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Modeling the effects of the contaminated environments on COVID-19 transmission in India

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A B S T R A C T

COVID-19 is an infectious disease caused by the SARS-CoV-2 virus that caused an outbreak of typical pneumonia first in Wuhan and then globally. Although researchers focus on the human-to-human transmission of this virus but not much research is done on the dynamics of the virus in the environment and the role of contaminated environments means there is a chance to get infected by the virus in the environment. We also calculated the threshold quantity $R_0$ to know the disease status and provide conditions that guarantee the local and global asymptotic stability of the equilibria using Volterra-type Lyapunov functions, LaSalle’s invariance principle, and the Routh–Hurwitz criterion. Furthermore, the sensitivity analysis is performed for the proposed model that determines the relative importance of the disease transmission parameters. Numerical experiments are performed to illustrate the effectiveness of the obtained theoretical results.

Introduction

COVID-19 disease is caused by SARS-COV-2 that represents a causative agent of a potentially fatal disease of great global public health concern. It is a disease that became a pandemic in a short period due to its accessible transmission routes as it transmits via respiratory droplets exhaled during sneezing, talking, or coughing. This deadly outbreak started in India on March 2, 2020, and is still uncontrolled. There are almost 30,585,229 confirmed cases and 402,758 deaths reported in India as of July 01, 2021. The virus belongs to a family of viruses that can cause illnesses such as the common cold, severe acute respiratory syndrome (SARS), Middle East respiratory syndrome (MERS), and even if left untreated can cause lung damage [1,2]. Some people may have only a few signs, and some people may have no symptoms at all. Fever, cough, and tiredness are common symptoms of infected people. However, loss of taste or even smell can also be found in such patients. Although the novel coronavirus that causes COVID-19 disease first appeared in mainland China’s Wuhan city in late 2019 before it rapidly spread globally, still the biological origin of the virus is not known. The symptoms of coronavirus disease from the people who got infected may appear 2–14 days after exposure. The time interval from exposure to developing symptoms is called the incubation period [3,4]. The virus spreads by respiratory droplets released when an infected individual cough, sneezes, breathes, sings, or talks. These droplets can be inhaled or land in the mouth, nose, or eyes of a person nearby. In some situations, the virus can spread by a person being exposed to small droplets or aerosols that stay in the air for several minutes or hours-called airborne transmission [1,4].

The mathematical modeling of infectious diseases is very useful in understanding epidemiological prototypes of diseases. It helps to take necessary measures of public health intrusions by controlling the spread of the diseases. In modeling COVID-19 dynamics, mathematical modeling helps cases and deaths by county and partners respond to the COVID-19 epidemic by informing decisions about epidemic planning, resource allocation, and implementation of social distancing measures and other interventions [5]. Numerous studies have been proposed to forecast the future of COVID-19 epemics in India and worldwide [6–16]. Borah et al. [17] analyzed Covid-19 progression in India concerning conditions on the spread. They found that the weather parameters are an important factor in determining the occurrence rate of Covid-19 in India. Their findings provided preliminary evidence that...
the Covid-19 pandemic may be partially suppressed with temperature and humidity increases in India. Kumar and Kumar [18] proposed a correlation study between meteorological parameters and the COVID-19 pandemic in Mumbai, India. In their study, they found that the relative humidity and pressure parameters had the most influencing effect out of all other significant parameters (obtained from Spearman’s method) on the active number of COVID-19 cases. Raza et al. [19], in their paper, investigated the impact of meteorological indicators (temperature, rainfall, and humidity) on total COVID-19 cases in Pakistan, its provinces, and administrative units from March 10, 2020, to August 25, 2020. The correlation analysis of the study showed that COVID-19 cases and temperature are positively correlated. Zu et al. [20] constructed a compartmental model a data- and model-driven approach for the study of transmission patterns of COVID-19 in the mainland of China. In their work, they have studied the efficacy of different control strategies and estimated the model parameters to the real reported data collected from January 10-February 17, 2020 from the National Health Commission of PR China. Rafiq et al. [21] developed a mathematical model to describe the spreading of epidemic disease in a human population with the emphasis on the study of the propagation of the coronavirus disease (COVID-19). They investigated the model analytically as well as numerically. Rohith and Devika [22] modeled COVID-19 dynamics using a susceptible-exposed-infectious–removed model with a nonlinear incidence rate. In their study, they performed the bifurcation analysis and studied the effect of varying reproduction numbers on the COVID-19 transmission. Shen et al. [23] developed a dynamic compartmental model of COVID-19 transmission in New York City to assess the effect of the executive order on face masks used on infections and deaths due to COVID-19 in the city. In their study, they provided the importance of implementing face mask policies in local areas as early as possible to control the spread of COVID-19 and reduce mortality. Bherwani et al. [24] explored the dependence of local areas as early as possible to control the spread of COVID-19 and they provided the importance of implementing face mask policies in China. In their work, they have studied the efficacy of different control strategies and estimated the model parameters to the real data for India. In Section "Sensitivity analysis", sensitivity analysis of the parameters involved in threshold parameter through PRCC to identify the key factors and the relative importance of the model parameters is discussed. Section "Numerical results and discussion" gives the numerical simulations of the proposed model to validate the analytical studies. The behavior of the obtained solutions is also discussed in this section. Finally, Section "Concluding remarks" concludes all the major findings of the present research study.

**Model formulation**

There are many classes of mathematical models used within epidemiology. Deterministic compartmental models divide the population into groups defined, at a minimum, by the possible disease states that one could be in over time. They are the foundation of mathematical epidemiology and provide a straightforward introduction to how models are built. Here, to develop a mathematical model for the transmission dynamics of the COVID-19 epidemic, we proposed a novel deterministic compartmental model contains the following seven classes of the population, namely susceptible class denoted by $S(t)$; Exposed class denoted by $E(t)$; Asymptomatic class denoted by $A(t)$; Symptomatic infected class denoted by $I(t)$; Confirmed class denoted by $C(t)$; Hospitalized and treated class denoted by $H(t)$; Recovered class denoted by $R(t)$ and Virus in the environment denoted by $V(t)$ at any time $t$. Hence, the total population $N(t)$ is given by $N(t) = S(t) + E(t) + A(t) + I(t) + C(t) + H(t) + R(t)$. To better understand the dynamic process, the population is mixing and interacting homogeneously means no lockdown, no quarantine, and no restrictions. Also, infection is spread due to the interaction of susceptible individuals with asymptomatic, symptomatic, and virus in the environment. Therefore, the proposed model describes the dynamics of COVID-19 transmission among different population classes can be represented through the following ordinary differential equations:

$$
\begin{align*}
\frac{dS(t)}{dt} &= \Delta - (\beta_E E(t) + \beta_A A(t) + \beta_I I(t) + \beta_V V(t)) S(t) - \mu S(t), \\
\frac{dE(t)}{dt} &= (\beta_E E(t) + \beta_A A(t) + \beta_I I(t) + \beta_V V(t)) S(t) - (\mu + \gamma) E(t), \\
\frac{dA(t)}{dt} &= \xi E(t) - (\mu + \gamma_1 + \omega) A(t), \\
\frac{dI(t)}{dt} &= (1 - \xi) E(t) - (\mu + \sigma + \omega) I(t), \\
\frac{dC(t)}{dt} &= \alpha I(t) - (\mu + \sigma + \omega + \gamma_2) C(t), \\
\frac{dH(t)}{dt} &= \sigma C(t) - (\mu + \omega + \gamma_3) H(t), \\
\frac{dR(t)}{dt} &= \gamma_1 A(t) + \gamma_2 C(t) + \gamma_3 H(t) - \mu R(t), \\
\frac{dV(t)}{dt} &= \alpha_1 A(t) + \alpha_2 E(t) + \alpha_3 I(t) - \theta V(t).
\end{align*}
$$

subject to non-negative initial conditions $S(0) = S_0 \geq 0$, $E(0) = E_0 \geq 0$, $A(0) = A_0 \geq 0$, $I(0) = I_0 \geq 0$, $C(0) = C_0 \geq 0$, $H(0) = H_0 \geq 0$, $R(0) = R_0 \geq 0$, $V(0) = V_0 \geq 0$.

The recruitment rate for the susceptible population is denoted by $\Delta$. The susceptible individuals become infected, due to effective contact with the exposed class at a rate $\beta_E$; asymptomatic class at a rate $\beta_A$; symptomatic class at a rate $\beta_I$ and with the virus in the environment at a rate $\beta_V$. $\mu$ is the natural death rate; $\sigma$ is the rate at which exposed individuals become infectious; $\xi$ is the rate at which exposed individuals become asymptomatic by a proportion $\alpha$; $\gamma_1$ is the recovery rate of asymptomatic individuals due to natural immunity; $\omega$ is the COVID-19 induced death rate; $\alpha$ is the rate at which infected individuals

\[\begin{align*}
\frac{dS(t)}{dt} &= \Delta - (\beta_E E(t) + \beta_A A(t) + \beta_I I(t) + \beta_V V(t)) S(t) - \mu S(t), \\
\frac{dE(t)}{dt} &= (\beta_E E(t) + \beta_A A(t) + \beta_I I(t) + \beta_V V(t)) S(t) - (\mu + \gamma) E(t), \\
\frac{dA(t)}{dt} &= \xi E(t) - (\mu + \gamma_1 + \omega) A(t), \\
\frac{dI(t)}{dt} &= (1 - \xi) E(t) - (\mu + \sigma + \omega) I(t), \\
\frac{dC(t)}{dt} &= \alpha I(t) - (\mu + \sigma + \omega + \gamma_2) C(t), \\
\frac{dH(t)}{dt} &= \sigma C(t) - (\mu + \omega + \gamma_3) H(t), \\
\frac{dR(t)}{dt} &= \gamma_1 A(t) + \gamma_2 C(t) + \gamma_3 H(t) - \mu R(t), \\
\frac{dV(t)}{dt} &= \alpha_1 A(t) + \alpha_2 E(t) + \alpha_3 I(t) - \theta V(t).
\end{align*}\]
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Positivity and boundedness of the solution for the proposed model (1) are given. After that, the existence conditions and the stability results for the equilibria are provided.

Analysis of the model

The stability analysis of an epidemic model determines its behavior in disease dynamics. In this section, the positivity and boundedness of the solution for the proposed model (1) are given. After that, the existence conditions and the stability results for the equilibria are provided.

Positivity and boundedness

From the system (1), we get the total differential equation as:

\[
\begin{align*}
\frac{dN(t)}{dt} &= \Delta - \mu N(t) - \omega (A(t) + I(t) + C(t) + H(t)), \\
\frac{dV(t)}{dt} &= \alpha_1 A(t) + \alpha_2 E(t) + \alpha_3 I(t) - \theta V(t).
\end{align*}
\]

(2)

are confirmed by testing; \(\sigma\) is the rate at which confirmed join the hospitalized class; \(\gamma_1\) is the recovery rate of confirmed individuals; \(\gamma_2\) is the recovery rate by treatment; \(a_1, a_2, \) and \(a_3\) are the virus released rates via the asymptomatic, exposed and symptomatic class respectively and \(\theta\) is the virus clearance rate. The transfer relationships between the eight compartments is shown in Fig.1 (see Fig. 1).

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\frac{dV(t)}{dt} &= \alpha_1 A(t) + \alpha_2 E(t) + \alpha_3 I(t) - \theta V(t).
\end{align*}
\]

(2)
Proof. To prove this, we have from first equation of (2),
\[
\frac{dN(t)}{dt} \leq \Delta - \mu N(t).
\]

Now, let us take this solution which is unique to the initial value problem
\[
\begin{aligned}
\frac{dX(t)}{dt} &= \Delta - \mu X(t), \text{ for } t > 0, \\
X(0) &= N(0),
\end{aligned}
\]

which on simplification yields
\[
X(t) = \frac{\Delta}{\mu} (1 - e^{-\mu t}) + N(0)e^{-\mu t}.
\]

Therefore, it follows from the comparison principle [35], that
\[
N(t) \leq \frac{\Delta}{\mu} (1 - e^{-\mu t}) + N(0)e^{-\mu t}.
\]

Let \( \zeta = (a_1 + a_2 + a_3) \) with the assumption that \( 0 < (A(t)+E(t)+I(t)) \leq \frac{\Delta}{\mu} \). Then, we have from the second equation of (2),
\[
\frac{dY(t)}{dt} \leq \frac{\Delta}{\mu} - \theta V(t).
\]

Now, let \( Y \) to be a solution which is unique to the initial value problem
\[
\begin{aligned}
\frac{dY(t)}{dt} &= \frac{\Delta}{\mu} - \theta V(t), \text{ for } t > 0, \\
Y(0) &= V(0),
\end{aligned}
\]

which on simplification yields
\[
Y(t) = \frac{\Delta}{\mu} (1 - e^{-\theta t}) + V(0)e^{-\theta t}.
\]

Therefore, it follows from the comparison principle [35], that
\[
V(t) \leq \frac{\Delta}{\mu} (1 - e^{-\theta t}) + V(0)e^{-\theta t}.
\]

Therefore, from (6) and (9), we confirm that all possible solution sets of the state variables \( S(t), E(t), A(t), I(t), C(t), H(t), R(t), V(t) \) are bounded and by the blow-up phenomena [35], the solution exist and is defined for all \( t \geq 0 \). Furthermore, for \( t \rightarrow +\infty \), we get
\[
0 \leq N(t) \leq \frac{\Delta}{\mu},
\]
\[
0 \leq V(t) \leq \frac{\Delta}{\mu} \theta.
\]

This shows that the total population \( N(t) \), i.e., the subpopulations \( S(t), E(t), A(t), I(t), C(t), H(t), R(t) \) and \( V(t) \), are bounded in \( \Omega \) making the model both mathematically and epidemiologically well posed. This proves the boundedness of the solution of system (1).

Equilibrium and their stability

For the equilibrium points, we consider the right-hand side of system (1) equal to zero first to get disease free equilibrium point as \( Y_0 = (\frac{\Delta}{\mu}, 0, 0, 0, 0, 0, 0) \) at this point the system is free from the disease. Now for the endemic equilibrium point, we solve the system
\[
\begin{aligned}
\Delta - (\beta_E E + \beta_A A + \beta_I I + \beta_V V) S(t) - \mu S(t) &= 0, \\
(\beta_E E + \beta_A A + \beta_I I + \beta_V V) S(t) - \mu S(t) &= 0, \\
\delta S(t) - (\mu + \gamma_1 + \omega) A(t) &= 0, \\
(1 - \zeta_2) \delta E(t) - (\mu + \omega + \omega I) I(t) &= 0, \\
\alpha I(t) - (\mu + \omega + \gamma_2 C(t) = 0, \\
\sigma C(t) - (\mu + \omega + \gamma_2 H(t) &- \mu R(t) = 0, \\
\gamma_1 A(t) + \gamma_2 C(t) + \gamma_3 H(t) - \mu R(t) = 0, \\
\alpha A(t) + \alpha A(t) + \alpha I(t) - \theta V(t) &= 0.
\end{aligned}
\]

with an assumption that \( E(i) \neq 0, A(i) \neq 0, I(i) \neq 0, C(i) \neq 0, H(i) \neq 0, R(i) \neq 0, V(i) \neq 0 \), i.e., the point at which virus persists in the population. The solution of system (10) yields the endemic equilibrium point \( Y_1 = (\gamma, \gamma, \gamma, \gamma, \gamma, \gamma, \gamma) \), where
\[
\begin{aligned}
\frac{\partial S}{\partial X} &= \frac{\partial S}{\partial Y} \gamma, \\
\frac{\partial S}{\partial E} &= \frac{\partial S}{\partial Y} \gamma, \\
\frac{\partial S}{\partial A} &= \frac{\partial S}{\partial Y} \gamma, \\
\frac{\partial S}{\partial I} &= \frac{\partial S}{\partial Y} \gamma, \\
\frac{\partial S}{\partial C} &= \frac{\partial S}{\partial Y} \gamma, \\
\frac{\partial S}{\partial H} &= \frac{\partial S}{\partial Y} \gamma, \\
\frac{\partial S}{\partial R} &= \frac{\partial S}{\partial Y} \gamma.
\end{aligned}
\]
Now, we prove the stability of the equilibria in the form of following theorems with proofs.

**Theorem 3.** The disease-free equilibrium $Y_0$ of the proposed COVID-19 epidemic model (1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

**Proof.** An easy substitution of the point $Y_0$, in the general Jacobian matrix $J$ readily yields the matrix of the form

$J(Y_0) = \begin{pmatrix}
-\mu & \frac{\beta_\omega\rho_M}{(\mu+\lambda)} & \frac{\beta_\omega\rho_M}{(\mu+\lambda)} & 0 & 0 & 0 & -\frac{\beta_\omega\rho_M}{\mu} \\
0 & \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)} & -\frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)} & 0 & 0 & 0 & \frac{\beta_\omega\rho_M}{\mu^2} \\
0 & \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)} & \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)} & 0 & 0 & 0 & \frac{\beta_\omega\rho_M}{\mu^2} \\
0 & \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)} & \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)} & 0 & 0 & 0 & \frac{\beta_\omega\rho_M}{\mu^2} \\
0 & 0 & 0 & \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)} & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)} & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)} & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)}
\end{pmatrix}$

As per Routh-Hurwitz criterion, for $R_0 < 1$ the disease-free equilibrium $Y_0$ of the proposed model (1) is locally asymptotically stable if all the eigenvalues $\lambda$, $i = 1, 2, \ldots, 8$ of the matrix $J(Y_0)$ are negative numbers or have negative real parts. We can evaluate these eigenvalues from the following characteristic polynomial

$$|J(Y_0) - \lambda I| = 0$$

where $I$ is an identity matrix of order eight and $\lambda$ is the eigenvalue. Therefore, we get a characteristic polynomial of the form

$$C^{Y_0}(\lambda) = (\mu + \lambda)^3 \lambda^2 + (-\beta_\omega\rho_M + m_2 + m_3)\lambda + (m_1\rho_m - \beta_\omega\rho_M m_1 + m_1\rho_\gamma\xi\sigma)$$

$$= \lambda^3 + (m_1 + m_2)\lambda + (m_1m_2) + (m_1m_2m_3).$$

Eq. (12) can be further written as

$$C^{Y_0}(\lambda) = C_{1}(\lambda) \times C_2(\lambda) \times C_3(\lambda) \times C_4(\lambda),$$

where

$$C_{1}(\lambda) = (\mu + \lambda)^3,$$

$$C_{2}(\lambda) = \lambda^2 + (-\beta_\omega\rho_M + m_2 + m_3)\lambda + (m_1\rho_m - \beta_\omega\rho_M m_1 + m_1\rho_\gamma\xi\sigma),$$

$$C_{3}(\lambda) = \lambda^2 + (m_1 + m_2)\lambda + (m_1m_2),$$

$$C_{4}(\lambda) = \lambda^2 + (m_1 + m_2)\lambda + (m_1m_2m_3),$$

and

$$m_1 = \frac{\beta_\omega\rho_M}{\mu(\mu+\lambda)}, m_2 = \mu + \lambda, m_3 = \mu + \lambda + \omega, m_4 = \mu + \alpha + \omega, m_5 = \mu + \alpha + \omega + \gamma_2, m_6 = \mu + \gamma_1 + \omega, m_7 = \mu + \alpha + \omega + \gamma_2, m_8 = \mu + \gamma_1 + \omega, m_9 = \mu + \alpha + \omega + \gamma_2, m_{10} = \mu + \gamma_1 + \omega, m_{11} = \mu + \alpha + \omega + \gamma_2.$$}

Theorem 4. The equilibrium point $Y_1$ of the proposed COVID-19 epidemic model (1) is locally asymptotically stable if $R_0 > 1$ and unstable otherwise.

**Proof.** The general Jacobian matrix $J$ at the endemic point $Y_1$ is obtained as

$$J(Y_1)$$

Let,

$$n_1 = \beta_\omega E^* + \beta_\omega A^* + \beta_I I^* + \beta_V V^*, n_2 = \beta_\omega E^*, n_3 = \beta_\omega A^*, n_4 = \beta_\omega I^*, n_5 = \beta_\omega V^*, n_6 = \mu + \gamma_1 + \omega, n_7 = \mu + \alpha + \omega, n_8 = \mu + \alpha + \omega + \gamma_1 + \omega, n_9 = \mu + \alpha + \omega + \gamma_1 + \omega + \gamma_2.$$}

As per Theorem 3, the eigenvalues satisfy

$$C^{Y_1}(\lambda) = (\mu + \lambda^3)\theta + (\lambda^3 + \lambda^2\alpha + \lambda\beta + \gamma)\lambda.$$}

Proof. In order to obtain the global stability of the point $Y_0$, we consider the Volterra-type Lyapunov functional approach to define a function $F(t) = \psi(t)$ with $F(t) = S + H + I + C + E + \rho I + \rho V + V$.

This function is defined, continuous and positive definite for all $t \geq 0$. It can be verified that the equality holds if and only if $S = S_0, E = A = I = C = H = R = V = 0.$

Now, we have on differentiation w.r.t. $t$

$$\frac{dF(t)}{dt} = (S - S_0)\frac{dS}{dt} + (H - H_0)\frac{dH}{dt} + (I - I_0)\frac{dI}{dt} + (V - V_0)\frac{dV}{dt}.$$

This implies on substitutions from Eq. (1)

$$\frac{dS}{dt} = \left(\frac{S - S_0}{S_0}\right)(\Delta - (\beta_\omega E + \beta_\omega A + \beta_I I + \beta_V V)S - \mu S)$$

$$+ (\beta_\omega E + \beta_\omega A + \beta_I I + \beta_V V)S - (\mu + \alpha + \omega) E + \xi\lambda E - (\mu + \gamma_1 + \omega)A$$

$$+ (\beta_\omega E + \beta_\omega A + \beta_I I + \beta_V V)S - (\mu + \alpha + \omega) I + \alpha I - (\mu + \gamma_1 + \omega)C$$

$$+ (\beta_\omega E + \beta_\omega A + \beta_I I + \beta_V V)S - (\mu + \gamma_2) V + \gamma_1 H + C + \gamma_1 H - \mu R$$

$$+ a_1 A + a_2 E + a_3 I - 0V.$$}

After simplification using the disease-free steady state condition of model (1), we have from Eq. (14) as

$$\frac{dE}{dt} = -\frac{(S - S_0)^2}{S_0} - (S - S_0)(\beta_\omega E + \beta_\omega A + \beta_I I + \beta_V V)S$$

$$+ (\beta_\omega E + \beta_\omega A + \beta_I I + \beta_V V)S - (\mu + \alpha + \omega) E + \xi\lambda E - (\mu + \gamma_1 + \omega)A$$

$$+ (\beta_\omega E + \beta_\omega A + \beta_I I + \beta_V V)S - (\mu + \alpha + \omega) I + \alpha I - (\mu + \gamma_1 + \omega)C$$

$$+ (\beta_\omega E + \beta_\omega A + \beta_I I + \beta_V V)S - (\mu + \gamma_2) V + \gamma_1 H + C + \gamma_1 H - \mu R$$

$$+ a_1 A + a_2 E + a_3 I - 0V.$$
\[ \frac{dE}{dt} = \left( 1 - \frac{\beta E S}{\mu} \right) - \omega E \]
\[ - \mu A \left( 1 - \frac{\beta A S}{\mu} \right) - \omega A \]
\[ - \omega I - \mu C - \mu H - \omega H \]
\[ - \mu R - \delta V. \]

(15)

Therefore,
\[ \frac{dE}{dt} \leq 0. \]

It can be noticed that if \( R_0 < 1 \), then the right-hand side of Eq. (15) is non-positive and it is equal to zero if \( S = S_0, E = A = I = C = H = R = V = 0 \). Therefore, the maximum invariant set for \( \{(S, E, A, I, C, H, R, V) \in \mathbb{Q}^8 : \frac{dE}{dt} = 0 \} \) is the singleton set \( Y_0 \). This means that the only trajectory of the system on which \( \frac{dE}{dt} = 0 \) is \( Y_0 \).

According to the LaSalle’s invariance principle [40–43], we know that all solutions in \( \mathbb{Q}^8 \) converge to \( Y_0 \). Therefore, the disease-free steady state of model (1) is globally asymptotically stable when \( R_0 < 1 \). This completes the proof of Theorem 5.

\[ \square \]

**Theorem 6.** The endemic equilibrium \( Y_1 \) of the proposed COVID-19 epidemic model (1) is globally asymptotically stable if \( R_0 > 1 \) and unstable otherwise.

**Proof.** To prove the global stability of the point \( Y_1 \), we consider the Volterra-type Lyapunov functional approach to define a function \( E_0(t) : y(t) \to R \), as

\[ E_0(t) = \frac{1}{b_1} \left( S - S^* \right) + \frac{1}{b_2} \left( A - A^* \right) + \frac{1}{b_3} \left( E - E^* \right) + \frac{1}{b_4} \left( I - I^* \right) + \frac{1}{b_5} \left( C - C^* \right) + \frac{1}{b_6} \left( H - H^* \right) + \frac{1}{b_7} \left( R - R^* \right) + \frac{1}{b_8} \left( V - V^* \right). \]

where \( b_1 = b_2 = \mu, b_3 = (\mu + \sigma), b_4 = (\mu + \alpha + \omega), b_5 = (\mu + \alpha + \gamma_1 + \omega), b_6 = (\mu + \gamma_2 + \omega), b_7 = (\mu + \gamma_1 + \omega). \) \( b_8 = \theta. \)

This function is defined, continuous and positive definite for all \( t \geq 0 \). It can be verified that the equality holds if and only if \( S = S^*, E = E^*, A = A^*, I = I^*, C = C^*, H = H^*, R = R^*, V = V^*. \)

Now, we have on differentiation w.r.t. \( t \)

\[ \frac{dE_0}{dt} = \frac{1}{b_1} \left( -S^* \right) + \frac{1}{b_2} \left( -A^* \right) + \frac{1}{b_3} \left( -E^* \right) + \frac{1}{b_4} \left( -I^* \right) + \frac{1}{b_5} \left( -C^* \right) + \frac{1}{b_6} \left( -H^* \right) + \frac{1}{b_7} \left( -R^* \right) + \frac{1}{b_8} \left( -V^* \right). \]

This further implies on substitutions from Eq. (1)

\[ \frac{dE_0}{dt} = \frac{1}{b_1} \left( -S^* \right) + \frac{1}{b_2} \left( -A^* \right) + \frac{1}{b_3} \left( -E^* \right) + \frac{1}{b_4} \left( -I^* \right) + \frac{1}{b_5} \left( -C^* \right) + \frac{1}{b_6} \left( -H^* \right) + \frac{1}{b_7} \left( -R^* \right) + \frac{1}{b_8} \left( -V^* \right). \]

(16)

On simplification using the endemic state condition of model (1), we have from Eq. (16) as

\[ \frac{dE_0}{dt} = \frac{1}{b_1} \left( 0 \right) \]

\[ \frac{dE_0}{dt} \leq 0. \]

It can be noticed that if \( R_0 > 1 \), then the right-hand side of Eq. (17) is non-positive and it is equal to zero if \( S = S^*, E = E^*, A = A^*, I = I^*, C = C^*, H = H^*, R = R^*, V = V^*. \) Therefore, the maximum invariant set for \( \{(S, E, A, I, C, H, R, V) \in \mathbb{Q}^8 : \frac{dE}{dt} = 0 \} \) is the singleton set \( Y_1 \). This means that the only trajectory of the system on which \( \frac{dE}{dt} = 0 \) is \( Y_1 \). According to the LaSalle’s invariance principle [40–43], we know that all solutions in \( \mathbb{Q}^8 \) converge to \( Y_1 \). Therefore, the endemic state of the model (1) is globally asymptotically stable when \( R_0 > 1 \). This completes the proof of Theorem 6.

\[ \square \]

**Fitting of the model to the real statistical data for India**

India is a country with a 1.3 billion population. There were some restrictions and lockdown phases in the initial stage of the virus, but it is not possible for the government to keep all populations under lockdown for long. It has been a year since this virus in India, but still, it is not controlled. Here, we attempt to fit and estimate our model parameters to the actual statistical data from India. To avoid the possibility of pitfalls described by the authors in [44], we do not fit the model to a cumulative number of cases or a cumulative number of deaths. We convert this data to a fraction of the population by taking total population data from The World Bank Group (about 602,979,360, in 2019). For the model fitting, we use updated data from Johns Hopkins University Center for Systems Science and Engineering (JHU CSSE) (https://github.com/CSSEGISandData/COVID-19) for India. We use recent data from the last six months of this virus in India, i.e., from 22nd October 2020 to 31st March 2021. The estimated parameter values are based on the data about the number of currently infected individuals that can be observed and roughly corresponding to \((i + c + h + (i + c + h)).\) Parameters except for recruitment rate of susceptible \((A)\) and natural death rate \((\mu)\) have been estimated from fitting the model to the data as shown in Fig. 2. In Fig. 2, the real COVID-19 cases for India are shown by blue circles, whereas the best-fitted curve of the model is shown by the red solid line. The summary of the biological parameters involved in the model is listed in Table 1 and their best-estimated values obtained via the non-linear least-squares method are listed in Table 2. The estimated values of the parameters for the real COVID-19 cases in India have produced the value of basic reproduction number \(R_0 = 3.0438\) from October 22, 2020, to March 31, 2021, as the epidemic is still on its peak in India and is uncontrolled. The obtained results are comparable with findings in [45].

The ordinary differential equation (ODE) system was solved using LSODA [46,47]. We use lmfit python package [44] for non-linear least-squares and minimize the sum of the squares of the errors using the trust region reflective method and obtaining goodness of fit measure of \(\chi^2 = 6.2555e^{-08}\) for India. The problem to minimize error is shown in the following equation for a fitting parameter set \(q:\)

\[ q \in \arg \min S(q) = \arg \min \sum_{i=1}^{n} [y_i - g_i(x_i, q)]^2 \]

(18)

where \(y_i\) are observations and \(g_i\) is the model output.
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Fig. 2. Parameter estimation and fitting active cases \((I(t) + C(t) + H(t))\) to actual data for India using lmfit. The plot shows actual data and best fit by minimizing sum of the squares of the errors.

Sensitivity analysis

Sensitivity analysis can aid in identifying influential model parameters and optimizing model structure. In the proposed COVID-19 epidemic model, due to uncertainties associated with the estimation of certain parameter values, it is useful to carry out the sensitivity analysis to determine the model’s robustness as the parameter values changes. Having analytical expression for the reproduction number \(R_0\), it is reasonable to employ the normalized forward sensitivity index of a variable \(R_0\), that depends on a parameter \(\eta\), that is defined as [48,49]

\[
\Phi_R \eta = \frac{\partial R_0}{\partial \eta} \times \frac{\eta}{R_0}
\]

(19)

where \(\eta\) is one of the parameters whose sensitivity on \(R_0\) is sought. This index implies that the higher the value in its magnitude, the more sensitive \(R_0\) is to the parameter. Also, the positive (or negative) sign indicates that \(R_0\) increases (or decreases) as \(\eta\) increases.

For our proposed COVID-19 epidemic model (1), all eight state variables and eighteen parameters were uncertain. Following the detailed analysis as done by Marino et al. [50], we use Latin-hypercube sampling-based method to quantify the uncertainty and sensitivity of all the model parameters. A positive partial rank correlation coefficient (PRCC) value in Fig. 3 indicates an increase in the parameter leads to an increase in \(R_0\), while a negative value shows increasing the parameter decreases \(R_0\). Among these parameters, \(\Delta, \beta_E, \beta_A, \beta_I, \beta_V, \xi, a_1\) have a positive influence on \(R_0\), while \(\mu, \gamma_1, \omega, a, a_2\) and \(\theta\) have a negative influence on \(R_0\). Thus, the sensitivity analysis results show \(\mu, \gamma_1, \omega, a, a_2\) and \(\theta\) are the most influential parameters for the proposed model (1), as the increase in the value of these parameters decreases the value of \(R_0\).

Numerical results and discussion

In this section, we discuss the behavior of obtained numerical results. The model parameters used in simulations are described in Table 2.

Fig. 4 shows the temporal variations of different population classes. It can be seen from Fig. 4 that as time increases, the number of infected individuals also increases. Also, Fig. 4 shows that the environmental virus goes on increasing by the movement of the asymptomatic as well as symptomatic individuals due to which the confirmed cases increase by getting infected from the virus in the environment.

Fig. 5 shows the impact of various model parameters on the dynamics of \(R_0\). It is visible from Fig. 5 that these parameters have a negative effect on the value of \(R_0\), i.e., for the higher values of these parameters, the \(R_0\) decreases. Further, it can be seen from Fig. 5 that these parameters are inversely proportional to the value of \(R_0\) means an increase in these parameters causes a decrease in the value of \(R_0\).

Fig. 6 shows the impact of the different transmission rates, including the environmental viral rate, on the dynamics of \(R_0\). It is visible from Fig. 6 that for the higher concentration of the virus, the infected population is higher than the lower values of the virus. Also, it can be seen that these parameters have a positive effect on the value of \(R_0\), i.e., the increase in these parameters increases the value of \(R_0\) means they are directly proportional to the value of \(R_0\) thus causes disease spread. Further, it can be seen from Fig. 6 that the transmission coefficient of virus in the environment to the susceptible is more significant than other transmission rates as an increase in its value makes the value of \(R_0\) very high.
Fig. 5. Dynamics of $R_3$ for $\mu$, $\gamma_1$, $\alpha$ and $\theta$.

Fig. 6. Dynamics of $R_3$ for $\beta_V$, $\beta_E$, $\beta_I$ and $\beta_A$. 
Fig. 7. 3D dynamics of $R_0$ for $\alpha_1$ and $\alpha$ along with contour plot.

Fig. 8. 3D dynamics of $R_0$ for $\beta_V$ and $\theta$ along with contour plot.

Fig. 9. 3D dynamics of $R_0$ for $\beta_I$ and $\gamma_1$ along with contour plot.

Figs. 7–10 shows the 3D dynamics of $R_0$ against different model parameters. It can be seen from the figures that each parameter contributes towards the dynamics of $R_0$ as the changes in the values of these parameters significantly affect the value of $R_0$. In Fig. 7, the variation of $R_0$ for $\alpha_1$ and $\alpha$ is presented. It can be seen from Fig. 7 that $\alpha_1$ contributes towards the increasing value of $R_0$, which is the virus releasing rate via asymptomatic individuals. This indicates freely movement of individuals without any restriction can spread this disease. In Fig. 8, the variation of $R_0$ for $\beta_V$ and $\theta$ is shown. From Fig. 8, it is clear that environmental viruses can make the infection spread in the population. In contrast, the spread can be controlled by decreasing the virus in the environment by restricting the free movement of individuals. Similar behavior is observed from Figs. 9–10 for the parameters $\beta_I$ and $\beta_E$, respectively.

While mathematical models do not provide a cure for a given infectious disease, they are, however, used to replicate possible scenarios of the dynamic at hand. In the modeling of COVID-19 disease, mathematical models may help to explore the transmission dynamics, understanding the trajectory of the epidemic, prediction, and design effective control measures for the spread of this fatal disease. However, any study is not ultimate in science, there must be some limitations and assumptions which are deviated from actual reality. There were some limitations to this study that must be considered. First, we ignored the effect of uneven population distribution and assumed that the total population was homogeneously distributed. Second, we ignored...
the differences in individual susceptibility and we assumed that infection susceptibility for all individuals in the free environment was the same; whereas, in actuality, adults and older people are more likely to be infected by SARS-CoV-2. Third, we did not take into account the limitation of medical resources, such as health care workers and medical protective equipment. Furthermore, since the study was constructed from reported data and some parameters were calculated based on preliminary studies, these data came from heterogeneous sources, which may have introduced biases. It was important to note that when we predicted the epidemic trend of COVID-19 without any control measures, due to the small amount of reported data, the estimated parameters might have certain errors and the predicted results might represent an over-prediction.

The current study is designed for the country India however the model can perform well if applied to other countries with high COVID-19 cases like Brazil or the USA and get results to depend on the reported cases for these countries. Many studies have already been reported in the literature [51–57] that studied the effect of environmental parameters like temperature, humidity, pollution, etc on the transmission dynamics of COVID-19. From these studies, it is clear that environmental parameters play important role in the dynamic process of COVID-19 disease.

Concluding remarks

In this paper, we have proposed and investigated a novel mathematical model for the transmission of COVID-19 under the influence of contaminated environments. This model has been used to describe the diverse transmission passages in the infection dynamics and affirms the role of the environmental reservoir in the transmission and outbreak of COVID-19 disease. The model is numerically simulated to signify the application of the study in India. We have presented a detailed analysis of the proposed model including, the derivation of equilibrium points, endemic and disease-free, the reproductive number \( R_0 \), and the positiveness of the model solutions. By using the threshold quantity \( R_0 \), the location and the existing conditions of the disease-free and endemic equilibrium point have been determined. The disease-free equilibrium point is proved to be globally asymptotically stable if \( R_0 < 1 \). Whereas, if \( R_0 > 1 \), the endemic equilibrium point exists and is locally asymptotically stable. In the segment of sensitivity analysis the importance of various parameters has been determined that highly affect the reproduction number and it signifies that to reduce the transmission rates, combined efforts towards the detection of undetected individuals, virus in the environment, and the treatment of infectives are required. The obtained analytical results explore that the proposed model provides a better fit to the real statistical data. The statistical results of this work may help the government and other proxies to reconfigure their strategies according to the expected situation.

From numerical simulations, it is observed that to control the disease, the reproduction number must be decreased below one. To reduce the reproduction number below unity, a continuous reduction in the transmission rate of COVID-19 is required by self-quarantine, isolation of infectives, and initiation of treatment for the infected individuals as early as possible. Based on the scientific evidence, the premature pulled out of these strategies influences the infection prevalence and implies that incautious decisions have been taken as can be visualized from the numerical simulations. Thus, we have concluded that if the reproduction number for the COVID-19 disease is reduced below unity by decreasing the transmission rates and increasing the detection rate, then the epidemic can be eradicated from the population.

CRediT authorship contribution statement

Parvaiz Ahmad Naik: Conceptualization, Analysis, Methodology, Writing - original draft. Jian Zu: Writing - review & editing, Funding acquisition, Supervision. Muhammad Bilal Ghori: Software, Validation, Numerical simulations. Mehraj-ud-din Naik: Writing - review & editing, Investigation, Resources.

Declaration of competing interest

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

Availability of data

The data used for the simulations and data fitting is collected from the Johns Hopkins University Center for Systems Science and Engineering (JHU CSSE) (https://github.com/CSSEGISandData/COVID-19).

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