Analyzing the Spread of COVID-19: A Mathematical Study on Transmission Dynamics

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Abstract - In this study, a dynamic mathematical model for COVID-19 was described. The analysis focuses on proving the existence of a disease-free equilibrium, which is locally asymptotically stable when the basic reproduction number is less than unity. Additionally, we determine the existence of an endemic equilibrium, providing insights into the long-term persistence of the disease. To further understand the behavior of the model, we delve into the global asymptotic stability of the disease-free equilibrium. This analysis involves the construction of a Lyapunov function, enabling us to assess the overall stability of the system through our comprehensive mathematical analysis, we aim to enhance our understanding of COVID-19 transmission dynamics, while offering a foundation for the development of effective strategies to mitigate the spread of the disease.

Index Terms - Mathematical model, COVID-19, basic reproduction number, local stability, Lyapunov function, global stability

I. INTRODUCTION

The coronavirus outbreak, which originated in Wuhan, China on December 31, 2019, quickly escalated into a global crisis and was declared a public health emergency of international concern [10]. The primary method of transmission of the disease was through respiratory droplets released during coughing and sneezing, which poses challenges to containment efforts due to transmission from individuals showing symptoms and those without symptoms [4]. This unprecedented epidemic has underscored the urgent need for effective models to comprehensively understand and predict the spread of the disease, and thus aid in prevention and control strategies. Mathematical models that describe epidemiological processes have emerged as valuable tools for researchers to simulate and analyze disease transmission dynamics, predict disease outbreak patterns, and evaluate control strategies [5] [6] [12].

The "SIR model" is a widely used mathematical method for modeling infectious diseases. It consists of three ordinary differential equations (ODEs) that divide the population into three compartments: susceptible (S), infected (I), and recovered (R). These equations describe the rates of change as individuals' transition between these compartments [11]. In addition to the SIR model, there are several generalizations such as the SEIR model, which incorporates an additional compartment for exposed individuals. The SEIR model involves four ODEs and is commonly used to simulate the outbreak of diseases like COVID-19 [2] [1] [7] [8]. These models play a crucial role in understanding the dynamics of disease transmission, predicting the spread of infections, and assessing the potential impact of intervention strategies.

In certain studies, the population of infected individuals has been further subdivided into three subclasses: asymptomatic, mild symptoms, and severe symptoms [9]. However, a numerical analysis conducted in one study found no significant distinction between the asymptomatic and mild symptom groups. As a result, it is deemed sufficient to divide the infected population into two compartments: asymptomatic and symptomatic. Consequently, we will construct an (SIR) model that incorporates this subdivision of the infected class into asymptomatic and symptomatic individuals. This approach will enable us to better understand the dynamics of the disease transmission and assess the impact of interventions within each subgroup.

The objective of this study is to develop a modified compartmental (SIR) model to accurately capture the transmission dynamics of Covid-19. We constructed SEIR model and discussed important preliminary concepts, including boundedness and the positivity of system (2.2). Furthermore, we presented the expressions for equilibria and the basic reproduction number, which are essential for understanding the spread of the disease. By establishing the global stability of the disease-free equilibrium, we gained insights into the long-term behavior of the disease. In conclusion, we emphasize the significance of the modified SIR model and discuss potential avenues for future research.

II. MODEL FORMULATION

The model formulation ($S\ E\ I_u I_d R$) used in this study focuses on human- to- human transmission of the COVID-19 within a closed population. At a given time (t), the total human population is denoted as N(t) and is further divided into different compartments: (Susceptible S(t), Exposed E(t), asymptomatic infections I_u(t), symptomatic infections I_d(t) and recovered population R(t)). It is assumed that a recovered individual becomes immunized and is no longer susceptible to reinfection. Figure (1) illustrates the developed mathematical model for Covid-19, which has been further analyzed in this study.

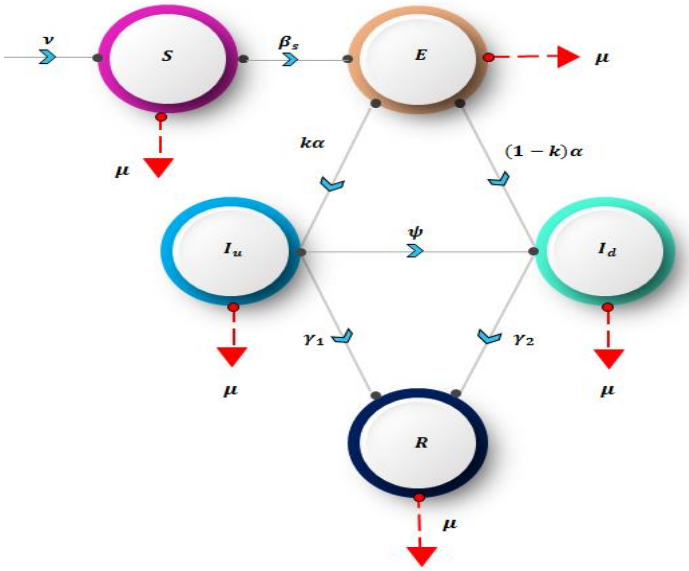

Figure 1 developed mathematical model for Covid-19

The population in this study was divided into five sections or categories, as shown in Table (1). These parts represent different groups based on their infection status. In addition, Table 1 provides an overview of the parameters used in formulating Model (1). These parameters play a crucial role in determining system dynamics and are essential for understanding disease behavior.

Table 1 Description of the variables and the Parameters for the model (1)

variables for model (1)		Parameters for the model (1)	
Variable	Description	Variable	Description
S(t)	Susceptible	ν	Recruitment rate into susceptible population
E(t)	Exposed	α	Progression rate from E to either I_u or I_d
I_u(t)	asymptomatic infections	k	Propagation of asymptomatic infections people
I_d(t)	symptomatic infections	β_u	Rate of transmission from S to E due to contract with I_u
R(t)	Recovered	β_d	Rate of transmission from S to E due to contract with I_d
		β_s	$\beta_s = \beta_u I_u + \beta_d I_d$
		ψ	Rate of transmission from I_u to I_d
		γ_1	Rate of recovery of people from I_u
		γ_2	Rate of recovery of people from I_d
		μ	Natural death rate

III. Model Constructs

A mathematical model can be constructed to describe the spread of COVID-19 in a population. In this study, we consider a homogeneous distribution of individuals. The dynamics of the disease are governed by the following system of ordinary differential equations. Based on Figure 1, we establish the following model:

$$\dot{S}(t) = \nu - (\mu + \beta_s)S \quad (1)$$

$$\dot{E}(t) = \beta_s S - (\mu + \alpha)E \quad (2)$$

$$\dot{I_u}(t) = k\alpha E - (\mu + \psi + \gamma_1)I_u \quad (3)$$

$$\dot{I_d}(t) = (1-k)\alpha E + \psi I_u - (\mu + \gamma_2)I_d \quad (4)$$

$$\dot{R}(t) = \gamma_1 I_u + \gamma_2 I_d - \mu R \quad (5)$$

The model assumes that individuals in the susceptible class, S(t), can become exposed to the Covid-19 infection at a rate represented by β_s. The state vectors and associated parameters of the system of equations (1-5) are described in detail in Tables 1. The initial conditions associated with the system are as follows $S(0) \geq 0$, $E(0) \geq 0$, $I_u(0) \geq 0$, $I_d(0) \geq 0, R(0) \geq 0$.

IV. Basic properties of the model

In this section, we will discuss some fundamental properties of the proposed model. First, we establish the existence and boundedness of solutions to demonstrate that the model has well-defined solutions. Furthermore, these solutions are unique and confined within a positive invariant region. These properties ensure the well- pawedness and epidemiological significance of the developed model.

Theorem 3.1.

Let D denote a rectangular region modeled with initial conditions $S(0) \geq 0$, $E(0) \geq 0$, $I_u(0) \geq 0$, $I_d(0) \geq 0, R(0) \geq 0$, the solution of the model exist and bounded for all $t > 0$.

Proof. Assume that F_i be the right hand sides of the equations in the system (1-5), then differentiate them with the respect to the state variable; we get that $\frac{\partial F_i}{\partial x_j}$ are continuous and bounded in D, where $i,j = 1,2,3,4,5$. It's obvious that the partial derivative of the whole system of equations exists, finite and bounded. For the model to be epidemiologically meaningful, it should to show that all its state variables are non-negative for all $t > 0$.

Theorem 3.2**.**

Given $S(0) \geq 0$, $E(0) \geq 0$, $I_u(0) \geq 0$, $I_d(0) \geq 0, R(0) \geq 0$, then the solutions of the model are positive for all $t > 0$.

Proof. Let $t^* = \sup\{ t > 0: S > 0, \quad E > 0, \ I_u > 0, \ I_d > 0, R > 0 \}$. From the second equation of the system (1-5), we have $\dot{E}(t) > \beta_u I_u S - (\mu + \alpha)E$. The integrating factor is given as $\exp\{(\mu + \alpha)\, t\}$. By multiplying the inequality by the integrating factor $(\mu + \alpha)\, t$ and integrating both sides with respect to time t from 0 to t^*, we can derive the following expression:

$$E(t^*) \geq E(0)\exp\{-(\mu+\alpha)t^*\} + \beta_u \exp\{-(\mu+\alpha)\, t^*\}\left[\int_0^{t^*} \exp\{(\mu+\alpha)\, z\}\ I_u(z)S(z)dz\right] \geq 0$$

Thus, we get $E(t) \geq 0$ when $E(0) \geq 0$. In a similar way, it can be shown that $S(t) \geq 0$, $I_u(t) \geq 0$, $I_d(t) \geq 0, R(t) \geq 0$ in the model; which implies that all state variables $S(t) \geq 0$, $E(t) \geq 0$, $I_u(t) \geq 0$, $I_d(t) \geq 0, R(t) \geq 0$ are all non- negative for all non-negative initial conditions.

Theorem 3.3**.**

The region D is positively-invariant, which indicates that all solutions of the system (1-5) with the initial conditions $S(0) \geq 0$, $E(0) \geq 0$, $I_u(0) \geq 0$, $I_d(0) \geq 0, R(0) \geq 0$ in D remain in D for all $t > 0$

Proof. By adding the two sides of the system (1-5) we have $\frac{dN(t)}{dt} + \mu N(t) = v$, which implies that $N(t) \leq N(0)e^{-\mu t} + \frac{v}{\mu}$, It can be written as

$$0 \leq N(t) \leq \frac{v}{\mu} + [S(0) + E(0) + I_u(0) + I_d(0) + R(0)]e^{-\mu t}.$$

If $N(0) < \frac{v}{\mu}$, then $\lim_{t\to\infty} \sup[S(t) + E(t)I_u(t) + I_d(t) + R(t)] \leq \frac{v}{\mu}$. Therefore, for all $t > 0$

$[S(t) + E(t) + I_u(t) + I_d(t) + R(t)] \leq \frac{v}{\mu}$. Therefore, all orbits of system (1-5) with initial conditions $S(0) \geq 0, E(0) \geq 0, I_u(0) \geq 0, I_d(0) \geq 0, R(0) \geq 0$ in , remain in D for all $t > 0$. Thus the region D is positively-invariant. Furthermore, if $N(0) \geq \frac{v}{\mu}$ then either $N(t)\ approaches\ \frac{v}{\mu}\ as\ t \to \infty$ and the infected variables E, I_u, I_d, R approaches zero, or the solution enters D in the finite time. Therefore, the region D attracts all solutions in $\mathbb{R}^5_+$ and all solutions of the system (1-5) are non-negative and epidemiological well posed.

Through this paper, we shall consider the dynamical behaviors of the system (1-5) on the region $D = \left\{(S, E, I_u\ ,\ I_d, R) \in \mathbb{R}^5_+ : 0 \leq S + E + I_u\ +\ I_d + R \leq \frac{v}{\mu}\right\}$

V. Model Analysis

This section focuses on the analysis of equilibria and the basic reproduction number. By setting the right-hand sides of the system to zero, we can determine that the system (1-5) has only one disease-free equilibrium (DFE), which is denoted by

$$\mathcal{X}_\circ = \left(\frac{v}{\mu}, \quad 0, \quad 0, \quad 0, \quad 0\right)$$

Next, we will calculate the basic reproduction number ($R_\circ$) of the system (1-5) using the Next Generation Matrix (NGM) method.

Basic reproduction number of the model.

To estimate the basic reproduction number for the COVID-19 infection, we employ the next generation matrix procedure [3]. Specifically, we focus on the infected subsystem (2-4), which can be represented as follows:

$$\begin{aligned} \dot{E}(t) &= \beta_s S - (\mu + \alpha)E \\ \dot{I_u}(t) &= k\alpha E - (\mu + \psi + \gamma_1)I_u \\ \dot{I_d}(t) &= (1-k)\alpha E + \psi I_u - (\mu + \gamma_2)I_d \end{aligned} \qquad (2\text{-}4)$$

Let $\mathcal{X} = (E,\ I_u,\ I_d)^T$, then the infected subsystem (2-4) can be written as

$\mathcal{X} = \mathcal{F}(x) - \mathcal{M}(x)$, where

$$\mathcal{F}(x) = \begin{bmatrix} \beta_s S \\ 0 \\ 0 \end{bmatrix} \quad , \quad \mathcal{M}(x) = \begin{bmatrix} (\mu+\alpha)E \\ -k\alpha E + (\mu+\psi+\gamma_1)I_u \\ -(1-k)\alpha E - \psi I_u + (\mu+\gamma_2)I_d \end{bmatrix}$$

We can obtain

$$F = \begin{bmatrix} 0 & \beta_s \frac{v}{\mu} & \beta_d \frac{v}{\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad , \quad M = \begin{bmatrix} (\mu+\alpha) & 0 & 0 \\ -k\alpha & (\mu+\psi+\gamma_1) & 0 \\ -(1-k)\alpha & -\psi & (\mu+\gamma_2) \end{bmatrix}$$

Then the next generation matrix for the system is taken by the spectral radians of $NGM = F\,M^{-1}$, where he spectral radius of the matrix FM^{-1} denoted by $\rho(\ FM^{-1})$.

$$M^{-1} = \begin{bmatrix} \frac{1}{(\mu+\alpha)} & 0 & 0 \\ \frac{k\alpha}{(\mu+\alpha)(\mu+\psi+\gamma_1)} & \frac{1}{(\mu+\psi+\gamma_1)} & 0 \\ \frac{k\alpha\psi + (1-k)\alpha(\mu+\alpha+\gamma_1)}{(\mu+\alpha)(\mu+\psi+\gamma_1)(\mu+\gamma_2)} & \frac{\psi}{(\mu+\psi+\gamma_1)(\mu+\gamma_2)} & \frac{1}{(\mu+\gamma_2)} \end{bmatrix}$$

Then

$$FM^{-1} = \begin{bmatrix} \frac{k\alpha v\beta_u}{\mu AB} + \frac{v}{\mu}\frac{\beta_d(k\alpha\psi + (1-k)\alpha B)}{ABC} & \frac{v\beta_u}{\mu B} + \frac{v\beta_d\psi}{\mu BC} & \frac{v\beta_d}{\mu C} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

Where A= $(\mu+\alpha)$, B= $(\mu+\psi+\gamma_1)$, C=$(\mu+\gamma_2)$.

$$\rho(\ FM^{-1}) = \frac{v}{\mu}\left[\frac{k\alpha\ (\mu+\gamma_2)\beta_u + \alpha[\psi\ k + (1-k)(\mu+\psi+\gamma_1)]\beta_d}{(\mu+\alpha)(\mu+\psi+\gamma_1)(\mu+\gamma_2)}\right].$$

Therefore, the basic reproduction number of the system is

$$R_\circ = \frac{v}{\mu}\left[\frac{k\alpha\ (\mu+\gamma_2)\beta_u + \alpha[\psi\ k + (1-k)(\mu+\psi+\gamma_1)]\beta_d}{(\mu+\alpha)(\mu+\psi+\gamma_1)(\mu+\gamma_2)}\right]$$

The Endemic Equilibrium.

The disease-endemic equilibrium (DEE) of the system can be expressed as $\mathcal{X}^* = (S^*, E^*, I^*_u, I^*_d, R^*)$, for simplicity, we put $I^*_1 = I^*_u$, $I^*_2 = I^*_d$, then $\beta_s = \sum_{j=1}^{2}\beta_j I_j$, where

$$S^* = \frac{v}{\mu + \sum_{j=1}^{2}\beta_j I^*_j} \tag{6}$$

$$E^* = \frac{\left(\sum_{j=1}^{2}\beta_j I^*_j\right)S^*}{A} \tag{7}$$

$$I^*_1 = \frac{k\alpha\left(\sum_{j=1}^{2}\beta_j I^*_j\right)S^*}{B} \tag{8}$$

$$I^*_2 = \frac{1}{C}\left[\frac{(1-k)\alpha}{A} + \psi\ \frac{k\alpha}{B}\right]\left(\sum_{j=1}^{2}\beta_j I^*_j\right)S^* \tag{9}$$

$$R^* = \left[\frac{\gamma_1 k\alpha}{\mu B} + \left(\frac{\gamma_2(1-k)\alpha}{\mu AC} + \frac{\gamma_2\psi k\alpha}{\mu BC}\right)\right]\left(\sum_{j=1}^{2}\beta_j I^*_j\right)S^* \tag{10}$$

Using the equation (8) and substituting the value of S^*, we get $I^*_1 = \frac{k\alpha}{B}\left(\sum_{j=1}^{2}\beta_j I^*_j\right)\frac{v}{\mu+\sum_{j=1}^{2}\beta_j I^*_j}$. Solving for I^*_1 we get

$$(I^*_1)\left(\mu B + \left\{(B - vk\alpha)\sum_{j=1}^{2}\beta_j I^*_j\right\}\right) = 0 \tag{11}$$

The Equation (11) is a quadratic equation. Thus solutions are $I^*_1 = 0$ or $\sum_{j=1}^{2}\beta_j I^*_j = \frac{\mu B}{B - vk\alpha}$,where $I^*_1 = 0$ corresponds to the disease-free equilibrium point, hence the system has a unique endemic equilibrium $\mathcal{X}^*$ when $\sum_{j=1}^{2}\beta_j I^*_j = \frac{\mu B}{B - vk\alpha}$. Hence the

expression of equation (9) can simplify as follows:

$I^*_2 = \frac{FDv}{\mu+F}$, Thus we obtain that

$$I^*_1 = \frac{\mu B}{\beta_1(vk\alpha - B)} - \frac{\beta_1}{\beta_2}\ \frac{FDv}{\mu + F}$$

Where, $D = \frac{(1-k)\alpha B + \psi k\alpha A}{ABC}$ $\quad if \quad (1-k)\alpha B + \psi k\alpha A > 0$

$F = \frac{\mu B}{vk\alpha - B}$ $\quad if \quad vk\alpha > \mu + \psi + \gamma_1$ Therefore, we have

$$I^*_1 = \left[\frac{\mu^2 B + (\mu B - \beta_2 v^2 k\alpha D + v\beta_2 DB)F}{(vk\alpha - B)(\mu + F)}\right] \tag{12}$$

$$I^*_2 = \left[\frac{(1-k)(\mu+\psi+\gamma_1) + \psi k(\mu+\alpha)}{k[vk\alpha - (\mu+\psi+\gamma_1)](\mu+\alpha)(\mu+\gamma_2)}\right] \tag{13}$$

VI. STEADY STATE ANALYSIS

We will demonstrate that the disease-free equilibrium (DFE) is both locally and globally asymptotically stable. For local stability, we will examine the negativity of the real parts of the associated Jacobian matrix. Additionally, we will construct a Lyapunov function to establish the globally asymptotic stability of the DFE. These analyses will provide insights into the stability properties of the model and the long-term behavior of the disease.

Local stability of the disease-free equilibrium of the model

The result of local stability of the disease- free equilibrium $\mathcal{X}_\circ = (S_\circ, 0, 0, 0, 0)$ for the system can is listed as following:

Theorem 5.1.

The disease-free equilibrium (DEF) of the model is locally asymptotically stable if $R_\circ < 1$, and unstable if $R_\circ > 1$.

Proof. To study the stability of (DFE) $\mathcal{X}_\circ = (S_\circ, 0, 0, 0, 0)$ locally, we find the Jacobin of the system.

$$J = \begin{bmatrix} -(\mu+\beta_s) & 0 & -\beta_u S & -\beta_d S & 0 \\ \beta_s & -(\mu+\alpha) & \beta_u S & \beta_d S & 0 \\ 0 & k\alpha & -(\mu+\psi+\gamma_1) & 0 & 0 \\ 0 & \alpha(1-k) & \psi & -(\mu+\gamma_2) & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & -\mu \end{bmatrix}$$

Evaluate the Jacobin of the system at (DFE) we get

$$J|_{\mathcal{X}_\circ} = \begin{bmatrix} -\mu & 0 & -\beta_u \frac{v}{\mu} & -\beta_d \frac{v}{\mu} & 0 \\ 0 & -(\mu+\alpha) & \beta_u \frac{v}{\mu} & \beta_d \frac{v}{\mu} & 0 \\ 0 & k\alpha & -(\mu+\psi+\gamma_1) & 0 & 0 \\ 0 & \alpha(1-k) & \psi & -(\mu+\gamma_2) & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & -\mu \end{bmatrix}$$

By solving the characteristic equation $|J(x_\circ) - \lambda I| = 0$, we get $\lambda_1 = -\mu$, $\lambda_2 = -\mu$. Consequently, the local stability of the disease free equilibrium (DFE) is determined by the eigenvalues of the equations for E, I_u and I_d . The Jacobian matrix for these equations is given by:

$$J_\circ = \begin{bmatrix} -(\mu+\alpha) - \lambda & \beta_u \frac{v}{\mu} & \beta_d \frac{v}{\mu} \\ k\alpha & -(\mu+\psi+\gamma_1) - \lambda & 0 \\ \alpha(1-k) & \psi & -(\mu+\gamma_2) - \lambda \end{bmatrix}$$

So the characteristic polynomial for the matrix is

$$\lambda^3 + (A + B + C)\lambda^2 + [AB + AC + B(-a_1 - a_3)]\lambda + ABC(R_\circ - 1) = 0$$

By applying the Routh-Hurwitz criteria, we can ensure that all the roots of the characteristic polynomial have negative real parts if the following conditions are met: $A + B + C > 0, \quad 1 - R_\circ > 0 \quad \Longrightarrow \quad R_\circ < 1,$

$(A + B + C)[AB + AC - B(-a_1 - a_3)] > ABC(1 - R_\circ)$.

Hence we have DFE is asymptotically stable when $R_\circ < 1$, and unstable otherwise.

Global stability of the disease-free equilibrium of the model

Theorem 5.1**.**

The disease free equilibrium $\mathcal{X}_\circ$ is globally asymptotically stable when $R_\circ < 1$,

Proof. Consider a Lyapunov function $V(t) = c_1E + c_2I_u + c_3I_d$, where c_1, c_2, c_3 are undetermined non- negative real numbers. The derivative of $V(t)$ along the solution curves of system has the following form

$$\frac{dV}{dt} = c_1\frac{dE}{dt} + c_2\frac{dI_u}{dt} + c_3\frac{dI_d}{dt}$$

$$\frac{dV}{dt} = c_1[(\beta_u I_u + \beta_u I_d)S - AE] + c_2[k\alpha E - BI_u] + c_3[(1-k)\alpha E + \psi I_u - CI_d],$$

$$\frac{dV}{dt} \leq c_1\left[(\beta_u I_u + \beta_d I_d)\frac{v}{\mu} - AE\right] + c_2[k\alpha E - BI_u] + c_3[(1-k)\alpha E + \psi I_u - CI_d],$$

$$\frac{dV}{dt} \leq [c_2k\alpha + c_3(1-k)\alpha - Ac_1]E + \left[\frac{v}{\mu}c_1\beta_u - Bc_2 + c_{3\psi}\right]I_u + \left[\frac{v}{\mu}c_1\beta_d - c_3C\right]I_d.$$

Now we select the coefficients c_1, c_2 and c_3 with the zero coefficients of I_u and I_d. Hence we obtain $c_1 = \frac{v}{\mu}$, $c_2 = \left[\frac{1}{B}\frac{\beta_u}{\mu} + \beta_d\frac{v}{\mu}\frac{\psi}{C}\right]\frac{v}{\mu}$ and $c_3 = \frac{v}{\mu}\left[\frac{\beta_d v}{\mu C}\right]$.

Substituting the values of c_1, c_2 and c_3 to $V(t)$, then the derivative of $V(t)$ can be expressed as

$$\frac{dV}{dt} \leq \frac{v}{\mu}A[R_\circ - 1]E \ .$$

Clearly, $\frac{dV}{dt} \leq 0$ *when* $R_\circ < 1$, also $V(t) = 0$ if and only if $E = I_u = I_d = 0$.

VII. Conclusion

In this study, we have investigated an $S\ E\ I_u I_d R$ f epidemic model to describe the transmission dynamics of COVID-19. The model incorporates various population compartments, including the susceptible, exposed, asymptomatic infections, symptomatic infections, and recovered individuals. We have shown the existence of a disease-free equilibrium and demonstrated its local and global asymptotic stability when the basic reproduction number, R_0, is less than 1. This implies that the disease can be effectively controlled and eventually eradicated under certain conditions. Additionally, we have explored the conditions for the existence of an endemic equilibrium, which represents a persistent level of infection in the population. These conditions provide insights into the factors that contribute to the sustained transmission of COVID-19. Overall, our findings contribute to the understanding of the transmission dynamics of COVID-19 and highlight the importance of effective control measures to prevent its spread. Further research can focus on extending the model to incorporate more complex factors and evaluating the impact of different intervention strategies on disease control.

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