**Theorem 2.** If \( R_0 \leq 1 \), the DFE is globally asymptotically stable in \( \Omega \).

**Proof.** To prove the global asymptotic stability (GAS) of the DFE, we construct the following Lyapunov function \( V: \Omega \to R \), where \( V(s,e,i) = i(t) \). Then the time derivative of \( V \) is given as

\[
 \frac{dV}{dt} = \frac{di^0}{dt} = \frac{de^0}{dt},
\]

since at the equilibrium points \( di^0/dt = de^0/dt = 0 \). Therefore

\[
 \frac{dV}{dt} = \beta s^0(i^0 + \kappa e^0) - (\mu + \sigma)e^0.
\]
Using (8) we get

\[
\frac{dV}{dt} = \beta \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) i^0 - \beta \left( \frac{\mu + \gamma + \delta}{\mu \sigma} \right) \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) (i^0)^2
\]

\[
- \frac{(\mu + \sigma)(\mu + \gamma + \delta) i^0}{\sigma},
\]

(14)

By factorization method

\[
\frac{dV}{dt} = \left( \frac{(\mu + \sigma)(\mu + \gamma + \delta)}{\sigma} \right) i^0 \left[ \frac{\sigma \beta}{(\mu + \sigma)(\mu + \gamma + \delta)} \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) - \frac{(\mu + \sigma)(\mu + \gamma + \delta)}{\sigma} \left( \frac{\sigma \beta}{(\mu + \sigma)(\mu + \gamma + \delta)} \left( 1 + \frac{\kappa (\mu + \gamma + \delta)}{\sigma} \right) i^0 - 1 \right) \right].
\]

Substituting $R_0$ we get

\[
\frac{dV}{dt} = \left( \frac{(\mu + \sigma)(\mu + \gamma + \delta)}{\sigma} \right) i^0 \left[ R_0 - \frac{(\mu + \sigma)(\mu + \gamma + \delta) R_0}{\mu \sigma} i^0 - 1 \right].
\]

(15)

Thus, for $dV/dt \leq 0$, if $i^0 = 0$, then $R_0 \leq 1$. According to the Lasalle invariance principle[13], the DFE point is GAS.

3.3 Stability analysis of the endemic equilibrium

Theorem 2: If $R_0 > 1$ the endemic equilibrium is locally asymptotically stable.

Proof. To prove the LAS of the endemic equilibrium we consider the Jacobian matrix associated with $E^*$, that is

\[
J_{E^*} = \begin{pmatrix}
-\mu - \beta (i^* + k e^*) & -\kappa \beta s^* & -\beta s^* \\
\beta (i^* + k e^*) & (\mu + \gamma + \delta) & \beta s^* \\
0 & \sigma & (\mu + \gamma + \delta)
\end{pmatrix},
\]

(16)

Substituting for $s^*$, $e^*$ and $i^*$ we get

\[
J_{E^*} = \begin{pmatrix}
\mu R_0 & -\frac{\kappa \beta}{R_0} & -\frac{\beta}{R_0} \\
\mu (R_0 - 1) & \frac{\kappa \beta}{R_0} & \frac{\beta}{R_0} \\
0 & \sigma & (\mu + \gamma + \delta)
\end{pmatrix},
\]

(17)

if $\lambda$ is the eigenvalues then
\[ J_{E^*} = \begin{pmatrix} \mu R_0 - \lambda & -\frac{\kappa \beta}{R_0} & -\frac{\beta}{R_0} \\ \mu (R_0 - 1) & \frac{\kappa \beta}{R_0} - (\mu + \sigma) - \lambda & -\frac{\beta}{R_0} \\ 0 & \sigma & -(\mu + \gamma + \delta) - \lambda \end{pmatrix} \]

We then get

\[ P(\lambda) = \lambda^3 + a \lambda^2 + b \lambda + c, \]

where

\[ a = \mu R_0 + (\mu + \gamma + \delta) + (\mu + \sigma) \left(1 - \frac{\kappa \beta}{(\mu + \sigma) R_0}\right). \]

From \( R_0 \) we get

\[ \frac{\sigma \beta}{(\mu + \gamma + \delta)(\mu + \sigma) R_0} = 1 - \frac{\kappa \beta}{(\mu + \sigma) R_0}, \tag{18} \]

hence

\[ a = \mu R_0 + (\mu + \gamma + \delta) + \frac{\sigma \beta}{(\mu + \gamma + \delta) R_0} > 0, \]

\[ b = \mu R_0 (\mu + \gamma + \delta) + \mu R_0 (\mu + \sigma) + (\mu + \gamma + \delta)(\mu + \sigma) - \mu \kappa \beta - \frac{\kappa \beta (\mu + \gamma + \delta)}{R_0} - \frac{\kappa \beta}{R_0}, \]

using (18) we get

\[ b = \mu R_0 (\mu + \gamma + \delta) + \mu R_0 (\mu + \sigma) \left(1 - \frac{\kappa \beta}{(\mu + \sigma) R_0}\right). \]

\[ c = \frac{\mu \kappa \beta (R_0 - 1)}{R_0} - \mu \kappa \beta (\mu + \gamma + \delta) + \mu (\mu + \sigma)(\mu + \gamma + \delta) R_0 - \mu \kappa \beta, \]

\[ c = \frac{\mu \kappa \beta (R_0 - 1)}{R_0} > 0, \]

and

\[ ab - c = \frac{\mu \kappa \beta}{R_0} \left(\frac{\beta}{(\mu + \gamma + \delta) + 1}\right) + \mu \beta \left(\frac{\mu R_0}{(\mu + \gamma + \delta) + 1}\right) + \mu R_0 (\mu + \gamma + \delta)(\mu R_0 + \mu + \gamma + \delta). \]

Since \( a > 0, b > 0, c > 0, \) and \( ab - c > 0, \) according to the Routh-Hurwitz criterion, the endemic equilibrium of system (4) is LAS.

**Theorem 3.** If \( R_0 \geq 1 \) the endemic equilibrium point is globally asymptotically stable in \( \Omega. \)
**Proof.** We prove the GAS for the system (4) EE by solving the EE equation of (11). As we did for (6), from (11), we get
\[ e^* = \frac{(\mu + \gamma + \delta)i^*}{\sigma}. \]
Substituting \( e^* \) into the first equation (11) and solve for \( i^* \) in terms of \( s^* \) we get
\[ i^* = \frac{\mu\sigma(1 - s^*)}{s^*\beta[\sigma + \kappa(\mu + \gamma + \delta)]}. \] (19)
Substituting (19) into the second equation of (11), we get
\[ \frac{\mu\sigma\beta s^*(1 - s^*)}{s^*\beta[\sigma + \kappa(\mu + \gamma + \delta)]} + \frac{\mu\kappa\beta(\mu + \gamma + \delta)s^*(1 - s^*)}{s^*\beta[\sigma + \kappa(\mu + \gamma + \delta)]} - \frac{\mu(\mu + \sigma)(\mu + \gamma + \delta)(1 - s^*)}{s^*\beta[\sigma + \kappa(\mu + \gamma + \delta)]} = 0, \]
By simplifying the left-hand side we get
\[ \frac{\beta}{(\mu + \gamma + \delta)} \left( \frac{\sigma}{\mu + \sigma} + \kappa \right)(s^* - s^{*2}) - (1 - s^*) = 0, \]
In terms of \( R_0 \) we get
\[ R_0 s^{*2} - (1 + R_0)s^* + 1 = 0. \]
For a positive solution,
\[ (1 + R_0)^2 - 4R_0 \geq 0 \implies R_0^2 - 2R_0 + 1 \geq 0, \]
hence
\[ (R_0 - 1)^2 \geq 0, \]
The proof of Theorem 4 is complete.

**4. Numerical simulations**

In this section, we illustrate the DFE and EE theorems numerically using the integration technique in R-software. The model parameter values are obtained from COVID-19 literature, and we focus our analysis in a small settlement approximately 1000 population. Using data of 10 June 2020, we estimate the global case fatality rate as the ratio of total deaths and total confirmed cases (\( \delta = \frac{408025}{7145539} = 0.057 \))[3], the incubation period has a mean average of 5.2 days and the recovery period is 5.8 days[14],i.e., (\( \sigma = 1/5.2 = 0.192, \gamma = 1/5.8 = 0.172 \)). The birth and death rate is assumed to be (\( \mu = 0.00005 \)), the virus asymptotic infection proportion \( \kappa = 0.5 \)[15]. Using
these parameters values together with $\beta = 0.533$ as in [16], we can see that our $R_0 = 3.71$, which is equivalent to its estimate in [12,16–18].

Firstly, we investigate the DFE by assuming $\beta = 0.0533$; we observe that when $R_0 = 0.371$ in Figure 2(a), Theorem 1 and Theorem 3 are satisfied for the DFE to asymptotically stable. Also, in Figure 2(b), we observe that when $\beta = 0.533$, before the first 20 days, the trajectories for the state proportion were in agreement with Theorem 2 and Theorem 4 for the EE of the model to be asymptotically stable. In general, the trajectories pattern is in strong agreement with the average global behavior of the COVID-19 pandemic. That is, in majority of the countries with COVID-19, the outbreak reach it peak at least the first two weeks after the disease become an outbreak and it started to decrease with time. The decrease of the trajectories in the case of the $E$ state is as results of the increase of asymptomatic individual to infectious state and natural death, whereas, for the $I$ state is as results of the increase in the recovered individual and those who might have died of natural or virus death.

![Figure 2. The SEIR model with asymptomatic infection asymptotic stability analyses](image)

Secondly, we investigate the effect of the asymptomatic infection by keeping the EE parameters values constant and regulating the degree of $\kappa$. The comparisons are made with the trajectories of Figure 2. That is, the positive effect when the magnitude of $\kappa$ reduce is noticeable when the curves in Figure 2(b) become similar to that of Figure 2(a). It is observed in Figure 3 that as $\kappa$
decreases the endemic trajectories patterns is similar to the DFE curves in Figure 2(a), indicating that the asymptomatic infection is detrimental to the COVID-19 pandemic.

Figure 3. The SEIR model with asymptomatic infection transmission effects

Similarly, we further investigate the effect of the direct transmission by regulating the magnitude of $\beta$, and we also observe the same curves pattern as Figure 3. That is, when $\beta$ is lower in magnitude, lesser susceptible individuals become infected as the curve tends to increase in proportion, indicating that direct transmission can enhance the persistence of the COVID-19 pandemic.

Figure 4. The SEIR model with asymptomatic infection direct transmission effects
5. Conclusion

In this paper, we formulate an asymptomatic infection SEIR model to investigate the stability analysis of the COVID-19 pandemic with demographic effects. We use simple algebraic procedures to describe the dynamics of the model theoretically. We showed that the model has two equilibrium states, which are disease-free and endemic equilibrium. The stability analyses show that the two equilibria states are locally and globally asymptotically stable, which are confirmed numerically using epidemiological data of COVID-19 pandemic. Also, we show numerically that the COVID-19 pandemic can be put to rest if both the direct and asymptomatic infections are control.

6. Declarations

Availability of data materials. Authors can confirm that all relevant data source are included in the article.

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Authors’ contributions. AAK designed, analysed and interpreted the results of this article; LNM analysed and interpreted the results; GB substantively revised the article. All authors read and approved the final manuscript.

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