RESEARCH ARTICLE • OPEN ACCESS

Qualitative analysis of a mathematical model of COVID-19 with intervention strategies in the Philippines

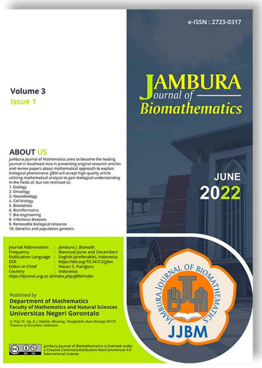
Rolly N. Apdo and Rolando N. Paluga

Volume 4, Issue 1, Pages 46–54, June 2023

Received 27 February 2023, Revised 20 April 2023, Accepted 10 May 2023, Published Online 26 June 2023

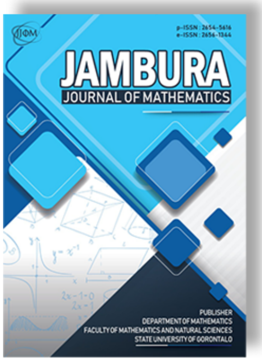
To Cite this Article : R. N. Apdo and R. N. Paluga,"Qualitative analysis of a mathematical model of COVID-19 with intervention strategies in the Philippines", *Jambura J. Biomath*, vol. 4, no. 1, pp. 46–54, 2023, *https://doi.org/10.34312/jjbm.v4i1.18990*

JOURNAL INFO • JAMBURA JOURNAL OF BIOMATHEMATICS



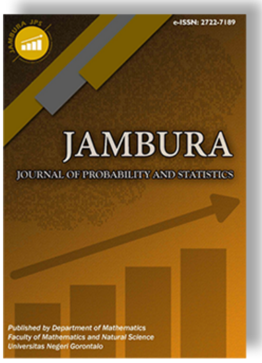
Homepage	:	http://ejurnal.ung.ac.id/index.php/JJBM/index
Journal Abbreviation	:	Jambura J. Biomath.
Frequency	:	Biannual (June and December)
Publication Language	:	English (preferable), Indonesia
DOI	:	https://doi.org/10.34312/jjbm
Online ISSN	:	2723-0317
Editor-in-Chief	:	Hasan S. Panigoro
Publisher	:	Department of Mathematics, Universitas Negeri Gorontalo
Country	:	Indonesia
OAI Address	:	http://ejurnal.ung.ac.id/index.php/jjbm/oai
Google Scholar ID	:	XzYgeKQAAAAJ
Email	:	editorial.jjbm@ung.ac.id

JAMBURA JOURNAL • FIND OUR OTHER JOURNALS

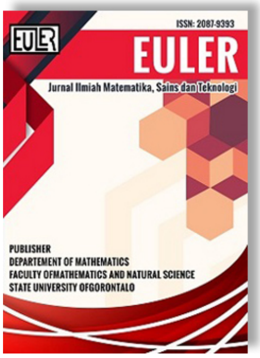


Jambura Journal of Mathematics

Jambura Journal of Mathematics Education



Jambura Journal of Probability and Statistics



EULER : Jurnal Ilmiah Matematika, Sains, dan Teknologi

Jambura Journal of Biomathematics, Volume 4, Issue 1, Pages 46–54, June 2023 *https://doi.org/10.34312/jjbm.v4i1.18990*

Research Article

Qualitative analysis of a mathematical model of COVID-19 with intervention strategies in the Philippines

Rolly N. Apdo[1,*] and **Rolando N. Paluga**[2]

[1,2] *Department of Mathematics, Caraga State University, Butuan City, Philippines*

ARTICLE HISTORY
Received 27 February 2023
Revised 20 April 2023
Accepted 10 May 2023
Published 26 June 2023

KEYWORDS
COVID-19
intervention
qualitative analysis
vaccination
contact tracing

ABSTRACT. *This paper focuses on the development of a mathematical model to analyze the transmission dynamics of COVID-19 in the Philippines, where the pandemic has significantly impacted the population despite several quarantine measures, testing, contact tracing, and vaccinations. The model considers the impact of contact tracing and vaccination campaigns on disease transmission. The model is analyzed qualitatively and numerically, and the results show that increasing the contact tracing rate and vaccination rate can effectively reduce the reproduction number of the virus. The disease-free equilibrium is found to be locally asymptotically stable when the basic reproduction number is less than one, and the disease-endemic equilibrium is locally asymptotically stable when the basic reproduction number is greater than one. The study suggests that a contact tracing rate greater than 0.08847694 is required to effectively manage the transmission of COVID-19 in the target population. These findings provide insights for policymakers and public health officials in developing effective strategies to mitigate the impact of the pandemic.*

1. Introduction

Until now, Coronavirus disease 2019 (COVID-19), caused by a novel coronavirus called Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), has remained a public health threat to all human beings since its emergence in late 2019. SARS-CoV-2 is continuously evolving, and to date, different variants of the virus have been identified and classified, which may pose an additional risk to the lives of millions of people. In fact, the World Health Organization (WHO) [1] reported that as of February 22, 2023, there had been $757,264,511$ confirmed cases, including $6,850,594$ deaths globally. Philippines is one of the countries that has been dramatically affected by COVID-19. To stop the virus' spread, the Philippines has been enforced with a number of quarantine measures, including heightened community quarantine. The government has also implemented measures such as testing and contact tracing, as well as vaccinations. However, the situation remains challenging, with rising cases nowadays. The Department of Health (DOH) reported $4,076,237$ confirmed cases and $66,108$ people died due to COVID-19, whereas $166,485,680$ doses of vaccine have been administered up to February 27, 2023 [2]. Therefore, research on the dynamics of COVID-19 after the vaccination campaigns is a significant concern in the Philippines.

Mathematical modeling plays a vital role in understanding, forecasting, and managing infectious disease transmission dynamics. Since the beginning of the COVID-19 pandemic, several models have been developed to study the transmission dynamics of the disease. In the context of the Philippines, there are several models related to the spread of COVID-19. Torres et al. [3] studied COVID-19 infection cases to forecast daily cases using numerous mathematical models, including the Susceptible-Exposed-Infected-Recovered (SEIR) model. Arcede et al. [4] also considered an SEIR-type model of COVID-19, emphasizing that the infected can be either symptomatic or not. Data on confirmed cases and deaths from numerous countries, including the Philippines, were used to calibrate the model. Results revealed that testing and isolation are required to stop the disease. Another study by Arcede et al. [5] utilized optimal control for the model proposed in [4]. Results indicate that the more capable the government is, the more it should undertake transmission reduction, testing, and improving patients' medical care without adding more hospital beds if all controls are implemented. Buhat et al. [6] developed an extended SEIR compartment model with two mutually exclusive populations: the general public and frontliners. They performed simulations and found that front liners and the general public should be protected against the disease. To simulate the first wave of the COVID-19 outbreak in the Philippines, Caldwell et al. [7] used an age-structured compartmental model that included time-varying mobility, testing, and personal preventive behaviors through a Minimum Health Standards (MHS) policy. They found that following MHS decreased the likelihood of transmission per encounter by 13-27%. These researchers, to our knowledge, did not consider vaccination in their model formulation. However, recently, researchers already included vaccination class in their models. For instance, in [8], they formulated six compartments differential equation model for the transmission of COVID-19 and analyzed the global stability of the equilibrium points. They found out that the disease-free equilibrium and endemic equilibrium are globally asymptotically stable when

---

[*] Corresponding Author.

Email : *rollyapdo92@gmail.com* (R. N. Apdo)

Homepage : http://ejurnal.ung.ac.id/index.php/JJBM/index / E-ISSN : 2723-0317

$R_0 < 1$ and $R_0 > 1$ respectively. In addition, Caga-anan et al. [9] developed a COVID-19 model with a delay-term for the vaccinated compartment. Their simulations provided compelling evidence that vaccination against COVID-19 has a significant and positive impact on reducing the number of future infections. Moreover, Campos et al. [10] also studied vaccination strategies considering the different COVID-19 variants. Their results have highlighted the importance of administering booster shots to increase vaccine-induced immunity duration against COVID-19. The studies mentioned did not consider the potential impact of contact tracing and vaccination campaigns on the spread of COVID-19, particularly regarding the severity of the infected individuals. To fulfill the gaps, we are inspired to conduct this research.

The rest of the paper is organized as follows. Section 2 discusses the construction of the model and is analyzed in Sections 3 and 4. The model is then subjected to numerical simulation in Section 5. Finally, Section 6 outlines the conclusion.

2. Methods

2.1. Mathematical Model

In this research work, we formulated a model that is an extension of the Susceptible-Exposed-Infection-Recovered ($SEIR$) COVID-19 model to study the transmission dynamics of the disease. In this model, we stressed the importance of contact tracing and vaccination campaigns. We considered nine compartments: susceptible individuals (S), exposed individuals (E), exposed and tested individuals (E_d), exposed and contact traced individuals (E_c), critical and severe infected individuals (I_c), moderate and mild infected individuals (I_m), asymptomatic infected individuals (I_a), recovered individuals (R), and vaccinated individuals (V). For any time t, the total population (assumed constant), $N(t)$ is given by

$$\begin{aligned} N(t) = S(t) + E(t) + E_d(t) + E_c(t) + I_c(t) + I_m(t) \\ + I_a(t) + R(t) + V(t). \end{aligned}$$

We made the following assumptions:

1. All dead individuals are considered as newborns and replaced in the susceptible compartment to keep the population size constant.
2. A natural death case is included in all compartments.
3. The critical and severe infected population die at a disease-induced death rate.
4. The individuals who do not contact traced remained in the exposed compartment.
5. Having recovered from the disease guaranteed lifelong immunity.
6. The susceptible compartment contained healthy individuals only never infected with the disease.
7. Vaccinated compartment contained at least partially vaccinated individuals. However, the vaccination campaign is not perfect that is vaccinated individuals may be reinfected with the disease.

Based on the above-mentioned assumptions, we developed the model, $(SE(E_dE_c)I(I_cI_mI_a)RV)$ as follows: The susceptible population is replenished by a birth rate θ. The natural death rate in each compartment is represented by μ. The susceptible individuals contract the virus after coming into contact with exposed individuals at the rate α_s. The susceptible individuals move to the vaccination compartment at a vaccination rate ν. The vaccinated individuals may also contract the virus after coming into contact with exposed individuals at the rate α_v. Individuals who have been exposed are tracked down and tested if necessary. Some exposed individuals undergo direct testing at a rate of β_d. While some exposed individuals are contact traced at a rate of β_c. The contact-traced individuals who exhibit symptoms will be tested at a rate of γ. The exposed and tested individuals are infected by the virus and move to the severe or critical compartment, mild or moderate compartment, and asymptomatic compartment at the incubation rates σ_c, σ_m, and σ_a respectively. The moderately and mildly infected individuals develop more serious symptoms and move to severe or critical compartment at the rate ϕ. The induced death rate due to COVID-19 to critically and severely infected individuals is represented by δ. Individuals who are critically and severely infected, moderately and mildly infected, and asymptomatic infected are recovered and moved to the recovery compartment at the rate ρ_c, ρ_m, and ρ_a respectively. The descriptions of the parameters are summarized in Table 1.

Table 1. Description of the parameters

Parameter	Description
θ	Birth rate
ν	Vaccination rate of S
α_s	Transmission rate from S to E from contact with E
α_v	Transmission rate from V to E from contact with E
β_d	Testing rate of E
β_c	Contact tracing rate of E
γ	Testing rate of E_c
σ_c	Incubation rate of E_d to I_c
σ_m	Incubation rate of E_d to I_m
σ_a	Incubation rate of E_d to I_a
ϕ	Transfer rate from I_m to I_c
ρ_c	Recovery rate of E_c
ρ_m	Recovery rate of E_m
ρ_a	Recovery rate of E_a
δ	Induced death rate by COVID-19
μ	Natural death rate

Adhering to the flow chart in Figure 1, the dynamics of the model are governed by the following system of ordinary differential equations:

$$\begin{cases} \dfrac{dS}{dt} = \theta N - (\alpha_s E + \nu + \mu) S, \\ \dfrac{dE}{dt} = (\alpha_s S + \alpha_v V) E - (\beta_d + \beta_c + \mu) E, \\ \dfrac{dE_d}{dt} = \beta_d E + \gamma E_c - (\sigma_c + \sigma_m + \sigma_a + \mu) E_d, \\ \dfrac{dE_c}{dt} = \beta_c E - (\gamma + \mu) E_c, \\ \dfrac{dI_c}{dt} = \sigma_c E_d + \phi I_m - (\rho_c + \delta + \mu) I_c, \\ \dfrac{dI_m}{dt} = \sigma_m E_d - (\rho_m + \phi + \mu) I_m, \\ \dfrac{dI_a}{dt} = \sigma_a E_d - (\rho_a + \mu) I_a, \\ \dfrac{dR}{dt} = \rho_c I_c + \rho_m I_m + \rho_a I_a - \mu R, \\ \dfrac{dV}{dt} = \nu S - (\alpha_v E + \mu) V, \end{cases} \tag{1}$$

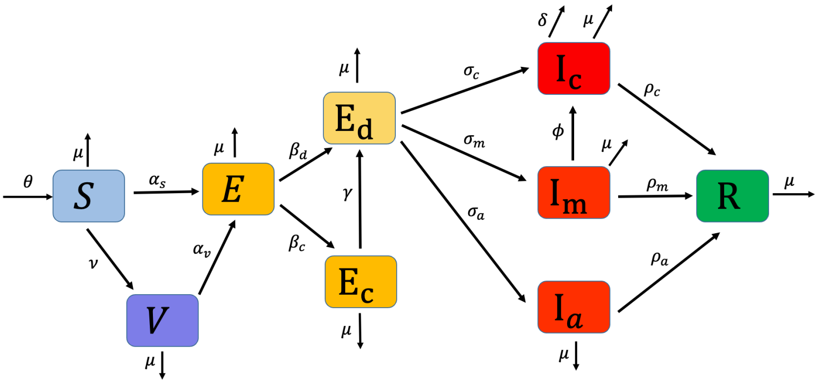


Figure 1. Schematic diagram of the COVID-19 model

with the positive initial conditions:

$$\begin{array}{rclrcl} S(0) &=& S_0 \geq 0, & E(0) &=& E_0 \geq 0, \\ E_d(0) &=& E_{d0} \geq 0, & E_c(0) &=& E_{c0} \geq 0, \\ I_c(0) &=& I_{c0} \geq 0, & I_m(0) &=& I_{m0} \geq 0, \\ I_a(0) &=& I_{a0} \geq 0, & R(0) &=& R_0 \geq 0, \\ V(0) &=& V_0 \geq 0. & & & \end{array} \qquad (2)$$

3. Qualitative Analysis

3.1. Well-posedness

In this subsection, we will prove that the system (1) is well-posed that is, positive and bounded since the system is dealing with human populations which cannot be negative and grow infinitely large.

> **Theorem 1.** *Under the initial conditions (2), the solution $(S(t), E(t), E_d(t), E_c(t), I_c(t), I_m(t), I_a(t), R(t), V(t))$ of the system (1) remains nonnegative for $t \geq 0$.*

Proof. Assume that $S(0) \geq 0$, $E(0) \geq 0$, $E_d(0) \geq 0$, $E_c(0) \geq 0$, $I_c(0) \geq 0$, $I_m(0) \geq 0$, $I_a(0) \geq 0$, $R(0) \geq 0$, $V(0) \geq 0$. Consider the first equation of model (1).

$$\frac{dS(t)}{dt} = \theta N - (\alpha_S E + \nu + \mu)\, S \Rightarrow \frac{dS(t)}{dt} + \tau S = \theta N,$$

where $\tau = \alpha_S E + \nu + \mu$. Using Leibniz' formula [11], the solution to this linear first-order equation in S is given by

$$\begin{aligned} S(t) =&\ \exp\left(-\int_0^t \tau(u)du\right)\left(\theta N \int_0^t \exp\left(\int_0^r \tau(v)dv\right) dr + S(0)\right) \\ =&\ S(0)\exp\left(-\int_0^t \tau(u)du\right) \\ &+ (\theta N)\exp\left(-\int_0^t \tau(u)du\right)\int_0^t \left(\exp\left(\int_0^r \tau(v)dv\right)\right) dr. \end{aligned}$$

By assumption, $S(0) \geq 0$. Further, all of the integrals in the equation are positive since the integrands are positive. Hence, $S(t) \geq 0$.

From the second equation of model (1), we get

$$\begin{aligned} \frac{dE(t)}{dt} &= (\alpha_s S + \alpha_v V)\, E - (\beta_d + \beta_c + \mu)\, E \\ &= (\alpha_s S + \alpha_v V - (\beta_d + \beta_c + \mu))\, E. \end{aligned}$$

Solving the above equation, we have

$$E(t) = E(0)\exp\left(\int_0^t (\alpha_s S + \alpha_v V - (\beta_d + \beta_c + \mu))\, du\right).$$

By assumption, $E(0) \geq 0$. Further, the integral in the equation is positive since the integrand is positive. Hence, $E(t) \geq 0$.

From the last equation of model (1), we have

$$\begin{aligned} \frac{dV(t)}{dt} &= \nu S - (\alpha_v E + \mu)\, V \\ &\geq -(\alpha_v E + \mu)\, V \text{ (since } S \geq 0). \end{aligned}$$

Solving the equation, we get the following solution

$$V(t) \geq V(0)\exp\left(-\int_0^t (\alpha_v E + \mu)\, du\right) > 0,$$

which shows that $V(t)$ is nonnegative for all t. Similar reasoning is used regarding the nonnegativity of the remaining variables. We have

$$\begin{aligned} E_d(t) &\geq E_d(0)\exp\left(-\int_0^t (\sigma_c + \sigma_m + \sigma_a + \mu)\, du\right) > 0, \\ E_c(t) &\geq E_c(0)\exp\left(-\int_0^t (\gamma + \mu)\, du\right) > 0, \\ I_c(t) &\geq I_c(0)\exp\left(-\int_0^t (\rho_c + \alpha + \mu)\, du\right) > 0, \\ I_m(t) &\geq I_m(0)\exp\left(-\int_0^t (\rho_m + \phi + \mu)\, du\right) > 0, \\ I_a(t) &\geq I_a(0)\exp\left(-\int_0^t (\rho_a + \mu)\, du\right) > 0, \\ R(t) &\geq R_{(0)}\exp\left(-\int_0^t \mu du\right) > 0. \end{aligned}$$

□

The boundedness of the system's solutions is guaranteed by the following theorem.

> **Theorem 2.** *Under the initial conditions (2), the solution $(S(t), E(t), E_d(t), E_c(t), I_c(t), I_m(t), I_a(t), R(t), V(t))$ of*

the system (1) remains bounded for $t \geq 0$.

Proof. Assume that $S(0) \geq 0, E(0) \geq 0, E_d(0) \geq 0, E_c(0) \geq 0, I_c(0) \geq 0, I_m(0) \geq 0, I_a(0) \geq 0, R(0) \geq 0, V(0) \geq 0$. Adding all the equations of the model (1), we have

$$\begin{aligned} \frac{dN}{dt} &= \frac{dS}{dt} + \frac{dE}{dt} + \frac{dE_d}{dt} + \frac{dE_c}{dt} + \frac{dI_c}{dt} \\ &\quad + \frac{dI_m}{dt} + \frac{dI_a}{dt} + \frac{dR}{dt} + \frac{dV}{dt} \\ &= \theta N - \mu N - \delta I_c \\ \Rightarrow \frac{dN}{dt} &= 0 \text{ since } \theta N = \mu N + \delta I_c \text{ by the first assumption.} \end{aligned}$$

Integrating both sides of the equation, we get

$$N(t) = \text{Constant}$$

which means the constant size of the population. Therefore, the solution $(S(t), E(t), E_d(t), E_c(t), I_c(t), I_m(t), I_a(t), R(t), V(t))$ of the system (1) remains bounded for $t \geq 0$. ☐

Combining Theorem 1 and Theorem 2 together with the trivial existence and uniqueness of a local solution for the system (1) which can be shown using the basic theory of dynamical systems as indicated in [12], and [13], the biologically feasible region for the system (1) is given by

$$\begin{aligned} \Omega = \{ &(S, E, E_d, E_c, I_c, I_m, I_a, R, V) \in \mathbb{R}^9_+ : \\ &S + E + E_d + E_c + I_c + I_m + I_a + R + V = N \}. \end{aligned}$$

3.2. Disease-Free Equilibrium Point

The disease-free equilibrium point of the system (1) is a point where the disease is not present in the population. It is obtained by setting the derivatives to zero and putting the disease compartments to zero.

Theorem 3. *The model (1) admits a disease-free equilibrium (DFE) given by $\mathcal{E}^0 = \left(S^0, 0, 0, 0, 0, 0, 0, 0, V^0\right)$ where $S^0 = \dfrac{\theta N}{\mu + \nu}$ and $V^0 = \dfrac{\nu \theta N}{(\mu + \nu)\mu}$.*

Proof. Let $\mathcal{E}^0 = \left(S^0, E^0, E^0_d, E^0_c, I^0_c, I^0_m, I^0_a, R^0, V^0\right)$ be an equilibrium point of the model (1), that is

$$\begin{aligned} \theta N - \left(\alpha_s E^0 + \nu + \mu\right) S^0 &= 0, \\ \left(\alpha_s S^0 + \alpha_v V^0\right) E^0 - \left(\beta_d + \beta_c + \mu\right) E^0 &= 0, \\ \beta_d E^0 + \gamma E^0_c - \left(\sigma_c + \sigma_m + \sigma_a + \mu\right) E^0_d &= 0, \\ \beta_c E^0 - \left(\gamma + \mu\right) E^0_c &= 0, \\ \sigma_c E^0_d + \phi I^0_m - \left(\rho_c + \delta + \mu\right) I^0_c &= 0, \\ \sigma_m E^0_d - \left(\rho_m + \phi + \mu\right) I^0_m &= 0, \\ \sigma_a E^0_d - \left(\rho_a + \mu\right) I^0_a &= 0, \\ \rho_c I^0_c + \rho_m I^0_m + \rho_a I^0_a - \mu R^0 &= 0, \\ \nu S^0 - \left(\alpha_v E^0 + \mu\right) V^0 &= 0. \end{aligned}$$

Suppose that the disease compartments are zero which means that the environment is COVID-19-free. If $E = E_d = E_c = I_c = I_m = I_a = 0$, we obtain

$$S^0 = \frac{\theta N}{\mu + \nu}, R^0 = 0, V^0 = \frac{\nu \theta N}{(\mu + \nu)\mu}.$$

This completes the proof. ☐

3.3. Basic Reproduction Number

Next, we will calculate the basic reproduction number of the model (1) using the next generation matrix method [14, 15]. The basic reproduction number denoted by R_0 is defined as the average number of secondary infections that occurs when one infective is introduced into a completely susceptible population [14, 15]. The disease compartments are Exposed E, Exposed and tested E_d, Exposed and contact traced E_c, Critical and severe infected I_c, Moderate and mild infected I_m, and Asymptomatic infected I_a compartments.

Then, the rate of appearance of new infections $\mathcal{F}$ and the rate of transfer of individuals by all other means $\mathcal{V}$ are given by the following matrices:

$$\mathcal{F} = \begin{bmatrix} \left(\alpha_s S + \alpha_v V\right) E \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and}$$

$$\mathcal{V} = \begin{bmatrix} \left(\beta_d + \beta_c + \mu\right) E \\ \left(\sigma_c + \sigma_m + \sigma_a + \mu\right) E_d - \beta_d E - \gamma E_c \\ \left(\gamma + \mu\right) E_c - \beta_c E \\ \left(\rho_c + \delta + \mu\right) I_c - \sigma_c E_d - \phi I_m \\ \left(\rho_m + \phi + \mu\right) I_m - \sigma_m E_d \\ \left(\rho_a + \mu\right) I_a - \sigma_a E_d \end{bmatrix}.$$

The Jacobian of $\mathcal{F}$ and $\mathcal{V}$ at E^0 are F and V, respectively.

$$F = \begin{bmatrix} \dfrac{\theta N \left(\mu \alpha_s + \nu \alpha_v\right)}{\left(\mu + \nu\right) \mu} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and}$$

$$V = \begin{bmatrix} v_{1,1} & 0 & 0 & 0 & 0 & 0 \\ -\beta_d & v_{2,2} & -\gamma & 0 & 0 & 0 \\ -\beta_c & 0 & v_{3,3} & 0 & 0 & 0 \\ 0 & -\sigma_c & 0 & v_{4,4} & -\phi & 0 \\ 0 & -\sigma_m & 0 & 0 & v_{5,5} & 0 \\ 0 & -\sigma_a & 0 & 0 & 0 & \end{bmatrix},$$

where $v_{1,1} = \beta_d + \beta_c + \mu$, $v_{2,2} = \sigma_c + \sigma_m + \sigma_a + \mu$, $v_{3,3} = \gamma + \mu$, $v_{4,4} = \rho_c + \delta + \mu$, $v_{5,5} = \rho_m + \phi + \mu$, and $v_{6,6} = \rho_a + \mu$. Then we get

$$FV^{-1} = \begin{bmatrix} \dfrac{\theta N \left(\mu \alpha_s + \nu \alpha_v\right)}{\mu \left(\mu + \nu\right) \left(\beta_d + \beta_c + \mu\right)} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

The basic reproduction number is the dominant eigenvalue of FV^{-1} which is $\mathcal{R}_0 = \rho\left(FV^{-1}\right)$. Thus,

$$\mathcal{R}_0 = \frac{\theta N\left(\mu\alpha_s + \nu\alpha_v\right)}{\mu\left(\mu+\nu\right)\left(\beta_d+\beta_c+\mu\right)}.$$

3.4. Disease-Endemic Equilibrium Point

The disease-endemic equilibrium point of the system (1) is a point where the disease is present in the population. It is obtained by making the system (1) equal to zero and then solving the values of the variables.

Theorem 4. *The model (1) admits a disease-endemic equilibrium (DEE) given by* $\mathcal{E}^* = (S^*, E^*, E_d^*, E_c^*, I_c^*, I_m^*, I_a^*, R^*, V^*)$ *with* $R_0 > 1$ *and* $N \geq \max\left\{\frac{6(\alpha_s+1)}{\alpha_s\theta}, \frac{12(\alpha_s+\alpha_v)}{\alpha_s\theta}, \frac{3}{\mu}\right\}$.

Proof. Let $\mathcal{E}^* = (S^*, E^*, E_d^*, E_c^*, I_c^*, I_m^*, I_a^*, R^*, V^*)$ be an endemic equilibrium point of the model (1), that is

$$\begin{aligned}
\theta N - \left(\alpha_s E^* + \nu + \mu\right) S^* &= 0,\\
\left(\alpha_s S^* + \alpha_v V^*\right) E^* - \left(\beta_d + \beta_c + \mu\right) E^* &= 0,\\
\beta_d E^* + \gamma E_c^* - \left(\sigma_c + \sigma_m + \sigma_a + \mu\right) E_d^* &= 0,\\
\beta_c E^* - \left(\gamma + \mu\right) E_c^* &= 0,\\
\sigma_c E_d^* + \phi I_m^* - \left(\rho_c + \delta + \mu\right) I_c^* &= 0,\\
\sigma_m E_d^* - \left(\rho_m + \phi + \mu\right) I_m^* &= 0,\\
\sigma_a E_d^* - \left(\rho_a + \mu\right) I_a^* &= 0,\\
\rho_c I_c^* + \rho_m I_m^* + \rho_a I_a^* - \mu R^* &= 0,\\
\nu S^* - \left(\alpha_v E^* + \mu\right) V^* &= 0.
\end{aligned} \tag{3}$$

Suppose that $\mathcal{E}^* = (S^*, E^*, E_d^*, E_c^*, I_c^*, I_m^*, I_a^*, R^*, V^*)$ are nonzero which means that the environment is not COVID-19-free. We express each compartment in terms of E^*. We solve for S^* from the first equation in (3).

$$\begin{aligned}
\theta N - \left(\alpha_s E^* + \nu + \mu\right) S^* &= 0\\
S^* &= \frac{\theta N}{\alpha_s E^* + \nu + \mu}
\end{aligned}$$

We solve for E_c^* from the fourth equation.

$$\begin{aligned}
\beta_c E^* - \left(\gamma + \mu\right) E_c^* &= 0\\
E_c^* &= \frac{\beta_c E^*}{\gamma + \mu}.
\end{aligned}$$

We substitute E_c^* to the third equation and solve for E_d^*.

$$\begin{aligned}
\beta_d E^* + \gamma E_c^* - \left(\sigma_c + \sigma_m + \sigma_a + \mu\right) E_d^* &= 0\\
\beta_d E^* + \frac{\gamma\beta_c E^*}{\gamma + \mu} - \left(\sigma_c + \sigma_m + \sigma_a + \mu\right) E_d^* &= 0\\
\Rightarrow E_d^* &= \frac{\left(\beta_c\gamma + \beta_d\gamma + \beta_d\mu\right) E^*}{\left(\gamma + \mu\right)\left(\sigma_c + \sigma_m + \sigma_a + \mu\right)}.
\end{aligned}$$

We substitute E_d^* to the sixth equation and solve for I_m^*.

$$\begin{aligned}
\sigma_m E_d^* - \left(\rho_m + \phi + \mu\right) I_m^* &= 0\\
\tfrac{\sigma_m(\beta_c\gamma+\beta_d\gamma+\beta_d\mu)E^*}{(\gamma+\mu)(\sigma_c+\sigma_m+\sigma_a+\mu)} - \left(\rho_m + \phi + \mu\right) I_m^* &= 0\\
\Rightarrow I_m^* &= \tfrac{\sigma_m(\beta_c\gamma+\beta_d\gamma+\beta_d\mu)E^*}{(\gamma+\mu)(\sigma_c+\sigma_m+\sigma_a+\mu)(\rho_m+\phi+\mu)}.
\end{aligned}$$

We substitute E_d^* to the seventh equation and solve for I_a^*.

$$\begin{aligned}
\sigma_a E_d^* - \left(\rho_a + \mu\right) I_a^* &= 0\\
\tfrac{\sigma_a(\beta_c\gamma+\beta_d\gamma+\beta_d\mu)E^*}{(\gamma+\mu)(\sigma_c+\sigma_m+\sigma_a+\mu)} - \left(\rho_a + \mu\right) I_a^* &= 0\\
\Rightarrow I_a^* &= \tfrac{\sigma_m(\beta_c\gamma+\beta_d\gamma+\beta_d\mu)E^*}{(\gamma+\mu)(\sigma_c+\sigma_m+\sigma_a+\mu)(\rho_a+\mu)}.
\end{aligned}$$

We substitute E_d^* and I_m^* to the fifth equation and solve for I_c^*.

$$\sigma_c E_d^* + \phi I_m^* - \left(\rho_c + \delta + \mu\right) I_c^* \quad = \quad 0.$$

After the substitution and quick algebraic manipulation, we get

$$I_c^* = \tfrac{(\beta_c\gamma+\beta_d\gamma+\beta_d\mu)(\mu\sigma_c+\phi\sigma_c+\phi\sigma_m+\rho_m\sigma_c)E^*}{(\gamma+\mu)(\sigma_c+\sigma_m+\sigma_a+\mu)(\rho_m+\phi+\mu)(\rho_c+\delta+\mu)}.$$

We substitute I_c^*, I_m^*, and I_a^* to the eighth equation and solve for R^*.

$$\rho_c I_c^* + \rho_m I_m^* + \rho_a I_a^* - \mu R^* = 0.$$

After the substitution and quick algebraic manipulation, we get

$$R^* = \tfrac{(\beta_c\gamma+\beta_d\gamma+\beta_d\mu)(e+f+g+h)E^*}{\mu(\gamma+\mu)(\sigma_c+\sigma_m+\sigma_a+\mu)(\rho_a+\mu)(\rho_m+\phi+\mu)(\rho_c+\delta+\mu)},$$

where

$$\begin{aligned}
e &= \rho_a\sigma_a\left(\rho_m + \phi + \mu\right)\left(\mu + \delta\right),\\
f &= \rho_a\rho_c\left(\sigma_a + \sigma_c + \sigma_m\right)\left(\phi + \rho_m\right),\\
g &= \mu\rho_c\left(\mu\sigma_c + \phi\sigma_c + \phi\sigma_m + \rho_a\sigma_a + \rho_a\sigma_c + \rho_m\sigma_c\right),\\
h &= \rho_m\sigma_m\left(\delta\mu + \delta\rho_a + \mu^2 + \mu\rho_a + \mu\rho_c\right).
\end{aligned}$$

We substitute S^* to the ninth equation and solve for V^*.

$$\begin{aligned}
\nu S^* - \left(\alpha_v E^* + \mu\right) V^* &= 0\\
\tfrac{\nu\theta N}{\alpha_s E^* + \nu + \mu} - \left(\alpha_v E^* + \mu\right) V^* &= 0\\
\Rightarrow V^* &= \tfrac{\nu\theta N}{(E^*\alpha_s+\mu+\nu)(E^*\alpha_v+\mu)}.
\end{aligned}$$

We substitute S^* and V^* to the equation and solve for E^*.

$$\left(\alpha_s S^* + \alpha_v V^*\right) E^* - \left(\beta_d + \beta_c + \mu\right) E^* = 0$$

After the substitution and lengthy algebraic manipulation, we get

$$E^* = \tfrac{\mu(\mu+\nu)\left(\theta N\alpha_s\alpha_v - ab + \sqrt{2\theta N\alpha_s\alpha_v ac + a^2 d^2}\right)(R_0-1)}{2\alpha_s\alpha_v(\theta N(\mu\alpha_s+\nu\alpha_v)-\mu(\mu+\nu)a)},$$

where

$$\begin{aligned}
a &= \beta_d + \beta_c + \mu,\\
b &= \mu\alpha_s + \mu\alpha_v + \nu\alpha_v,\\
c &= \mu\alpha_s - \mu\alpha_v - \nu\alpha_v + 2\alpha_v,\\
d &= \mu\alpha_s - \mu\alpha_v - \nu\alpha_v.
\end{aligned}$$

Note that E^* exists when the basic reproduction number $R_0 > 1$ and $N \geq \max\left\{\frac{6(\alpha_s+1)}{\alpha_s\theta}, \frac{12(\alpha_s+\alpha_v)}{\alpha_s\theta}, \frac{3}{\mu}\right\}$. This completes the proof. □

4. Stability Analysis

In this section, we will show the local stability of the equilibrium points. Stability analysis sheds light on a system's long-term behavior when it is close to equilibrium.

Theorem 5. *The disease-free equilibrium,* $\mathcal{E}^0 = (S^0, 0, 0, 0, 0, 0, 0, 0, V^0)$ *is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

Proof. The Jacobian of the system (1) is given by

$$J = \begin{bmatrix} a_{1,1} & -\alpha_s S & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ E\alpha_s & a_{2,2} & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_v E \\ 0 & \beta_d & a_{3,3} & \gamma & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_c & 0 & a_{4,4} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_c & 0 & a_{5,5} & \phi & 0 & 0 & 0 \\ 0 & 0 & \sigma_m & 0 & 0 & a_{6,6} & 0 & 0 & 0 \\ 0 & 0 & \sigma_a & 0 & 0 & 0 & a_{7,7} & 0 & 0 \\ 0 & 0 & 0 & 0 & \rho_c & \rho_m & \rho_a & -\mu & 0 \\ \nu & -V\alpha_v & 0 & 0 & 0 & 0 & 0 & 0 & a_{9,9} \end{bmatrix}, \tag{4}$$

where $a_{1,1} = -E\alpha_s - \mu - \nu$, $a_{2,2} = \alpha_s S + V\alpha_v - \beta_c - \beta_d - \mu$, $a_{3,3} = -\sigma_c - \sigma_m - \sigma_a - \mu$, $a_{4,4} = -\gamma - \mu$, $a_{5,5} = -\rho_c - \delta - \mu$, $a_{6,6} = -\rho_m - \phi - \mu$, $a_{7,7} = -\rho_a - \mu$, and $a_{9,9} = -\alpha_v E - \mu$.

The Jacobian matrix (4) evaluated at the disease-free equilibrium is of the form

$$J(\mathcal{E}^0) = \begin{bmatrix} a_{1,1} & -\alpha_s S^0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a_{2,2} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_d & a_{3,3} & \gamma & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_c & 0 & a_{4,4} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_c & 0 & a_{5,5} & \phi & 0 & 0 & 0 \\ 0 & 0 & \sigma_m & 0 & 0 & a_{6,6} & 0 & 0 & 0 \\ 0 & 0 & \sigma_a & 0 & 0 & 0 & a_{7,7} & 0 & 0 \\ 0 & 0 & 0 & 0 & \rho_c & \rho_m & \rho_a & -\mu & 0 \\ \nu & -\alpha_v V^0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu \end{bmatrix},$$

where $a_{1,1} = -\mu - \nu$, $a_{2,2} = \alpha_s S^0 + \alpha_v V^0 - \beta_c - \beta_d - \mu$, $a_{3,3} = -\sigma_c - \sigma_m - \sigma_a - \mu$, $a_{4,4} = -\gamma - \mu$, $a_{5,5} = -\rho_c - \delta - \mu$, $a_{6,6} = -\rho_m - \phi - \mu$, and $a_{7,7} = -\rho_a - \mu$.

To show that all the eigenvalues of $J(\mathcal{E}^0)$ are negative, we can first note that the eighth and ninth columns of $J(\mathcal{E}^0)$ contain only the diagonal element $-\mu$, indicating that $-\mu$ is a negative eigenvalue. The remaining eigenvalues can be determined from the sub-matrix $J_1(\mathcal{E}^0)$, which can be obtained by eliminating the eighth and ninth rows, and eight and ninth columns of $J(\mathcal{E}^0)$. This gives us the following matrix:

$$J_1(\mathcal{E}^0) = \begin{bmatrix} a_{1,1} & -\alpha_s S^0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a_{2,2} & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_d & a_{3,3} & \gamma & 0 & 0 & 0 \\ 0 & \beta_c & 0 & a_{4,4} & 0 & 0 & 0 \\ 0 & 0 & \sigma_c & 0 & a_{5,5} & \phi & 0 \\ 0 & 0 & \sigma_m & 0 & 0 & a_{6,6} & 0 \\ 0 & 0 & \sigma_a & 0 & 0 & 0 & a_{7,7} \end{bmatrix},$$

where $a_{1,1} - \mu - \nu$, $a_{2,2} = \alpha_s S^0 + \alpha_v V^0 - \beta_c - \beta_d - \mu$, $a_{3,3} = -\sigma_c - \sigma_m - \sigma_a - \mu$, $a_{4,4} = -\gamma - \mu$, $a_{5,5} = -\rho_c - \delta - \mu$, $a_{6,6} = -\rho_m - \phi - \mu$, and $a_{7,7} = -\rho_a - \mu$.

The first, fifth, and seventh columns of the matrix $J_1(\mathcal{E}^0)$ only contain diagonal elements that form the negative eigenvalues $-(\mu + v)$, $-(\rho_c + \delta + \mu)$, and $-(\rho_a + \mu)$ respectively. To find the remaining eigenvalues, we can create a reduced sub-matrix, $J_2(\mathcal{E}^0)$, by removing the first, fifth, and seventh rows and corresponding columns from $J_1(\mathcal{E}^0)$.

$$J_2(\mathcal{E}^0) = \begin{bmatrix} b_{1,1} & 0 & 0 & 0 \\ \beta_d & b_{2,2} & \gamma & 0 \\ \beta_c & 0 & b_{3,3} & 0 \\ 0 & \sigma_m & 0 & b_{4,4} \end{bmatrix},$$

where $b_{1,1} = \alpha_s S^0 + \alpha_v V^0 - \beta_c - \beta_d - \mu$, $b_{2,2} = -\sigma_c - \sigma_m - \sigma_a - \mu$, $b_{3,3} = -\gamma - \mu$, and $b_{4,4} = -\rho_m - \phi - \mu$.

In a similar manner, the fourth column of the matrix $J_2(\mathcal{E}^0)$ contains only the diagonal element $-(\rho_m + \phi + \mu)$, which means that $-(\rho_m + \phi + \mu)$ is a negative eigenvalue. The remaining eigenvalues can be determined using the sub-matrix $J_3(\mathcal{E}^0)$ which can be obtained by removing the fourth row and fourth column of the matrix $J_2(\mathcal{E}^0)$.

$$J_3(\mathcal{E}^0) = \begin{bmatrix} c_{1,1} & 0 & 0 \\ \beta_d & c_{2,2} & \gamma \\ \beta_c & 0 & -\gamma - \mu \end{bmatrix},$$

where $c_{1,1} = \alpha_s S^0 + \alpha_v V^0 - \beta_c - \beta_d - \mu$ and $c_{2,2} = -\sigma_c - \sigma_m - \sigma_a - \mu$.

Looking at $J_3(\mathcal{E}^0)$, we can see that the second column contains only the diagonal element that forms the negative eigenvalue $-(\sigma_c + \sigma_m + \sigma_a + \mu)$. We can form $J_4(\mathcal{E}^0)$ by removing the second row and second column of the matrix $J_3(\mathcal{E}^0)$ to find the remaining eigenvalues.

$$J_4(\mathcal{E}^0) = \begin{bmatrix} \alpha_s S^0 + \alpha_v V^0 - \beta_c - \beta_d - \mu & 0 \\ \beta_c & -\gamma - \mu \end{bmatrix}$$

In a similar way, the second column of $J_4(\mathcal{E}^0)$ contains only the diagonal element which form the negative eigenvalue $-(\gamma + \mu)$. The remaining eigenvalue is $\alpha_s S^0 + \alpha_v V^0 - \beta_c - \beta_d$. We need to show that this is a negative eigenvalue. Now,

$$\begin{aligned} &\alpha_s S^0 + \alpha_v V^0 - \beta_c - \beta_d - \mu \\ &\quad = \tfrac{\alpha_s \theta N}{\mu + \nu} + \tfrac{\alpha_v \nu \theta N}{(\mu + \nu)\mu} - \beta_c - \beta_d - \mu \\ &\quad = (\beta_c + \beta_d + \mu)\left(\tfrac{\theta N(\alpha_s \mu + \alpha_v \nu)}{\mu(\mu + \nu)(\beta_c + \beta_d + \mu)} - 1\right) \\ &\quad = (\beta_c + \beta_d + \mu)(R_0 - 1) < 0 \text{ for any values of } R_0 < 1. \end{aligned}$$

Hence, the disease-free equilibrium $\mathcal{E}^0$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. ☐

Theorem 6. *The disease-endemic equilibrium,* $\mathcal{E}^* = (S^*, E^*, E_d^*, E_c^*, I_c^*, I_m^*, I_a^*, R^*, V^*)$ *is locally asymptotically stable if* $R_0 > 1$.

Proof. At the disease-endemic equilibrium, the Jacobian matrix (4) of the system (1) can be expressed as

$$J(\mathcal{E}^*) = \begin{bmatrix} a_{1,1} & -\alpha_s S^* & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a_{2,2} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_d & a_{3,3} & \gamma & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_c & 0 & a_{4,4} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_c & 0 & a_{5,5} & \phi & 0 & 0 & 0 \\ 0 & 0 & \sigma_m & 0 & 0 & a_{6,6} & 0 & 0 & 0 \\ 0 & 0 & \sigma_a & 0 & 0 & 0 & a_{7,7} & 0 & 0 \\ 0 & 0 & 0 & 0 & \rho_c & \rho_m & \rho_a & -\mu & 0 \\ \nu & -\alpha_v V^* & 0 & 0 & 0 & 0 & 0 & 0 & -\mu \end{bmatrix}$$

where $a_{1,1} = -(\mu + \nu)$, $a_{2,2} = \alpha_s S^* + \alpha_v V^* - \beta_c - \beta_d - \mu$, $a_{3,3} = -(\sigma_c + \sigma_m + \sigma_a + \mu)$, $a_{4,4} = -(\gamma + \mu)$, $a_{6,6} = -(\rho_m + \phi + \mu)$, $a_{7,7} = -(\rho_a + \mu)$ and $a_{5,5} = -(\rho_c + \delta + \mu)$.

To demonstrate that all the eigenvalues of $J(\mathcal{E}^*)$ are negative, we can see that the eighth and ninth columns of $J(\mathcal{E}^*)$ only include the diagonal element $-\mu$, proving that $-\mu$ is a negative eigenvalue. The sub-matrix $J_1(\mathcal{E}*)$, which can be created by removing the eighth and ninth rows, as well as the eighth and ninth columns of $J(\mathcal{E}^*)$, can be used to calculate the remaining eigenvalues. This results in the following matrix

$$J_1(\mathcal{E}^*) = \begin{bmatrix} a_{1,1} & -\alpha_s S^* & 0 & 0 & 0 & 0 & 0 \\ 0 & a_{2,2} & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_d & a_{3,3} & \gamma & 0 & 0 & 0 \\ 0 & \beta_c & 0 & a_{4,4} & 0 & 0 & 0 \\ 0 & 0 & \sigma_c & 0 & a_{5,5} & \phi & 0 \\ 0 & 0 & \sigma_m & 0 & 0 & a_{6,6} & 0 \\ 0 & 0 & \sigma_a & 0 & 0 & 0 & a_{7,7} \end{bmatrix},$$

where $a_{1,1} = -\mu - \nu$, $a_{2,2} = \alpha_s S^* + \alpha_v V^* - \beta_c - \beta_d - \mu$, $a_{3,3} = -\sigma_c - \sigma_m - \sigma_a - \mu$, $a_{4,4} = -\gamma - \mu$, $a_{5,5} = -\rho_c - \delta - \mu$, $a_{6,6} = -\rho_m - \phi - \mu$, and $a_{7,7} = -\rho_a - \mu$. Likewise, the first, fifth, and seventh columns of the matrix $J_1(\mathcal{E}^*)$ comprise only the diagonal terms $-(\mu + \nu)$, $-(\rho_c + \delta + \mu)$, and $-(\rho_a + \mu)$, respectively, which make up the negative eigenvalues. By deleting the first, fifth, and seventh rows and associated columns from $J_1(\mathcal{E}^*)$, we may create a smaller sub-matrix, $J_2(\mathcal{E}^*)$, in order to locate the remaining eigenvalues.

$$J_2(\mathcal{E}^*) = \begin{bmatrix} b_{1,1} & 0 & 0 & 0 \\ \beta_d & b_{2,2} & \gamma & 0 \\ \beta_c & 0 & -\gamma - \mu & 0 \\ 0 & \sigma_m & 0 & b_{3,3} \end{bmatrix},$$

where $b_{1,1} = \alpha_s S^* + \alpha_v V^* - \beta_c - \beta_d - \mu$. $b_{2,2} = -\sigma_c - \sigma_m - \sigma_a - \mu$, and $b_{3,3} = -\rho_m - \phi - \mu$. Similar to this, only the diagonal element $-(\rho_m + \phi + \mu)$ is present in the fourth column of the matrix $J_2(\mathcal{E}^*)$, indicating that $-(\rho_m + \phi + \mu)$ is a negative eigenvalue. The sub-matrix $J_3(\mathcal{E}^*)$, which is created by eliminating the fourth row and fourth column of the matrix $J_2(\mathcal{E}^*)$, can be used to calculate the remaining eigenvalues.

$$J_3(\mathcal{E}^*) = \begin{bmatrix} c_{1,1} & 0 & 0 \\ \beta_d & c_{2,2} & \gamma \\ \beta_c & 0 & -\gamma - \mu \end{bmatrix},$$

where $c_{1,1} = \alpha_s S^* + \alpha_v V^* - \beta_c - \beta_d - \mu$ and $c_{2,2} = -\sigma_c - \sigma_m - \sigma_a - \mu$. As we can see from $J_3(\mathcal{E}^*)$, the second column only contains the diagonal element that makes up the negative eigenvalue $-(\sigma_c + \sigma_m + \sigma_a + \mu)$. By eliminating the second row and second column of the matrix $J_3(\mathcal{E}^*)$, we may create $J_4(\mathcal{E}^*)$ and identify the remaining eigenvalues.

$$J_4(\mathcal{E}^*) = \begin{bmatrix} \alpha_s S^* + \alpha_v V^* - \beta_c - \beta_d - \mu & 0 \\ \beta_c & -\gamma - \mu \end{bmatrix}.$$

In a similar way, the 2^{nd} column of $J_4(\mathcal{E}^*)$ contains only the diagonal element which form the negative eigenvalue $-(\gamma + \mu)$. The remaining eigenvalue is $\alpha_s S^* + \alpha_v V^* - \beta_c - \beta_d - \mu$. Now,

$$\begin{aligned} \alpha_s S^* + \alpha_v V^* - \beta_c - \beta_d - \mu = & \tfrac{\alpha_s \theta N}{\alpha_s E^* + \mu + \nu} \\ & + \tfrac{\alpha_v \nu \theta N}{(\alpha_s E^* + \mu + \nu)(\alpha_v E^* + \mu)} \\ & - \beta_c - \beta_d - \mu. \end{aligned}$$

When $\theta = 2.93 \times 10^{-5}$, $\nu = 9.97 \times 10^{-4}$, $\alpha_s = 2.02 \times 10^{-9}$, $\alpha_v = 4.05 \times 10^{-10}$, $\beta_d = 4.78 \times 10^{-4}$, $\beta_c = 4.32 \times 10^{-2}$, $\mu = 1.56 \times 10^{-5}$, $E^* = 7 \times 10^7$ and $N = 110.5 \times 10^6$, we get

$$\alpha_s S^* + \alpha_v V^* - \beta_c - \beta_d - \mu = -0.0436910971278318$$

Hence, the disease-endemic equilibrium $\mathcal{E}^*$ is locally asymptotically stable if $R_0 > 1$. □

5. Numerical Simulations

In this section, we performed numerical simulations on the COVID-19 model (1) using the parameter values in Table 1 using Python 3.9 to determine the behavior of the infected population over time and evaluate the effects of various control measures that we took into account when developing the model. Particularly to assess the impact of the vaccination rate ν and contact tracing rate β_c to the basic reproduction number R_0.

Table 2. Parameter values for the model simulation

Parameter	Value	Source
δ	1.80×10^{-3}	[16]
α_s	2.02×10^{-9}	[4]
α_v	4.05×10^{-10}	Estimated
ρ_c	1.23×10^{-1}	[4]
ρ_m	1.23×10^{-1}	[4]
ρ_a	1.23×10^{-1}	[4]
θ	2.93×10^{-5}	[17]
μ	1.56×10^{-5}	[17]
ν	9.97×10^{-4}	Estimated
ϕ	5.00×10^{-2}	Assumed
σ_c	1.60×10^{-3}	Estimated
σ_m	1.29×10^{-1}	Estimated
σ_a	4.33×10^{-3}	Estimated
γ	2.00×10^{-1}	Assumed
β_d	4.78×10^{-4}	Assumed
β_c	4.32×10^{-2}	Assumed

5.1. *Behavior of Infected Population*

Figure 2 provides a visual representation of the temporal behavior of the infected population, categorizing individuals into three distinct groups based on the severity of their symptoms - Critical and severe infected individuals I_c, Moderate and mild infected individuals I_m and Asymptomatic infected individuals I_a. The figure showcases how the number of individuals in each group changes over time. After approximately 800 days, the infected population will converge to a certain value. Plugging in the values in Table 1 into the basic reproduction number, we have $R_0 = 2.03 > 1$ which means the disease will spread in the population even after the vaccination.

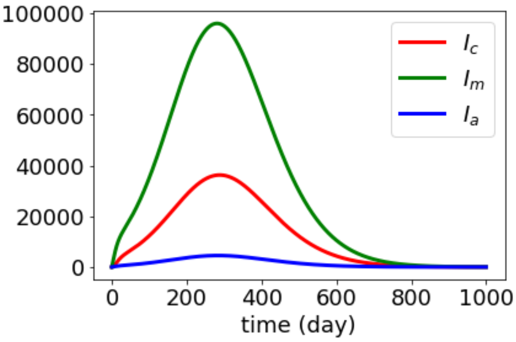


Figure 2. Behavior of Infected Population over Time

5.2. *Impact of ν to R_0*

Figure 3 illustrates the impact of the vaccination rate ν on the basic reproduction number R_0. According to Figure 3, as the vaccination rate ν increases, the basic reproduction number R_0 decreases. Therefore, the government should aim to increase

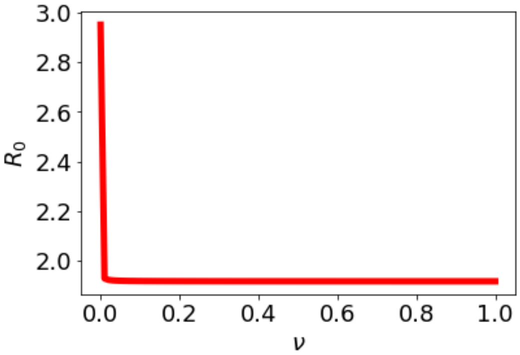


Figure 3. Impact of vaccination rate on R_0

the vaccination rate to control the spread of the pandemic to the community. This result is supported by the following theorems.

Theorem 7. *The function* $R_0(\nu) = \dfrac{\theta N\left(\mu\alpha_s + \nu\alpha_v\right)}{\mu\left(\mu+\nu\right)\left(\beta_d+\beta_c+\mu\right)}$ *is decreasing for all* $\nu \in [0,1]$.

Proof. The function $R_0(\nu)$ is a rational function which is not defined when $\nu = -\mu < 0$. It follows that this function is continuous and differentiable for all $\nu \in [0,1]$.
Observe that $R_0'(\nu) = \dfrac{\theta N\mu\left(\alpha_v - \alpha_s\right)}{\mu\left(\beta_d+\beta_c+\mu\right)} < 0$ since $\alpha_s < \alpha_v$. Hence, $R_0(\nu)$ is decreasing for all $\nu \in [0,1]$. ☐

Theorem 8. *The disease will eventually die out as long as the vaccination rate* $\nu > \dfrac{\theta N\mu\alpha_s - \mu^2\left(\beta_d+\beta_c+\mu\right)}{\mu\left(\beta_d+\beta_c+\mu\right) - \theta N\alpha_v} > 0$. *Otherwise, the spread of the disease will persist.*

Proof. Let $\nu > \nu_0$ where $\nu_0 = \dfrac{\theta N\mu\alpha_s - \mu^2\left(\beta_d+\beta_c+\mu\right)}{\mu\left(\beta_d+\beta_c+\mu\right) - \theta N\alpha_v} > 0$. By Theorem 7, $R_0(\nu)$ is decreasing for all $\nu \in [0,1]$. It follows that

$$\begin{aligned} R_0(\nu) &< R_0(\nu_0) \\ &= \frac{\theta N\left(\mu\alpha_s + \left(\frac{\theta N\mu\alpha_s - \mu^2(\beta_d+\beta_c+\mu)}{\mu(\beta_d+\beta_c+\mu)-\theta N\alpha_v}\right)\alpha_v\right)}{\mu\left(\mu + \left(\frac{\theta N\mu\alpha_s - \mu^2(\beta_d+\beta_c+\mu)}{\mu(\beta_d+\beta_c+\mu)-\theta N\alpha_v}\right)\right)(\beta_d+\beta_c+\mu)} \\ &= 1 \end{aligned}$$

The disease will eventually die out as long as the vaccination rate $\nu > \dfrac{\theta N\mu\alpha_s - \mu^2\left(\beta_d+\beta_c+\mu\right)}{\mu\left(\beta_d+\beta_c+\mu\right) - \theta N\alpha_v}$. The spread of the disease will persist if $\nu < \dfrac{\theta N\mu\alpha_s - \mu^2\left(\beta_d+\beta_c+\mu\right)}{\mu\left(\beta_d+\beta_c+\mu\right) - \theta N\alpha_v}$. ☐

The disease is likely to persist in the population based on the given parameter values. However, if the parameter ν is increased, which reflects the effectiveness of measures that reduce disease transmission, the value of R_0 is expected to decrease.

5.3. *Impact of β_c to R_0*

Figure 4 shows the impact of the contact tracing rate β_c to the basic reproduction number R_0.

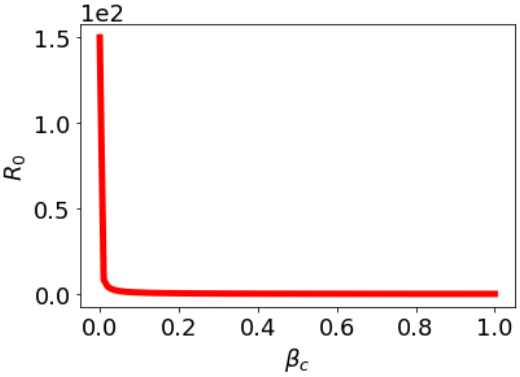


Figure 4. Impact of contact tracing rate on R_0

Based on Figure 4, as the contact tracing rate β_c increases, the basic reproduction number R_0 decreases. Therefore, the government should aim to increase the contact tracing rate to control the spread of the pandemic to the community. This result is supported by the following theorems.

Theorem 9. *The function* $R_0(\beta_c) = \dfrac{\theta N\left(\mu\alpha_s + \nu\alpha_v\right)}{\mu\left(\mu+\nu\right)\left(\beta_d+\beta_c+\mu\right)}$ *is decreasing for all* $\beta_c \in [0,1]$.

Proof. The function $R_0(\beta_c)$ is a rational function which is not defined when $\nu = -\left(\beta_d+\mu\right) < 0$. It follows that this function is continuous and differentiable for all $\beta_c \in [0,1]$.
Observe that $R_0'(\beta_c) = -\dfrac{\theta N\left(\mu\alpha_s + \nu\alpha_v\right)}{\mu\left(\mu+\nu\right)\left(\beta_d+\beta_c+\mu\right)^2} < 0$. Hence, $R_0(\beta_c)$ is decreasing for all $\beta_c \in [0,1]$. ☐

Theorem 10. *The disease will eventually die out as long as the contact tracing rate* $\beta_c > \dfrac{\theta N\left(\mu\alpha_s + \nu\alpha_v\right)}{\mu\left(\mu+\nu\right)} - \left(\beta_d+\mu\right) > 0$. *Otherwise, the spread of the disease will persist.*

Proof. Let $\beta_c > \beta_c^0$ where $\beta_c^0 = \dfrac{\theta N\left(\mu\alpha_s + \nu\alpha_v\right)}{\mu\left(\mu+\nu\right)} - \left(\beta_d+\mu\right)$. By Theorem 9, $R_0(\beta_c)$ is decreasing for all $\beta_c \in [0,1]$. It follows that

$$\begin{aligned} R_0(\beta_c) &< R_0(\beta_c^0) \\ &= \frac{\theta N(\mu\alpha_s+\nu\alpha_v)}{\mu(\mu+\nu)\left(\beta_d + \left(\frac{\theta N(\mu\alpha_s+\nu\alpha_v)}{\mu(\mu+\nu)} - (\beta_d+\mu)\right) + \mu\right)} \\ &= 1. \end{aligned}$$

This means that the disease will eventually die out as long as $\beta_c > \dfrac{\theta N\left(\mu\alpha_s + \nu\alpha_v\right)}{\mu\left(\mu+\nu\right)} - \left(\beta_d+\mu\right)$. The spread of the disease will persist if $\beta_c < \dfrac{\theta N\left(\mu\alpha_s + \nu\alpha_v\right)}{\mu\left(\mu+\nu\right)} - \left(\beta_d+\mu\right)$. ☐

If the contact tracing rate β_c is greater than 0.08847694, the disease outbreak is likely to be controlled and eventually eliminated. On the other hand, if β_c is less than this threshold value, the disease outbreak is likely to persist and continue to spread.

6. Conclusion

We have developed a model to better understand the transmission dynamics of COVID-19. We focused on the importance of contact tracing and vaccination campaigns in controlling the spread of the disease. The qualitative analysis showed that the solutions are positive and bounded, with the disease-free equilibrium being locally asymptotically stable when the basic reproduction number is less than one, and the disease-endemic equilibrium being locally asymptotically stable when the basic reproduction number is greater than one. From the numerical result, we get the value of the basic reproduction number which is $R_0 = 2.31$. The numerical simulations also demonstrated that the reproduction number decreases with an increase in both the vaccination rate and contact tracing rate. It is worthy to note that when the contact tracing rate $\beta_c > 0.08847694, R_0 < 1$. Hence, to stop and manage COVID-19 transmission in the target population, public health authorities should focus on increasing the value of the contact tracing rate to more than 0.08847694. These findings highlight the effectiveness of contact tracing and vaccination in reducing the spread of COVID-19. Overall, this study provides useful insights for policymakers and public health officials in developing strategies to mitigate the impact of the pandemic.

Author Contributions. **Rolly N. Apdo:** Conceptualization, methodology, validation, formal analysis, data curation, writing–original draft preparation, writing–review and editing. **Rolando N. Paluga:** Conceptualization, methodology, validation, formal analysis and supervision. All authors have read and agreed to the published version of the manuscript.

Acknowledgement. The authors express their sincere gratitude to the editors and reviewers who have provided invaluable support and feedback, contributing significantly to the enhancement of this manuscript.

Funding. This research received no external funding.

Conflict of interest. The authors declare no conflict of interest.

Data availability. Several parameter values are cited from some references. See Table 2.

References

[1] World Health Organization, “COVID-19 Tracker,” `https://doh.gov.ph/covid19tracker`, accessed: 2023-02-27.

[2] Department of Health, “WHO Coronavirus (COVID-19) Dashboard,” `https://covid19.who.int/`, accessed: 2023-02-22.

[3] M. C. Torres, C. A. H. Buhat, B. P. B. Dela Cruz, E. F. O. Felix, E. B. Gemida, and J. B. Mamplata, “Forecasting covid-19 cases in the philippines using various mathematical models,” 2020. DOI: 10.1101/2020.10.07.20208421

[4] Arcede, Jayrold P., Caga-anan, Randy L., Mentuda, Cheryl Q., and Mammeri, Youcef, “Accounting for symptomatic and asymptomatic in a seir-type model of covid-19,” *Math. Model. Nat. Phenom.*, vol. 15, p. 34, 2020. DOI: 10.1051/mmnp/2020021

[5] J. Macalisang, M. Caay, J. Arcede, and R. Caga-anan, “Optimal control for a covid-19 model accounting for symptomatic and asymptomatic,” *Computational and Mathematical Biophysics*, vol. 8, pp. 168–179, 11 2020. DOI: 10.1515/cmb-2020-0109

[6] C. A. H. Buhat, M. C. Torres, Y. H. Olave, M. K. A. Gavina, E. F. O. Felix, G. B. Gamilla, K. V. B. Verano, A. L. Babierra, and J. F. Rabajante, “A mathematical model of covid-19 transmission between frontliners and the general public,” 2020. DOI: 10.1101/2020.03.27.20045195

[7] J. M. Caldwell, E. de Lara-Tuprio, T. R. Teng, M. R. J. E. Estuar, R. F. R. Sarmiento, M. Abayawardana, R. N. F. Leong, R. T. Gray, J. G. Wood, L.-V. Le, E. S. McBryde, R. Ragonnet, and J. M. Trauer, “Understanding covid-19 dynamics and the effects of interventions in the philippines: A mathematical modelling study,” *The Lancet Regional Health - Western Pacific*, vol. 14, p. 100211, 2021. DOI: 10.1016/j.lanwpc.2021.100211

[8] M. C. T. Lagura, R. J. A. David, and E. P. de Lara-Tuprio, *Mathematical Modelling for COVID-19 Dynamics with Vaccination Class*. Cham: Springer International Publishing, 2022, pp. 355–375. ISBN 978-3-031-04028-3. DOI: 10.1007/978-3-031-04028-3_23

[9] R. L. Caga-anan, M. N. Raza, G. S. G. Labrador, E. B. Metillo, P. del Castillo, and Y. Mammeri, “Effect of vaccination to covid-19 disease progression and herd immunity,” *Computational and Mathematical Biophysics*, vol. 9, no. 1, pp. 262–272, 2021. DOI: 10.1515/cmb-2020-0127

[10] H. J. Campos, M. N. Raza, J. P. Arcede, J. G. T. Martinez, and R. L. Caga-anan, “Vaccination and variants: A covid-19 multi-strain model evolution for the philippines,” *Frontiers in Applied Mathematics and Statistics*, vol. 9, 2023. DOI: 10.3389/fams.2023.1029018

[11] S. Lang, *A First Course in Calculus: Chapter 6: Applications of Differentiation*, ser. Springer Science, 2001.

[12] L. Perko, *Differential equations and dynamical systems*, 3rd ed., vol. 7. New York, NY: Springer New York, 2001. DOI: 10.1007/978-1-4613-0003-8

[13] V. Hutson and K. Schmitt, “Permanence and the dynamics of biological systems,” *Mathematical Biosciences*, vol. 111, no. 1, pp. 1–71, Sep. 1992. DOI: 10.1016/0025-5564(92)90078-B

[14] 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,” *Journal of Mathematical Biology*, vol. 28, no. 4, 1990. DOI: 10.1007/BF00178324

[15] P. van den Driessche and J. Watmough, “Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission,” *Mathematical Biosciences*, vol. 180, no. 1–2, pp. 29–48, 2002. DOI: 10.1016/S0025-5564(02)00108-6

[16] C. D. S. Estadilla, J. Uyheng, E. P. de Lara-Tuprio, T. R. Teng, J. M. R. Macalalag, and M. R. J. E. Estuar, “Impact of vaccine supplies and delays on optimal control of the covid-19 pandemic: mapping interventions for the philippines,” *Infectious Diseases of Poverty*, vol. 10, no. 1, p. 107, 2021. DOI: 10.1186/s40249-021-00886-5

[17] Philippine Statistics Authority, “Birth, Marriage, and Death Statistics for 2022,” `https://psa.gov.ph/content/birth-marriage-and-death-statistics-2022-preliminary-31-january-2023`, accessed: 2023-02-27.