APPLICATIONES MATHEMATICAE
Online First version

Youcef Belgaid

GLOBAL STABILITY OF THE DYNAMICS OF A NEW MODEL OF CORONAVIRUS TRANSMISSION WITH VIRUSES IN THE ENVIRONMENT

Abstract. In the present study, we develop a novel mathematical model for COVID-19 transmission by explicitly incorporating the influence of viruses in the environment. Epidemiological evidence suggests that regions with high environmental viral concentration tend to exhibit higher infection rates, although this factor is often neglected in classical transmission models. To address this limitation, we propose a compartmental model consisting of susceptible individuals, symptomatic infected individuals, asymptomatic infected individuals, recovered individuals, and a distinct compartment representing viruses in the environment. The system dynamics are formulated using ordinary differential equations. The dynamical behavior of the model is investigated by analyzing the equilibrium points and deriving the basic reproduction number $\mathcal{R}_0$. Using Lyapunov's direct method and LaSalle's invariance principle, sufficient conditions for the local and global stability of both disease-free and endemic equilibria are established. Conditions for disease extinction and persistence are also derived. Similarly, it is inferred that the presence of viruses in the environment is responsible for disease occurrence and is associated with infection severity and threshold levels. Numerical simulations are performed to validate the analytical results and to illustrate the impact of environmental viral concentration on disease transmission.

1. Introduction. Coronavirus, specifically COVID-19, is a respiratory illness caused by the SARS-COV-2 virus. Its initial case was reported in De-

2020 *Mathematics Subject Classification*: Primary 34D20; Secondary 34C60, 34C23, 92B05.
Key words and phrases: epidemiology model, covid-19, compartment model, numerical stability and simulation, basic reproduction number.
Received 2 February 2025; revised 24 June 2025 and 2 March 2026.
Published online 28 April 2026.

DOI: 10.4064/am2565-10-2025

cember 2019 in Wuhan, Hubei Province, China [6]. Subsequently, the virus swiftly disseminated worldwide, prompting its classification as a global pandemic and necessitating stringent measures by governments to mitigate its transmission. COVID-19 symptoms manifest in diverse ways, encompassing fever, cough, breathing difficulties, and fatigue [25]. These symptoms can range from mild to severe, with severe cases potentially resulting in critical complications and mortality. The transmission of the virus is influenced by factors such as proximity to an infected individual, social interactions, and adherence to healthy practices [20, 32].

A vital and simple tool for analyzing and forecasting the dynamic behavior of different epidemics is mathematical modeling. In fact, in the last two decades, researchers have made significant efforts to identify the factors responsible for the spread of deadly diseases in society. They have developed certain types of epidemiological models to fix the reasons for such diseases and optimize the spread in the community. Various models with diverse features and characteristics of infectious diseases have been formulated and analyzed in the literature [4, 5, 10, 11, 13, 14].

Epidemic models are well established, and a comprehensive bibliography on the topic is presented in [10]. The SI model is the simplest epidemiological model, consisting of two classes: susceptible (S) and ineffective (I). Disease transmission occurs through direct or indirect contact, making it a communicable disease. If the disease allows for recovery with potential relapses, the recovered individuals form the class R, providing temporary immunity. This extends the model to the SIR type. If recovered individuals can lose their immunity and become susceptible again, allowing for disease relapses, the model becomes SIRS.

More advanced versions of the model incorporate features such as quarantine, exposed individuals, and asymptomatic cases [11, 35]. In this study, we will consider some of these additional classes, and their details will be presented before proceeding with the model formulation.

In order to mitigate the spread of the virus, numerous countries have implemented various measures including partial or complete lock downs, quarantine protocols, the promotion of social distancing, the utilization of protective masks, and the emphasis on personal hygiene [1, 21, 22]. These measures are implemented with the intention of reducing the transmission of the virus and safeguarding public health.

Multiple vaccines have been developed to combat the coronavirus [12]. These vaccines have undergone distribution and administration on a global scale, with the objective of curtailing the transmission of the disease and minimizing associated fatalities. The widespread vaccination efforts aim to provide protection against COVID-19 and contribute to the overall mitigation of the pandemics impact.

The transmission and spread of the virus have been extensively studied [4, 31]. Research has focused on analyzing how the virus is transmitted between individuals and across different locations [9]. Mathematical models have been employed to predict the future spread of the virus [3].

Furthermore, the analysis of injury and mortality data has provided insights into the patterns of viral spread across various regions [24, 26]. Additionally, studies have examined the impact of restrictions and closures on economic and social aspects [19, 30]. These investigations aim to understand the consequences of containment measures and their implications for societies as a whole.

Many classical models focus primarily on symptomatic cases as the main source of transmission, often overlooking the contributions of asymptomatic carriers and environmental factors. However, it is crucial to explore the role of asymptomatic individuals and the presence of the virus in the environment to gain a comprehensive understanding of infectious disease transmission and effectively manage outbreaks.

Asymptomatic individuals, who do not display symptoms, can unknowingly spread the virus to others, leading to what is known as "hidden transmission". This hidden transmission poses a significant challenge in controlling outbreaks since these individuals are more likely to interact with the general population and may not seek medical attention or take necessary precautions, allowing the virus to propagate silently.

Additionally, identifying areas with a higher concentration of the virus in the environment can be instrumental in implementing targeted preventive measures. Environmental factors, such as contaminated surfaces or aerosolized particles, can contribute to the spread of infectious diseases but are often overlooked in existing models. Beside other features, stochastic perturbation factors (such as absolute humidity, precipitation and temperature, etc.) greatly affect the force of infection specifically in human viral diseases. Recognizing this effect enables us to add randomness to deterministic biological models and therefore expose the effects of environmental variability. Stochastic population dynamics has been the subject of much research by several writers (see [2, 23]).

To address these limitations, our proposed model explicitly considers the dynamics of asymptomatic individuals, capturing their role in transmission chains. By incorporating the influence of environmental factors, we aim to provide a more comprehensive and accurate understanding of how the disease spreads, enabling better strategies for disease control and prevention.

This paper aims to examine the significance of asymptomatic individuals and the presence of viruses in the surrounding environment. Specifically, we investigate the potential progression of asymptomatic individuals to symptomatic cases, which are recognized as infected. In this context, the situa-

tion can be compared to the aquatic infection model presented by Kuang and Beretta [5]. The objective of our study is to conduct a mathematical analysis of a modified version of the COVID model that incorporates an explicit equation for viral particles. In the initial phase of our investigation, we focus on the theoretical examination of the proposed system. Subsequently, we conduct preliminary simulations utilizing realistic parameter values.

It is essential to examine mathematical models that clarify covid transmission pathways in order to understand the diseases progress and devise control measures. These models provide a compromise between the robustness of data links and biological scenarios by depicting the infections natural course. It is important to highlight that there is a strong correlation between the spread and persistence of bacteria and changes in the surrounding environment.

The paper is structured as follows. In Section 2, we present the formulation of the model. We provide a detailed mathematical analysis, including the boundedness of the trajectories and the derivation of an expression for the basic reproduction number of the disease. Additionally, we determine the equilibria of the model and assess their local stability. Furthermore, we investigate the global stability of the equilibria in Section 3.

In Section 4, we present the results of a numerical simulation. We provide a comprehensive overview of the simulation findings. Finally, in the concluding section, we offer a brief discussion summarizing the key points of our study.

2. Model formulation. Let $\mathcal{P}(t)$ denote the total population. Thus, $\mathcal{P}(t) = \mathcal{C}_S(t) + \mathcal{C}_I(t) + \mathcal{C}_A(t) + \mathcal{C}_R(t)$. Here:

- $\mathcal{C}_S(t)$: The susceptible class comprises individuals who have not been exposed to the virus and thus have no immunity or infection. They are at risk of acquiring the virus.
- $\mathcal{C}_I(t)$: The symptomatic infectious class represents individuals who display disease symptoms and have the ability to transmit it to others. These individuals have contracted the virus and are experiencing noticeable symptoms that are characteristic of the disease.
- $\mathcal{C}_A(t)$: The asymptomatic infectious class represents individuals who are infected with the disease but do not show any noticeable symptoms. Despite not showing explicit symptoms, individuals in this class can still transmit the disease to others, making them capable of spreading the infection.
- $\mathcal{C}_R(t)$: represents the removed class, which comprises individuals who have recovered from the disease. These individuals have undergone the necessary treatment and have either developed immunity to the disease or are no longer capable of transmitting it.

In addition to the various classes of individuals, we also consider the presence of free viruses in the environment, denoted by $\mathcal{C}_V(t)$, which represents the concentration of viruses in the surrounding environment or air. These free viruses are relevant because they have the potential to infect individuals who come into contact with them. For instance, if a person touches a surface where the viruses are present and then touches their mouth, nose, or eyes, they can be infected. Moreover, in highly polluted environments, viruses can attach to aerosol particles and be inhaled by individuals. Infected individuals can release these viruses into the air through sneezing or coughing.

For the first equation we have

$$\frac{d\mathcal{C}_S}{dt} = \Pi - \zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) - \zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H} + \mathcal{C}_V} - m\mathcal{C}_S.$$

Susceptibles are recruited under the steady state assumption at a rate of Π. They can become infected through contact with infected and asymptomatic individuals at a rate of ζ_S, with k representing the infectivity factor of the latter (where $k < 1$). Additionally, susceptible individuals can become infected through direct exposure to viruses present in the air at a rate of ζ_V. In this scenario, a Holling type II response is utilized, as assuming a higher quantity of viruses does not contribute to new infections. However, the probability of infection does increase, but is capped at a maximum value of 1. Finally, the natural mortality rate is denoted by m. We have

$$\frac{d\mathcal{C}_A}{dt} = (1-p)\zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) + (1-p)\zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} - (\epsilon_A + m + \delta_A)\mathcal{C}_A - \Upsilon\mathcal{C}_A,$$
$$\frac{d\mathcal{C}_I}{dt} = p\zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) + p\zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} - (\epsilon_I + m + \delta_I)\mathcal{C}_I + \Upsilon\mathcal{C}_A.$$

Asymptomatic infected individuals can arise from the two infection processes mentioned earlier, with a probability of $1-p$. In other words, p represents the probability of transitioning to symptomatic infection. Both classes of infected individuals, asymptomatic and symptomatic, are susceptible to disease-related mortality δ_A and δ_I respectively and in addition to natural mortality. Furthermore, they can recover at respective rates of ϵ_A and ϵ_I. Finally, we consider the possibility of a progression from asymptomatic to symptomatic infection at a rate of Υ. Next, we have

$$\frac{d\mathcal{C}_R}{dt} = \epsilon_I \mathcal{C}_I + \epsilon_A \mathcal{C}_A - m\mathcal{C}_R.$$

Recovered individuals are recruited from both the asymptomatic and symptomatic infected classes. However, like other population groups, they are also subject to natural mortality. Consider

$$\frac{d\mathcal{C}_V}{dt} = \xi_I \mathcal{C}_I + \xi_A \mathcal{C}_A - n\mathcal{C}_V - \zeta_V[\mathcal{C}_S + \mathcal{C}_I + \mathcal{C}_A + \mathcal{C}_R]\frac{\mathcal{C}_V}{\mathcal{H} + \mathcal{C}_V}.$$

Viruses are released into the air by symptomatic infected individuals at a rate of ξ_I and by asymptomatic infected individuals at a rate of ξ_A. These viruses cannot survive in the environment and decay with a rate of n. They can be taken up by individuals from all classes, but they do not contribute to new infections if the individual taking them up is already infected or has recovered. As a result, some of the terms in the virus equation are specific to the equation for susceptibles and do not appear in the equations for other classes. Finally, we have

$$\frac{d\mathcal{C}_D}{dt} = \delta_I \mathcal{C}_I + \delta_A \mathcal{C}_A,$$

where the class $\mathcal{C}_D$ represents a sink, which corresponds to disease-related deaths. This class is included in the model to allow for potential comparison with data, but it can be excluded from the analysis if desired. The mechanisms underlying the model are depicted in Figure 1.

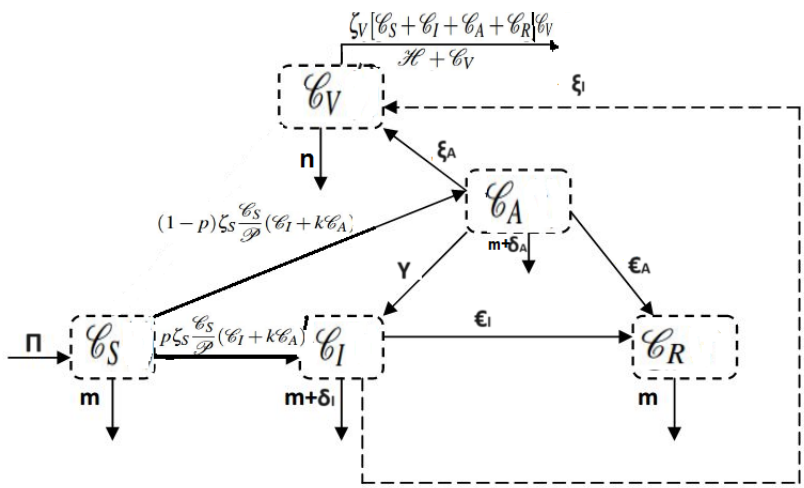

Fig. 1. The basic interactions among the compartments

The dynamics of the proposed model are governed by the following system of nonlinear ordinary differential equations:

$$\begin{cases} \frac{d\mathcal{C}_S}{dt} = \Pi - \zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) - \zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} - m\mathcal{C}_S, \\ \frac{d\mathcal{C}_A}{dt} = [(1-p)\mathcal{C}_S]\left[\frac{\zeta_S(\mathcal{C}_I+k\mathcal{C}_A)}{\mathcal{P}} + \frac{\zeta_V \mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V}\right] - (\epsilon_A + m + \delta_A)\mathcal{C}_A - \Upsilon\mathcal{C}_A, \\ \frac{d\mathcal{C}_I}{dt} = p\zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) + p\zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} - (\epsilon_I + m + \delta_I)\mathcal{C}_I + \Upsilon\mathcal{C}_A, \\ \frac{d\mathcal{C}_R}{dt} = \epsilon_I \mathcal{C}_I + \epsilon_A \mathcal{C}_A - m\mathcal{C}_R, \\ \frac{d\mathcal{C}_V}{dt} = \xi_I \mathcal{C}_I + \xi_A \mathcal{C}_A - n\mathcal{C}_V - \zeta_V[\mathcal{C}_S + \mathcal{C}_I + \mathcal{C}_A + \mathcal{C}_R]\frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V}, \\ \frac{d\mathcal{C}_D}{dt} = \delta_I \mathcal{C}_I + \delta_A \mathcal{C}_A. \end{cases} \tag{2.1}$$

The positive parameters used in the model are defined as follows (Table 1):

Table 1. Model parameters and their meaning

-	Description
Π	Susceptibles recruitment rate
ζ_S	Disease transmission rate by direct contact with infected individuals $\mathcal{C}_I$ and $\mathcal{C}_A$ to susceptible individuals $\mathcal{C}_S$
k	The ratio of transmissibility between individuals with asymptomatic infections and those with symptomatic infections
ζ_V	Disease transmission rate by contact with viruses in the environment
m	Natural death rate of people
δ_A	Disease-related mortality rate for asymptomatic individuals
δ_I	Disease-related mortality rate for infected individuals
Υ	Progression rate from asymptomatic to symptomatic infection
ϵ_A	Recovery rate from asymptomatic infection
ϵ_I	Recovery rate from symptomatic infection
ξ_I	Shedding coefficient from infected individuals $\mathcal{C}_I$ to viruses $\mathcal{C}_V$
ξ_A	Shedding coefficient from individuals $\mathcal{C}_A$ to viruses $\mathcal{C}_V$
$1/n$	Lifetime of the virus $\mathcal{C}_V$
p	Probability of symptomatic infection transition
$\mathcal{H}$	Maximal transmission efficiency

Regarding the initial conditions, we establish the following:

$$\mathcal{C}_S > 0, \quad \mathcal{C}_I \geq 0, \quad \mathcal{C}_A \geq 0, \quad \mathcal{C}_R \geq 0, \quad \mathcal{C}_V \geq 0.$$

3. Fundamental characteristics of the model

3.1. Biological feasible region and positivity of the solution. The application of the classical theory of ordinary differential equations implies that for every set of initial data, $(\mathcal{C}_{S0}, \mathcal{C}_{A0}, \mathcal{C}_{I0}, \mathcal{C}_{R0}, \mathcal{C}_{V0})$, there exists a solution $(\mathcal{C}_S(t), \mathcal{C}_A(t), \mathcal{C}_I(t), \mathcal{C}_R(t), \mathcal{C}_V(t))$ defined in the maximal open interval $(0, T)$ with $(T > 0)$ (see [7, 33, 34]).

3.1.1. *Positivity of the solution.* It is clear that any solution to (2.1) with nonnegative initial conditions remains nonnegative. It follows that if there exists $t_0 \in (0, T)$ such that $\mathcal{C}_i(t_0) = 0$, then we have $\frac{d\mathcal{C}_i(t_0)}{dt} \geq 0$ for all $i = S, A, I, R, V$.

For $\mathcal{C}_S$ we suppose that there exists $t_0 \in (0, T)$ such that $\mathcal{C}_S(t_0) = 0$, $\mathcal{C}'_S(t_0) \leq 0$ and $\mathcal{C}_S(t) > 0$ for $t \in [0, t_0)$. Additionally, from the first equation of system (2.1), $\mathcal{C}'_S(t_0) = \Pi > 0$, but this leads to a contradiction to the supposition that $\mathcal{C}'_S(t_0) \leq 0$, which completes the proof. Following the same method used to demonstrate that $\mathcal{C}_A \geq 0$, $\mathcal{C}_I \geq 0$, $\mathcal{C}_R \geq 0$, $\mathcal{C}_V \geq 0$, we infer

that $\mathcal{C}_S = 0$, $\mathcal{C}_A = 0$, $\mathcal{C}_I = 0$, $\mathcal{C}_R = 0$, $\mathcal{C}_V = 0$ are invariant sets showing that $\mathcal{C}_S \geq 0$, $\mathcal{C}_A \geq 0$, $\mathcal{C}_I \geq 0$, $\mathcal{C}_R \geq 0$, $\mathcal{C}_V \geq 0$ whenever

$$\mathcal{C}_S(0) > 0, \quad \mathcal{C}_A(0) > 0, \quad \mathcal{C}_I(0) > 0, \quad \mathcal{C}_R(0) > 0, \quad \mathcal{C}_V(0) > 0.$$

3.1.2. *Biological feasible region.* Summing the equations, assuming the absence of disease related mortality, we obtain

$$\frac{d\mathcal{P}}{dt} = \Pi - m\mathcal{P}.$$

Then the population reaches a state of equilibrium at Π/m. In order to avoid the demographic concerns, we introduce a modification in our model by setting $\Pi = m\mathcal{P}$, hence

$$\frac{d\mathcal{P}}{dt} = -\delta_A \mathcal{C}_A - \delta_I \mathcal{C}_I.$$

As a result of this modification, the sum of disease-related deaths and the population, denoted by $\mathcal{P} + \mathcal{C}_D$, remains constant. Since the initial value of disease related deaths is zero ($\mathcal{C}_D(0) = 0$), we can conclude that $\mathcal{P}(t) + \mathcal{C}_D(t) = \mathcal{P}(0)$ holds true at all times.

By summing the four equations of the system, we obtain

$$\frac{d\mathcal{P}}{dt} = \Pi - m\mathcal{P} - \delta_I \mathcal{C}_I - \delta_A \mathcal{C}_A = \Pi - m\mathcal{P} - \frac{d\mathcal{C}_D}{dt} \leq \Pi - m\mathcal{P}.$$

And

$$\frac{d\mathcal{C}_V}{dt} \leq \xi_I \mathcal{C}_I + \xi_A \mathcal{C}_A - n\mathcal{C}_V \leq (\xi_I + \xi_A)\frac{\Pi}{m} - n\mathcal{C}_V \leq \Pi_V - n\mathcal{C}_V.$$

Thus, the biologically feasible region implies that the set

$$\begin{aligned}\Sigma = \big\{(\mathcal{C}_S, \mathcal{C}_A, \mathcal{C}_I, \mathcal{C}_R, \mathcal{C}_V) \in \mathbb{R}^5_+ : \mathcal{C}_S + \mathcal{C}_A + \mathcal{C}_I + \mathcal{C}_R \leq \mathcal{M} = \max\{\mathcal{P}(0), \Pi/m\} \\ \text{and } \mathcal{C}_V \leq \mathcal{M}_\mathcal{V} = \max\{\mathcal{C}_V(0), \Pi_V/n\},\ \mathcal{C}_S > 0,\ \mathcal{C}_A,\ \mathcal{C}_I,\ \mathcal{C}_R,\ \mathcal{C}_V \geq 0\big\}\end{aligned}$$

is positively invariant and attractive for (2.1).

As a result, we can focus on solutions with initial conditions within the feasible region for all $t > 0$, taking into account the existence and uniqueness of solutions (see [7, 33]).

For all parameter values, the system (2.1) possesses a disease-free equilibrium denoted by $\mathcal{E}_0$, which is characterized by the state $\mathcal{E}_0 = (\mathcal{C}_{S_0}, 0, 0, 0, 0)$, where $\mathcal{C}_{S_0} = \Pi/m$. This equilibrium represents the level of susceptible population in the absence of infection.

3.2. The basic reproduction number. The basic reproduction number, denoted by $\mathcal{R}_0$, is a fundamental epidemiological metric that quantifies the average number of secondary infections produced by a single infection introduced into a completely susceptible population. It serves as a crucial

threshold quantity that mathematically characterizes the spread of the disease. This measure is valuable in determining whether an infectious disease has the potential to propagate within a population [8].

In the context of the disease-free equilibrium, the condition $\mathcal{R}_0 < 1$ indicates that the threshold quantity is below unity. In this case, the equilibrium is stable, and there is no spread of the disease within the community. However, if the value of $\mathcal{R}_0$ surpasses 1, the disease becomes endemic and persists in the population, leading to an epidemic. When the threshold quantity exceeds 1, the endemic equilibrium is both locally and globally stable [8, 27].

We utilized the next-generation matrix method [28] to determine the basic reproduction number $\mathcal{R}_0$ for the system (2.1).

The reduced form of the system (2.1) can be expressed as

$$\dot{\mathfrak{X}} = \mathcal{F}(\mathfrak{X}) - \mathcal{V}(\mathfrak{X}),$$

where $\mathfrak{X}(\mathcal{C}_A, \mathcal{C}_I, \mathcal{C}_V)$ denotes the state vector of the system, and

$$\mathcal{F}(\mathcal{C}_A, \mathcal{C}_I, \mathcal{C}_V) = \begin{pmatrix} (1-p)\zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) + (1-p)\zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} \\ p\zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) + p\zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} \\ 0 \end{pmatrix},$$

$$\mathcal{V}(\mathcal{C}_A, \mathcal{C}_I, \mathcal{C}_V) = \begin{pmatrix} (\epsilon_A + m + \delta_A + \Upsilon)\mathcal{C}_A \\ (\epsilon_I + m + \delta_I)\mathcal{C}_I - \Upsilon \mathcal{C}_A \\ -\xi_I \mathcal{C}_I - \xi_A \mathcal{C}_A + n\mathcal{C}_V + \zeta_V \mathcal{P} \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} \end{pmatrix}.$$

The Jacobian matrices of $\mathcal{F}(\mathfrak{X})$ and $\mathcal{V}(\mathfrak{X})$ at the disease-free equilibrium point $\mathcal{E}_0$ are

$$\mathfrak{J}_{\mathcal{F}}(\mathcal{E}_0) = \begin{pmatrix} (1-p)k\zeta_S & (1-p)\zeta_S & (1-p)\frac{\zeta_V \mathcal{C}_{S_0}}{\mathcal{H}} \\ pk\zeta_S & p\zeta_S & p\frac{\zeta_V \mathcal{C}_{S_0}}{\mathcal{H}} \\ 0 & 0 & 0 \end{pmatrix},$$

$$\mathfrak{J}_{\mathcal{V}}(\mathcal{E}_0) = \begin{pmatrix} B_0 & 0 & 0 \\ -\Upsilon & C_0 & 0 \\ -\xi_A & -\xi_I & n + \frac{\zeta_V \mathcal{C}_{S_0}}{\mathcal{H}} \end{pmatrix},$$

where

$$\begin{cases} B_s = \epsilon_A + m + \delta_A + \Upsilon - (1-p)k\zeta_S, \\ C_s = \epsilon_I + \delta_I + m - p\zeta_S, \\ B_0 = \epsilon_A + m + \delta_A + \Upsilon, \\ C_0 = \epsilon_I + \delta_I + m. \end{cases} \tag{3.1}$$

Consequently, we have

$$\mathfrak{J}_{\mathcal{V}}^{-1}(\mathcal{E}_0) = \frac{1}{\Delta} \times \begin{pmatrix} \Delta/B_0 & 0 & 0 \\ \Upsilon[n + \zeta_V \mathcal{C}_{S_0}/\mathcal{H}] & \Delta/C_0 & 0 \\ \xi_I \Upsilon + \xi_A C_0 & \xi_I B_0 & \frac{\Delta}{n+\zeta_V \mathcal{C}_{S_0}/\mathcal{H}} \end{pmatrix},$$

where

$$\Delta = [n + \zeta_V \mathcal{C}_{S_0}/\mathcal{H}] B_0 C_0.$$

The next generation matrix $\mathfrak{J}_{\mathcal{F}}(\mathcal{E}_0)\mathfrak{J}_{\mathcal{V}}^{-1}(\mathcal{E}_0)$ is given by

$$\mathfrak{J}_{\mathcal{F}}(\mathcal{E}_0)\mathfrak{J}_{\mathcal{V}}^{-1}(\mathcal{E}_0) = \frac{1}{\Delta} \begin{pmatrix} (1-p)k\zeta_S & (1-p)\zeta_S & (1-p)\zeta_V \mathcal{C}_{S_0}/\mathcal{H} \\ pk\zeta_S & p\zeta_S & p\zeta_V \mathcal{C}_{S_0}/\mathcal{H} \\ 0 & 0 & 0 \end{pmatrix} \times \begin{pmatrix} \Delta/B_0 & 0 & 0 \\ \Upsilon[n + \zeta_V \mathcal{C}_{S_0}/\mathcal{H}] & \Delta/C_0 & 0 \\ \xi_I \Upsilon + \xi_A C_0 & \xi_I B_0 & \frac{\Delta}{n+\zeta_V \mathcal{C}_{S_0}/\mathcal{H}} \end{pmatrix}.$$

Hence, the next-generation matrix can be written in compact form as

$$\mathfrak{J}_{\mathcal{F}}(\mathcal{E}_0)\mathfrak{J}_{\mathcal{V}}^{-1}(\mathcal{E}_0) = \begin{pmatrix} \mathcal{A}_{11} & \mathcal{A}_{12} & \mathcal{A}_{13} \\ \mathcal{A}_{21} & \mathcal{A}_{22} & \mathcal{A}_{23} \\ 0 & 0 & 0 \end{pmatrix}$$

with

$$\begin{aligned}
\mathcal{A}_{11} &= (1-p)\frac{\mathcal{H}k\zeta_S\Delta + \zeta_S\Upsilon[\mathcal{H}n + \zeta_V\mathcal{C}_{S_0}]B_0 + \zeta_V\mathcal{C}_{S_0}[\xi_I\Upsilon + \xi_A C_0]B_0}{\mathcal{H}B_0\Delta}, \\
\mathcal{A}_{12} &= (1-p)\frac{\mathcal{H}\zeta_S\Delta + \zeta_V\mathcal{C}_{S_0}\xi_I C_0 B_0}{\mathcal{H}C_0\Delta}, \\
\mathcal{A}_{13} &= (1-p)\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}n + \zeta_V\mathcal{C}_{S_0}}, \\
\mathcal{A}_{21} &= p\frac{\mathcal{H}k\zeta_S\Delta + \zeta_S\Upsilon[\mathcal{H}n + \zeta_V\mathcal{C}_{S_0}]B_0 + \zeta_V\mathcal{C}_{S_0}[\xi_I\Upsilon + \xi_A C_0]B_0}{\mathcal{H}B_0\Delta}, \\
\mathcal{A}_{22} &= p\frac{\mathcal{H}\zeta_S\Delta + \zeta_V\mathcal{C}_{S_0}\xi_I C_0 B_0}{\mathcal{H}C_0\Delta}, \\
\mathcal{A}_{23} &= p\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}n + \zeta_V\mathcal{C}_{S_0}}.
\end{aligned}$$

Thus

$$\mathcal{R}_0 = \rho(\mathfrak{J}_F(\mathcal{E}_0)\mathfrak{J}_V^{-1}(\mathcal{E}_0)) = \rho\begin{pmatrix} \mathcal{A}_{11} & \mathcal{A}_{12} & \mathcal{A}_{13} \\ \mathcal{A}_{21} & \mathcal{A}_{22} & \mathcal{A}_{23} \\ 0 & 0 & 0 \end{pmatrix} = \rho\begin{pmatrix} \mathcal{A}_{11} & \mathcal{A}_{12} \\ \mathcal{A}_{21} & \mathcal{A}_{22} \end{pmatrix},$$

where $\rho(\cdot)$ denotes the spectral radius of a square matrix. Hence

$$\mathcal{R}_0 = \max\{|\lambda| : \lambda \text{ is a root of } \lambda^2 - (\mathcal{A}_{11} + \mathcal{A}_{22})\lambda + \mathcal{A}_{11}\mathcal{A}_{22} - \mathcal{A}_{12}\mathcal{A}_{21} = 0\}.$$

However, it is easy to verify that $p\mathcal{A}_{11} = (1-p)\mathcal{A}_{21}$, and $p\mathcal{A}_{12} = (1-p)\mathcal{A}_{22}$.

So, we have

$$\mathcal{A}_{11}\mathcal{A}_{22} - \mathcal{A}_{12}\mathcal{A}_{21} = 0.$$

And

$$\begin{aligned}\mathcal{A}_{11} + \mathcal{A}_{22} &= (1-p)\frac{k\zeta_S C_0[n\mathcal{H} + \zeta_V \mathcal{C}_{S_0}] + \zeta_S \Upsilon[\mathcal{H}n + \zeta_V \mathcal{C}_{S_0}]}{\mathcal{H}\Delta} \\ &\quad + (1-p)\frac{\zeta_V \mathcal{C}_{S_0}[\xi_I \Upsilon + \xi_A C_0]}{\mathcal{H}\Delta} \\ &\quad + p\frac{\zeta_S B_0[n\mathcal{H} + \zeta_V \mathcal{C}_{S_0}] + \zeta_V \mathcal{C}_{S_0}\xi_I B_0}{\mathcal{H}\Delta} \\ &= \frac{\zeta_V \mathcal{C}_{S_0}[[\xi_I + \zeta_S](pB_0 + (1-p)\Upsilon) + (1-p)C_0(k\zeta_S + \xi_A)]}{\mathcal{H}\Delta} \\ &\quad + \frac{n\mathcal{H}\zeta_S[(1-p)(kC_0 + \Upsilon) + pB_0]}{\mathcal{H}\Delta}.\end{aligned}$$

Finally, $\mathcal{R}_0 = \mathcal{A}_{11} + \mathcal{A}_{22}$, where

$$\begin{aligned}\mathcal{R}_0 &= \frac{\zeta_V \Pi[[\xi_I + \zeta_S](pB_0 + (1-p)\Upsilon) + (1-p)C_0(k\zeta_S + \xi_A)]}{m\mathcal{H}\Delta} \\ &\quad + \frac{n\zeta_S[(1-p)(kC_0 + \Upsilon) + pB_0]}{\Delta}.\end{aligned}$$

3.3. Equilibria and their stability

3.3.1. *Existence of equilibria.* The existence of the disease-free equilibrium $\mathcal{E}_0$ for System (2.1) has been previously confirmed for all parameter values. Our objective now is to demonstrate that the existence of at least one endemic equilibrium is $\mathcal{E}^*$. We can state the following theorem.

THEOREM 3.1. *The system* (2.1) *possesses the following equilibria:*

(1) *The coronavirus disease-free equilibrium* $\mathcal{E}_0(\Pi/m, 0, 0, 0, 0)$ *always exists.*
(2) *The coronavirus endemic equilibrium* $\mathcal{E}^*(\mathcal{C}_S^*, \mathcal{C}_A^*, \mathcal{C}_I^*, \mathcal{C}_R^*, \mathcal{C}_V^*)$.

Proof. At an equilibrium point $(\mathcal{C}_S, \mathcal{C}_A, \mathcal{C}_I, \mathcal{C}_R, \mathcal{C}_V) \in \Sigma$ of system (2.1), the following equalities hold:

$$\begin{cases} \Pi - \zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) - \zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} - m\mathcal{C}_S = 0, \\ (1-p)\mathcal{C}_S[\frac{\zeta_S(\mathcal{C}_I + k\mathcal{C}_A)}{\mathcal{P}} + \frac{\zeta_V \mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V}] - (\epsilon_A + m + \delta_A)\mathcal{C}_A - \Upsilon\mathcal{C}_A = 0, \\ p\zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) + p\zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} - (\epsilon_I + m + \delta_I)\mathcal{C}_I + \Upsilon\mathcal{C}_A = 0, \\ \epsilon_I \mathcal{C}_I + \epsilon_A \mathcal{C}_A - m\mathcal{C}_R = 0, \\ \xi_I \mathcal{C}_I + \xi_A \mathcal{C}_A - n\mathcal{C}_V - \zeta_V[\mathcal{C}_S + \mathcal{C}_I + \mathcal{C}_A + \mathcal{C}_R]\frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} = 0. \end{cases} \tag{3.2}$$

From the first equality we have $\Pi - m\mathcal{C}_S = \zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) + \zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V}$. By substituting this expression in the second and third equalities, we obtain

$$\mathcal{C}_A = \frac{(1-p)[\Pi - m\mathcal{C}_S]}{B_0}$$

and

$$\mathcal{C}_I = [\Pi - m\mathcal{C}_S]\frac{pB_0 + (1-p)\Upsilon}{B_0 C_0}.$$

Furthermore, we have

$$\mathcal{C}_R = [\Pi - m\mathcal{C}_S]\frac{\epsilon_I(pB_0 + (1-p)\Upsilon) + \epsilon_A(1-p)C_0}{mB_0C_0},$$

which implies

$$n\mathcal{C}_V^2 + [(n\mathcal{H} + \zeta_V\mathcal{P}) - (\xi_I\mathcal{C}_I + \xi_A\mathcal{C}_A)]\mathcal{C}_V - \mathcal{H}(\xi_I\mathcal{C}_I + \xi_A\mathcal{C}_A) = 0. \tag{3.3}$$

By straightforward verification, we can confirm that (3.3) has a positive root; let us denote it by $\mathcal{C}_V = \psi(\mathcal{C}_S)$.

Therefore, to find the positive equilibria of (2.1), we need to solve the equation

$$\phi(\mathcal{C}_S) = \Pi - \zeta_S\frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) - \zeta_V\mathcal{C}_S\frac{\psi(\mathcal{C}_S)}{\mathcal{H} + \psi(\mathcal{C}_S)} - m\mathcal{C}_S = 0. \tag{3.4}$$

We observe that the function ϕ is continuous, and $\frac{\partial\phi}{\partial\mathcal{C}_S} < 0$. Additionally, we have

$$\lim_{\mathcal{C}_S \to \Pi/m} \phi(\mathcal{C}_S) < 0 \quad \text{and} \quad \lim_{\mathcal{C}_S \to 0^+} \phi(\mathcal{C}_S) > 0.$$

By applying the intermediate value theorem, we can conclude that there exists at least one value $0 < \mathcal{C}_S < \Pi/m$ for which $\phi(\mathcal{C}_S) = 0$. Consequently, the system (2.1) possesses at least one endemic equilibrium. ∎

3.3.2. *Local stability.* In this subsection, we will examine the local stability of the coronavirus-free equilibrium $\mathcal{E}_0$ and the coronavirus-endemic equilibrium $\mathcal{E}^*$. We present the following theorems.

THEOREM 3.2. *We have the following results:*

(1) *The coronavirus-free equilibrium* $\mathcal{E}_0 = (\mathcal{C}_{S_0}, 0, 0, 0, 0)$ *of the system* (2.1) *is locally asymptotically stable if* $\mathcal{R}_0 < 1$ *and*

$$B_0 + C_0 > (1-p)[k\zeta_S + \xi_A] + p[\zeta_S + \xi_I], \quad \xi_I > \zeta_S. \tag{3.5}$$

(2) *If* $\mathcal{R}_0 > 1$, *the coronavirus-free equilibrium* $\mathcal{E}_0$ *of the system* (2.1) *is not stable.*

Proof. By applying the linearization method, we can obtain the Jacobian matrix of the system (2.1) at the coronavirus-free equilibrium $\mathcal{E}_0$,

$$J(\mathcal{E}_0) = \begin{pmatrix} -m & -k\zeta_S & -\zeta_S & 0 & -\zeta_V\mathcal{C}_{S_0}/\mathcal{H} \\ 0 & -B_s & (1-p)\zeta_S & 0 & (1-p)\zeta_V\mathcal{C}_{S_0}/\mathcal{H} \\ 0 & pk\zeta_S+\Upsilon & -C_s & 0 & p\zeta_V\mathcal{C}_{S_0}/\mathcal{H} \\ 0 & \epsilon_A & \epsilon_I & -m & 0 \\ 0 & \xi_A & \xi_I & 0 & -[n+\zeta_V\mathcal{C}_{S_0}/\mathcal{H}] \end{pmatrix}.$$

At the point $\mathcal{E}_0$, the eigenvalue $-m$ has multiplicity 2, and the remaining eigenvalues are the roots of the characteristic polynomial of the corresponding minor matrix of J, which is determined by

$$\zeta_V^3 + \alpha_2\zeta_V^2 + \alpha_1\zeta_V + \alpha_0 = 0, \tag{3.6}$$

where α_i, $i = 0, 1, 2$, are given by

$$\begin{aligned}
\alpha_2 &= B_s + C_s + n + \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}} \\
\alpha_1 &= B_sC_s + n[B_s + C_s] - (1-p)\zeta_S[pk\zeta_S + \Upsilon] \\
&\quad + \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}[B_s + C_s - (1-p)\xi_A - p\xi_I] \\
\alpha_0 &= \left[n + \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right]B_sC_s - (1-p)n\zeta_S[pk\zeta_S + \Upsilon] \\
&\quad - \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\left(\xi_I[pB_s + (1-p)[pk\zeta_S + \Upsilon]] + (1-p)\xi_A[C_s + p\zeta_S]\right).
\end{aligned}$$

It is evident that $a_2 > 0$. Additionally, the following equations are satisfied, based on condition (3.5):

$$\begin{aligned}
\alpha_0 &= \left[n + \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right]B_sC_s - (1-p)n\zeta_S[pk\zeta_S + \Upsilon] \\
&\quad - \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\big(\xi_I[pB_s + (1-p)[pk\zeta_S + \Upsilon]] + (1-p)\xi_A[C_s + p\zeta_S]\big) \\
&= \Delta - p\zeta_S\left[n + \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right]B_0 - (1-p)k\zeta_S\left[n + \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right]C_0 \\
&\quad + p\zeta_S(1-p)k\zeta_S\left[n + \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right] - (1-p)n\zeta_S[pk\zeta_S + \Upsilon] \\
&\quad - \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\big(\xi_I[pB_0 + (1-p)[pk\zeta_S + \Upsilon]] - p(1-p)k\zeta_S\xi_I\big) \\
&\quad - \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\big((1-p)\xi_A[C_0 + p\zeta_S] - p\zeta_S(1-p)\xi_A\big) \\
&= \Delta - \frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\big(\xi_I[pB_0 + (1-p)\Upsilon] + (1-p)C_0[k\zeta_S + \xi_A]\big) \\
&\quad - n\zeta_S[(1-p)[kC_0 + \Upsilon] + pB_0] \\
&= \Delta(1 - R_0) > 0,
\end{aligned}$$

$$
\begin{aligned}
\alpha_1 &= B_0C_0-(1-p)k\zeta_SC_0-\zeta_S[pB_0+(1-p)\Upsilon]\\
&\quad+\left[n+\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right][B_s+C_s]-\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}[(1-p)\xi_A+p\xi_I]\\
&= B_0C_0-\zeta_S[(1-p)[kC_0+\Upsilon]+pB_0]+n[B_s+C_s]\\
&\quad+\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}[B_s+C_s-(1-p)\xi_A-p\xi_I],\\
\alpha_1\left[n+\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right] &= \Delta-n\zeta_S[(1-p)[kC_0+\Upsilon]+pB_0]\\
&\quad-\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\zeta_S[(1-p)[kC_0+\Upsilon]+pB_0]\\
&\quad+n\left[n+\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right]n[B_s+C_s]\\
&\quad+\left[n+\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right]\left[\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}[B_s+C_s-(1-p)\xi_A-p\xi_I]\right]\\
&= \alpha_0+\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}([\xi_I-\zeta_S][pB_0+(1-p)\Upsilon]+(1-p)\xi_AC_0)\\
&\quad+\left[n+\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right]n[B_s+C_s]\\
&\quad+\left[n+\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right]\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}[B_0+C_0-(1-p)[k\zeta_S+\xi_A]-p[\zeta_S+\xi_I]]\\
&= \alpha_0+P\left(\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right)>0,
\end{aligned}
$$

where

$$
\begin{aligned}
P(X) &= \big(B_0+C_0-(1-p)[k\zeta_S+\xi_A]-p[\zeta_S+\xi_I]\big)X^2\\
&\quad+\big(B_0(p[\xi_I-\zeta_S]+2n)+C_0((1-p)\xi_A+2n)\\
&\quad+(1-p)([\xi_I-\zeta_S]\Upsilon-n[2k\zeta_S+\xi_A])-np[2\zeta_S+\xi_I]\big)X\\
&\quad+n^2[B_s+C_s],
\end{aligned}
$$

and

$$
\begin{aligned}
\alpha_2\alpha_1 &= [B_s+C_s]\alpha_1+\left[n+\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right]a_1\\
&= [B_s+C_s]\alpha_1+\alpha_0+P\left(\frac{\zeta_V\mathcal{C}_{S_0}}{\mathcal{H}}\right)\geq\alpha_0.
\end{aligned}
$$

Thus, $\alpha_i>0$, $i=0,1,2$ and $\alpha_2\alpha_1>\alpha_0$. Based on the Routh–Hurwitz criterion, all the roots of the characteristic equation (3.6) have negative real parts. As a result, the coronavirus-free equilibrium point $\mathcal{E}_0$ is locally asymptotically stable under the condition (3.5). ∎

THEOREM 3.3. *If $\mathcal{R}_0 > 1$, then the coronavirus endemic equilibrium $\mathcal{E}^*$ of the system* (2.1) *is locally asymptotically stable if*

$$\beta_3\beta_2 > \beta_1, \quad \beta_1(\beta_3\beta_2 - \beta_1) > \beta_3^2\beta_0, \quad \beta_i > 0, \quad i = 0, \ldots, 3. \tag{3.7}$$

The coefficients β_0, β_1, β_2, and β_3 are defined in the proof.

Proof. The Jacobian matrix $J(\mathcal{E}^*)$ of system (2.1) at the coronavirus endemic equilibrium $\mathcal{E}^*$ is given by

$$\begin{pmatrix} -J_{11}^* & -J_{12}^* & -J_{13}^* & 0 & -J_{15}^* \\ (1-p)[J_{11}^* - m] & (1-p)J_{12}^* - B_0 & (1-p)J_{13}^* & 0 & (1-p)J_{15}^* \\ p[J_{11}^* - m] & pJ_{12}^* + \Upsilon & pJ_{13}^* - C_0 & 0 & pJ_{15}^* \\ 0 & \epsilon_A & \epsilon_I & -m & 0 \\ -J_{51}^* & \xi_A - J_{51}^* & \xi_I - J_{51}^* & -J_{51}^* & -\left(n + \frac{\mathcal{P}}{\mathcal{C}_S} J_{15}^*\right) \end{pmatrix}$$

with

$$\begin{aligned} J_{11}^* &= \zeta_S(\mathcal{C}_I^* + k\mathcal{C}_A^*)\frac{\mathcal{C}_A^* + \mathcal{C}_I^* + \mathcal{C}_R^*}{\mathcal{P}^{*2}} + \zeta_V \frac{\mathcal{C}_V^*}{\mathcal{H} + \mathcal{C}_V^*} + m, \\ J_{12}^* &= \mathcal{C}_S^*\zeta_S \frac{k(\mathcal{C}_S^* + \mathcal{C}_I^* + \mathcal{C}_R^*) - \mathcal{C}_I^*}{\mathcal{P}^{*2}}, \\ J_{13}^* &= \mathcal{C}_S^*\zeta_S \frac{(\mathcal{C}_S^* + \mathcal{C}_A^* + \mathcal{C}_R^*) - k\mathcal{C}_A^*}{\mathcal{P}^{*2}}, \\ J_{15}^* &= \zeta_V \mathcal{C}_S^* \frac{\mathcal{H}}{(\mathcal{H} + \mathcal{C}_V^*)^2}, \\ J_{51}^* &= \zeta_V \frac{\mathcal{C}_V^*}{\mathcal{H} + \mathcal{C}_V^*}. \end{aligned}$$

One eigenvalue, $-m$, satisfies $-m < 0$. The remaining eigenvalues are the roots of the quartic characteristic equation given by

$$\zeta_V^4 + \beta_3\zeta_V^3 + \beta_2\zeta_V^2 + \beta_1\zeta_V + \beta_0 = 0, \tag{3.8}$$

where

$$\begin{aligned} \beta_3 &= B_0 + k_2 - (1-p)J_{12}^*, \\ \beta_2 &= k_2B_0 + k_1 - (1-p)(mJ_{12}^* + l_1), \\ \beta_1 &= J_{15}^*J_{51}^*[(1-p)\epsilon_A + p\epsilon_I] - [(1-p)(ml_1 + l_0) - k_1B_0 - k_0], \\ \beta_0 &= J_{15}^*J_{51}^*[(1-p)C_0\epsilon_A + \epsilon_I[pB_0 + (1-p)\Upsilon]] - [(1-p)ml_0 - k_0B_0], \end{aligned}$$

and

$$\begin{aligned} k_2 &= J_{11}^* - pJ_{13}^* + \frac{N}{S}J_{15}^* + n + C_0, \\ k_1 &= J_{11}^*\left(n + \frac{N}{S}J_{15}^*\right) - J_{15}^*J_{51}^* + C_0\left(J_{11}^* + \left(n + \frac{N}{S}J_{15}^*\right)\right) \\ &\quad - mpJ_{13}^* - p\left[J_{13}^*\left(n + \frac{N}{S}J_{15}^*\right) + J_{15}^*(\xi_I - J_{15}^*)\right], \end{aligned}$$

$$k_0 = C_0\left[J_{11}^*\left(n+\frac{N}{S}J_{15}^*\right)-J_{15}^*J_{51}^*\right]-pm\left[J_{13}^*\left(n+\frac{N}{S}J_{15}^*\right)+J_{15}^*(\xi_I-J_{15}^*)\right],$$

$$l_1 = J_{13}^*\Upsilon+C_0J_{12}^*+J_{12}^*\left(n+\frac{N}{S}J_{15}^*\right)+J_{15}^*(\xi_A-J_{51}^*),$$

$$l_0 = \Upsilon\left[J_{13}^*\left(n+\frac{N}{S}J_{15}^*\right)+J_{15}^*(\xi_I-J_{51}^*)\right] + C_0\left[J_{12}^*\left(n+\frac{N}{S}J_{15}^*\right)+J_{15}^*(\xi_A-J_{51}^*)\right].$$

According to the Routh–Hurwitz criterion, stability is achieved if the conditions (3.7) hold. ∎

3.3.3. *Global stability.* In this section, we will demonstrate the global stability of $\mathcal{E}_0$ for System (2.1) utilizing LaSalle's invariance principle [15, 16]. Furthermore, we will establish the global stability of the endemic equilibrium $\mathcal{E}^*$ through the application of Lyapunov's direct method [18, 17].

Global stability of disease-free equilibrium

THEOREM 3.4. *If the coronavirus-free equilibrium $\mathcal{E}_0$ of model (2.1) is locally asymptotically stable, then the equation $\mathcal{E}_0$ is globally asymptotically stable if*

$$\zeta_S \leq \min\left\{\frac{m+\delta_A-\xi_A}{k}, m+\delta_I-\xi_I\right\}. \tag{3.9}$$

Proof. Let us consider the following Lyapunov–LaSalle function:

$$\mathcal{Z} = \mathcal{C}_I+\mathcal{C}_A+\mathcal{C}_R+\mathcal{C}_V. \tag{3.10}$$

It is evident that $\mathcal{Z} \geq 0$. Furthermore, $\mathcal{Z} = 0$ if and only if $\mathcal{C}_A = 0$, $\mathcal{C}_I = 0$, $\mathcal{C}_R = 0$, and $\mathcal{C}_V = 0$. Moreover, upon evaluating the time derivative of $\mathcal{Z}$ along the positive solutions of (2.1), we get

$$\frac{d\mathcal{Z}}{dt} = \zeta_S\frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I+k\mathcal{C}_A)-(m+\delta_A)\mathcal{C}_A-(m+\delta_I)\mathcal{C}_I-m\mathcal{C}_R+\xi_I\mathcal{C}_I + \xi_A\mathcal{C}_A-n\mathcal{C}_V-\zeta_V[\mathcal{C}_I+\mathcal{C}_A+\mathcal{C}_R]\frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V}.$$

Since $\mathcal{E}_0 \in \Sigma$, we get $\mathcal{C}_S \leq \mathcal{P} \leq \Pi/m$. And we have

$$\frac{d\mathcal{Z}}{dt} \leq -(m+\delta_A-k\zeta_S-\xi_A)\mathcal{C}_A-(m+\delta_I-\zeta_S-\xi_I)\mathcal{C}_I-m\mathcal{C}_R - n\mathcal{C}_V-\zeta_V[\mathcal{C}_I+\mathcal{C}_A+\mathcal{C}_R]\frac{\mathcal{C}_V}{\mathcal{H}+\mathcal{C}_V} \leq 0.$$

The final negativity result follows from the assumption (3.9). Therefore, the largest invariant set contained in the set Σ where $d\mathcal{Z}/dt = 0$ is reduced to $\mathcal{E}_0$. Since we are dealing with a compact positively invariant set, we can

apply LaSalle's invariance principle, which implies that $\mathcal{E}_0$ is globally asymptotically stable within Σ. ∎

Global stability of endemic equilibrium. We will now establish the global stability of $\mathcal{E}^*$ by constructing a Lyapunov function and using Lyapunov's direct method. We introduce the following assumptions:

(**H1**) $\epsilon_I(2m+\delta_A) = \epsilon_A(2m+\delta_I)$ and $2(m-\epsilon_I)+\delta_I \geq 0$.

(**H2**) Let $c_1 \geq \max\{c_{H1}, c_{H2}, x\}$, where

$$c_{H1} = \frac{(1-p)[9(2m+\delta_A)^2 - 4m(m+\delta_A)]}{4B_0},$$
$$c_{H2} = \frac{p[9(2m+\delta_I)^2 - 4m(m+\delta_I)]}{4C_0},$$

and x is the positive root of the equation

$$\left[\frac{4C_0B_0}{p(1-p)} - 9\frac{\Upsilon^2}{p^2}\right]x^2 + \left[\frac{4C_0(m+\delta_A)}{p} + \frac{4B_0(m+\delta_I)}{1-p} - 9\frac{\Upsilon}{p}\right]x - 9[(m+\delta_I)^2 + (m+\delta_A)^2] - 14(m+\delta_I)(m+\delta_A)] = 0.$$

(**H3**) Let $\mathcal{E}^*$ be an endemic equilibrium of (2.1) and define the functions

$$\alpha_{\mathcal{C}_I^*}(u,y,v) = \frac{\mathcal{G}(u,v) - \mathcal{G}(\mathcal{P}^*, \mathcal{C}_V^*)}{y - \mathcal{C}_I^*},$$
$$\alpha_{\mathcal{C}_A^*}(u,z,v) = \frac{\mathcal{G}(u,v) - \mathcal{G}(\mathcal{P}^*, \mathcal{C}_V^*)}{z - \mathcal{C}_A^*},$$
$$\phi_{\mathcal{C}_V^*}(u,x,y,z,v) = \frac{F(u,x,y,z,v) - F(\mathcal{P}^*, \mathcal{C}_S^*, \mathcal{C}_I^*, \mathcal{C}_A^*, \mathcal{C}_V^*)}{v - \mathcal{C}_V^*},$$

for all $y \neq \mathcal{C}_{I^*}$, $z \neq \mathcal{C}_{A^*}$ and $0 \leq u,x,y,z,v \leq \Pi/m$, where

$$F(u,x,y,z,v) = \zeta_S\frac{x}{u}(y+kz) - \zeta_V x\frac{v}{\mathcal{H}+v}, \quad \mathcal{G}(u,v) = -u\frac{v}{\mathcal{H}+v}.$$

Then there exists a positive constant c_3 such that

(3.11)
$$\frac{2nc_3}{3}\left(m+\delta_I+\frac{c_1C_0}{p}\right) \leq \left[\frac{c_3\zeta_V}{2}\alpha_{\mathcal{C}_{I^*}}(u,w,v) - c_1\phi_{\mathcal{C}_{V^*}}(u,w,v) - c_3\xi_I\right]^2,$$

(3.12)
$$\frac{2nc_3}{3}\left(m+\delta_A+\frac{c_1B_0}{1-p}\right) \leq \left[\frac{c_3\zeta_V}{2}\alpha_{\mathcal{C}_{A^*}}(u,w,v) - c_1\phi_{\mathcal{C}_{V^*}}(u,w,v) - c_3\xi_A\right]^2,$$

THEOREM 3.5. *Under assumptions* (**H1**)–(**H3**)*, the endemic equilibrium* $\mathcal{E}^*$ *of system* (2.1) *is globally asymptotically stable in* Σ.

Proof. Consider the function

$$P = \frac{1}{2}[\mathcal{C}_S - \mathcal{C}_{S^*} + \mathcal{C}_I - \mathcal{C}_{I^*} + \mathcal{C}_A - \mathcal{C}_{A^*} + \mathcal{C}_R - \mathcal{C}_{R^*}]^2 + \frac{c_1}{2p}[\mathcal{C}_I - \mathcal{C}_{I^*}]^2 + \frac{c_1}{2(1-p)}[\mathcal{C}_A - \mathcal{C}_{A^*}]^2 + \frac{c_2}{2}[\mathcal{C}_R - \mathcal{C}_{R^*}]^2 + \frac{c_3}{2}[\mathcal{C}_V - \mathcal{C}_{V^*}]^2,$$

where c_1, c_3 are the positive constants defined in (**H2**), (**H3**), and $c_2 > 0$ will be determined later.

It is clear that $P \geq 0$, and $P = 0$ if and only if $\mathcal{C}_S = \mathcal{C}_{S^*}$, $\mathcal{C}_I = \mathcal{C}_{I^*}$, $\mathcal{C}_A = \mathcal{C}_{A^*}$, $\mathcal{C}_R = \mathcal{C}_{R^*}$. Therefore, P is a Lyapunov function.

Now, we can rewrite (2.1) as

(3.13)

$$\begin{aligned}
\frac{d\mathcal{C}_S}{dt} &= -m(\mathcal{C}_S - \mathcal{C}_{S^*}) - [F(\mathcal{P}, \mathcal{C}_S, \mathcal{C}_I, \mathcal{C}_A, \mathcal{C}_V) - F(\mathcal{P}^*, \mathcal{C}_{S^*}, \mathcal{C}_{I^*}, \mathcal{C}_{A^*}, \mathcal{C}_{V^*})],\\
\frac{d\mathcal{C}_A}{dt} &= (1-p)[F(\mathcal{P}, \mathcal{C}_S, \mathcal{C}_I, \mathcal{C}_A, \mathcal{C}_V) - F(\mathcal{P}^*, \mathcal{C}_{S^*}, \mathcal{C}_{I^*}, \mathcal{C}_{A^*}, \mathcal{C}_{V^*})]\\
&\quad - (\epsilon_A + m + \delta_A + \Upsilon)(\mathcal{C}_A - \mathcal{C}_{A^*}),\\
\frac{d\mathcal{C}_I}{dt} &= p[F(\mathcal{P}, \mathcal{C}_S, \mathcal{C}_I, \mathcal{C}_A, \mathcal{C}_V) - F(\mathcal{P}^*, \mathcal{C}_{S^*}, \mathcal{C}_{I^*}, \mathcal{C}_{A^*}, \mathcal{C}_{V^*})]\\
&\quad - (\epsilon_I + m + \delta_I)(\mathcal{C}_I - \mathcal{C}_{I^*}) + \Upsilon(\mathcal{C}_A - \mathcal{C}_{A^*}),\\
\frac{d\mathcal{C}_R}{dt} &= \epsilon_I(\mathcal{C}_I - \mathcal{C}_{I^*}) + \epsilon_A(\mathcal{C}_A - \mathcal{C}_{A^*}) - m(\mathcal{C}_R - \mathcal{C}_{R^*}),\\
\frac{d\mathcal{C}_V}{dt} &= \xi_I(\mathcal{C}_I - \mathcal{C}_{I^*}) + \xi_A(\mathcal{C}_A - \mathcal{C}_{A^*}) - n(\mathcal{C}_V - \mathcal{C}_{V^*}) - \zeta_V[\mathcal{G}(\mathcal{P}, \mathcal{C}_V) - \mathcal{G}(\mathcal{P}^*, \mathcal{C}_{V^*})],
\end{aligned}$$

with

$$F(\mathcal{P}, \mathcal{C}_S, \mathcal{C}_I, \mathcal{C}_A, \mathcal{C}_V) = \zeta_S \frac{\mathcal{C}_S}{\mathcal{P}}(\mathcal{C}_I + k\mathcal{C}_A) - \zeta_V \mathcal{C}_S \frac{\mathcal{C}_V}{\mathcal{H} + \mathcal{C}_V},$$

$$\mathcal{G}(\mathcal{P}, \mathcal{C}_V) = -\mathcal{P}\frac{\mathcal{C}_V}{\mathcal{H} + \mathcal{C}_V}.$$

Calculating the time derivative of P along the positive solutions of (3.13), we have

$$\begin{aligned}
\frac{dP}{dt} &= [\mathcal{C}_S - \mathcal{C}_{S^*} + \mathcal{C}_I - \mathcal{C}_{I^*} + \mathcal{C}_A - \mathcal{C}_{A^*} + \mathcal{C}_R - \mathcal{C}_{R^*}]\frac{d\mathcal{P}}{dt} + \frac{c_1}{p}[\mathcal{C}_I - \mathcal{C}_{I^*}]\frac{d\mathcal{C}_I}{dt}\\
&\quad + \frac{c_1}{1-p}[\mathcal{C}_A - \mathcal{C}_{A^*}]\frac{d\mathcal{C}_A}{dt} + c_2[\mathcal{C}_R - \mathcal{C}_{R^*}]\frac{d\mathcal{C}_R}{dt} + c_3[\mathcal{C}_V - \mathcal{C}_{V^*}]\frac{d\mathcal{C}_V}{dt}\\
&= [\mathcal{C}_S - \mathcal{C}_{S^*} + \mathcal{C}_I - \mathcal{C}_{I^*} + \mathcal{C}_A - \mathcal{C}_{A^*} + \mathcal{C}_R - \mathcal{C}_{R^*}][-m(\mathcal{C}_S - \mathcal{C}_{S^*})\\
&\quad - (m + \delta_I)(\mathcal{C}_I - \mathcal{C}_{I^*}) - (m + \delta_A)(\mathcal{C}_A - \mathcal{C}_{A^*}) - m(\mathcal{C}_R - \mathcal{C}_{R^*})]\\
&\quad + \frac{c_1}{p}[\mathcal{C}_I - \mathcal{C}_{I^*}][p[F(\mathcal{P}, \mathcal{C}_S, \mathcal{C}_I, \mathcal{C}_A, \mathcal{C}_V) - F(\mathcal{P}^*, \mathcal{C}_S^*, \mathcal{C}_{I^*}, \mathcal{C}_{A^*}, \mathcal{C}_{V^*})]\\
&\quad - (\epsilon_I + m + \delta_I)(\mathcal{C}_I - \mathcal{C}_{I^*}) + \Upsilon(\mathcal{C}_A - \mathcal{C}_{A^*})]
\end{aligned}$$

$$
\begin{aligned}
&+ \frac{c_1}{(1-p)}[\mathcal{C}_A - \mathcal{C}_{A^*}][(1-p)[F(\mathcal{P}, \mathcal{C}_S, \ldots) - F(\mathcal{P}^*, \mathcal{C}_{S^*}, \ldots)] \\
&- (\epsilon_A + m + \delta_A + \Upsilon)(\mathcal{C}_A - \mathcal{C}_{A^*})] + c_2[\mathcal{C}_R - \mathcal{C}_{R^*}][\epsilon_I(\mathcal{C}_I - \mathcal{C}_{I^*}) \\
&+ \epsilon_A(\mathcal{C}_A - \mathcal{C}_{A^*}) - m(\mathcal{C}_R - \mathcal{C}_{R^*})] \\
&+ c_3[\mathcal{C}_V - \mathcal{C}_{V^*}][\xi_I(\mathcal{C}_I - \mathcal{C}_{I^*}) + \xi_A(\mathcal{C}_A - \mathcal{C}_{A^*}) - n(\mathcal{C}_V - \mathcal{C}_{V^*}) \\
&- \zeta_V[G(N,V) - G(N^*, V^*)]].
\end{aligned}
$$

So

$$
\begin{aligned}
\frac{dP}{dt} = &-m(\mathcal{C}_S - \mathcal{C}_{S^*})^2 - m(1+c_2)(\mathcal{C}_R - \mathcal{C}_R^*)^2 - nc_3(\mathcal{C}_V - \mathcal{C}_{V^*})^2 \\
&- \left(m + \delta_I + \frac{c_1(\epsilon_I + m + \delta_I)}{p}\right)(\mathcal{C}_I - \mathcal{C}_{I^*})^2 \\
&- (2m + \delta_I)(\mathcal{C}_S - \mathcal{C}_{S^*})(\mathcal{C}_I - \mathcal{C}_{I^*}) \\
&- \left(m + \delta_A + \frac{c_1(\epsilon_A + m + \delta_A + \Upsilon)}{1-p}\right)(\mathcal{C}_A - \mathcal{C}_{A^*})^2 \\
&- (2m + \delta_A)(\mathcal{C}_S - \mathcal{C}_{S^*})(\mathcal{C}_A - \mathcal{C}_{A^*}) \\
&- 2m(\mathcal{C}_S - \mathcal{C}_{S^*})(\mathcal{C}_R - \mathcal{C}_{R^*}) \\
&- \left(2m + \delta_I + \delta_A - \frac{c_1\Upsilon}{p}\right)(\mathcal{C}_I - \mathcal{C}_{I^*})(\mathcal{C}_A - \mathcal{C}_{A^*}) \\
&- (2m + \delta_I - c_2\epsilon_I)(\mathcal{C}_I - \mathcal{C}_{I^*})(\mathcal{C}_R - \mathcal{C}_{R^*}) \\
&- (2m + \delta_A - c_2\epsilon_A)(\mathcal{C}_A - \mathcal{C}_{A^*})(\mathcal{C}_R - \mathcal{C}_{R^*}) \\
&+ \xi_I c_3(\mathcal{C}_V - \mathcal{C}_{V^*})(\mathcal{C}_I - \mathcal{C}_{I^*}) + \xi_A c_3(\mathcal{C}_V - \mathcal{C}_{V^*})(\mathcal{C}_A - \mathcal{C}_{A^*}) \\
&+ c_1(\mathcal{C}_I - \mathcal{C}_{I^*})[F(\mathcal{P}, \mathcal{C}_S, \mathcal{C}_I, \mathcal{C}_A, \mathcal{C}_V) \\
&- F(\mathcal{P}^*, \mathcal{C}_{S^*}, \mathcal{C}_{I^*}, \mathcal{C}_{A^*}, \mathcal{C}_{V^*})] \\
&+ c_1(\mathcal{C}_A - \mathcal{C}_{A^*})[F(\mathcal{P}, \mathcal{C}_S, \mathcal{C}_I, \mathcal{C}_A, \mathcal{C}_V) - F(\mathcal{P}^*, \mathcal{C}_{S^*}, \mathcal{C}_{I^*}, \mathcal{C}_{A^*}, \mathcal{C}_{V^*})] \\
&- c_3\zeta_V(\mathcal{C}_V - \mathcal{C}_{V^*})[\mathcal{G}(\mathcal{P}, V) - \mathcal{G}(\mathcal{P}^*, V^*)].
\end{aligned}
$$

We can take

$$
c_2 = \frac{2m + \delta_A}{\epsilon_A} = \frac{2m + \delta_I}{\epsilon_I},
$$

so that the terms in $(\mathcal{C}_I - \mathcal{C}_I^*)(\mathcal{C}_R - \mathcal{C}_R^*)$ and $(\mathcal{C}_A - \mathcal{C}_A^*)(\mathcal{C}_R - \mathcal{C}_R^*)$ cancel out. Thus,

$$
\begin{aligned}
\frac{dP}{dt} = &-\left\{\frac{m}{3}(\mathcal{C}_S - \mathcal{C}_S^*)^2 + (2m + \delta_I)(\mathcal{C}_S - \mathcal{C}_S^*)(\mathcal{C}_I - \mathcal{C}_I^*)\right\} \\
&- \left\{\frac{m + \delta_I + \frac{c_1(\epsilon_I + m + \delta_I)}{p}}{3}(\mathcal{C}_I - \mathcal{C}_I^*)^2\right\} \\
&- \left\{\frac{m}{3}(\mathcal{C}_S - \mathcal{C}_S^*)^2 + (2m + \delta_A)(\mathcal{C}_S - \mathcal{C}_S^*)(\mathcal{C}_A - \mathcal{C}_A^*)\right\}
\end{aligned}
$$

$$
\begin{aligned}
&- \left\{ \frac{m+\delta_A+\frac{c_1(\epsilon_A+m+\delta_A+\Upsilon)}{1-p}}{3}(\mathcal{C}_A-\mathcal{C}_A^*)^2 \right\} \\
&- \left\{ \frac{m}{3}(\mathcal{C}_S-\mathcal{C}_S^*)^2 + 2m(\mathcal{C}_S-\mathcal{C}_S^*)(\mathcal{C}_R-\mathcal{C}_R^*) + m(1+c_2)(\mathcal{C}_R-\mathcal{C}_R^*)^2 \right\} \\
&- \left\{ \frac{m+\delta_I+\frac{c_1(\epsilon_I+m+\delta_I)}{p}}{3}(\mathcal{C}_I-\mathcal{C}_I^*)^2 \right. \\
&+ \left(2m+\delta_I+\delta_A-\frac{c_1\Upsilon}{p} \right)(\mathcal{C}_I-\mathcal{C}_I^*)(\mathcal{C}_A-\mathcal{C}_A^*) \\
&\left. + \frac{m+\delta_A+\frac{c_1(\epsilon_A+m+\delta_A+\Upsilon)}{1-p}}{3}(\mathcal{C}_A-\mathcal{C}_A^*)^2 \right\} \\
&- \left\{ \frac{nc_3}{2}(\mathcal{C}_V-\mathcal{C}_V^*)^2 + \frac{m+\delta_I+\frac{c_1(\epsilon_I+m+\delta_I)}{p}}{3}(\mathcal{C}_I-\mathcal{C}_I^*)^2 \right. \\
&+ \left[\frac{c_3\zeta_V}{2}\,\frac{\mathcal{G}(\mathcal{P},\mathcal{C}_V)-\mathcal{G}(\mathcal{P}^*,\mathcal{C}_V^*)}{\mathcal{C}_I-\mathcal{C}_I^*} \right] \times (\mathcal{C}_V-\mathcal{C}_V^*)(\mathcal{C}_I-\mathcal{C}_I^*) \\
&+ \left[-c_1\frac{F(\mathcal{P},\mathcal{C}_S,\mathcal{C}_I,\mathcal{C}_A,\mathcal{C}_V)-F(\mathcal{P}^*,\mathcal{C}_S^*,\mathcal{C}_I^*,\mathcal{C}_A^*,\mathcal{C}_V^*)}{\mathcal{C}_V-\mathcal{C}_V^*} - c_3\xi_I \right] \\
&\left. \times (\mathcal{C}_V-\mathcal{C}_V^*)(\mathcal{C}_I-\mathcal{C}_I^*) \right\} \\
&- \left\{ \frac{nc_3}{2}(\mathcal{C}_V-\mathcal{C}_V^*)^2 + \frac{m+\delta_A+\frac{c_1(\epsilon_A+m+\delta_A+\Upsilon)}{1-p}}{3}(\mathcal{C}_A-\mathcal{C}_A^*)^2 \right. \\
&+ \left[\frac{c_3\zeta_V}{2}\,\frac{\mathcal{G}(\mathcal{P},\mathcal{C}_V)-\mathcal{G}(\mathcal{P}^*,\mathcal{C}_V^*)}{\mathcal{C}_A-\mathcal{C}_A^*} \right] \times (\mathcal{C}_V-\mathcal{C}_V^*)(\mathcal{C}_I-\mathcal{C}_I^*) \\
&+ \left[-c_1\frac{F(\mathcal{P},\mathcal{C}_S,\mathcal{C}_I,\mathcal{C}_A,\mathcal{C}_V)-F(\mathcal{P}^*,\mathcal{C}_S^*,\mathcal{C}_I^*,\mathcal{C}_A^*,\mathcal{C}_V^*)}{\mathcal{C}_V-\mathcal{C}_V^*} - c_3\xi_A \right] \\
&\left. \times (\mathcal{C}_V-\mathcal{C}_V^*)(\mathcal{C}_I-\mathcal{C}_I^*) \right\}.
\end{aligned}
$$

So

$$
\begin{aligned}
\frac{dP}{dt} = &-[\mathcal{C}_S-\mathcal{C}_S^*,\mathcal{C}_I-\mathcal{C}_I^*]Q_{\mathcal{C}_S\mathcal{C}_I}[\mathcal{C}_S-\mathcal{C}_S^*,\mathcal{C}_I-\mathcal{C}_I^*]^t \\
&- [\mathcal{C}_S-\mathcal{C}_S^*,\mathcal{C}_A-\mathcal{C}_A^*]Q_{\mathcal{C}_S\mathcal{C}_A}[\mathcal{C}_S-\mathcal{C}_S^*,\mathcal{C}_A-\mathcal{C}_A^*]^t \\
&- [\mathcal{C}_S-\mathcal{C}_S^*,\mathcal{C}_R-\mathcal{C}_R^*]Q_{\mathcal{C}_S\mathcal{C}_R}[\mathcal{C}_S-\mathcal{C}_S^*,\mathcal{C}_R-\mathcal{C}_R^*]^t \\
&- [\mathcal{C}_I-\mathcal{C}_I^*,\mathcal{C}_A-\mathcal{C}_A^*]Q_{\mathcal{C}_I\mathcal{C}_A}[\mathcal{C}_I-\mathcal{C}_I^*,\mathcal{C}_A-\mathcal{C}_A^*]^t \\
&- [\mathcal{C}_V-\mathcal{C}_V^*,\mathcal{C}_I-\mathcal{C}_I^*]Q_{\mathcal{C}_V\mathcal{C}_I}[\mathcal{C}_V-\mathcal{C}_V^*,I-\mathcal{C}_I^*]^t \\
&- [\mathcal{C}_V-\mathcal{C}_V^*,\mathcal{C}_A-\mathcal{C}_A^*]Q_{\mathcal{C}_V\mathcal{C}_A}[\mathcal{C}_V-\mathcal{C}_V^*,A-\mathcal{C}_A^*]^t,
\end{aligned}
$$

where

$$Q_{\mathcal{C}_S\mathcal{C}_I} = \begin{pmatrix} m/3 & (2m+\delta_I)/2 \\ (2m+\delta_I)/2 & \big(m+\delta_I+\frac{c_1(\epsilon_I+m+\delta_I)}{p}\big)/3 \end{pmatrix},$$

$$Q_{\mathcal{C}_S\mathcal{C}_A} = \begin{pmatrix} m/3 & (2m+\delta_A)/2 \\ (2m+\delta_A)/2 & \big(m+\delta_A+\frac{c_1(\epsilon_A+m+\delta_A+\Upsilon)}{1-p}\big)/3 \end{pmatrix},$$

$$Q_{\mathcal{C}_I\mathcal{C}_A} = \begin{pmatrix} (m+\delta_I)(p+c_1)+c_1\epsilon_I/3p & 2m+\delta_I+\delta_A-c_1\Upsilon/p/2 \\ 2m+\delta_I+\delta_A-c_1\Upsilon/p/2 & \big(m+\delta_A+\frac{c_1(\epsilon_A+m+\delta_A+\Upsilon)}{1-p}\big)/3 \end{pmatrix},$$

$$Q_{\mathcal{C}_S\mathcal{C}_R} = m\begin{pmatrix} 1/3 & 1 \\ 1 & (1+c_2) \end{pmatrix},$$

$$Q_{\mathcal{C}_V\mathcal{C}_I} = \begin{pmatrix} nc_3/2 & L_{\mathcal{C}_I}/2 \\ L_{\mathcal{C}_I}/2 & \big(m+\delta_I+\frac{c_1(\epsilon_I+m+\delta_I)}{p}\big)/3 \end{pmatrix},$$

$$Q_{\mathcal{C}_V\mathcal{C}_A} = \begin{pmatrix} nc_3/2 & L_{\mathcal{C}_A}/2 \\ L_{\mathcal{C}_A}/2 & \big(m+\delta_A+\frac{c_1(\epsilon_A+m+\delta_A+\Upsilon)}{1-p}\big)/3 \end{pmatrix}$$

with

$$\begin{aligned} L_{\mathcal{C}_I} &= \frac{c_3\zeta_V}{2}\,\frac{\mathcal{G}(\mathcal{P},\mathcal{C}_V)-\mathcal{G}(\mathcal{P}^*,\mathcal{C}_V^*)}{\mathcal{C}_I-\mathcal{C}_I^*} \\ &\quad - c_1\frac{F(\mathcal{P},\mathcal{C}_S,\mathcal{C}_I,\mathcal{C}_A,\mathcal{C}_V)-F(\mathcal{P}^*,\mathcal{C}_S^*,\mathcal{C}_I^*,\mathcal{C}_A^*,\mathcal{C}_V^*)}{\mathcal{C}_V-\mathcal{C}_V^*} - c_3\xi_I, \\ L_{\mathcal{C}_A} &= \frac{c_3\zeta_V}{2}\,\frac{\mathcal{G}(\mathcal{P},\mathcal{C}_V)-\mathcal{G}(\mathcal{P}^*,\mathcal{C}_V^*)}{\mathcal{C}_A-\mathcal{C}_A^*} \\ &\quad - c_1\frac{F(\mathcal{P},\mathcal{C}_S,\mathcal{C}_I,\mathcal{C}_A,\mathcal{C}_V)-F(\mathcal{P}^*,\mathcal{C}_S^*,\mathcal{C}_I^*,\mathcal{C}_A^*,\mathcal{C}_V^*)}{\mathcal{C}_V-\mathcal{C}_V^*} - c_3\xi_A. \end{aligned}$$

Under the assumptions (**H1**)–(**H3**), it follows that all leading principal minors of $Q_{\mathcal{C}_S\mathcal{C}_I}$, $Q_{\mathcal{C}_S\mathcal{C}_A}$, $Q_{\mathcal{C}_S\mathcal{C}_R}$, $Q_{\mathcal{C}_I\mathcal{C}_A}$, $Q_{\mathcal{C}_V\mathcal{C}_I}$, and $Q_{\mathcal{C}_V\mathcal{C}_A}$ are positive. Consequently, $Q_{\mathcal{C}_S\mathcal{C}_I}$, $Q_{\mathcal{C}_S\mathcal{C}_A}$, $Q_{\mathcal{C}_S\mathcal{C}_R}$, $Q_{\mathcal{C}_I\mathcal{C}_A}$, $Q_{\mathcal{C}_V\mathcal{C}_I}$, and $Q_{\mathcal{C}_V\mathcal{C}_A}$ are positive-definite matrices.

Let

$$a_{\mathcal{C}_{SI}} = [\mathcal{C}_S-\mathcal{C}_S^*, \mathcal{C}_I-\mathcal{C}_I^*],\ a_{\mathcal{C}_{SA}} = [\mathcal{C}_S-\mathcal{C}_S^*, \mathcal{C}_A-\mathcal{C}_A^*],\ a_{\mathcal{C}_{SR}} = [\mathcal{C}_S-\mathcal{C}_S^*, \mathcal{C}_R-\mathcal{C}_R^*],$$
$$a_{\mathcal{C}_{IA}} = [\mathcal{C}_I-\mathcal{C}_I^*, \mathcal{C}_A-\mathcal{C}_A^*],\ a_{\mathcal{C}_{VI}} = [\mathcal{C}_V-\mathcal{C}_V^*, \mathcal{C}_I-\mathcal{C}_I^*],\ a_{\mathcal{C}_{VA}} = [\mathcal{C}_V-\mathcal{C}_V^*, \mathcal{C}_A-\mathcal{C}_A^*].$$

Then

$$\begin{aligned} \frac{dP}{dt} &\le -a_{\mathcal{C}_{SI}}Q_{\mathcal{C}_S\mathcal{C}_I}a_{\mathcal{C}_{SI}}^t - a_{\mathcal{C}_{SA}}Q_{\mathcal{C}_S\mathcal{C}_A}a_{\mathcal{C}_{SA}}^t - a_{\mathcal{C}_{SR}}Q_{\mathcal{C}_S\mathcal{C}_R}a_{\mathcal{C}_{SR}}^t \\ &\quad - a_{\mathcal{C}_{IA}}Q_{\mathcal{C}_I\mathcal{C}_A}a_{\mathcal{C}_{IA}}^t - a_{\mathcal{C}_{VI}}Q_{\mathcal{C}_V\mathcal{C}_I}a_{\mathcal{C}_{VI}}^t - a_{\mathcal{C}_{VA}}Q_{\mathcal{C}_V\mathcal{C}_A}a_{\mathcal{C}_{VA}}^t \le 0. \end{aligned}$$

Suppose that $\frac{dP}{dt} = 0$ for some $(\mathcal{C}_S, \mathcal{C}_A, \mathcal{C}_I, \mathcal{C}_R, \mathcal{C}_V) \in \Sigma$. From the positive-definiteness of $Q_{\mathcal{C}_S\mathcal{C}_I}$, $Q_{\mathcal{C}_S\mathcal{C}_A}$, $Q_{\mathcal{C}_S\mathcal{C}_R}$, $Q_{\mathcal{C}_I\mathcal{C}_A}$, $Q_{\mathcal{C}_V\mathcal{C}_I}$, and $Q_{\mathcal{C}_V\mathcal{C}_A}$, it follows that $a_{\mathcal{C}_{SI}} = a_{\mathcal{C}_{SA}} = a_{\mathcal{C}_{SR}} = a_{\mathcal{C}_{IA}} = a_{\mathcal{C}_{VI}} = a_{\mathcal{C}_{VA}} = 0$.

Consequently, we have

$$\begin{aligned}[\mathcal{C}_S - \mathcal{C}_S^*, \mathcal{C}_I - \mathcal{C}_I^*] &= [\mathcal{C}_S - \mathcal{C}_S^*, \mathcal{C}_A - \mathcal{C}_A^*] = [\mathcal{C}_S - \mathcal{C}_S^*, \mathcal{C}_R - \mathcal{C}_R^*] \\ &= [\mathcal{C}_I - \mathcal{C}_I^*, \mathcal{C}_A - \mathcal{C}_A^*] = [\mathcal{C}_V - \mathcal{C}_V^*, \mathcal{C}_I - \mathcal{C}_I^*] \\ &= [\mathcal{C}_V - \mathcal{C}_V^*, \mathcal{C}_A - \mathcal{C}_A^*] = 0,\end{aligned}$$

so $(\mathcal{C}_S, \mathcal{C}_A, \mathcal{C}_I, \mathcal{C}_R, \mathcal{C}_V) = \mathcal{E}^*$. Therefore, by applying Lyapunov's direct method [18, 17], we can conclude that the endemic equilibrium $\mathcal{E}^*$ is globally asymptotically stable. ∎

4. Discussion and simulation

4.1. Simulation methodology and data acquisition. We ran some computer simulations using the parameter values provided in Table 2. Although the simulations may not accurately represent real world scenarios, our main objective is to study their general characteristics and behavior. To conduct these simulations, we utilized a custom-built driver code that calls the ode45 routine in Matlab. This routine implements the well known Runge–Kutta 45 integration method for solving ordinary differential equations. We gathered data on the spread of the epidemic from official websites,

Table 2. Parameter values

Parameter	Value	Parameter	Value
Π	5000	m	0.044
δ_A	0.0009	δ_I	0.08
ϵ_A	0.06	ϵ_I	0.05
ζ_S	0.09	ζ_V	0.01
ξ_I	1/3	k	$0.1 \in [0.005, 0.2]$
ξ_A	1/18	Υ	$0.15 \in [0.01, 0.3]$
p	0.6	n	0.05
$\mathcal{H}$	100		

and we supplemented it with additional information from [4]. We used days as the fundamental unit of time, and we introduced a correction factor, denoted by k, to account for the transmission of the disease by asymptomatic individuals. The value of k falls within the range of $[0.005, 0.2]$.

Since our model does not consider age structure, we assumed a fixed mortality rate for the disease to be 0.08. We set the final mortality rate denoted by m, which provides a reasonable estimation of the overall losses, in approximate agreement with the recorded data. However, mortality among

asymptomatic individuals is not completely ignored and is assumed to be 0.0009.

By analyzing the published data from [4, 29], we estimated the parameter values listed in Table 2. Additionally, we adjusted the susceptible recruitment rate, denoted by Π, to ensure that the total population, denoted by $\mathcal{P}$, remains relatively stable throughout the simulation period.

4.2. Discussion. The proposed epidemic model for COVID-19, which includes a virus compartment, has been thoroughly analyzed. The model takes into consideration the contribution of asymptomatic individuals and the presence of viruses in the environment. By implementing effective quarantine measures, it is possible to observe a decline and eventual elimination of the disease.

The results of the analysis indicate that the model is biologically plausible and valid. The trajectories of the subpopulations are confined to a compact set, and the subpopulations themselves remain bounded. The model exhibits two equilibrium points: the endemic equilibrium $\mathcal{E}^*$ where the disease persists, and the disease-free equilibrium $\mathcal{E}_0$ where the pandemic is ultimately eradicated. The disease-free equilibrium $\mathcal{E}_0$ should be the objective of policies aimed at containing and preventing the spread of the epidemic.

In Section 3.3.3, the global stability of each equilibrium under appropriate conditions is demonstrated. Figures 2 and 3 illustrate the theoretical types of possible model dynamics. It is important to note that the purpose of these results is not to directly compare them with publicly available COVID-19 datasets.

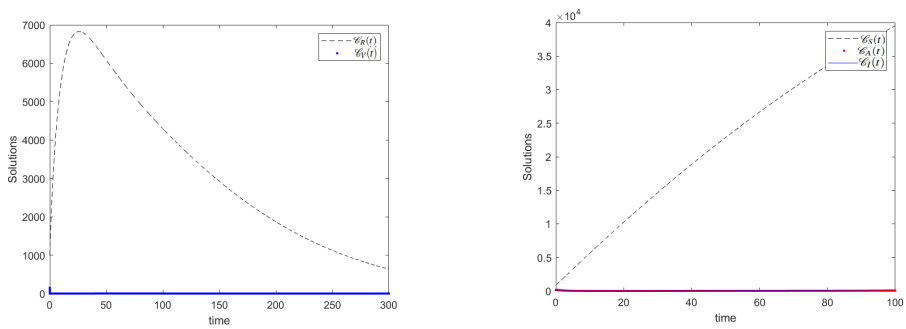

Fig. 2. Left: Susceptible individuals $\mathcal{C}_S$ reach the level of 39000; infected $\mathcal{C}_I$ and asymptomatic $\mathcal{C}_A$ individuals are reduced to the single digits, essentially vanishing, Right: Recovered and infected individuals remain confined respectively around the levels 100 and 05. In this situation the system approaches the disease-free equilibrium $\mathcal{E}_0$ at $\mathcal{R}_0 = 0.36$.

The simulations' goals are to learn more about the models dynamics and investigate the implications of changing the value of $\mathcal{R}_0$. Utilizing MATLAB

2019, the simulation process has a time range of 0 to 600 steps. We thoroughly investigated how various $\mathcal{R}_0$ values affect the dynamics of the model through the executed numerical simulations. In particular, for $\mathcal{R}_0 < 1$, we examined the behavior of each class within the system (2.1).

The ensuing Figures 2 depict these dynamics. We can see an increase in the $\mathcal{C}_R$ and $\mathcal{C}_S$, with a corresponding decrease in the rest of the compartments. Moreover, the paths of the systems approach $\mathcal{E}_0$ over time.

Figures 3 show the dynamics of the model. For $\mathcal{R}_0 > 1$, all compartments initially increase except for the $\mathcal{C}_V$ compartment, which exhibits a rapid decline before starting to increase again. Consequently, the system trajectories eventually approach the endemic equilibrium $\mathcal{E}^*$ over time.

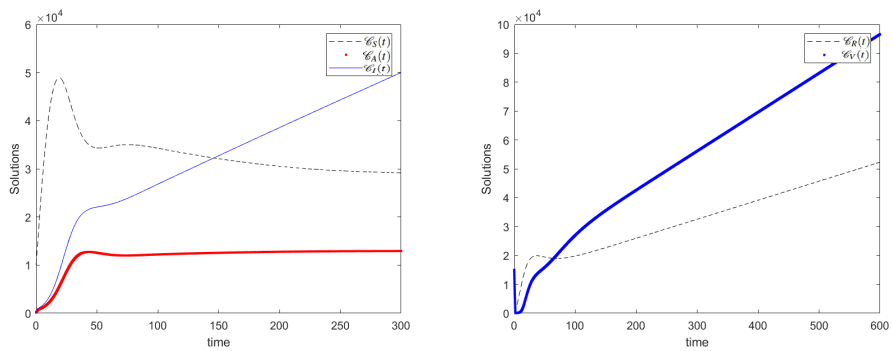

Fig. 3. The system approaches the endemic equilibrium $\mathcal{E}^* = (29210, 12920, 49690, 52180, 96380)$ at $\mathcal{R}_0 = 1.6$.

Acknowledgements. The author would like to thank the reviewers and the editor for their constructive comments and valuable suggestions that improved the quality of our paper.

References

[1] V. Alfano and S. Ercolano, *The efficacy of lockdown against COVID-19: A cross-country panel analysis*, Appl. Health Econ. Health Policy 18 (2020), 509–517.

[2] S. M. Ali Shah, H. Tahir, A. Khan, W. A. Khan and A. Arshad, *Stochastic model on the transmission of worms in wireless sensor network*, J. Math. Techniques in Modeling 1 (2024), 75–88.

[3] Y. Belgaid, M. Helal, A. Lakmeche and E. Venturino, *A mathematical study of a coronavirus model with the Caputo fractional-order derivative*, Fractal and Fractional 5 (2021), art. 87, 23 pp.

[4] Y. Belgaid, M. Helal and E. Venturino, *Analysis of a model for coronavirus spread*, Mathematics 8 (2020), art. 820, 30 pp.

[5] E. Beretta and Y. Kuang, *Modeling and analysis of a marine bacteriophage infection*, Math. Biosci. 149 (1998), 57–76.

[6] T. M. Chen, J. Rui, Q. P. Wang, Z. Y. Zhao, J. A. Cui and L. Ayin, *Mathematical model for simulating the transmission of Wuhan novel coronavirus*, Infectious Diseases of Poverty 9 (2020), art. 24, 8 pp.

[7] E. A. Coddington, *An Introduction to Ordinary Differential Equations*, Dover Publ., New York, 1961.

[8] O. Diekmann, J. A. P. Heesterbeek and J. A. J. Metz, *On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations*, J. Math. Biol. 28 (1990), 365–382.

[9] D. W. Eyre et al., *Effect of Covid-19 vaccination on transmission of alpha and delta variants*, New England J. Medicine 386 (2022), 744–756.

[10] A. Herbert and W. Hethcote, *The mathematics of infectious diseases*, SIAM Rev. 42 (2000), 599–653.

[11] L. Hongfan, D. Yuting, G. Silin and W. Shishi, *Mathematical modeling and dynamic analysis of SIQR model with delay for pandemic COVID-19*, Math. Biosci. Engrg. 18 (2021), 3197–3214.

[12] S. Kashte, A. Gulbake, S. F. El-Amin III and A. Gupta, *COVID-19 vaccines: rapid development, implications, challenges and future prospects*, Human Cell 34 (2021), 711–733.

[13] F. M. Khan and Z. U. Khan, *Numerical analysis of fractional order drinking mathematical model*, J. Math. Techniques in Modeling 1 (2024), 11–24.

[14] W. A. Khan, R. Zarin, Z. Aurang, Y. Khan and A. Khan, *Navigating food allergy dynamics via a novel fractional mathematical model for antacid-induced allergies*, J. Math. Techniques in Modeling 1 (2024), 25–51.

[15] J. P. LaSalle, *Stability of nonautonomous systems*, Nonlinear Anal. 1 (1976), 83–91.

[16] J. P. LaSalle, *Stability theory for ordinary differential equations*, J. Differential Equations 41 (1968), 57–65.

[17] J. P. LaSalle, *The Stability of Dynamical Systems*, SIAM, Philadelphia, PA, 1976.

[18] J. P. LaSalle and S. Lefschetz, *Stability by Liapunov's Direct Method with Applications*, Academic Press, New York, 1961.

[19] E. Miguel and A. M. Mobarak, *The economics of the COVID-19 pandemic in poor countries*, Ann. Rev. Economics 14 (2022), 253–285.

[20] W. Msemburi, A. Karlinsky, V. Knutson, S. Aleshin-Guendel, S. Chatterji and J. Wakefield, *The WHO estimates of excess mortality associated with the COVID-19 pandemic*, Nature 613 (2023), 130–137.

[21] T. Oraby et al., *Modeling the effect of lockdown timing as a COVID-19 control measure in countries with differing social contacts*, Sci. Rep. 11 (2021), art. 3354, 13 pp.

[22] M. B. Ortigoza et al., *Efficacy and safety of COVID-19 convalescent plasma in hospitalized patients: a randomized clinical trial*, JAMA Internal Medicine 182 (2022), 115–126.

[23] A. Qura tul, *Nonlinear stochastic cholera epidemic model under the influence of noise*, J. Math. Techniques in Modeling 1 (2024), 52–74.

[24] M. A. Rois, T. Trisilowati and U. Habibah, *Dynamic analysis of covid-19 model with quarantine and isolation*, JTAM (Jurnal Teori dan Aplikasi Matematika) 5 (2021), 418–433.

[25] C. N. S. Romero et al., *Clinical signs and symptoms associated with COVID-19: a cross sectional study*, Int. J. Odontostomat. 16 (2022), 112–119.

[26] S. Sharma and M. Banerjee, *Challenges and innovations in mathematical modelling of COVID-19*, in: COVID-19 and SARS-Cov-2: The Science and Clinical Application of Conventional and Complementary Treatments, Chapter 6, CRC Press, 2022.

[27] P. Van den Driessche, *Reproduction numbers of infectious disease models*, Infectious Disease Modelling 2 (2007), 288–303.
[28] P. Van den Driessche and J. A. Watmough, *Simple SIS epidemic model with a backward bifurcation*, J. Math. Biol. 40 (2000), 525–540.
[29] R. Verity et al., *Estimates of the severity of coronavirus disease 2019: A model-based analysis*, Lancet Infectious Diseases 20 (2020), 669–677.
[30] T. A. Vo, M. Mazur and A. Thai, *The impact of COVID-19 economic crisis on the speed of adjustment toward target leverage ratio: An international analysis*, Finance Res. Lett. 45 (2022), art. 102157, 8 pp.
[31] V. Volpert, M. Banerjee and S. Sharma, *Epidemic progression and vaccination in a heterogeneous population, application to the covid-19 epidemic*, Ecological Complexity 47 (2021), art. 100940, 12 pp.
[32] A. Wilder-Smith, *COVID-19 in comparison with other emerging viral diseases: risk of geographic spread via travel*, Trop. Dis. Travel Med. Vaccines 7 (2021), art. 3, 11 pp.
[33] N. Yoshida, *Exact solution of the Susceptible-Infectious-Recovered-Deceased (SIRD) epidemic model*, Electron. J. Qual. Theory Differential Equations 2022, art. 38, 24 pp.
[34] N. Yoshida, *Exact solution of the Susceptible-Exposed-Infectious-Recovered-Deceased (SEIRD) epidemic model*, Electron. J. Qual. Theory Differential Equations 2024, art. 8, 37 pp.
[35] H. M. Youssef, N. A. Alghamdi, M. A. Ezzat, A. A. El-Bary and A. M. Shawky, *A new dynamical modeling SEIR with global analysis applied to the real data of spreading COVID-19 in Saudi Arabia*, Math. Biosci. Engrg. 17 (2020), 7018–7044.

Youcef Belgaid
Department of Common Core in Exact Sciences and Informatics
Hassiba Benbouali University
Chlef, 02000, Algeria
and
Biomathematics Laboratory
University Sidi Bel-Abbes
Sidi Bel-Abbes, 22000, Algeria
E-mail: y.belgaid@univ-chlef.dz