COVID-19 outbreak: a predictive mathematical study incorporating shedding effect

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Abstract
In this paper, a modified SEIR epidemic model incorporating shedding effect is proposed to analyze transmission dynamics of the COVID-19 virus among different individuals’ classes. The direct impact of pathogen concentration over susceptible populations through the shedding of COVID-19 virus into the environment is investigated. Moreover, the threshold value of shedding parameters is computed which gives information about their significance in decreasing the impact of the disease. The basic reproduction number ($R_0$) is calculated using the next-generation matrix method, taking shedding as a new infection. In the absence of disease, the condition for the equilibrium point to be locally and globally asymptotically stable with $R_0 < 1$ are established. It has been shown that the unique endemic equilibrium point is globally asymptotically stable under the condition $R_0 > 1$. Bifurcation theory and center manifold theorem imply that the system exhibit backward bifurcation at $R_0 = 1$. The sensitivity indices of $R_0$ are computed to investigate the robustness of model parameters. The numerical simulation is demonstrated to illustrate the results.

Keywords Epidemiological model · Shedding effect · Disease threshold · Backward bifurcation

1 Introduction
The mathematical models describing the dynamics of contagious diseases are now throughout. These models play a significant role in aid to realize techniques to control infectious diseases and alleviate their potential effects. Several research papers and
online resources are available in the frame of mathematical models discussing the dynamics of infectious disease spread in the population. Mathematical models played a crucial role and have a positive history of applications to help humans, such as in dengue [1, 2], malaria [3–5], tuberculosis [6, 7], and a lot more. Presently, many authors explored various research works related to COVID-19, discussing its transmission dynamics and other vital factors [8–10]. The Coronaviruses are a pervasive group of pathogens that contains microorganism that may emerge as a group of diseases in humans, resulting in hyposmia (decreased sense of smell)/anosmia (inability to smell anything), loss of the sense of taste, poor appetite and cold to the acute respiratory syndrome.

Coronaviruses are highly transmissible and are morbific viruses of the 21st century worldwide. Latterly, COVID-19 overwhelmed the whole world briefly and drastically took the face of a pandemic. According to the International Committee on Taxonomy of Viruses (ICTV) report, coronaviruses belong to the sub-family Coronavirinae, a member of the family Coronaviridae and the order Nidovirales. The sub-family Coronavirinae consists of four biological groups; $\alpha$, $\beta$, $\gamma$, and $\delta$-coronavirus [11, 12]. It has been found that all coronaviruses are animal originated [12, 13]. $\alpha$-coronaviruses involve HumanCoV-NL63 and HumanCoV-229E that show mild infections in humans. Further, SADS-CoV (Swine acute diarrhea syndrome coronavirus), having swine as an intermediate carrier, does not show infectious symptoms in humans. HCoV-OC43 and HCoV-HKU1 both are a type of $\beta$-coronaviruses and are also found harmless to the human body in most cases. The highly pathogenic coronaviruses have not taken seriously until the outrage of SARS-CoV (Severe acute respiratory syndrome coronavirus) in 2003 and MERS-CoV (Middle East respiratory syndrome coronavirus) in 2012.

The compartment models are critical tools to characterize the dynamics of an ongoing pandemic and include various challenges. Generally, the SEIR modeling approach is applied retrospectively when the epidemic has ended and most clinical and epidemiological information about the disease is revealed. It is difficult to characterize the beginning of the ongoing COVID-19 pandemic with the SEIR model, given that many aspects of the disease remain unclear. Therefore, the lack of a comprehensive understanding of the COVID-19 pandemic makes current modeling efforts inconsistent and sometimes confusing. Apart from this, SEIR-type mechanistic modeling also allows little flexibility for new evidence and insights without substantially changing the model structure compared to other alternative approaches. Nowadays, a lot of mathematical models are available investigating the transmission dynamics of COVID-19 disease. These mathematical models are based on a system of differential equations that provides complete dynamics of transmitting a disease [14]. Moreover, these models accommodate several factors in the spread of a disease, such as symptomatic and asymptomatic cases, presence of a disease vector, relapse, and reinfection. The whole population is divided into sub-populations such as susceptible, exposed, infected, and recovered compartments in the form of the SEIR model [15–18]. Liang [19] proposed a mathematical model analysing the infection kinetics of COVID-19. The author compared the spread of COVID-19 with that of SARS and MERS infectious disease. In his work, it is concluded that the infection rate of SARS-COV-2 is nearly double compared to that of SARS and MERS viruses. The dynamics of an SEIR epidemic model.
accounting infectivity in incubation period is discussed by Jiao et al. [20]. Further, Sardar et al. [21] investigated a modified SEIR model under the impact of lockdown in some states of India. According to their study, an effective and planned lockdown is necessary to eradicate COVID-19 disease in India. Also, a case study of SARS-COV-2 disease transmission in the USA is done by Badr et al. [22], incorporating mobility patterns.

The modeling of infectious diseases is not only limited to such compartment models; several other mathematical works are investigating the spread of the virus. In the past few decades, Markovian stochastic systems have played a vital role in the modeling of biological population models based on time-inhomogeneous birth-death processes; that is, all individuals at a given time have the same birth and death rates. The exact solution of such systems is likely to be solved using the Lie-theoretic approach [23]. Shang [24] derives an analytical solution for a viral model with time-dependent rates and time-inhomogeneous Markov chain formalism using Lie’s approach. It is found that this methodology may offer useful insights for other biological and ecological applications [25, 26] but in the current study Lie’s approach remains a challenge due to its time-inhomogeneity. Recently, a mathematical framework targeting the immunization of networks with limited knowledge and temporary immunity is also developed in [27] in the field of statistical physics. Further, Nguyen et al. [28] discussed the long-term dynamics of a stochastic SIRS model incorporating a general incidence rate. The results for the extinction and permanence of disease are established concerning the threshold parameter.

The COVID-19 outbreak is assumed to be the world’s most considerable discord in terms of each country’s economics and medical health system, which has been pushed into a crucial stage. Moreover, it caused millions of confirmed infections accompanied thousands of deaths worldwide. According to the Worldometer [29], the last statistics as of September 7, 2021, is more than two hundred twenty-two million, while the number of deaths has exceeded four million worldwide.

Like many respiratory viruses, the novel coronavirus SARS-CoV-2 can be shed in the form of tiny droplets released from an infected individual’s nose and mouth. The shedding of virus is the expulsion and release of virus offspring following successful reproduction during a host cell infection. The T-zones tissue areas of the face, including the eyes, nose, and mouth, are the main entry points of the virus into the human body. When the infected person sneezes and coughs, the virus is released from their mouth and reaches other people’s clothes and nearby surfaces. It can also be shed into the environment in the form of micro-particles in the air. The individual who coughs and sneezes without taking any safety measures can also touch the T-zone areas of their face and gets infected. Therefore, the role of viral shedding in transmitting the disease is a significant factor in the modeling of COVID-19 disease [30–33]. Doremalen et al. [34] investigated the stability of SARS-CoV-2 and SARS-CoV-1 in aerosols and on various surfaces (plastic, stainless steel, copper, and cardboard). Their experimental measurements show that SARS-CoV-2 was more substantial on stainless steel and plastic compared to cardboard and copper. Also, the microbe survives on the stainless steel and plastic surface up to 72h after application, whereas the virus titer was greatly reduced on plastic and after 48h on stainless steel. According to their study, the stability kinetics of SARS-CoV-1 were similar to SARS-CoV-2. The susceptible individuals

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can be infected through either direct contact with infected individuals and indirect exposure with the COVID-19 infected environment as it is discussed in Kassa et al. [35].

In this paper, a SEIHRP (Susceptible-Exposed-Infected-Hospitalized-Recovered-Pathogen) model incorporating the shedding effect is accounted to discuss the transmission dynamics of the COVID-19 pandemic. Viral shedding is an essential factor in the modeling of COVID-19 disease. Several researchers incorporate shedding parameters in their mathematical models to study the dynamics of SARS-CoV-2 [31, 32, 35]. Consideration of the shedding effect and its direct impact on the susceptible population are the main contribution of this work. This model has introduced a new parameter to measure the indirect transmission rate of the infectious disease over the susceptible one directly. Further, the biological significance of shedding parameters and their threshold values gives relevant information about their role in eradicating the disease. Lastly, this work includes theoretical analyses and numerical findings with some pre-assumed scenarios, along with concluding remarks of the study.

2 Motivation

The coronavirus SARS-COV-2 is a highly transmissible disease. It can spread via direct (through contaminated air by tiny droplets and airborne particles containing the virus) and indirect (through contaminated surfaces) transmission. The T-zones tissue areas of the face are the main entry points of the virus. When the infected person sneezes and coughs, the virus is released from their mouth, reaches other people’s clothes and nearby surfaces, and is shed into the environment in the form of micro-particles in the air. Therefore, the role of viral shedding in transmitting the disease is a significant factor in modeling COVID-19 disease. We know that currently, rapid diagnostic tests and effective vaccine treatments are available to control the disease impact, yet the importance of standard hygiene practices, face masks and chemical disinfectants in public spaces cannot be ignored. According to the infectious disease specialist Kristin Englund, MD, vaccine is just another protection layer against the COVID-19 virus until we achieve some level of herd immunity; that is, 50% to 80% of the population will need to be vaccinated. According to the Our World in Data report [36], as of April 24, 2022, only 61.6% population of India is fully vaccinated and 59.4% worldwide. Undoubtedly the vaccines are incredibly effective and provide 94−−95% protection from disease, but the remaining 5% will still be at risk of infection through shedding of virus. Therefore, it is essential to maintain a standard hygiene level by using hand sanitizers and chemical disinfectants in public spaces.

3 Contribution

Coronaviruses are a pervasive group of pathogens for which transmission through the contaminated surface is a concern. The transmission of the virus occurs primarily through person-person contact, with contaminated surfaces providing a secondary transmission route. Several researchers approach the shedding effect of the virus for
modeling COVID-19 disease transmission [31, 32]. This work is motivated by one of the works of Tien and Earn [31], in which they investigated multiple transmission pathways in a waterborne pathogen model. In their work, both person-person and person-water-person transmissions are allowed. Also, they consider that the susceptible individuals become infected either by contact with infected individuals or through contact with contaminated water. Our model uses this for the disease transmission modeling of COVID-19 in the susceptible population. The uninfected population can get infected by direct and indirect contact with the exposed or infected class is the novel assumption of this work. Infected and exposed individuals can contaminate the environment by shedding pathogens. The basic reproduction number ($R_0$) is computed for the SEIHRP model, and the impact of viral shedding over $R_0$ is examined. The bifurcation theory and central manifold theorem are employed to exhibit the conditions for backward bifurcation at $R_0 = 1$. It is observed that an increase in the transmission rate of virus from infected surfaces to uninfected individuals leads the system to an endemic state resulting in backward bifurcation. The numerical simulation shows that an increase in person-contaminated surface-person transmission parameter leads the system to endemicity. Therefore, healthy hygiene practices i.e., using alcohol-based hand sanitizers, face masks and effective chemical disinfectants for public areas, are necessary steps to break the chain of COVID-19.

### 4 Model formulation

The total human population $N(t)$, is divided into five mutually-exclusive compartments, say susceptible $S(t)$, exposed $E(t)$, infected $I(t)$, hospitalized $H(t)$ and recovered $R(t)$ at time $t$. Here, the standard SEIR-model is extended by the incorporation of $H(t)$ class. It follows that

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

To explore the shedding impact of the virus, a distinct compartment for contaminated surfaces, namely pathogen class $P(t)$, is further added. The description of variables is given in Table 1.

The mathematical model is considered under the following assumptions:
1. The transmission dynamics of the COVID-19 model is similar to the SEIR model.
2. The exposed (asymptomatic but infectious) and infected (symptomatic and infectious) individuals can transmit the virus particles into the environment during talking, sneezing, exhaling, etc. Therefore, their contribution to the transmission of the virus is considered in the form of shedding parameters.
3. The susceptible individual can get infected by contacting a pathogen-contaminated surface. So, the direct impact of pathogen concentration on the susceptible class due to the shedding of COVID-19 virus into the environment is accounted.
4. It is also assumed that asymptomatic and symptomatic individuals with strong immunity can recover naturally.
5. According to the WHO report [37], the mild or asymptomatic COVID cases can be recovered without requiring any special medical treatment even lack of awareness, less number of testing and limited resources can also be a reason. Therefore, with no loss of generality, it can be assumed that only symptomatic and COVID-19 confirmed cases are hospitalized.
6. A fraction of COVID-19 pathogen is assumed to be removed from the environment due to sanitization.

The total number of susceptible individuals \( \alpha \) decreases, followed by the infection of SARS-CoV-2, which can be acquired either by direct contact from person to person (at the rate \( \beta \)) or indirectly through contaminated surfaces (at the rate \( \beta_e \)). On the basis of above, the rate of change of susceptible population is given by

\[
\frac{dS}{dt} = \alpha - \beta SI - \beta_e SP - \mu S.
\]

Here, \( \mu \) represents the natural death rate of susceptible population.

The exposed compartment of the human population consists of those individuals who are asymptotically infectious. The individuals exposed to the COVID-19 virus increase the population of the exposed class at the rate \( \beta \) and \( \beta_e \). The exposed population falls off as the asymptomatic infectious person shows symptoms and moves to the symptomatic-infectious class at the rate \( \sigma \delta \). SARS-CoV-2 is a highly transmissible disease; followed by a WHO report [37], most people infected with this virus will show mild to moderate illness and can be recovered without requiring medical attention (however, some will become serious and require special medical treatment). Therefore, it can be considered that some of the exposed population can recover (at the rate \( \eta \)) without being hospitalized. It follows that

\[
\frac{dE}{dt} = \beta SI + \beta_e SP - (\mu + \eta + \sigma \delta) E.
\]

The population of symptomatic-infected individuals (with clinical symptoms of COVID-19) in compartment \( I \) increases as the individual in the exposed class develops clinical signs of infection at the rate \( \sigma \delta \). The infected individuals who show serious illness are transferred to hospitalized compartment \( H(t) \) for medical treatment at the rate \( \tau \). It is assumed that those with mild to moderate symptoms recovered (at the rate
\( \lambda \) without being hospitalized. This gives

\[
\frac{dI}{dt} = \sigma \delta E - (\tau + \lambda + \mu + \phi)I.
\]

Here, \( \phi \) denotes the death rate due to COVID-19.

Infected individuals with severe disease symptoms increase the population in the hospitalized compartment (at the rate \( \tau \)). It decreased as the hospitalized individual recovers at the rate \( \xi \). Thus,

\[
\frac{dH}{dt} = \tau I - (\xi + \mu + \phi)H.
\]

The recovered individuals from exposed \( E(t) \), infected \( I(t) \) and hospitalized \( H(t) \) compartments give rise to recovered class \( R(t) \) at the rates \( \eta, \lambda \) and \( \xi \), respectively. Hence,

\[
\frac{dR}{dt} = \eta E + \xi H + \lambda I - \mu R.
\]

Infectious individuals in exposed and infected classes contaminate the environment via viral shedding at the rates \( p_1 \) and \( p_2 \), respectively. The virus is removed from the contaminated surface at the rate \( d \). It follows that

\[
\frac{dP}{dt} = p_1 E + p_2 I - dP.
\]

In essence, the following set of differential equations represents the COVID-19 transmission model:

\[
\begin{align*}
\frac{dS}{dt} &= \alpha - \beta SI - \beta_e SP - \mu S \\
\frac{dE}{dt} &= \beta SI + \beta_e SP - (\mu + \eta + \sigma \delta)E \\
\frac{dI}{dt} &= \sigma \delta E - (\tau + \lambda + \mu + \phi)I \\
\frac{dH}{dt} &= \tau I - (\xi + \mu + \phi)H \\
\frac{dR}{dt} &= \eta E + \xi H + \lambda I - \mu R \\
\frac{dP}{dt} &= p_1 E + p_2 I - dP 
\end{align*}
\]

with the following non-negative initial conditions:

\[
S(0) = S_0, \quad E(0) = E_0, \quad I(0) = I_0, \quad H(0) = H_0, \quad R(0) = R_0, \quad P(0) = P_0.
\]

The flow diagram for the disease transmission dynamics of the COVID-19 model is depicted in Fig. 1. All model parameters are defined in Table 2 and are assumed to be non-negative.
Fig. 1 Flow diagram of the model 2

Table 2 Parameters of the model

| Parameters | Description of parameters |
|------------|----------------------------|
| $\alpha$   | Birth rate of the susceptible individuals |
| $\mu$      | Natural death rate |
| $\beta$    | Direct transmission rate of virus |
| $\beta_e$  | Indirect transmission rate of virus from the environment |
| $\delta$   | Rate of transfer of exposed individuals to the infected class |
| $\eta$     | Natural recover rate of exposed |
| $\lambda$  | Natural recover rate of infected |
| $\frac{1}{\sigma}$ | Incubation period |
| $\tau$     | Transfer rate of infected individual to the hospital |
| $\phi$     | Death rate due to COVID-19 |
| $\xi$      | Recovery rate of hospitalized individuals |
| $p_1$      | Shedding rate of virus from the exposed class to the environment |
| $p_2$      | Shedding rate of virus from the infected class to the environment |
| $d$        | Removal rate of the COVID-19 virus from the environment due to sanitization |

5 Preliminaries

In this section, the qualitative nature of the solutions of the system (2) is discussed.

Let $intR^6_+ = \{(S, E, I, H, R, P) \in R^6 : S > 0, E > 0, I > 0, H > 0, R > 0, P > 0\}$ be the positive cone.

5.1 Positivity of the solution

The following theorem implies that the solutions of the system (2) are positive with respect to non-negative initial conditions.

Theorem 1 The cone $intR^6_+$ of the solution is invariant.
Proof. Firstly, we show that the variables $S(t)$ and $E(t)$ of the system (2) are positive, for all $t \geq 0$. Let us consider a trajectory crosses one of the positive cone either at time $t_1$ or $t_2$ such that [35]:

- $t_1$: $S(t_1) = 0$, $\left(\frac{dS}{dt}\right)_{t_1} < 0$, $E(t) > 0$, $I(t) > 0$, $H(t) > 0$, $R(t) > 0$ and $P(t) > 0$ for $t \in (0, t_1)$ or
- $t_2$: $E(t_2) = 0$, $\left(\frac{dE}{dt}\right)_{t_2} < 0$, $E(t) > 0$, $I(t) > 0$, $H(t) > 0$, $R(t) > 0$ and $P(t) > 0$ for $t \in (0, t_2)$.

Using the first assumption, the first equation of (2) leads to

$$\left(\frac{dS}{dt}\right)_{t_1} = \alpha > 0,$$

which contradicts the assumption, $S'(t_1) < 0$. Therefore, $S(t) > 0$ for all $t \geq 0$.

Similarly, the second equation of (2) gives

$$\left(\frac{dE}{dt}\right)_{t_2} = \beta S(t)I(t) + \beta eS(t)P(t) > 0,$$

which also contradicts the assumption, $E'(t_2) < 0$.

Choosing $t_2$ such that the point lies on the positive axis of $E(t)$ so that $I(t_2)$ and $P(t_2)$ are positive. Thus, $E(t)$ is positive for all $t \geq 0$.

From the third equation of (2) it is obtained

$$I' = \sigma \delta E - (\tau + \lambda + \mu + \phi)I \geq -(\tau + \lambda + \mu + \phi)I, \quad (3)$$

since $E(t)$ is non-negative for all $t \geq 0$. Therefore, Eq. (3) yields

$$I(t) \geq I(0)e^{-(\tau + \lambda + \mu + \phi)t} \geq 0.$$

Likewise, from the fourth equation of (2)

$$H' = \tau I - (\xi + \mu + \phi)H \geq -(\xi + \mu + \phi)H. \quad (4)$$

On solving equation (4), we get

$$H(t) \geq H(0)e^{-(\xi + \mu + \phi)t} \geq 0.$$

Similarly, the last two equations of (2) leads to

$$R' = \eta E + \xi H + \lambda I - \mu R \geq -\mu R \quad (5)$$

and

$$P' = p_1 E + p_2 I - dP \geq -dP. \quad (6)$$
Above two equations give

\[ R(t) \geq R(0)e^{-\mu t} \geq 0 \]

and

\[ P(t) \geq P(0)e^{-dt} \geq 0. \]

Hence the cone of solution is invariant for the system (2). \( \square \)

### 5.2 Boundedness of the solution

In this subsection, a positively invariant feasible region is determined. The solutions of the system (2) are bounded in this region.

**Theorem 2** Solutions of the system (2) with specified initial conditions are bounded in the domain

\[ \mathcal{D} = \{ (S, E, I, H, R) \in \mathbb{R}^6_{\geq 0} : 0 \leq (S + E + I + H + R) \}
\]

\[ = N(t) \leq \frac{\alpha}{\mu}, 0 \leq P(t) \leq \frac{(p_1 + p_2)\alpha}{\mu d} \]

for all \( t \geq 0 \).

**Proof** The total number of the population \( N(t) \) at time \( t \) is

\[ N = S + E + I + H + R. \]

The rate of change of the total population is given by

\[ \frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dH}{dt} + \frac{dR}{dt} \]

\[ = \alpha - \mu N - \phi(I + H) \]

or

\[ \frac{dN}{dt} \leq \alpha - \mu N. \]

Using Gronwall inequality, it can be resulted that

\[ 0 \leq N(t) \leq \frac{\alpha}{\mu} \]

for the initial data \( N(0) \leq \frac{\alpha}{\mu} \).

Now, for the pathogen parameter \( P(t) \):

\[ \frac{dP}{dt} \leq (p_1 + p_2)\frac{\alpha}{\mu} - dP. \]  \hspace{1cm} (7)
Again we apply Gronwall inequality for the equation (7), it is obtained

$$0 \leq P(t) \leq \frac{(p_1 + p_2)\alpha}{\mu d}$$

with $P(0) \leq \frac{(p_1 + p_2)\alpha}{\mu d}$.

Hence, any solution of the system (2) is bounded in $\mathcal{D}$. $\square$

6 Model analysis

6.1 Trivial equilibria and basic reproduction number

The system (2) possess a unique disease-free equilibrium (DFE) point, $M_0 = \left( \frac{\alpha}{\mu}, 0, 0, 0, 0, 0 \right)$.

Basic reproduction number

It measures the average number of secondary cases caused by a primary infectious individual in a totally susceptible population.

Considering the following first three equations of system (2):

\[
\begin{align*}
\frac{dE}{dt} &= \beta SI + \beta_e SP - (\mu + \eta + \sigma\delta)E \\
\frac{dI}{dt} &= \sigma\delta E - (\tau + \lambda + \mu + \phi)I \\
\frac{dP}{dt} &= p_1 E + p_2 I - dP.
\end{align*}
\]

On writing system (8) in the matrix form, we get

\[
\dot{X} = \mathcal{F}_i(X) - \mathcal{V}_i(X) \quad i = 1, 2, 3
\]

such that

\[
\mathcal{F}_i(X) = \begin{pmatrix} \beta SI + \beta_e SP \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V}_i(X) = \begin{pmatrix} (\mu + \eta + \sigma\delta)E \\ -\sigma\delta E + (\tau + \lambda + \mu + \phi)I \\ -p_1 E - p_2 I + dP \end{pmatrix}
\]

Here $\mathcal{F}_i(X)$ represents the rate of appearance of new infection in $i^{th}$ compartment, $\mathcal{V}_i(X)$ denotes the rate of other transitions between compartment $i^{th}$ and other infected compartments and shedding is considered as a new infection.

Evaluate $\mathcal{F}$ and $\mathcal{V}$ at DFE($M_0$), it is obtained

\[
\mathcal{F} = \left( \frac{\partial \mathcal{F}_i}{\partial x_j} \right)_{M_0} = \begin{pmatrix} 0 & \alpha\beta & \beta_e\alpha \\ \mu & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}
\]

and
\[
V = \left(\frac{\partial V_i}{\partial x_j}\right)_{M_0} = \begin{pmatrix}
(\mu + \eta + \sigma \delta) & 0 & 0 \\
-\sigma \delta & (\tau + \lambda + \mu + \phi) & 0 \\
-p_1 & -p_2 & d
\end{pmatrix}.
\]

Now, the next generation matrix \(FV^{-1}\) is given by
\[
FV^{-1} = \begin{pmatrix}
\frac{\alpha \beta \sigma \delta d + \beta_e \alpha (\sigma \delta p_2 + p_1 (\tau + \lambda + \mu + \phi))}{\mu d (\tau + \lambda + \mu + \phi) (\mu + \eta + \sigma \delta)} & \frac{\alpha \beta d + \beta_e p_2 \alpha}{\mu (\tau + \lambda + \mu + \phi)} & \frac{\alpha \beta_e}{\mu d} \\
0 & 0 & 0
\end{pmatrix}
\]

Each \((i, j)\)th entry of \(FV^{-1}\) gives the expected number of secondary infections in \(i^{th}\) compartment produced by an infected introduced in \(j^{th}\) compartment. Therefore, the basic reproduction number is
\[
R_0 = \rho(FV^{-1}),
\]
where \(\rho\) is the spectral radius. \(R_1\) represents the endowment of direct human-to-human contact (susceptible-to-infected) routes, \(R_2\) and \(R_3\) measures the impact of the indirect transmission route of virus from contaminated surfaces to humans via viral shedding (exposed-to-environment and infected-to-environment, respectively).

### 6.2 Endemic equilibrium point

The endemic equilibrium point \(M^* = (S^*, E^*, I^*, H^*, R^*, P^*)\) exists where
\[
S^* = \frac{1}{\beta \sigma \delta d + \beta_e (p_1 (\tau + \lambda + \mu + \phi) + p_2 \sigma \delta)},
\]
\[
E^* = \frac{1}{(\mu + \eta + \sigma \delta)} \left[ \alpha - \frac{\mu d (\tau + \lambda + \mu + \phi) (\mu + \eta + \sigma \delta)}{\beta \sigma \delta d + \beta_e (p_1 (\tau + \lambda + \mu + \phi) + p_2 \sigma \delta)} \right],
\]
\[
I^* = \frac{1}{(\tau + \lambda + \mu + \phi)}, \quad H^* = \frac{\xi}{(\xi + \mu + \phi) (\tau + \lambda + \mu + \phi) + \lambda \sigma \delta} E^*,
\]
\[
R^* = \frac{1}{\mu} \left[ \frac{\eta + \xi (\eta + \phi) (\tau + \lambda + \mu + \phi) + \lambda \sigma \delta}{(\xi + \mu + \phi) (\tau + \lambda + \mu + \phi)} \right] E^*,
\]
\[
P^* = \frac{1}{\mu} \left[ \frac{p_2 \sigma \delta}{(\tau + \lambda + \mu + \phi)} \right] E^*
\]

provided that
\[
A = \alpha (\beta \sigma \delta d + \beta_e (p_1 (\tau + \lambda + \mu + \phi) + p_2 \sigma \delta)) - \mu d (\tau + \lambda + \mu + \phi) (\mu + \eta + \sigma \delta) > 0.
\]
The corresponding quadratic equation in terms of $I$ is given by

$$a_0 I^2 + a_1 I + a_2 = 0. \quad (10)$$

Where,

$$a_0 = \beta d (\mu + \eta + \sigma\delta)(\tau + \lambda + \mu + \phi)A,$$
$$a_1 = \beta_\epsilon \alpha (p_1 (\tau + \lambda + \mu + \phi) + p_2 \sigma\delta)(A - \mu d)(\tau + \lambda + \mu + \phi)(\mu + \eta + \sigma\delta) - \alpha \beta \sigma\delta A,$$
$$a_2 = -(R_0 - 1)\mu d\sigma\delta (A - \alpha \beta \sigma\delta d)),$$

Here, $a_0$ is always positive and $a_2$ is positive for $R_0 < 1$ and negative for $R_0 > 1$. Let $I_{\pm}$ be the root of quadratic Eq. (10),

$$I_{\pm} = \frac{-a_1 \pm \sqrt{\Delta}}{2a_0},$$

where the discriminant, $\Delta = a_1^2 - 4a_0a_2$.

Introducing a new variable $\tilde{R}$ corresponding to $\Delta = 0$, it is obtained

$$\tilde{R} = 1 - \frac{a_1^2}{4a_0\mu d\sigma\delta\alpha (A - \alpha \beta \sigma\delta d)}. \quad (11)$$

It can be easily seen that

| $\Delta$   | $R_0$       |
|-----------|-------------|
| $< 0$     | $< \tilde{R}$|
| $> 0$     | $> \tilde{R}$|
| $= 0$     | $= \tilde{R}$|

Now, depending on the sign of the coefficients of quadratic equation (10) and its discriminant $\Delta$, it can be summarized that the equation (10) has unique endemic, no endemic, and two endemic equilibria. The following remarks are noted for system (2):

**Remark 4.1** *No endemic state*, the system (2) possess no endemic equilibrium point for $a_1 > 0$ along with any one of the following condition:

1. $R_0 < \tilde{R}$
2. $R_0 = \tilde{R}$
3. $\tilde{R} < R_0 < 1$.

**Remark 4.2** *Unique endemic state*, the system (2) has a unique positive endemic equilibrium point if:

1. $\tilde{R} < R_0 = 1, a_1 < 0$
2. $R_0 > 1$.

**Remark 4.3** *Two positive endemic state*, for $a_1 < 0$, the two real endemic equilibria exists only if $\tilde{R} < R_0 < 1$. 
6.3 Biological significance

The shedding parameters, $p_1$, $p_2$ and their indirect impact over the susceptible population $\beta_e$ are the two main concerns of this work. In this subsection, the significance of these parameters and their role in eradicating the disease are discussed. Consequently, a threshold value of parameters $p_1$, $p_2$ and $\beta_e$ is calculated, which helps in reducing the size of the epidemic.

1. **Shedding parameters** : From the expression of $R_0$,

$$\frac{\partial R_0}{\partial p_1} = \frac{\alpha \beta_e}{\mu d (\mu + \eta + \sigma \delta)}$$

$$\frac{\partial R_0}{\partial p_2} = \frac{\alpha \beta_e \sigma \delta}{\mu d (\tau + \lambda + \mu + \phi)(\mu + \eta + \sigma \delta)}.$$ 

Therefore, $R_0$ is an increasing function of shedding parameters, $p_1$ and $p_2$. The critical value of $p_1$, $p_2$ at $R_0 = 1$ are given by

$$p_1^* = \frac{\mu d (\tau + \lambda + \mu + \phi)(\mu + \eta + \sigma \delta) - \alpha \sigma \delta (\beta d + \beta_e p_2)}{\alpha \beta_e (\tau +\lambda + \mu + \phi)}$$

$$p_2^* = \frac{\mu d (\tau + \lambda + \mu + \phi)(\mu + \eta + \sigma \delta) - \alpha \sigma \delta (\beta d + \beta_e p_1 (\tau + \lambda + \mu + \phi))}{\alpha \beta_e \sigma \delta}.$$ 

If $p_1 < p_1^*$, $p_2 < p_2^*$ then $R_0 < 1$ and $R_0 > 1$ for $p_1 > p_1^*$, $p_2 > p_2^*$. Thus, the burden of pandemic can be lowered if the value of shedding parameters is less than their critical values.

2. **Indirect transmission rate of pathogen from environment through shedding** : The basic reproduction number, $R_0$ increases with respect to parameter, $\beta_e$ as

$$\frac{\partial R_0}{\partial \beta_e} = \frac{\alpha (\sigma \delta p_2 + p_1 (\tau + \lambda + \mu + \phi))}{\mu d (\tau + \lambda + \mu + \phi)(\mu + \eta + \sigma \delta)}.$$ 

Further, the threshold value of $\beta_e$ is

$$\beta_e^* = \frac{\mu d (\tau + \lambda + \mu + \phi)(\mu + \eta + \sigma \delta) - \alpha \sigma \delta \beta d}{\alpha (\sigma \delta p_2 + p_1 (\tau + \lambda + \mu + \phi))}.$$ 

Therefore, in order to nether the value of $R_0$ to 1, $\beta_e$ must be less than $\beta_e^*$ otherwise the disease will persist.

6.4 Stability analysis

In this subsection, the stability properties of disease free state and endemic state of system (2) are discussed.
The variational matrix $J$ at $(S^*, E^*, I^*, H^*, R^*, P^*)$ is given by

$$
J = \begin{pmatrix}
-\beta I^*-\beta_e P^*-\mu & 0 & -\beta S^* & 0 & 0 & -\beta_e S^* \\
\beta I^*+\beta_e P^* & -(\mu+\eta+\sigma\delta) & -\beta S^* & 0 & 0 & \beta_e S^* \\
0 & \sigma\delta & -\tau+\lambda+\mu+\phi & 0 & 0 & 0 \\
0 & 0 & \tau & -(\xi+\mu+\phi) & 0 & 0 \\
0 & \eta & \lambda & \xi & -\mu & 0 \\
0 & p_1 & p_2 & 0 & 0 & -d \\
\end{pmatrix}.
$$

At DFE ($M_0$),

$$
J = \begin{pmatrix}
-\mu & 0 & -\beta\alpha & 0 & 0 & -\beta_e\alpha \\
0 & -(\mu+\eta+\sigma\delta) & -\beta\alpha & 0 & 0 & \beta_e\alpha \\
0 & \sigma\delta & -\tau+\lambda+\mu+\phi & 0 & 0 & 0 \\
0 & 0 & \tau & -(\xi+\mu+\phi) & 0 & 0 \\
0 & \eta & \lambda & \xi & -\mu & 0 \\
0 & p_1 & p_2 & 0 & 0 & -d \\
\end{pmatrix}.
$$

(12)

6.4.1 Local stability at disease free equilibria

**Theorem 3** The disease free state of system (2) is locally stable for $R_0 < 1$ and unstable for $R_0 > 1$ provided $(\tau+\lambda+\mu+\phi) > d$.

**Proof** The variational matrix at $M_0$ gives the following characteristic equation,

$$
(\lambda'+\mu)^2(\lambda'+(\xi+\mu+\phi)) \left( \lambda'^3 + (d+\tau+\lambda+2\mu+\phi+\eta+\sigma\delta)\lambda'^2 + \left(d(\tau +\lambda+2\mu+\phi+\eta+\sigma\delta)+(\mu+\eta+\sigma\delta)(\tau+\lambda+\mu+\phi) - \frac{\alpha\beta\sigma\delta}{\mu} - \frac{\alpha\beta_e p_1}{\mu} \right) \right)
+ \lambda' \left(d(\mu+\eta+\sigma\delta)(\tau+\lambda+\mu+\phi) - \frac{\alpha\beta_e(\sigma\delta p_2 + (\tau+\lambda+\mu+\phi)p_1)}{\mu} \right) = 0.
$$

(13)

Clearly, the linear factors of the Eq. (13) are negative and the other linear factors of the cubic equation

$$
\lambda'^3 + (d+\tau+\lambda+2\mu+\phi+\eta+\sigma\delta)\lambda'^2 + \left(d(\tau +\lambda+2\mu+\phi+\eta+\sigma\delta) \right) + (\mu+\eta+\sigma\delta)(\tau+\lambda+\mu+\phi) - \frac{\alpha\beta\sigma\delta}{\mu} - \frac{\alpha\beta_e p_1}{\mu} \right) \right)
+ \lambda' \left(d(\mu+\eta+\sigma\delta)(\tau+\lambda+\mu+\phi) - \frac{\alpha\beta_e(\sigma\delta p_2 + (\tau+\lambda+\mu+\phi)p_1)}{\mu} \right) = 0.
$$

(14)
are negative followed by Routh-Hurwitz criterion as follows:

The Eq. (14) can be rewritten as

\[
\lambda'^3 + A_1\lambda'^2 + A_2\lambda' + A_3 = 0
\]

where

\[
A_1 = d + \tau + \lambda + 2\mu + \phi + \eta + \sigma \delta,
\]

\[
A_2 = d(\tau + \lambda + 2\mu + \phi + \eta + \sigma \delta) + (\mu + \eta + \sigma \delta)(\tau + \lambda + \mu + \phi) - \frac{\alpha\beta\sigma\delta}{\mu}
\]

\[
= \frac{\alpha\beta\sigma\delta}{\mu},
\]

\[
A_3 = d(\mu + \eta + \sigma \delta)(\tau + \lambda + \mu + \phi) - \frac{\alpha\beta\sigma\delta p_2 + (\tau + \lambda + \mu + \phi)p_1}{\mu}
\]

\[
= \frac{\alpha\beta\sigma\delta d}{\mu},
\]

According to Routh–Hurwitz criterion, the system (2) is locally stable if and only if

\[A_1, A_2, A_3 > 0 \text{ and } A_1A_2 - A_3 > 0.\]

Now, for \( R_0 < 1 \) i.e.

\[
\alpha\beta\sigma\delta d + \beta_e\alpha(\sigma\delta p_2 + p_1(\tau + \lambda + \mu + \phi)) < \mu d (\tau + \lambda + \mu + \phi)(\mu + \eta + \sigma \delta)
\]

\[A_1 = (d + \tau + \lambda + 2\mu + \phi + \eta + \sigma \delta) > 0,
\]

\[
A_2 = d(\tau + \lambda + 2\mu + \phi + \eta + \sigma \delta) + (\mu + \eta + \sigma \delta)(\tau + \lambda + \mu + \phi) - \frac{\alpha\beta\sigma\delta}{\mu}
\]

\[
= \frac{\alpha\beta\sigma\delta}{\mu},
\]

\[
\geq d(\tau + \lambda + 2\mu + \phi + \eta + \sigma \delta) + (\mu + \eta + \sigma \delta)(\tau + \lambda + \mu + \phi) - \frac{\alpha\beta\sigma\delta}{\mu}
\]

\[
= \frac{\alpha\beta\sigma\delta}{\mu} (\tau + \lambda + \mu + \phi) - \frac{\beta_e\alpha\sigma\delta P_2}{\mu d} (1 - R_0)
\]

provided that \((\tau + \lambda + \mu + \phi) > d,\)

\[
A_2 = d(\tau + \lambda + 2\mu + \phi + \eta + \sigma \delta) + (\mu + \eta + \sigma \delta)(\tau + \lambda + \mu + \phi)(1 - R_0) > 0,\quad (16)
\]

and

\[
A_3 = d(\mu + \eta + \sigma \delta)(\tau + \lambda + \mu + \phi)(1 - R_0) > 0.\quad (17)
\]

Using expressions (15), (16) and (17), it follows that

\[
A_1A_2 - A_3 > (d + \tau + \lambda + 2\mu + \phi + \eta + \sigma \delta)(d(\tau + \lambda + 2\mu + \phi + \eta + \sigma \delta) + (\mu + \eta + \sigma \delta)(\tau + \lambda + \mu + \phi)(1 - R_0)) - d(\mu + \eta + \sigma \delta)(\tau + \lambda + \mu + \phi)(1 - R_0),
\]
\[ d^2 (\tau + \lambda + 2\mu + \phi + \eta + \sigma\delta) + d(\tau + \lambda + 2\mu + \phi + \eta + \sigma\delta)^2 + \]
\[ (\tau + \lambda + 2\mu + \phi + \eta + \sigma\delta)(\mu + \eta + \sigma\delta)(\tau + \lambda + \mu + \phi)(1 - R_0), \]
\[ > 0 \]

for \( R_0 < 1 \).

Therefore, the disease free state \((M_0)\) of system (2.1) is locally stable.

### 6.4.2 Global stability

**Theorem 4** The equilibrium point \( M_0 \) is globally stable in \( \Omega \) iff \( R_0 < 1 \).

**Proof** Consider a real valued Lyapunov function defined as,

\[ V = \int_{S(t)}^{S_0} \left( 1 - \frac{\alpha}{\mu S} \right) dz + E + \frac{(\mu + \eta + \sigma\delta)I}{\sigma\delta}. \]  

(18)

Differentiating equation (18) for all \( t \geq 0 \), it is obtained

\[ V' = - \frac{(\alpha - \mu S)^2}{\mu S} + \frac{\alpha(\beta I + \beta e P)}{\mu} - \frac{(\mu + \eta + \sigma\delta)(\tau + \lambda + \mu + \phi)I}{\sigma\delta} \]
\[ - \frac{(\beta I + \beta e P)}{(\tau + \lambda + \mu + \phi)I} \left( 1 - \frac{\sigma\delta\alpha}{\mu(\mu + \eta + \sigma\delta)} \right) \]
\[ = - \frac{(\alpha - \mu S)^2}{\mu S} - \frac{(\mu + \eta + \sigma\delta)(\tau + \lambda + \mu + \phi)I}{\sigma\delta} \left( 1 - R_0 \right). \]

(19)

Since all the parameters and variables of the system (2) are positive (see Theorem 1), \( V' \leq 0 \) for \( R_0 < 1 \). Equality holds if and only if \( S(t) = S_0, I(t) = 0 \) and \( E(t) = 0 \). Thus, \( V \) is a Lyapunov function on \( \Omega \) and the largest invariant subset in the region \( \{(S(t), E(t), I(t)) \in R_3^+ : V' = 0\} \) is \( \left\{ \left( \frac{\alpha}{\mu}, 0, 0 \right) \right\} \). Therefore, by using LaSalle’s invariance principle [38], it follows that

\[ (S(t), E(t), I(t)) \to \left( \frac{\alpha}{\mu}, 0, 0 \right) \text{ as } t \to \infty. \]

(20)

From equation (20), \( \lim_{t \to \infty} I = 0 \) then for arbitrarily small \( \delta > 0 \) there exist a constant \( q > 0 \) such that \( \lim \sup_{t \to \infty} I \leq \delta \) for all \( t > q \). Hence, from the fourth equation of system (2) it follows that

\[ H' \leq \tau \delta - (\xi + \mu + \phi)H. \]

(21)
Now, by comparison theorem \[39\]

\[
H^\infty = \limsup_{t \to \infty} H \leq \frac{\tau \vartheta}{(\xi + \mu + \phi)}
\]  

(22)

and for \(\vartheta \to 0\),

\[
H^\infty = \limsup_{t \to \infty} H \leq 0.
\]  

(23)

Similarly, for \(\liminf_{t \to \infty} I = 0\), it is obtained

\[
H^\infty = \liminf_{t \to \infty} H \geq 0.
\]  

(24)

From equation (23) and (24), it can be concluded that

\[
H^\infty \geq 0 \geq H^\infty.
\]

Therefore,

\[
\lim_{t \to \infty} H = 0.
\]  

(25)

Similarly, it can be shown that

\[
\lim_{t \to \infty} R = 0,
\]  

(26)

and

\[
\lim_{t \to \infty} P = 0.
\]  

(27)

Thus, from Eqs. (20, 25, 26 and 27), it can be concluded that all the solutions of the system (2) converges to \(M_0\) as \(t \to \infty\) in \(\mathcal{D}\). Therefore, \(M_0\) is globally stable.  

\[\square\]

**Theorem 5** The unique endemic equilibria \((S^*, E^*, I^*, H^*, R^*, P^*)\) of the system (2) is globally stable if and only if \(R_0 > 1\).

**Proof** Define a positive definite function as follows

\[
\mathcal{V}_1 = S - S^* - S^* \ln \frac{S}{S^*} + E - E^* - E^* \ln \frac{E}{E^*} + \frac{(\mu + \eta + \sigma \delta)}{\sigma \delta} \left( I - I^* - I^* \ln \frac{I}{I^*} \right).
\]

On differentiating, it is obtained

\[
\mathcal{V}'_1 = \left(1 - \frac{S}{S^*}\right) \frac{dS}{dt} + \left(1 - \frac{E}{E^*}\right) \frac{dE}{dt} + \frac{(\mu + \eta + \sigma \delta)}{\sigma \delta} \left(1 - \frac{I}{I^*}\right) \frac{dI}{dt}
\]

\[
= \left(1 - \frac{S}{S^*}\right) (\alpha - \beta SI - \beta_e SP - \mu S) + \left(1 - \frac{E}{E^*}\right) (\beta SI + \beta_e SP - (\mu + \eta)
\]

\[
+ \sigma \delta) E) + \left(1 - \frac{I}{I^*}\right) (\sigma \delta E - (\tau + \lambda + \mu + \phi) I)
\]

\[
= -\frac{\mu(S - S^*)^2}{S} + \left(1 - \frac{S}{S^*}\right) (\mu + \eta + \sigma \delta) E^* \left(1 - \frac{S}{S^*} (\beta I + \beta_e P)\right)
\]

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\[
+ (\mu + \eta + \sigma \delta) E^* \left( \frac{S}{S^*} (\beta I + \beta e P) \right) + 2 - \frac{S}{E} \left( \frac{\beta I + \beta e P}{\mu + \eta + \sigma \delta} - \frac{I}{I^*} - \frac{I^* E}{I E^*} \right)
\]
\[
= - \frac{\mu (S - S^*)^2}{S} + (\mu + \eta + \sigma \delta) E^* \left[ 3 - \frac{S^*}{S} - \frac{E I^*}{E^* I} + \left( - \frac{S I E^*}{S^* I E^*} \right) \right]
\]
\[
+ \left( \frac{\beta I + \beta e P}{\beta I^* + \beta e P^*} \right) \frac{I}{I^*} \left( 1 - \frac{S^* E}{S E^*} \right) \right]
\]

Here, it is noted that at \((S^*, E^*, I^*, H^*, R^*, P^*)\)
\[
\frac{(\beta I + \beta e P)}{I} = \frac{(\beta I^* + \beta e P^*)}{I^*}.
\]
\[
\mathcal{V}'_1 = - \frac{\mu (S - S^*)^2}{S} + (\mu + \eta + \sigma \delta) E^* \left( 3 - \frac{S^*}{S} - \frac{E I^*}{E^* I} - \frac{S I E^*}{S^* I E^*} \right).
\]

Since the arithmetic mean exceeds geometric mean,
\[
\frac{S^*}{S} + \frac{E I^*}{E^* I} + \frac{S I E^*}{S^* I E^*} \geq 3.
\]

Therefore,
\[
\mathcal{V}'_1 \leq 0,
\]
\[
d\mathcal{V}_1 \leq 0 \text{ if and only if } S(t) = S^*, E(t) = E^* \text{ and } I(t) = I^*. \text{ Hence, } \mathcal{V}_1 \text{ is a Lyapunov function on } \mathcal{D} \text{ and } (S^*, E^*, I^*) \text{ is the largest invariant set for } \mathcal{V}_1 = 0. \text{ Applying LaSalle’s theorem [38], it is obtained}
\]
\[
(S(t), E(t), I(t)) \to (S^*, E^*, I^*) \text{ as } t \to \infty. \quad (28)
\]

Now, since \(\lim \sup_{t \to \infty} I = I^*\) thus for arbitrarily small \(\theta > 0\) there exist a constant \(r > 0\) such that \(\lim \sup_{t \to \infty} I \leq I^* + \theta\) for all \(t > r\). Then, the fourth equation of system (2) gives
\[
H' \leq \tau(I^* + \theta) - (\xi + \mu + \phi)H. \quad (29)
\]

Using comparison theorem [39]
\[
H^\infty = \lim \sup_{t \to \infty} H \leq \frac{\tau(I^* + \theta)}{\xi + \mu + \phi}. \quad (30)
\]

Letting \(\theta \to 0\),
\[
H^\infty = \lim \sup_{t \to \infty} H \leq \frac{\tau I^*}{\xi + \mu + \phi}. \quad (31)
\]

Similarly, for \(\lim \inf_{t \to \infty} I = I^*\), it yields
\[
H^\infty = \lim \inf_{t \to \infty} H \geq \frac{\tau I^*}{\xi + \mu + \phi}. \quad (32)
\]
From equation (31) and (32), it can be concluded that

\[ H_{\infty} \geq \frac{\tau I^*}{(\xi + \mu + \phi)} \geq H^\infty. \]

Therefore,

\[ \lim_{t \to \infty} H = \frac{\tau I^*}{(\xi + \mu + \phi)} = H^*. \] (33)

Similarly, it can be shown that

\[ \lim_{t \to \infty} R = R^*, \] (34)

and

\[ \lim_{t \to \infty} P = P^*. \] (35)

Thus, from Eqs. (28, 33, 34 and 35), it can be concluded that every solution of the system (2) approaches \( M^* \) as \( t \to \infty \) in \( \mathcal{D} \). Therefore, \( M^* \) is globally stable. \( \square \)

## 7 Backward bifurcation

Backward bifurcation is a bifurcation at which a stable epidemic equilibria and a stable endemic equilibria co-exists with \( R_0 < 1 \). Basic reproduction number, \( R_0 \) is a threshold parameter that helps us to determine the condition under which the disease can be managed. Usually, \( R_0 < 1 \) guarantees that the infection is under control. However, a mathematical model that shows backward bifurcation, the condition \( R_0 < 1 \) does not guarantee the elimination of the infection.

This section gives result for the existence of backward bifurcation using center manifold theorem [38, 40]. The following proof is a direct application of Castillo-Chavez and Song theorem mentioned in [40].

**Theorem 6** The system (2) undergoes backward bifurcation at \( R_0 = 1 \) if

\[ p_2 < \min \left\{ \left( \tau + \lambda + \mu + \phi \right) - \frac{(p_1 + \sigma \delta)\alpha \beta}{(\mu + \eta + \sigma \delta)\mu}, d - \frac{p_1(\tau + \lambda + \mu + \phi)}{\sigma \delta} \right\}. \]

**Proof** Let us assume that

\[ S = z_1, \ E = z_2, \ I = z_3, \ H = z_4, \ R = z_5, \ P = z_6. \]

Rewriting the system in the form \( \dot{Z}(t) = (h_1, h_2, h_3, h_4, h_5, h_6) \), it is obtained

\[
\begin{align*}
\frac{dz_1}{dt} &= h_1 = \alpha - \beta z_1 z_3 - \beta_c z_1 z_6 - \mu z_1 \\
\frac{dz_2}{dt} &= h_2 = \beta z_1 z_3 + \beta_c z_1 z_6 - (\mu + \eta + \sigma \delta) z_2 \\
\frac{dz_3}{dt} &= h_3 = \sigma \delta z_2 - (\tau + \lambda + \mu + \phi) z_3
\end{align*}
\]
\[
\begin{align*}
\frac{dz_4}{dt} &= h_4 = \tau z_3 - (\xi + \mu + \phi)z_4 \\
\frac{dz_5}{dt} &= h_5 = \eta z_2 + \xi z_4 + \lambda z_3 - \mu z_5 \\
\frac{dz_6}{dt} &= h_6 = p_1 z_2 + p_2 z_3 - d z_6.
\end{align*}
\] (36)

Taking \(\beta_e\) as a bifurcation parameter and solving \(R_0 = 1\) with respect to \(\beta_e\) yields

\[
\beta_e = \beta_e^* = \frac{\mu d(\tau + \lambda + \mu + \phi)(\mu + \eta + \sigma \delta) - \alpha \sigma \delta \beta d}{\alpha(\sigma \delta p_2 + p_1(\tau + \lambda + \mu + \phi))}.
\] (37)

Substituting bifurcation parameter \(\beta = \beta_e^*\) in the Jacobian matrix (12) obtained at the disease free equilibria,

\[
J_B = \begin{pmatrix}
-\mu & 0 & -\beta_e^* \alpha & 0 & 0 & -\beta_e^* \alpha \\
0 & -(\mu + \eta + \sigma \delta) & -\beta_e^* \alpha & \mu & 0 & -\beta_e^* \alpha \\
0 & \sigma \delta & -\tau - (\xi + \mu + \phi) & 0 & 0 & \mu \\
0 & 0 & \tau & -\xi & 0 & 0 \\
0 & \eta & \lambda & \xi & -\mu & 0 \\
0 & p_1 & p_2 & 0 & 0 & -d
\end{pmatrix}.
\] (38)

The transformed system Eq. (36), with \(\beta_e = \beta_e^*\), has a non-hyperbolic equilibrium point. Hence, the centre manifold theory [40] can be used to analyse the dynamics of the model equation (36) near \(\beta_e = \beta_e^*\).

Let \(X = (x_1, x_2, x_3, x_4, x_5, x_6)\) be the right-eigenvector of \(J_B\) corresponding to the zero eigenvalue such that

\[J_B X = 0,
\]

where

\[
\begin{align*}
x_1 &= \frac{\alpha(\mu + \eta + \sigma \delta)(\beta d + \beta_e(p_2 - (\tau + \lambda + \mu + \phi)))}{\mu}, \\
x_2 &= -\frac{d \alpha \beta}{\mu} - \frac{\beta_e \alpha(p_2 - (\tau + \lambda + \mu + \phi))}{\mu}, \\
x_3 &= (\sigma \delta + p_1)\beta_e \alpha - (\mu + \eta + \sigma \delta)d, \\
x_4 &= \frac{\tau}{(\xi + \mu + \phi)} x_3, \\
x_5 &= \frac{1}{\mu} \left( \eta \left( -\frac{d \alpha \beta}{\mu} - \frac{\beta_e \alpha}{\mu}(p_2 - (\tau + \lambda + \mu + \phi)) \right) + \lambda x_3 + \frac{\xi \tau}{(\xi + \mu + \phi)} x_3 \right), \\
x_6 &= -(\mu + \eta + \sigma \delta)(p_2 - (\tau + \lambda + \mu + \phi)) - \frac{\beta_e \alpha(\sigma \delta + p_1)}{\mu}.
\end{align*}
\]

Similarly, the left-eigenvector \(Y = (y_1, y_2, y_3, y_4, y_5, y_6)\) associated with the zero eigenvalue of \(J_B\) such that

\[Y J_B = 0
\]

is given by

\[
y_1 = 0, \quad y_2 = (\sigma \delta(p_2 - d) + (\tau + \lambda + \mu + \phi)p_1),
\]
\[ y_3 = (\mu + \eta + \sigma \delta)(p_2 - d) + \frac{p_1 \alpha (\beta_e + \beta)}{\mu}, \quad y_4 = y_5 = 0, \]

\[ y_6 = (\mu + \eta + \sigma \delta)(\tau + \lambda + \mu + \phi) - \frac{\sigma \delta \alpha (\beta_e + \beta)}{\mu}. \]

It is to be noted that the right and left eigenvector need to satisfy \( Y \cdot X = 1. \)

Therefore, the bifurcation coefficients \( K \) and \( Q \) at the DFE \((M_0)\) are given by

\[
K = \sum_{k,i,j=1}^{6} y_k x_i x_j \frac{\partial^2 h_k}{\partial z_i \partial z_j} (M_0, \beta_e^*)
\]

\[
= \sum_{i,j=1}^{6} \left( y_2 x_i x_j \frac{\partial^2 h_2}{\partial z_i \partial z_j} + y_3 x_i x_j \frac{\partial^2 h_3}{\partial z_i \partial z_j} + y_6 x_i x_j \frac{\partial^2 h_6}{\partial z_i \partial z_j} \right)
\]

\[
= \sum_{i=1}^{6} \left( y_2 x_i x_1 \frac{\partial^2 h_2}{\partial z_i \partial z_1} + y_2 x_i x_3 \frac{\partial^2 h_2}{\partial z_i \partial z_3} + y_2 x_i x_6 \frac{\partial^2 h_2}{\partial z_i \partial z_6} \right)
\]

\[
= 2y_2 x_1 x_3 \beta + 2y_2 x_1 x_6 \beta_e^*
\]

\[
= \frac{2\alpha (\mu + \eta + \sigma \delta)^2}{\mu^2} (\beta d + \beta_e(p_2 - (\tau + \lambda + \mu + \phi)))^2 (\sigma \delta (d - p_2)
\]

\[
+ p_1 (\tau + \lambda + \mu + \phi))
\]

\[
> 0.
\]

And

\[
Q = \sum_{k,j=1}^{6} y_k x_j \frac{\partial^2 h_k}{\partial z_j \partial \beta} (M_0, \beta_e^*)
\]

\[
= \sum_{j=1}^{6} \left( y_2 x_j \frac{\partial^2 h_2}{\partial z_j \partial \beta} (M_0, \beta_e^*) + y_3 x_j \frac{\partial^2 h_3}{\partial z_j \partial \beta} (M_0, \beta_e^*) + y_6 x_j \frac{\partial^2 h_6}{\partial z_j \partial \beta} (M_0, \beta_e^*) \right)
\]

\[
= y_2 x_6 \frac{\partial^2 h_2}{\partial z_6 \partial \beta} (M_0, \beta_e^*)
\]

\[
= \frac{\alpha}{\mu} (\sigma \delta (d - p_2) - p_1 (\tau + \lambda + \mu + \phi)) ((\mu + \eta + \sigma \delta)((\tau + \lambda + \mu + \phi) - p_2)
\]

\[
- (\sigma \delta + p_1) \frac{\beta \alpha}{\mu})
\]

\[
> 0.
\]

Since \( K > 0 \) and \( Q > 0 \), the system \((2)\) undergoes backward bifurcation at \( R_0 = 1. \) \(\square\)

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8 Numerical simulation

In this section, numerical analysis is performed to substantiate the analytical findings for the system (2) with the help of MATLAB software.

The information regarding the infection dynamics of coronaviruses is still evolving, and the biological studies of parameter values representing the infection dynamics are still in progress.

In COVID-19, after exposure to an infected individual, the onset of symptoms is observed to range between 2 – 14 days [41] and 2 – 24 days [37] and we take the incubation period of 14 days.

According to our model assumptions, only COVID-19 confirmed individuals are hospitalized and the hospitalization rate of symptomatic infection is estimated to be \((0 - 1) \text{ day}^{-1}\), ranges between \((0.002 - 0.009)\) [21].

The recovery time of COVID-19 disease highly depends on the severity of the infection. The infected individuals with mild/critical illness were observed to be recovered in an average period of 5 – 42 days, subject to the disease’s seriousness. For our simulation, we consider a nominal value of recovery rate \(\lambda = 0.098\) (approximately 1/11 per day) [9].

The members of SARS-COV-2 can survive on various surfaces in the environment for up to 3 days [30, 31, 34]. For simulation purpose, the values of virus removal rate \((d)\) and shedding parameters \((p_1, p_2)\) have been taken from the literature [9].

Furthermore, the simulation is performed for the different sets of parametric values [9, 21] (some parameters are mentioned above), supporting the analytical results for the system (2), as:

\[
\begin{align*}
\alpha &= 130, \quad \beta = 0.011, \quad \beta_e = 0.025, \quad \mu = 0.0395, \quad \eta = 15.7, \quad \sigma = 0.07, \quad \delta = 0.98, \\
\tau &= 0.009, \quad \lambda = 0.098, \quad \phi = 0.015, \quad p_1 = 0.001, \quad p_2 = 0.000398, \quad d = 0.016.
\end{align*}
\]  

(39)

For this set of parameters, the condition for the global stability of endemic equilibrium point \(M^*\) is satisfied, and the basic reproduction number \(R_0 = 1.35307 > 1\). Specifically, it is obtained that

\[
R_1 = 0.972772, \quad R_2 = 0.325302, \quad R_3 = 0.0549948
\]

which shows the risk of infection through multiple transmission pathways in a pathogen model. Among these three values of \(R_0\), the largest component \((R_1)\) represents the person-to-person transmission risk. Further, \(R_2\) shows a significant contribution of the exposed class in increasing the overall infection risk via infected surfaces to susceptible populations. The lowest one \((R_3)\) measures the infection risk from infected to susceptible individuals through the contaminated environment. It can be seen that \(R_2 > R_3\) since the asymptomatic infectious person can easily spread the disease as they are unaware of having the disease.

The time-series plot is drawn for the parameters \(S, E, I\) and \(P\) in Fig. 2 which shows that the system (2) converges to endemic state. Next, for \(\beta_e = 0.00001231\), the value of \(R_0\) turns to less than 1 i.e. \(R_0 = 0.97279\). Thus, the condition for the
existence of global stability at disease free equilibrium is satisfied, depicted in Fig. 3. The time-series plotted in Fig. 3 described the situation for $M_0$ converges to 0 at $R_0 < 1$.

Moreover, the time-series plotted in Fig. 2 (at $\beta_e = 0.025$) and Fig. 3 (at $\beta_e = 0.00001231$) confirms that for higher value of indirect disease transmission parameter $\beta_e$ generated through shedding, the infection level persists in the system.

At $R_0 = 1$, disease-free state becomes non-hyperbolic. Applying the centre manifold theory, taking $\beta_e$ as a bifurcation parameter leads to backward bifurcation under certain conditions. The bifurcation diagram corresponding to $\alpha = 80$, $\beta =
Fig. 4 Backward bifurcation in $R_0$-I plane for parameter values $\alpha = 80$, $\beta = 0.002$, $\eta = 14.7$, $p_1 = 0.105$, $d = 21$.

0.002, $\eta = 14.7$, $p_1 = 0.105$, $d = 21$ (without changing other parameters) is drawn in Fig. 4. Thus, a stable endemic equilibrium co-exists with a stable disease-free equilibrium which leads to the existence of multiple endemic equilibria.

In Figs. 5 and 6, time-series plots have been drawn for different classes and pathogen concentration by varying the shedding parameter $p_1$ and $p_2$, respectively. Subfigure 5(a) shows an increase in the shedding rate of virus from exposed class to the environment ($p_1$) leads to falling off susceptible population. Moreover, the subfigures 5(b, c, d) exhibits that enlarging $p_1$ magnifies the exposed and infected population and also pathogen concentration. Similar explanations can be given for the subfigures 6(a, b, c, d) concerning the shedding parameter $p_2$. These figures show the significant role of the shedding parameters in spreading the disease.

Meanwhile, Figs. 5 and 6 also help in identifying the waves of COVID-19. A slight increase in $p_1$ and $p_2$ raises the population of infected individuals along with a noticeable variation in the pathogen population, showing the sensitivity of the system (2) variables over these parameters. The shackles of SARS-COV-2 can only be smashed if we adopt standard hygiene practices in our daily life.

9 Sensitivity analysis

Sensitivity analysis is used to investigate the robustness of predictions to model parameters. To reduce the disease spreading and human mortality rate due to COVID-19, we must determine the significance of some epidemiologically essential parameters of our system(2).
In this section, the sensitivity indices of the basic reproduction number $R_0$ to variation in parameters are calculated using the normalized forward sensitivity index scheme [42]. The normalized forward sensitivity index of a variable $x$ is the ratio of the relative change in the variable to the relative change in the parameter $q$, defined as

$$\gamma^x_q = \frac{\partial x}{\partial q} \times \frac{q}{x}.$$ 

The sensitivity analysis of $R_0$ at the parameter values (39) are depicted in table (3).

For all the parameters (described in Table (3)), the sign of the sensitivity indices of $R_0$ supports the intuitive expectations of the system (2).

The basic reproductive number increases substantially as the disease transmission parameter $\beta$ from infected to susceptible via direct contact increases. Further, since $\gamma^R_\beta = +0.281062$, increasing (or decreasing) $\beta_e$ by 10% increases (or decreases) $R_0$ by 2.8%. From table (3), it is to be noted that

$$\gamma^{R_0}_\beta = \gamma^{R_0}_{p_1} + \gamma^{R_0}_{p_2}.$$
COVID-19 outbreak: a predictive mathematical study...

\[ \text{Fig. 6} \quad \text{Effect of shedding parameter } p_2 \text{ on different individual classes and pathogen concentration} \]

\[ \text{Table 3} \quad \text{Sensitivity indices of } R_0 \text{ to parameters for the COVID-19 model, evaluated at the parameters mentioned in (39)} \]

| Parameter | Sensitivity index |
|-----------|------------------|
| $\beta$   | +0.718938        |
| $\beta e$ | +0.281062        |
| $p_1$     | +0.240418        |
| $p_2$     | +0.0406445       |
| $\tau$    | -0.0423296       |
| $\lambda$ | -0.460923        |
| $\eta$    | -0.993162        |
| $\delta$  | -0.755242        |
| $\phi$    | -0.0705494       |
| $d$       | -0.281062        |

which clearly indicates that indirect disease transmission is directly proportional to the viral shedding. Also, the sensitivity table of $R_0$ shows the positive impact of $p_1$, $p_2$ over $R_0$.

Further, the $R_0$ decreases substantially as the asymptomatic-infectious person shows symptoms and moves to infectious class since when the person is diagnosed...
with COVID-19, their probability of spreading infection decreases (due to isolation and medical treatments). Similar explanations can be given for other parameters.

10 Conclusion

In this work, the transmission dynamics of COVID-19 disease are represented by a modified SEIR model incorporating the shedding effect. The symptomatic and asymptomatic individuals can relieve the virus particles into the environment while talking, sneezing, exhaling, etc. Consequently, the indirect transmission rate of the pathogen from the atmosphere through shedding is an essential factor to be elucidated. The total human population is divided into five categories: Susceptible, Exposed, Infected, Hospitalized, and Recovered, along with Pathogen compartment. The primary characteristics of the model including positivity and boundedness, are investigated. To observe the impact of disease, the threshold value of shedding and indirect disease transmission parameters are also computed. The infection-free equilibrium point is locally and globally stable if the basic reproduction number $R_0$, is less than one, otherwise unstable. It has been shown the system analytically achieves endemic equilibrium whenever $R_0 > 1$. The bifurcation theory and central manifold theorems are applied to explore the bifurcation. The system undergoes backward bifurcation at $R_0 = 1$. It means that $R_0 < 1$ alone is not sufficient to control the disease, but $R_0 < 1$ along with lower viral shedding value may reduce the size of the pandemic. The aforementioned numerical simulation gives evidence for our analytical results. The numerical exercise shows that an increase in indirect disease transmission coefficient through shedding leads to an endemic state. The sensitivity indices of $R_0$ are computed to determine the significance of epidemiologically essential parameters of our system. Therefore, to break the chain of COVID-19, it is necessary to decrease the possibilities of disease transmission via hand sanitizers, masks, etc., whereas chemical disinfectants for public spaces.

Every community has a limited capacity for the treatment of a disease. Our model does not include the condition of fixed treatment assets such as effective medicines, vaccination and hospital facilities. Therefore, a future model incorporating these factors as mitigation strategies can be investigated to control the spread of disease. In COVID-19, it is observed that the disease affects different age groups differently. Thus, it is also plausible to explore an age-structured model to better describe disease dynamics in the respective age groups.

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Declarations

Conflict of interest There are no conflicts of interest to this work.
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