How do the contaminated environment influence the transmission dynamics of COVID-19 pandemic?

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Abstract COVID-19 is an infectious disease caused by the SARS-CoV-2 virus that first appeared in Wuhan city and then globally. The COVID-19 pandemic exudes public health and socio-economic burden globally. Mathematical modeling plays a significant role to comprehend the transmission dynamics and controlling factors of rapid spread of the disease. Researchers focus on the human-to-human transmission of the virus but the SARS-CoV-2 virus also contaminates the environment. In this study we proposed a nonlinear mathematical model for the COVID-19 pandemic to analyze the transmission dynamics of the disease in India. We have also incorporated the environment contamination by the infected individuals as the population density is very high in India. The model is fitted and parameterized using daily new infection data from India. Analytical study of the proposed COVID-19 model, including feasibility of critical points and their stability reveals that the infection-free steady state is stable if the basic reproduction number is less than unity otherwise the system shows significant outbreak. Numerical illustrations demonstrates that if the rate of environment contamination increased then the number of infected persons also increased. But if the environment is disinfected by sanitization then the number of infected persons cannot drastically increase.

1 Introduction

The ongoing novel coronavirus disease is initiated from the Wuhan city of the Republic of China in the end of December 2019 and spread throughout the world. This COVID-19 epidemic has became global pandemic with more than 273,330,018 coronavirus cases, 5,355,749 deaths and 245,501,110 recovered as of December 17, 2021 [1]. The first COVID-19 positive cases in India was reported on January 30, 2020 when a student came from China, and at present 34,726,049 total individuals are reported with positive cases, with 476,869 fatalities and 34,162,765 recoveries [1]. A series of intervention strategies have been taken into account by the Govt. of different countries to maintain the spread of the novel coronavirus diseases, including nationwide lockdown, social distancing, contact tracing and identifying the cluster of cases.

SARS-CoV-2 firstly transfer from animals to humans [2]. Respiratory droplets and contact transmission is the most important way for the transmission dynamics of SRAS-CoV-2 virus. In general, the COVID-19 infected patients suffers from high fever, sneeze, dry cough, tiredness, runny nose, breathing complication, lung penetration to severely ill & dead. Shared surfaces are a serious unsafe. Because the COVID-19 virus may alive or it may remain infectious from 2 hours up to one week on different kind of metals such as Aluminum, Metal, Wood, Silicon, Steel, Glass, Rubber and Paper [3].

This pandemic has brought massive organizational alters throughout the world, with frequent nationwide lockdown and the utilization of distant solutions for work, learning and daily life style. It has also a remarkable affects on hospitals and public health policies and to increase the number of hospitals bed, isolation ward and put on hold operations for the patients who are not infected by Covid-19. In spite of these changes, the pandemic has ended the life of million of human population world-wide. The latent period is greater than 10 days [4] causing in a lag among the administration of intervention strategies and their affect on hospital admissions, and created the usefulness of limitations more difficult to quantify. Predicting the number of infected cases and modeling the number of beds in hospital is essential to manage the novel coronavirus epidemic and

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has been the subject of various research papers over the last year. Investigations in the existing publications indicate that SARS-CoV & MERS-CoV initiated in bats, additionally both spreading in civet cats & camels, independently. Still the pool for the novel coronavirus is not familiar to us, although pangolins & bats are accepted to be the origin. Actually, many things are familiar about the transmission kinetics of SARS-CoV as well as MERS-CoV that presented to maintain and control the related infections. On the other hand, the understanding about the transmission kinetics and medical characteristics (for example, immune response) of COVID-19 pandemic is very poor. The relevant alikes and dissimilarities among SARS-CoV, MERS-CoV and novel coronavirus can be obtained in [5].

Howbeit, it is well accepted that the novel coronavirus is very diffusive in nature, spread through droplets, contact tracing as well as direct and indirect interactions are as stated: (a) direct interaction with infected populations and (b) indirect transmission with contaminated things. Due to absences specific vaccinations or proper antivirals as well as rapid diagnosis, the major maintain against novel coronavirus are social-distancing, contact-tracing, use classical hygienic precautions (for example, hand sanitizer, frequently cleaning hands by soap), use face-mask, quarantine of the populations feared exposed or recognized with COVID-19 [7, 8].

The modeling strategy has been investigated to explore the interactive kinetics of infectious diseases, and to estimate different control and reduction policies. After the pioneering work of Kermack-McKendric SIR epidemic model (in the year 1927) [9], a series of mathematical models have been formulated or modified the basic SIR model by several researchers to study the evolution of the epidemics. Particularly, modeling strategy is a viable tool in giving rational insight into the interactive kinetics and to mitigate the speedy spreading infectious diseases including SARS-CoV-2. Numerous mathematical models have been represented to explore the transmission kinetics of the earlier two relatives of SARS-CoV-2 (SERS & MERS) in Ontario, Hong-Kong, Singapore, Beijing [10–12], Korea [13, 14], and Middle East [14]. In an extended version of the classical SEIR (susceptible-exposed-infected-recovered) model has been studied for the infectious diseases incorporates to investigate the role of asymptomatic and pre-symptomatic infected individuals that are plays a key role in the transmission dynamics of the infectious diseases [15, 16].

The ongoing novel coronavirus has started at the end of December 2019, various mathematical models have been studied by several authors to describe successful ways to combat against COVID-19 pandemic [17–36]. Eikenberry and his colleagues [18] designed mathematical models to estimate the potentiality of face masks utilized by human population to reduce the novel coronavirus epidemic. In [17], the authors designed an agent-based mathematical model to study the influences of non-pharmaceutical intervention (NPI) strategies to mitigate the SARS-CoV-2 fatality and public health care demand. Ngonghala and his colleagues [21] also developed a mathematical to estimate the potentiality of non-pharmaceutical intervention (NPI) strategies in curtailing the SARS-CoV-2 virus. In [20], Muzimoto & Chowell established a mathematical model to study the interaction capability of the SARS-CoV-2 virus in the Diamond Princess Cruises Ship, 2020 while the epidemic is developed. Giordano et al. [22] designed a mathematical model for the novel coronavirus outbreak and explained that combining both strict lockdown and testing can control the severity of infectious coronavirus disease within human. To estimate or evaluate the clinical severity of the transmission dynamics of novel coronavirus pandemic Wu et al. [23] designed a stochastic model. Sarkar & Khajanchi [24] designed a mathematical model to describe the transmission kinetics and forecasting the SARS-CoV-2 virus in various states of India and India as a whole. Predicting the number of infections due to SARS-CoV-2 has been investigated by Liu et al. [25] by considering a 3-step procedure on the contact rate throughout the outbreak with linear, next exponential increase in diseases and at the end an exponential mitigate the contact rate.

Recently studied research work [6] demonstrates that the infected patients shed COVID-19 in their stool, reflect that SARS-CoV-2 can be transmitted indirectly following contact with contaminated environment. This is the essence of the campaign to disinfect objects, surfaces, buttons, hands, knobs and places touched often. Due to over-crowded family homes and local communities in India, we are more challenged by the following scenario: soon after COVID-19 infected persons cough or sneeze and release droplets from their mouths and noses, people touch objects and surfaces on which these droplets have landed, and then touch their eyes, noses or mouths. Furthermore, the vulnerable situation of India’s healthcare facility is a cause for concern about high concentration of the virus in the environments. For these reasons, we introduce a separate compartment of contaminated environment.

Our main aim of the present article is to design a new mathematical model which is capable of computing recovery rate, basic reproduction number and predicting the mode of interactive dynamics of the novel coronavirus infected patients. In this work, we proposed an extended Susceptible-Exposed-Infectious-Recovered (SEIR) model by inclusion of clinically ill classes, hospitalized or isolated classes and the environmental effect. Good number of studies on the dynamics of COVID-19 proposed by many researchers so far but still there is a need to develop feasible and more accurate models for the control and prevention of COVID-19 disease. Thus the introduction of environmental contamination class into the proposed model influences the basic reproduction number in a considerable amount, thereby extending the correctness of the quantification of the disease. To investigate the control policies, our main aim are as follows:
(a) Estimation of the most effective parameters as identified by the PRCC sensitivity analysis.
(b) Knowing the severity of the epidemic through theoretical analysis of the basic reproduction number.
(c) We included two parameters, such as $\beta_s$ due to environmental contamination and $\tau_e$ due to clearing rate of diseases from the environment.

We organized the remaining part of this paper as follows. We designed the mathematical model for the transmission dynamics of novel coronavirus pandemic in the Sect. 2. The qualitative behavior of the model is discussed in the Section 3. Qualitative behavior includes positivity of the solutions, boundedness of the system, computation of basic reproduction number with environmental contamination, sensitivity analysis with reference to the basic reproduction number, determination of the biologically feasible equilibrium points and their stability analysis, persistence of the proposed model for novel coronavirus. We performed an extensive numerical simulations in the Sect. 4. Numerical simulations include parameter estimation of the effective parameters and explorations regarding epidemiological point of view of the diseases and the effect of accessible control measures. At the end, Sect. 5 describes the conclusion of this work and some future directions.

2 Model derivation

In this section, we used the standard susceptible-exposed-infectious-removed (SEIR) model clarified by incorporating isolation techniques and the contaminated environment to investigate the transmission dynamics of novel coronavirus. We studied our proposed model by utilizing data obtained from the daily new confirmed COVID-19 cases of India and calculated basic reproduction number for the coronavirus infection. To construct the proposed model more convincing, we assume the followings

- the model includes a constant source rate of uninfected population $\Lambda_s$ per unit time,
- the proposed model for COVID-19 transmitted for human population only,
- the proposed model includes several demographical affects by considering proportional decay rate of all the compartments.

The total human individuals at any instant $t$, represented by $N(t)$ is classified into six sub-populations, namely susceptible $S(t)$, exposed $E(t)$, asymptomatic $A(t)$, clinically ill $I(t)$, isolated $H(t)$ and recovered $R(t)$ individuals. Therefore,

$$N(t) = S(t) + E(t) + A(t) + I(t) + H(t) + R(t).$$

As our proposed model is concerned for human individuals, we take an expansion of the classical SEIR model [9], refined by the introduction of $A$ & $H$ classes to take into account of asymptomatic and isolated individuals. Recent studies [8, 37, 38] has been demonstrated that the SARS-CoV-2 virus can be infected obliquely following interaction with the contaminated environment. Very recent investigation by Goldman [39] focused on the contaminated environment and shows that the transmission of COVID-19 can be minimized by maintaining the contaminated environment. Due to over populated country, crowded cities as well as different communities in India, we are interested to consider the following phenomena: soon after novel coronavirus infected individuals sneeze or cough and emancipate droplets for their noses & mouth, human touches their necessary things and use same places on which these droplets are remained. Moreover, the vulnerable conditions of public health service is a very good reason due to high density of the COVID-19 in the environments. Due to the reasons, we incorporate another important sub-class of contaminated environment, $C(t)$. There are some investigations focused on the contaminated environment to better understand the transmission dynamics novel coronavirus. A flow-diagram of the above biological mechanism for the interactive dynamics of COVID-19 has been shown in the Fig. 1. The dynamics of SARS-CoV-2 model is given by the following system of coupled ordinary differential equations.

2.1 Dynamics of susceptible individuals $S(t)$

The uninfected population is enlisted into the individuals with a source rate $\Lambda_s$ and diminished by a death rate $\delta_s$. The term $\beta_s \alpha_s$ describes the disease transmissibility constant of the asymptomatic populations to the susceptible, $\beta_s \alpha_i$ is the disease transmission coefficient of the clinically ill compartment to the susceptible compartment, and $\beta_s \alpha_h$ denotes the disease transmissibility term of the isolated populations from the susceptible individuals. Here, $\beta_s$ represents the infection transmissibility coefficient rate for COVID-19 infection rate, with modification parameters $\alpha_a$ (for asymptomatic compartments), $\alpha_i$ (for clinically ill populations) and $\alpha_h$ (for isolated populations).

However, the novel coronavirus is considered to be epidemic entirely due to symptomatic individuals with a lower rate of infection due to asymptomatic individuals that can be neglected for our proposed model. The modification term $\alpha_a \geq 0$ is considered to describe altering levels of hygiene precautions via asymptomatic, a similar conclusion can be described due to the modification terms $\alpha_i$ and $\alpha_h$ during clinically ill and isolated individuals, respectively. As the asymptomatic, clinically ill and isolated populations and hygiene precautions were continued and increased successively the outbreak of COVID-19, the terms $\alpha_a \beta_s$, $\alpha_i \beta_s$ and $\alpha_h \beta_s$, asymptomatic, clinically ill and isolation rates could be considered as time-dependents in numerical illustrations. Furthermore, the interaction between uninfected
and infected individuals (asymptomatic, clinically ill and isolated) is modeled using classical homogeneous mixing terms \[40, 42\], in terms of the entire population. Due to the incorporation of social-distancing and contact tracing strategy (for example, lockdown or home-quarantine), it is essential to consider that the interaction rate is a non-increasing function with time. Herein the parameter \(\beta_c\) denotes the contaminated environmental factor, representing the contact rate of individuals who touch with the necessary things everyday.}

Depends on the above biological framework, the equation for susceptible individuals is described by the following ordinary differential equation (ODE):

\[
\frac{dS}{dt} = \Lambda_s - \beta_s S (\frac{\alpha_a A + \alpha_i I + \alpha_h H}{N}) - \beta_c SC - \delta_s S. \tag{1}
\]

### 2.2 Dynamics of exposed populations E(t)

The individuals known as exposed who are infected by the virus but not yet progressed the medical signs of SARS-CoV-2 virus. The exposed population can be diminished due to asymptomatic and symptomatic population with rate \(\gamma_e\) & innate mortality rate \(\delta_e\). The constant per-capita rate \(\gamma_e\) is modeled via exposed class \(E\) to asymptomatic class \(A\) or clinically ill class \(I\); here \(q_a(0 < q_a < 1)\) describes the rate of progression of individuals who develop to the asymptomatic class \(A\) or clinically ill class \(I\) at the rate \(\gamma_e\). The rate of change of exposed individuals is described by

\[
\frac{dE}{dt} = \beta_s S (\frac{\alpha_a A + \alpha_i I + \alpha_h H}{N}) - \delta_e E - \gamma_e E. \tag{2}
\]

### 2.3 Dynamics of asymptomatic individuals A(t)

Asymptomatic individuals were exposed to the SARS-CoV-2 virus but are not able to spread the diseases among the population. The latent individuals developed to asymptomatic individuals with rate \(\gamma_e\) with a fraction \((1 - q_a)\). The asymptotic population develop to recovered individuals with rate \(\xi_a\) and death rate \(\delta_a\). Hence, the equation for asymptomatic individuals is given by

\[
\frac{dA}{dt} = (1 - q_a) \gamma_e E - \delta_a A - \xi_a A. \tag{3}
\]

### 2.4 Dynamics of symptomatic populations I(t)

The clinically ill populations are generated after the development of medical signs COVID-19 virus due to asymptomatic populations. The exposed populations develop the symptomatic populations at the rate \(\gamma_e\) a constant portion \(q_a\). The clinically ill populations develop to the hospitalized class \(H\) with rate \(\gamma_i\) with death rate \(\delta_i\). We assumed that \(\gamma_i\), the rate represents the clinically ill individuals needs medical treatment and thus move to the isolated compartment, which is greater that \(\gamma_e\), the rate represents the latent populations develop to asymptomatic and symptomatic compartments. The symptomatic individuals \(I\) are developed to the recovered individuals without being diagnosed at a rate \(\xi_i\). The dynamics of clinically ill populations is described by the given ODE

\[
\frac{dI}{dt} = q_a \gamma_e E - \delta_i I - \xi_i I - \gamma_i I. \tag{4}
\]
2.5 Dynamics of isolated populations \( H(t) \)

The individuals who have developed medical signs for the COVID-19 virus and has been isolated to the hospital or nursing home or intensive care unit (ICU) for the treatment. The isolated populations are came from the symptomatic individuals \( (I) \) at the rate \( \gamma_i \) with natural death rate \( \delta_h \). Hospitalized individuals recovered at the rate \( \xi_h \). We considered that \( \xi_h > \xi_i \), as an isolated individuals are most likely to get an intensive care during isolation. The kinetics of isolated populations are described by the following equation

\[
\frac{dH}{dt} = \gamma_i I - \delta_h H - \xi_h H. \tag{5}
\]

2.6 Dynamics of recovered populations \( R(t) \)

We considered that the recovered individuals have acquired immunity to combat COVID-19 virus in spite of this has neither been confirmed nor contradicted. The asymptomatic, clinically ill and isolated populations recovered from the SARS-CoV-2 virus with rates \( \xi_a, \xi_i \) and \( \xi_h \), respectively. The recovered population decays with the rate \( \delta_r \). The rate of change of the recovered population is described by

\[
\frac{dR}{dt} = \xi_a A + \xi_i I + \xi_h H - \delta_r R. \tag{6}
\]

2.7 Environmental contamination \( C(t) \)

The symptomatic and asymptomatic individuals have the potential to spread the COVID-19 virus among others through direct transmission by close contact. The infected individual spreads COVID-19 virus in the environment by sneezing and coughing. The COVID-19 virus can survive for several days in damp, dark and cold environment \cite{3,43}. Thus the virus transmitted indirectly to the people who stay in such a contaminated environment for a longer time. In this study we incorporate COVID-19 contamination of the environment by the asymptomatic \( (A) \) and symptomatic \( (I) \) individuals. The virus in the contaminated environment is denoted by \( C(t) \) at any time \( t \). The environment is contaminated due to COVID-19 virus by the asymptomatic individuals \( (A) \) at the rate \( \tau_1 \) and the symptomatic individual \( (I) \) contaminates the environment at the rate \( \tau_2 \). The coronavirus cannot survive in the environment for an infinite time due to sanitization and self-life time of the virus. Hence we assume that the novel coronavirus is cleared from the contaminated environment at the rate \( \tau_c \). Therefore the environmental contamination is modeled by the given equation

\[
\frac{dC}{dt} = \tau_1 A + \tau_2 I - \tau_c C. \tag{7}
\]

Bringing together all the above equations, the COVID-19 dynamics is represented by the system of coupled ordinary differential equations (the schematic representation of the mathematical model is described in the Fig. 1 and the description of the model parameters are explained in the Table 2):

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda_s - \beta_s \left( \frac{\alpha_a A + \alpha_i I + \alpha_h H}{N} \right) S - \beta_c SC - \delta_s S, \\
\frac{dE}{dt} &= \beta_s \left( \frac{\alpha_a A + \alpha_i I + \alpha_h H}{N} \right) S + \beta_c SC - (\delta_c + \gamma_e)E, \\
\frac{dA}{dt} &= (1 - q_a)\gamma_e E - (\delta_a + \xi_a)A, \\
\frac{dI}{dt} &= q_a\gamma_e E - (\delta_i + \xi_i + \gamma_i)I, \\
\frac{dH}{dt} &= \gamma_i I - (\delta_h + \xi_h)H, \\
\frac{dR}{dt} &= \xi_a A + \xi_i I + \xi_h H - \delta_r R, \\
\frac{dC}{dt} &= \tau_1 A + \tau_2 I - \tau_c C,
\end{align*}
\]

with non-negative initial values

\[
\begin{align*}
S(t_0) &= S_{t_0} \geq 0, & E(t_0) &= E_{t_0} \geq 0, & A(t_0) &= A_{t_0} \geq 0, \\
I(t_0) &= I_{t_0} \geq 0, & H(t_0) &= H_{t_0} \geq 0, & R(t_0) &= R_{t_0} \geq 0, & C(t_0) &= C_{t_0} \geq 0.
\end{align*}
\tag{9}
\]

3 Qualitative properties of the model

In this section, we performed the qualitative behavior of the proposed model (8) including positivity of the solutions, boundedness of the system, determination of the biologically realistic equilibrium points and their stability analysis and the persistent of the diseases.

3.1 Positive invariance

In this subsection, we shall investigate the positivity of the solutions of (8) with respect to the initial values \( (S(0), E(0), A(0), I(0), H(0), R(0), C(0)) \in \mathbb{R}^+_0 \). To prove the positivity, we consider the theorem as follows.

**Theorem 1** If the solutions of (8) with initial values (9) satisfying \( S(t) > 0, E(t) > 0, A(t) > 0, I(t) > 0, H(t) > 0, R(t) > 0, \) and \( C(t) > 0 \) for all \( t > 0 \). Then the system (8) is positively invariant and attracting in \( \mathbb{R}^+_0 \).

**Proof** First equation of (8) we can be written as

\[
\frac{dS}{dt} = \Lambda_s - \varnothing(t)S,
\]
where
\[ \vartheta(t) = \frac{\beta_s}{N} \left( \frac{\alpha_d A + \alpha_t I + \alpha_h H}{N} + \beta_e C + \delta_s \right). \]

It follows that
\[ S(t) = S_{t_0} e^{-\int_0^t \vartheta(s) \, ds} + \Lambda_s e^{-\int_0^t \vartheta(s) \, ds} \int_0^t e^{\int_0^s \vartheta(s) \, ds} \, ds, \]
which is positive as long as \( S_{t_0} \geq 0 \). From the second equation of (8), we get
\[ \frac{dE}{dt} \geq - (\delta_e + \gamma_e) E, \]
which leads to
\[ E(t) \geq E_{t_0} \exp \left[ - \int_0^t (\delta_e + \gamma_e) \, ds \right] > 0. \]

In similar way, from the third equation of (8), we have
\[ \frac{dA}{dt} \geq - (\delta_a + \xi_a) A, \]
which gives
\[ A(t) \geq A_{t_0} \exp \left[ - \int_0^t (\delta_a + \xi_a) \, ds \right] > 0. \]

In the following way, we can easily show that \( I(t) \), \( H(t) \), \( R(t) \), and \( C(t) \) are all strictly non-negative for all finite time or \( t > 0 \). Thus, all the solutions of (8) remain nonnegative for all finite time, that is, \( \forall \ t > 0 \).

### 3.2 Boundedness

In this subsection, we study the boundedness of the system (8) as none of the populations can not grow unboundedly. To do this, we state the theorem that assured that the solutions of (8) is bounded if we start with non-negative initial conditions.

**Theorem 2** All the solutions of (8) with nonnegative initial values (9) that starts in \( R^+_0 \), are uniformly bounded in the \( \Theta \), where \( \Theta \) is defined in the proof.

**Proof** Now, we verify that all the solutions are bounded in \( \Theta \). From the positivity, it is clear that
\[ \frac{dS}{dt} \leq \Lambda_s - \delta_s S, \]
which implies that
\[ \limsup_{t \to \infty} S(t) \leq \frac{\Lambda_s}{\delta_s}. \]

Adding the first six equations (\( N = S + E + A + I + H + R \)) yields
\[ \frac{dN}{dt} \leq \Lambda_s - \delta N, \]
where \( \delta = \min \{ \delta_s, \delta_e, \delta_a, \delta_i, \delta_h, \delta_r \} \), which implies that
\[ \limsup_{t \to \infty} N \leq \frac{\Lambda_s}{\delta}. \]

From the last equation of (8), we have
\[ \frac{dC}{dt} = \tau_1 A + \tau_2 I - \tau_e C \leq \frac{\Lambda_s (\tau_1 + \tau_2)}{\delta \tau_e}, \]
this implies that
\[ \limsup_{t \to \infty} C \leq \frac{\Lambda_s (\tau_1 + \tau_2)}{\delta \tau_e}. \]

Therefore, we get the positively invariant set as
\[ \Theta = \left\{ (S, E, A, I, H, R, C) \in R^+_0 : S \leq \frac{\Lambda_s}{\delta}, \quad S + E + A + I + H + R \leq \frac{\Lambda_s (\tau_1 + \tau_2)}{\delta \tau_e} \right\}. \]

Therefore, the solutions in \( R^+_0 \) will enter and remain in the region \( \Theta \) for all finite time. Thus, the dynamics of the system (8) can be considered in \( \Theta \).

### 3.3 Basic reproduction number

It can be noticed that the compartments identified as infected individuals for the system (8) are \( E, A, I, H, R \), and \( C \). By decomposing the system (8), we may rewrite it as \( \frac{dX}{dt} = G - K \), where the matrix \( G \) stands for inclusion of infections in the system and the matrix \( K \) is set for transmission of infection indicating the behavioral changes in the infected states. The matrices \( G \) and \( K \) can be considered as follows
\[ G = \begin{pmatrix} \beta_s \left( \frac{\alpha_d A + \alpha_t I + \alpha_h H}{N} \right) & S + \beta_e SC \\ 0 & 0 \\ 0 & 0 \\ 0 & 0 \end{pmatrix} \quad \text{and} \quad K = \begin{pmatrix} (\delta_e + \gamma_e) E \\ - (1 - q_a) \gamma_e E + (\delta_a + \xi_a) A \\ -q_a \gamma_e E + (\delta_i + \xi_i + \gamma_i) I \\ -\gamma_i I + (\delta_h + \xi_h) H \\ -\tau_1 A + \tau_2 I + \tau_e C \end{pmatrix}. \]

Now using the next-generation matrix method [44], the Jacobians \( G \) and \( K \) of the matrices \( G \) and \( K \) respectively...
are computed as

\[ G = \begin{pmatrix}
0 & \beta_s \alpha_a & \beta_s \alpha_i & \beta_s \alpha_h & \frac{\Delta_s}{\beta_s} \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0
\end{pmatrix}, \]

and

\[ K = \begin{pmatrix}
\delta_c + \gamma_e & 0 & 0 & 0 & 0 \\
-1 - q_a \gamma_e & \delta_i + \xi_i + \gamma_i & 0 & 0 & 0 \\
-q_a \gamma_e & 0 & \delta_i + \xi_i + \gamma_i & 0 & 0 \\
0 & 0 & -\gamma_i & \delta_h + \xi_h & 0 \\
0 & -\tau_1 & -\tau_2 & 0 & \tau_c
\end{pmatrix}. \]

Due to the method developed in [44], we determine that \( GK^{-1} \) is the next-generation matrix of (8) expressing the expected number of secondary infections and \( R_0 \) is the spectral radius, that is, the largest eigenvalue of the next-generation matrix \( GK^{-1} \) defined as basic reproduction number of (8). Therefore, we have

\[ \rho(GK^{-1}) = R_0 = \frac{(1 - q_a)\gamma_e \beta_s \alpha_a}{\delta_e + \gamma_e}(\delta_i + \xi_i + \gamma_i) + \frac{q_a \gamma_e \beta_s \alpha_i}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)} \]

\[ + \frac{q_a \gamma_e \beta_s \alpha_h \gamma_i}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i + (\delta_h + \xi_h) \delta_i + \xi_i + \gamma_i} + \frac{\Lambda_s \beta_s \alpha_a \tau_1}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} + \frac{\Lambda_s \beta_s \alpha_i \tau_2}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i}. \]

### 3.4 Sensitivity analysis of \( R_0 \)

In this section, we performed normalized forward sensitivity analysis for \( R_0 \) to investigate the importance of each system parameters in disease transmission. Sensitivity indices gives a measure of the relative alteration of \( R_0 \), when a system parameter is varied. Sensitivity analysis is primarily utilized to investigate the robustness of the system outputs to the parameter values related to \( R_0 \), as there are errors in collected data and considered parameters. Hence, we use the following definition.

**Definition 1** If the basic reproduction number \( R_0 \) is differentiable with respect to parameter \( \sigma \), then the forward sensitivity indices of \( R_0 \) with reference to \( \sigma \) is stated as

\[ \Gamma_{R_0}^{\sigma} = \frac{\partial R_0}{\partial \sigma} \times \frac{\sigma}{R_0}. \]

Now we calculate sensitivity indices of all parameters associated to \( R_0 \) are as follows

\[ \Gamma_{R_0}^{\beta_s} = \frac{\partial R_0}{\partial \beta_s} \times \frac{\beta_s}{R_0} = 1, \]

\[ \Gamma_{R_0}^{\beta_c} = \frac{\Lambda_s \beta_s \alpha_a \tau_1}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} + \frac{\Lambda_s \beta_s \alpha_i \tau_2}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} \]

\[ \Gamma_{R_0}^{\alpha_a} = \frac{(1 - q_a) \gamma_e \beta_s}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} + \frac{q_a \gamma_e \beta_s \alpha_i}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} \]

\[ \Gamma_{R_0}^{\alpha_i} = \frac{\Lambda_s \beta_s \alpha_a \tau_1}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} + \frac{\Lambda_s \beta_s \alpha_i \tau_2}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} \]

\[ \Gamma_{R_0}^{\alpha_h} = \frac{q_a \gamma_e \beta_s \alpha_a}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} \times \frac{\alpha_h}{R_0}, \]

\[ \Gamma_{R_0}^{\delta_c} = \frac{\delta_e + \gamma_e}{R_0}, \quad \Gamma_{R_0}^{\delta_i} = 0, \]

\[ \Gamma_{R_0}^{\alpha_a} = \frac{(1 - q_a) \gamma_e \beta_s \alpha_a}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} + \frac{q_a \gamma_e \beta_s \alpha_i \gamma_i}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} \]

\[ \Gamma_{R_0}^{\alpha_h} = \frac{q_a \gamma_e \beta_s \alpha_h \gamma_i}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} \times \frac{\alpha_h}{R_0}, \]

\[ \Gamma_{R_0}^{\gamma_e} = \frac{(1 - q_a) \beta_s \alpha_a}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} + \frac{q_a \beta_s \alpha_i}{(\delta_e + \gamma_e) \delta_i + \xi_i + \gamma_i} \]

\[ + \frac{\alpha_h}{R_0} \times \gamma_e - \frac{\delta_e + \gamma_e}{R_0}. \]
\[
\Gamma^R_{\xi_i} = \left[ -\frac{q_a \gamma_e \beta_s \alpha_i}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)^2} + \frac{q_a \gamma_e \beta_s \alpha_h}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)(\delta_h + \xi_h)} - \frac{q_a \gamma_e \beta_s \alpha_h \gamma_i}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)^2(\delta_h + \xi_h)} - \frac{\Lambda_s \beta_s \alpha_i \tau_2}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)(\delta_h + \xi_h)} \right] \times \frac{\gamma_i}{R_0},
\]
\[
\Gamma^R_{\xi_h} = -\frac{q_a \gamma_e \beta_s \alpha_h \gamma_i}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)(\delta_h + \xi_h)^2} \times \frac{\xi_h}{R_0},
\]
\[
\Gamma^R_{\tau_1} = \frac{\Lambda_s \beta_s \alpha_i}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)(\delta_h + \xi_h)} \times \frac{\tau_1}{R_0},
\]
\[
\Gamma^R_{\tau_2} = \frac{\Lambda_s \beta_s \alpha_i}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)(\delta_h + \xi_h)} \times \frac{\tau_2}{R_0},
\]
\[
\Gamma^R_{\tau_c} = -\left[ \frac{\Lambda_s \beta_s \alpha_i \tau_1}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)^2(\delta_h + \xi_h)} + \frac{\Lambda_s \beta_s \alpha_i \tau_2}{(\delta_e + \gamma_e)(\delta_i + \xi_i + \gamma_i)(\delta_h + \xi_h)} \right] \times \frac{\tau_c}{R_0}.
\]

From the above expressions we obtain that \(\Gamma^R_{\beta_s}, \Gamma^R_{\beta_c}, \Gamma^R_{\alpha_s}, \Gamma^R_{\alpha_i}, \Gamma^R_{\alpha_h}, \Gamma^R_{\gamma_e}, \Gamma^R_{\gamma_i}, \Gamma^R_{\gamma_h}, \Gamma^R_{\delta_s}, \Gamma^R_{\delta_i}, \Gamma^R_{\delta_h}, \Gamma^R_{\delta_c}, \Gamma^R_{\xi_a}, \Gamma^R_{\xi_i}, \Gamma^R_{\xi_h}, \Gamma^R_{\tau_1}, \Gamma^R_{\tau_2}, \Gamma^R_{\tau_c} > 0\), which implies that the increase of \(R_0\) produces equal and opposite changes in the parameters. \(\alpha_s, \alpha_i, \alpha_h\) will increase the value of \(R_0\). Again, \(\gamma_e, \gamma_i, \gamma_h\) and \(\delta_s, \delta_i, \delta_h\) will cause the decrease of \(R_0\) as \(\Gamma^R_{\gamma_e}, \Gamma^R_{\gamma_i}, \Gamma^R_{\gamma_h}, \Gamma^R_{\delta_s}, \Gamma^R_{\delta_i}, \Gamma^R_{\delta_h} < 0\). Again the rate of recovery \((\xi_a, \xi_i, \xi_h)\) decreases increases then the reproduction number \(R_0\) decreases. Here \(\Gamma^R_{\gamma_e} < 0\) and \(\Gamma^R_{\gamma_i} < 0\), which implies that increase in rate of conversion from asymptomatic to hospitalized \((\gamma_i)\) and from exposed to asymptomatic \((\gamma_e)\) will cause \(R_0\) to decrease. Since \(\Gamma^R_{\delta_c} < 0\) interprets that the increase in clearance of the virus from the environment by sanitization will cause \(R_0\) to decrease. The numerical values of the sensitivity indices are computed and presented in the Table 1 and graphically presented in the Fig. 2.

We observe that \(\Gamma^R_{\beta_s} = -\Gamma^R_{\beta_c}\), which implies that the perturbations in the parameter \(\beta_s\) (or \(\beta_c\)) and \(\tau_c\) produces equal and opposite changes in \(R_0\). Also, \(\Gamma^R_{\alpha_s} = -\Gamma^R_{\xi_a}\), which implies that the perturbation in the parameters \(\alpha_s\) and \(\xi_a\) produces equal and opposite changes in \(R_0\). The absolute value of the normalized forward sensitivity indices become larger for the parameters \(\beta_s, \beta_c\) and \(\tau_c\). Here \(\Gamma^R_{\beta_s} = \Gamma^R_{\beta_c} = 1\) implies that

\begin{table}
\centering
\caption{Sensitivity indices of \(R_0\) is calculated at the baseline parameters described in Table 2 and 3}
\begin{tabular}{ccc}
\hline
Parameters & Sensitivity index & Sensitivity index (without environment contamination) \\
\hline
\(\beta_s\) & 1.0000 & 1.0000 \\
\(\alpha_a\) & 0.4711 & 0.9963 \\
\(\alpha_i\) & 0.5289 & 0.0021 \\
\(\alpha_h\) & 0.3496 \times 10^{-11} & 0.0016 \\
\(\delta_s\) & -1.0000 & 0 \\
\(\delta_e\) & -0.1361 \times 10^{-3} & -0.1361 \times 10^{-3} \\
\(\delta_h\) & -0.8328 \times 10^{-4} & -0.1761 \times 10^{-3} \\
\(\delta_i\) & -0.0360 & -0.2511 \times 10^{-3} \\
\(\xi_a\) & -0.3177 \times 10^{-15} & -0.1416 \times 10^{-6} \\
\(\xi_i\) & 0.9999 & 0.1361 \times 10^{-3} \\
\(\xi_h\) & -0.4710 & -0.9691 \\
\(\beta_c\) & -0.2443 \times 10^{-12} & -0.1089 \times 10^{-3} \\
\(\tau_1\) & 1.0000 & - \\
\(\tau_2\) & 0.5289 & - \\
\(\tau_c\) & -1.0000 & - \\
\hline
\end{tabular}
\end{table}
if the parameter $\beta_s$ (or $\beta_c$) is increased by 10% then the value of $R_0$ will also increased by 10%. Again if $\Gamma_{R_0} = -1$ implies that if the parameter $\tau_c$ is increased by 10% then the value of $R_0$ will also decreased by 10%.

We have also considered the case without considering the effect of environment contamination. The magnitudes of the sensitivity indices are presented in the third column of Table 1 and graphically represented in the Fig. 3. In this scenario, we observed that $\Gamma_{R_0}$ is positive, that is, the rate of conversion from exposed to asymptomatic individuals ($\gamma_e$) has opposite impact in $R_0$, if the effect of environment contamination does not considered. So, in this scenario if $\gamma_e$ is increased then $R_0$ will also increased as $\Gamma_{R_0}^{\gamma_e} > 0$.

### 3.5 Stability analysis of the steady states

We carried out the local stability analysis for the system (8) by using the techniques of linearization, in this section. We linearized the system (8) around each of the biologically feasible equilibrium points and perturb the model system by a very small quantity, and determine whether the system return to its original equilibrium points or converge to any other equilibrium points. The local stability analysis provides an idea to comprehend the qualitative dynamics of the model under consideration.

To study the local stability analysis, we determine the biologically feasible equilibrium points. The system (8) has the following equilibria, namely

**Fig. 2** The plot represents the forward sensitivity indices of the reproduction number $R_0$ with respect to the system parameter values specified in the Table 2.

**Fig. 3** The plot represents the forward sensitivity indices of the reproduction number $R_0$ without considering the environmental contamination effect. The system parameters are defined in Tables 2 and 3.
(i) a unique infection-free equilibrium point $P_0(S_0, 0, 0, 0, 0, 0)$, where $S_0 = \frac{\beta_c}{\eta}$ represents the level of susceptible population in absence of any infection, and

(ii) a unique chronic infection equilibrium $P^*(S^*, E^*, A^*, I^*, H^*, R^*, C^*)$ where

$$S^* = \frac{\Lambda_s}{\eta + \delta_s}, \quad E^* = \frac{\eta}{\omega_1} S^*, A^* = \frac{(1 - q_a)\gamma_c E^*}{\omega_2},$$

$$I^* = \frac{q_a \gamma_c E^*}{\omega_3},$$

$$H^* = \frac{q_a \gamma_c \gamma_i E^*}{\omega_4},$$

$$R^* = \left(\frac{(1 - q_a)\gamma_c \xi_a}{\omega_2} + \frac{q_a \gamma_c \gamma_i \xi_h}{\omega_3} + \frac{q_a \gamma_c \gamma_i \xi_h}{\omega_4}\right) E^*,$$

$$C^* = \frac{1}{\tau_c} \left(\frac{(1 - q_a)\gamma_c \tau_1}{\omega_2} + \frac{q_a \gamma_c \tau_2}{\omega_3}\right) E^*.$$

Considering the above result as

$$\eta(t) = \frac{\beta_s (\alpha_A + \alpha_I + \alpha_H)}{N} + \beta_c C,$$

then, we have

$$m_1\eta^2 + m_2\eta + m_3 = 0,$$

where

$$m_1 = \omega_1 \omega_2 \omega_3 \omega_4 \delta_s \tau_c (\omega_1 \omega_2 \omega_3 + (1 - q_a)\gamma_c \omega_3 \omega_4 (1 + \xi_a) + q_a \gamma_c \omega_2 \omega_4 (1 + \xi) + q_a \gamma_c \gamma_i \omega_2 (1 + \xi h)), $$

$$m_2 = \omega_1 \omega_2 \omega_3 \omega_4 \delta_s \tau_c (\omega_1 \omega_2 \omega_3 + (1 - q_a)\gamma_c \omega_3 \omega_4 (1 + \xi_a) + q_a \gamma_c \omega_2 \omega_4 (1 + \xi) + q_a \gamma_c \gamma_i \omega_2 (1 + \xi h)) + \Lambda_s \beta_s \beta_c \omega_4 (\alpha \xi_1 \omega_3 + \alpha \tau_2 \omega_2) + (1 - \Lambda_s) \omega_1 \omega_2 \omega_3 \omega_4 \delta_s \tau_c (\omega_1 \omega_2 \omega_3 + (1 - q_a)\gamma_c \omega_3 \omega_4 (1 + \xi_a) + q_a \gamma_c \omega_2 \omega_4 (1 + \xi) + q_a \gamma_c \gamma_i \omega_2 (1 + \xi h)), $$

$$m_3 = (1 - \Lambda_s) \omega_1^2 \omega_2^2 \omega_3^2 \omega_4^2 \delta_s \tau_c.$$

Here, $m_1 > 0$ and $m_3$ depends on the sign of $R_0$, which is positive when $R_0 < 1$ and negative when $R_0 > 1$. We summarized the above results are as follows.

**Theorem 3** The model system (8) has the following properties:

(i) if $m_3 < 0$ and $R_0 > 1$, then there exists a unique endemic equilibrium point;

(ii) if $m_2 < 0$ and $m_3 = 0$, then we have a unique endemic equilibrium point;

(iii) if $m_3 > 0$, $m_2 < 0$ and their discriminant is positive then two endemic equilibria exist;

and (iv) otherwise, no possibilities of equilibrium point.

It can be observed from the first point of the Theorem 3 that for $R_0 > 1$, we obtained a unique positive endemic equilibrium point. The third point of the Theorem 3 assured that the possibility of backward bifurcation when $R_0 < 1$.

To study the stability of disease-free steady state $P_0$ and chronic infection steady state $P^*$, use the following theorem.

**Theorem 4** The infection-free equilibrium point $P_0(S_0, 0, 0, 0, 0, 0, 0)$, where $S_0 = \frac{\beta_c}{\eta}$ is locally asymptotically stable in the feasible region $\Theta$ for $R_0 < 1$, otherwise unstable.

Proof To determine the local asymptotic stability of the infection-free equilibrium point $P_0(\Lambda_s/\delta_s, 0, 0, 0, 0, 0, 0)$, we calculate the variational matrix of (8) is

$$J_{P_0} =
\begin{pmatrix}
\delta_s & 0 & -\beta_s \alpha_a & -\beta_s \alpha_i & -\beta_s \alpha_h & 0 & -\beta_s \frac{\Lambda_s}{\delta_s} \\
0 & -\omega_1 & \beta_s \alpha_a & \beta_s \alpha_i & \beta_s \alpha_h & 0 & \beta_s \frac{\Lambda_s}{\delta_s} \\
0 & 0 & 0 & -\omega_2 & 0 & 0 & 0 \\
0 & q_a \gamma_c & 0 & 0 & -\omega_3 & 0 & 0 \\
0 & 0 & 0 & 0 & -\omega_4 & 0 & 0 \\
0 & 0 & \xi_a & \xi_i & \xi_h & -\tau_c & 0 \\
0 & \tau_1 & \tau_2 & 0 & 0 & 0 & -\tau_c
\end{pmatrix},$$

where $\omega_1 = (\delta_c + \gamma_c),$ $\omega_2 = \delta_a + \xi_a, \omega_3 = \delta_i + \xi_i + \gamma_i, \omega_4 = \delta_h + \xi_h.$ Corresponding characteristics polynomial is given by $|J_{P_0} - \lambda I| = 0$,

$$(\lambda + \delta_s)(\lambda + \delta_i)[(\omega_1 + \lambda)(\omega_2 + \lambda)(\omega_3 + \lambda)(\omega_4 + \lambda)(\tau_c + \lambda) - \Lambda_s(1 - q_a)(\omega_3 + \lambda)(\omega_4 + \lambda)] = 0.$$
Remark The aforementioned explanation establishes the fact that the basic reproduction number for viral infection $R_0$ acts as a threshold parameter, which characterized the local asymptotic stability for the infection-free fixed point $P_0$.

Moreover, a more interesting explanation can be drawn about the stability of an infection-free fixed point $P_0$ for $R_0 < 1$, namely that it is the global asymptotic stability of $P_0$ in the biologically feasible region $\Theta$. In the following theorem, we prove that the infection free fixed point $P_0$ is globally asymptotically stable by constructing suitable Lyapunov function.

**Theorem 5** The infection-free fixed point $P_0$ is globally asymptotically stable for the system (8) in the feasible region $\Theta$ when $\Lambda_s \beta_e < \tau_e \delta_s$, $\tau_1 < \delta_a$ and $\tau_2 < \delta_i$ holds.

**Proof** First, we consider the Lyapunov function for the infection-free fixed point as

$$\mathcal{L}_{P_0} = \left( S - S^0 - S^0 \ln \frac{S}{S^0} \right) + E + A + I + H + R + C.$$

Here, we choose a Lyapunov function $\mathcal{L}_{P_0}$ is positive definite function for all $(S, E, A, I, H, R, C)$ other than the infection free fixed point $P_0$. Now Compute the time derivative of $\mathcal{L}_{P_0}$ along with the solutions of system (8) as follows

$$\frac{d\mathcal{L}_{P_0}}{dt} = \left( 1 - \frac{S^0}{S} \right) \left( \Lambda_s - \beta_s \left( \frac{\alpha_a A + \alpha_s I + \alpha_h H}{N} \right) \right)$$

$$+ \beta_s \left( \frac{\alpha_a A + \alpha_s I + \alpha_h H}{N} \right) S$$

$$+ \beta_e SC - \delta_i S$$

$$+ \delta_a S^0 - \delta_e E - \delta_s A - \delta_i I - \delta_h H - \delta_r R$$

$$+ \tau_1 A + \tau_2 I - \tau_e C$$

$$= \Lambda_s - \delta_i S - \Lambda_s \frac{S^0}{S} + \beta_s S^0 C$$

$$+ \delta_s S^0 - \delta_e E - \delta_s A - \delta_i I - \delta_h H - \delta_r R$$

$$+ \tau_1 A + \tau_2 I - \tau_e C$$

$$\leq \left( \frac{\Lambda_s}{S} - \delta_s \right) (S - S^0) + \frac{1}{\delta_s} (\Lambda_s \beta_e - \tau_e \delta_s) C$$

$$+ (\tau_1 - \delta_a) A + (\tau_2 - \delta_i) I$$

$$= \frac{\delta_s}{\delta_s} (S - S^0) + \frac{1}{\delta_s} (\Lambda_s \beta_e - \tau_e \delta_s) C$$

$$+ (\tau_1 - \delta_a) A + (\tau_2 - \delta_i) I.$$
Furthermore, \( \exists \) an interior steady state in this case.

**Proof** To prove the disease persistence, we employ the theorem by H.R. Thieme [46]. To do this, we set

\[
V = (S, E, A, I, H, R, C), \quad \dot{V} = (E, A, I, H, C),
\]

where \( V_i \) is the \( i \)-th component of \( V \).

\[
B = \{ V \in R^7_+ : V_i \geq 0, i = 1, ..., 7, \}
\]

where \( B \) is the equilibrium manifold for the equilibrium \( P \).

\[
B_0 = \{ V \in B : V_i > 0, i = 2, ..., 5, 7, \}
\]

\[
D = B / B_0 = \{ V \in B : V_i = 0, \text{ for some } i = 2, ..., 5, 7 \}
\]

Now, we prove that the model (8) is uniformly permanence with reference to \( (B_0, D) \). As \( D \) represents a single singular point \( P_0 \), it is enough to prove that \( U^* (P_0) \cap B_0 = \phi \), where \( U^* (P_0) \) represents the stable manifold for the equilibrium \( P_0 \). To prove by contradiction, we assume that the condition is false. There is a solution \( (S, E, A, I, H, R, C) \in B_0 \) for the system (8) such that the initial point satisfies

\[
\lim_{t \to \infty} (S(t), E(t), A(t), I(t), H(t), R(t), C(t)) = \left( \frac{A_s}{\delta_s}, 0, 0, 0, 0, 0, 0 \right).
\]

Then for any \( \varrho > 0 \), we obtain

\[
\frac{A_s}{\delta_s} - \varrho \leq S \leq \frac{A_s}{\delta_s} + \varrho, \quad 0 \leq V_i \varrho, \quad i = 2, ..., 5, 7,
\]

for sufficiently large \( t \). From the system (8), it follows that

\[
\frac{dE}{dt} = \left( \frac{\beta_s S}{T} (\alpha_a A + \alpha_I I + \alpha_h H) + \beta_s C \right) E - \omega_1 0 0 0 0 0
\]

\[
+ \left( \begin{array}{cccccc}
-\varrho_e & 0 & 0 & 0 & 0 & 0 \\
q_a \varrho_e & 0 & -\varrho_1 & 0 & 0 & 0 \\
0 & 0 & \gamma_I & -\varrho_4 & 0 & 0 \\
0 & \tau_1 & \tau_2 & 0 & -\tau_c & 0 \\
0 & \varrho_e & 0 & -\varrho_1 & 0 & 0 \\
0 & 0 & \gamma_I & -\varrho_4 & 0 & 0 \\
0 & \tau_1 & \tau_2 & 0 & -\tau_c & 0 \\
\end{array} \right)
\]

\[
= J(\varrho) \dot{V},
\]

where

\[
\dot{S}(\varrho) = \frac{A_s}{\delta_s} - \varrho, \quad \delta_s + \varrho \]

and

\[
\tilde{J}(0) = \left( \begin{array}{cccccc}
-\omega_1 & \beta_s \alpha_a & \beta_s \alpha_I & \beta_s \alpha_h & \beta_s S_0 \\
(1 - q_a) \gamma_e & -\varrho_1 & 0 & 0 & 0 \\
q_a \gamma_e & 0 & -\varrho_3 & 0 & 0 \\
0 & 0 & \gamma_I & -\varrho_4 & 0 \\
0 & \tau_1 & \tau_2 & 0 & -\tau_c \\
\end{array} \right).
\]

It can be observed that \( \tilde{J}(0) \) is same as the expression of \( F - V \), has at least 1 eigenvalue with nonnegative real part for \( R_0 > 1 \). As \( \varrho > 0 \) chosen at arbitrary and sufficiently small in such a way that \( q(\tilde{J}(\varrho)) \) become positive, where \( h(P) \) is the maximal real part for the eigenvalues of the matrix \( P \). Thus, \( \exists \) solutions for the linear system

\[
\frac{d\dot{V}}{dt} = \tilde{J}(\varrho) \dot{V},
\]

that can proliferate exponentially. Due to comparison argument, solutions of \( V \) never be bounded for \( t \to \infty \), which provides a contradiction to the reality that the solutions of (8) are uniformly bounded. Thus, we have \( U^* (M_0) \cap B_0 = \phi \). By using the Theorem 4.6 [46], we can conclude that the model (8) is uniformly persistent [41, 47].

Moreover, the model (8) is dissipative (verified in Theorem 2), therefore due to the Theorem 3.3 in [48], implies that the system (8) has an endemic steady state (i.e., all components are nonnegative). This gives the theorem.

To determine the local asymptotic stability of a unique chronic infection equilibrium point \( P^* (S^*, E^*, A^*, I^*, H^*, R^*, C^*) \), we compute the following variation matrix around the endemic equilibrium point \( P^* \) is given by

\[
J(P^*) = \left( \begin{array}{cccccccc}
-p_{11} & 0 & -p_{13} & -p_{14} & -p_{15} & 0 & -p_{17} \\
p_{21} & -\varrho_1 & p_{13} & p_{14} & p_{15} & 0 & p_{17} \\
0 & p_{32} & -\varrho_2 & 0 & 0 & 0 & 0 \\
0 & p_{42} & 0 & -\varrho_3 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \gamma_I & -\varrho_4 & 0 \\
0 & 0 & 0 & 0 & \tau_1 & \tau_2 & 0 \\
0 & 0 & 0 & 0 & 0 & -\tau_c \\
\end{array} \right),
\]

where \( p_{11} = \beta_s \alpha_a A^* + \alpha_I I^* + \alpha_h H^* + \beta_c C^* + \delta_s, \quad p_{13} = \beta_s \alpha_a S^*, \quad p_{14} = \beta_s \alpha_I S^*, \quad p_{15} = \beta_s \alpha_h S^*, \quad p_{17} = \beta_s S^*, \quad p_{21} = \beta_s \alpha_a A^* + \alpha_I I^* + \alpha_h H^* + \beta_c C^*, \quad p_{32} = (1 - q_a) \gamma_e, \quad p_{42} = q_a \gamma_e. \)

To investigate the local asymptotically stability of an unique chronic-infection equilibrium point \( P^* \), one eigenvalue of the Jacobian matrix \( J(P^*) \) is \( -\delta_r \) and the
remaining six eigenvalues are the roots of the following polynomial

$$\varphi^6 + \kappa_1 \varphi^5 + \kappa_2 \varphi^4 + \kappa_3 \varphi^3 + \kappa_4 \varphi^2 + \kappa_5 \varphi + \kappa_6 = 0. \quad (11)$$

Coefficients for the characteristic equation (11) in the expressions for the $\kappa'_i$s for $i = 1, \ldots, 6$ are given by

$$\kappa_1 = p_{11} + r_c + \omega_1 + \omega_2 + \omega_3 + \omega_4,$$

$$\kappa_2 = p_{11} r_c + p_{11} \omega_1 + r_c \omega_1 + p_{11} \omega_2 + r_c \omega_2 + p_{11} \omega_3 + r_c \omega_3 + p_{11} \omega_4 - p_{13} p_{32} - p_{14} p_{42} + p_{14} \omega_4 + p_{13} \omega_4,$$

$$\kappa_3 = p_{13} p_{21} p_{32} - p_{11} p_{14} p_{42} + p_{14} p_{21} p_{42} - p_{15} p_{32} p_{64} - p_{17} p_{32} p_{71} - p_{17} p_{21} p_{42} - p_{13} p_{21} r_c - p_{14} p_{42} r_c + p_{11} r_c \omega_1 + p_{17} r_c \omega_2 + p_{11} r_c \omega_3 + p_{12} r_c \omega_2 + p_{13} p_{32} r_c + p_{11} p_{13} p_{32} + p_{11} \omega_1 r_c + \omega_2 r_c + \omega_3 r_c + \omega_4 - p_{13} p_{32} \omega_3 + p_{11} \omega_1 \omega_3 + p_{11} \omega_2 \omega_3 + r_c \omega_2 \omega_3 + \omega_1 \omega_2 \omega_3 - p_{13} p_{32} \omega_4 + p_{14} p_{42} \omega_4 + p_{11} r_c \omega_4 + \omega_1 \omega_3 \omega_4 + \omega_2 \omega_3 \omega_4.$$

$$\kappa_4 = p_{15} p_{21} p_{32} p_{64} - p_{11} p_{17} p_{32} p_{71} + p_{17} p_{21} p_{32}$$

$$\kappa_5 = p_{15} p_{21} p_{32} p_{64} r_c - p_{11} p_{17} p_{42} p_{72} + p_{17} p_{21} p_{42} p_{72} + p_{15} p_{32} p_{64} r_c - p_{11} p_{17} p_{32} p_{71} + p_{17} p_{21} p_{32} p_{71}$$

$$\kappa_6 = p_{15} p_{21} p_{32} p_{64} r_c \omega_3 - p_{11} p_{17} p_{32} p_{72} + p_{17} p_{21} p_{42} p_{72} + p_{15} p_{32} p_{64} r_c \omega_4 - p_{11} p_{17} p_{32} p_{71} + p_{17} p_{21} p_{32} p_{71}$$

We performed the numerical computations for the local asymptotic stability of the chronic infection fixed point $P^*$, in the numerical section, for the set of parameters are specified in the Table 2.

**4 Numerical simulation**

We have supported our proposed model illustration with daily novel coronavirus cases in India. The daily new SARS-CoV-2 viruses in India are obtained from the web portal of WHO for the time window March 02, 2020 to June 24, 2021. The data shows two COVID-19 waves in India. The proposed mathematical model for the COVID-19 pandemic simulated for the first COVID-19 wave in India from March 02, 2020 to January 31, 2021. The model is simulated without considering the effect of environment contamination as the data not available for environment contamination. The initial populations size and estimated parameters are given in the Table 3.

The observed novel coronavirus cases are shown in the Fig. 4 by the red dots and the solid blue graph denotes finest fitting curve for our proposed model illustrations. The value of the reproduction number, $R_0$ for the first COVID-19 wave in India is 1.8174 that represents significant outbreak of the SRAS-CoV-2 virus.

Again for the second wave of novel coronavirus in India, the proposed mathematical model simulated for the time window February 01, 2021 to June 24, 2021. The initial number of infected cases $I(t)$ are assumed the value of $I(t)$ on January 31, 2021 for the first wave. The observed novel coronavirus outbreak for second wave are also shown in the Fig. 4 by the red dots and the solid blue graph describes the finest fitting curve for our
proposed model illustrations. The value of the reproduction number, $R_0$ for the second COVID-19 wave in India is 2.7580, which also shows substantial outbreak of the novel coronavirus. The crest of the first COVID-19 wave in India is nicely captured by the model simulation. The crest for the second COVID-19 wave in India is higher than the model simulation. Though the model simulation nicely fitted with the second COVID-19 wave in India.

### 4.1 PRCC sensitivity analysis

The mathematical model for the transmission dynamics of novel coronavirus described in the equations (8) is studied so that the model parameter values has been obtained from diverse sources incorporating research papers, real data estimation due to their broad changeability. Therefore, a sensitivity analysis is performed by utilizing Partial Rank Correlation Coefficient (PRCC) procedure to investigate how the coronavirus system output is influenced by alters in a definite parameters whatever the unpredictability over the other system parameter values. In our coronavirus system has 21 parameters for that altered all the parameters consecutively, PRCC identifies the relation among a state variable of fascination & each of the system parameter. For that PRCC aid in finding the most effective parameters that gives most influence to the system changeability. For our model illustration, we have taken the PRCC values from $-1$ to $+1$ [49].

Due to the procedure established by Marino et al. [50], we conducted Latin hypercube sampling & created 3000 samples to calculate PRCC & p-values with respect to clinically ill or symptomatic classes for the time 60 days to investigate which parameters affect the system output significantly. The indices are calculated at time point 60 days prior to steady state. The PRCC outcomes has plotted in Fig. 5 which implies that the parameters for source rate of susceptible population $\Lambda_i$; disease transmission rate, $\beta_a$; adjustment factor for asymptomatic classes, $\alpha_a$; proportion of exposed individuals, $q_a$; recovery rate from asymptomatic individuals, $\xi_a$; recovery rate from symptomatic individuals, $\xi_i$; conversion rate from exposed individuals to asymptomatic individuals, $\gamma_c$; rate at which symptomatic individuals become hospitalized individuals, $\gamma_i$. The PRCC result shows in the Fig. 5 which implies that the symptomatic individuals over time is primarily affected due to alteration of the parameter values $\Lambda_s$, $\beta_s$, $\alpha_q$, $q_a$, $\bar{\xi}_s$, $\xi_i$, $\gamma_c$, $\gamma_i$ and other parameters are less sensitive with reference to the clinically ill classes. Thus, we estimated eight most sensitive parameters from the real data for COVID-19 epidemic.

### 4.2 Parameter estimation

The performance of the COVID-19 mathematical model is highly dependent on the numerical values of the system parameters and thus the estimation of the numerical values of system parameters is of utmost challenge. The numerical illustrations of (8) rely on the initial values of system variables and the parameter values of the system. The system parameter values have been calculated by considering initial size of the population and the simulations are fitted with daily new coronavirus cases. The considered initial populations size are described in Table 3. We calibrated eight system parameters, $\Lambda_s$, $\beta_s$, $\alpha_q$, $q_a$, $\bar{\xi}_s$, $\xi_i$, $\gamma_c$, $\gamma_i$ and $q_a$ as these parameters are most effective in PRCC technique (see Fig. 5). Parameters may also be estimated by the least square method [51]. The daily new coronavirus infection are fitted with the numerical illustrations of the model by utilizing the least square method that minimizes the sum of the square of errors. The sum of the square of the computation error are estimated as

$$
\sum_{i=1}^{n}(O(i) - P(i))^2,
$$

where $O(i)$ describes the daily novel coronavirus infected cases at i-th day, $P(i)$ represents the model illustration at i-th day and $n$ indicates the total number of days. The estimated numerical parameters are given in Table 3.

### 4.3 Impact of environment contamination

The government policies to sanitize publicly access places such as offices, academic institutions, shopping malls and other places touched often as the places may contaminated by the COVID-19 virus. The virus can remain viable for up to four hours on copper, up to 24 hours on cardboard, and up to 72 hours on stainless steel and plastic [3]. As the population density in
India is very high, which expose people to the contaminated environment, the situation required special attention in India. The mathematical model (8) is incorporated the role of COVID-19 virus on environmental contamination by the infected individuals in the class \(A\) and \(I\). Theoretically we have studied the effect of environment contamination in our proposed model by the infected population. The time series solutions shows in the Figs. 6, 7, 8 and 9 demonstrates the effect of the parameters related to environment contamination. If the rate of infection \(\beta_c\) due to the contaminated environment increases then the number of infected populations (classes \(A\) and \(I\)) also increases (see the Fig. 6) but the duration of the pandemic decreases, which is a good agreement with our study and the real scenarios. Again if the rate of environment contamination \((\tau_1\) and \(\tau_2\)) increases, then also the number of infected populations (classes \(A\) and \(I\)) also increases (see the time series Figs. 7 and 8). Also, in both the cases if \(\tau_1\) and \(\tau_2\) are increased, then the duration of the pandemic decreases.
Table 2 Table of biologically realistic parameters and their delineation for the COVID-19 model (8)

| Parameter | Description | Values (Unit) | Source |
|-----------|-------------|---------------|--------|
| $\Lambda_s$ | Source rate of uninfected individual | - | Estimated |
| $\beta_s$ | Infection transmissibility coefficient | $day^{-1}$ | Estimated |
| $\beta_c$ | Disease transmission coefficient due to environmental contamination | $(0, 10.5 \times 10^{-5})$ | Assumed |
| $\alpha_a$ | Modification term for asymptomatic classes | $day^{-1}$ | Estimated |
| $\alpha_i$ | Modification term for symptomatic classes | 0.45 $day^{-1}$ | [52] |
| $\alpha_h$ | Adjustment factor for hospitalized individuals | 0.35 $day^{-1}$ | [53] |
| $\delta_s$ | Natural death rate of susceptible classes | $0.1945 \times 10^{-4} day^{-1}$ | [52] |
| $\delta_e$ | Mortality rate of exposed individuals | $0.1945 \times 10^{-4} day^{-1}$ | [52] |
| $\delta_a$ | Mortality rate of asymptomatic individuals | $0.1945 \times 10^{-4} day^{-1}$ | [52] |
| $\delta_i$ | Mortality rate of symptomatic individuals | 0.03 $day^{-1}$ | [54] |
| $\delta_h$ | Mortality rate of hospitalized individuals | $0.1945 \times 10^{-4} day^{-1}$ | [52] |
| $\delta_r$ | Mortality rate of recovered individuals | $0.1945 \times 10^{-4} day^{-1}$ | [52] |
| $\gamma_e$ | Conversion rate from latent class to asymptomatic class | $day^{-1}$ | Estimated |
| $\gamma_i$ | Rate at which symptomatic individuals become hospitalized | $day^{-1}$ | Estimated |
| $q_a$ | Proportion of exposed individuals | - | Estimated |
| $\xi_a$ | Recovery rate from asymptomatic population | $day^{-1}$ | Estimated |
| $\xi_i$ | Rate of recovery from symptomatic individuals | $day^{-1}$ | Estimated |
| $\xi_h$ | Rate of recovery from hospitalized individuals | 0.2141 $day^{-1}$ | [53] |
| $\tau_1$ | Rate of environmental contamination by asymptomatic individuals | $(0, 2.1 \times 10^{-6}) day^{-1}$ | [8] |
| $\tau_2$ | Rate of environmental contamination by infected individuals | $(0, 1.3 \times 10^{-4}) day^{-1}$ | [8] |
| $\tau_c$ | Virus clearance rate from the environment | $(0, 0.1) day^{-1}$ | [8] |

decreases, which is also a good agreement with the proposed model outcome with the real situation. Now if the infected environment is disinfected due to the sanitization (that is, $\tau_c$ is increased) then the number of infected populations (classes $A$ and $I$) can not be drastically increases (see the time series Fig. 9). Thus, to clear/eliminate the novel coronavirus from the contaminated environment not only decrease the duration of the pandemic but also control the number of infected populations.

5 Conclusions

At the starting of March 2020, India has knocked due to the novel coronavirus epidemic in a more intricated way than the Republic of China, where the COVID-19 begun, as most of the cases were brought and culminated into demanding types of interaction of the diseases in super-spread events, hotspot transmission regions, wide contact regions, and so on. Due to absence of specific therapeutics, proper treatments, vaccination that could reduce or eliminate the COVID-19 epidemic, Indian Govt. responsibility to novel coronavirus is to curtail the graph of diseases early and to mitigate the number of viruses at the peak time, while enlarging our public health facility and better arranging our hospitals equipments including number of beds, sufficient

Table 3 Estimated parameters values and the assumed initial population size

| Parameters | First wave | Source |
|------------|------------|--------|
| $S(0)$ | 1380004385 | [55] |
| $E(0)$ | 150000 | Estimated |
| $A(0)$ | 1500 | Estimated |
| $I(0)$ | 5 | Data |
| $H(0)$ | 5 | Estimated |
| $R(0)$ | 0 | Data |
| $\Lambda_s$ | 35000 | Estimated |
| $\beta_s$ | 1.0019 | Estimated |
| $\alpha_a$ | 0.2001 | Estimated |
| $\xi_a$ | 0.1091 | Estimated |
| $\gamma_e$ | 0.1429 | Estimated |
| $q_a$ | 0.0038 | Estimated |
| $\tau_1$ | 0.2011 | Estimated |
| $\xi_i$ | 0.2100 | Estimated |
nurses, life-saving medicines, etc. for the worst situation to come.

In this article, we studied the transmission dynamics of novel coronavirus in India, which is an addition of classical SEIR model where the infected populations are latent, asymptomatic, clinically ill infections and hospitalized or isolated components. We have included a most important component, namely environmental contamination that is primarily happened due to seafood market where most of the people visiting market to purchase their necessary things. The mathematical model is being utilized to estimated the influence of different non-pharmaceutical intervention strategies to control or maintain the COVID-19 pandemic in India. Theoretical analysis has been performed including positivity of the
solutions and boundedness of the system. We calculate $R_0$ using next generation matrix. We compute the normalized forward sensitivity analysis with environmental contamination and without environmental contamination to recognize the most sensitive parameters with reference to the reproduction number $R_0$. Without environmental contamination the normalized forward sensitivity analysis indicates that infection transmission rate $\beta_s$, adjustment parameter for asymptomatic population $\alpha_a$ and the recovery rate from asymptomatic individuals $\xi_a$ are the most effective parameters with respect to $R_0$. But with environmental contamination it can be observed that the following parameters $\beta_s, \beta_c, \delta_s, \gamma_e, \tau_c, \alpha_a, \alpha_i, \xi_a, \tau_1$ and $\tau_2$ are most sensitive with reference to $R_0$. We obtained two biologically feasible equilibrium points, namely infection free equilibrium point $P_0$ and the interior steady state $P^*$. We investigate the local asymptotic stability of the biologically feasible equilibrium points. The disease free equilibrium point $P_0$ is globally asymptotically stable, which has been shown using Lyapunov functional. To investigate the long-run kinetics of our proposed coronavirus model, we performed uniform persistence of the system.

We calibrated our COVID-19 model based on the daily new observed SARS-CoV-2 cases for India. Our mathematical model have 21 system parameters among which we estimated 8 most significant parameters with reference to the clinically ill infected classes. We have identified 8 most important parameters based on the PRCC sensitivity analysis. Other 13 parameters are not too important with reference to the clinically ill infected cases. After recognized the sensitive parameters, we estimated the parameters based on the real data from India from March 02, 2020 to June 24, 2021.

Based on the estimated parameters for the first novel coronavirus wave we obtained $R_0 = 1.8174$ and for the second COVID-19 wave we have $R_0 = 2.7580$. In both the cases, the basic reproduction number is greater than unity which shows substantial outbreak of COVID-19.

Our model simulations mainly focused on the effect of environmental contamination. We included two important parameters, namely $\beta_c$ for environmental transmission & $\tau_c$ for the elimination rate of coronavirus in the environment. It can be noticed that for increasing the value of $\beta_c$ the susceptible population decreases whereas the environmental contamination become higher. It can be observed that for increasing the value of $\tau_c$ susceptible population decreases and the environmental population become higher.

In the future, our proposed model can be extended by considering fractional order differential equations with extensive numerical simulations about the transmission dynamics of novel coronavirus. Moreover, the role of saturated incidence rate can be taken into account to extend the proposed model. Few mathematical models has been developed on the influence of humidity or temperature. Thus, in future we can extend our work by considering the role of climate change and environmental heterogeneity. The recorded data for the novel coronavirus infected patients are altering with time. As a result, we obtain a poor quality of data for comparison purposes. Once we obtain the proper data about the coronavirus infected population, we can ameliorate our analytical result and the comparison become more methodical.
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Declarations

Conflict of interest The authors declare that they have no conflict of interest.

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