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

© 2023 by author(s)
Qualitative analysis of a mathematical model of COVID-19 with intervention strategies in the Philippines

Rolly N. Apdo¹,²* and Rolando N. Paluga²

Department of Mathematics, Caraga State University, Butuan City, Philippines

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 have 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 frontliners 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...
We made the following assumptions: constant), \( N \) (individuals), moderate and mild infected individuals \( t \), critical and severe infected individuals \( E \), exposed and tested individuals \( E \), exposed and contact traced individuals \( E \), critical and severe infected individuals \( I \), moderate and mild infected individuals \( I \), asymptomatic infected individuals \( I \), recovered individuals \( R \), and vaccinated individuals \( V \). For any time \( t \), the total population (assumed constant), \( N(t) \) is given by

\[
N(t) = S(t) + E(t) + E_d(t) + E_c(t) + I_c(t) + I_m(t) + I_a(t) + R(t) + V(t).
\]

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, \( (SEIR) \) as follows: The susceptible population is replenished by a birth rate \( \theta \). The natural death rate in each compartment is represented by \( \mu \). The susceptible individuals contract the virus after coming into contact with exposed individuals at the rate \( \alpha_s \). The susceptible individuals move to the vaccination compartment at a vaccination rate \( \nu \). The vaccinated individuals may also contract the virus after coming in contact with exposed individuals at the rate \( \alpha_v \). Individuals who have been exposed are tracked down and tested if necessary. Some exposed individuals undergo direct testing at a rate of \( \beta_d \). While some exposed individuals are contact traced at a rate of \( \beta_c \). The contact-traced individuals who exhibit symptoms will be tested at a rate of \( \gamma \). 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 \( \sigma_c, \sigma_m \), and \( \sigma_a \) respectively. The moderately and mildly infected individuals develop more serious symptoms and move to severe or critical compartment at the rate \( \phi \). The induced death rate due to COVID-19 to critically and severely infected individuals is represented by \( \delta \). 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 \( \rho_c, \rho_m \), and \( \rho_a \) respectively. The descriptions of the parameters are summarized in Table 1.

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

\[
\begin{align*}
\frac{dS}{dt} &= \theta \, N - (\alpha_s \, E + \nu + \mu) \, S, \\
\frac{dE}{dt} &= (\alpha_s \, S + \alpha_v \, V) \, E - (\beta_d + \beta_c + \mu) \, E, \\
\frac{dE_d}{dt} &= \beta_d \, E + \gamma \, E_c - (\sigma_c + \sigma_m + \sigma_a + \mu) \, E_d, \\
\frac{dE_c}{dt} &= \beta_c \, E - (\gamma + \mu) \, E_c, \\
\frac{dI_c}{dt} &= \sigma_c \, E_d + \phi \, I_m - (\rho_c + \delta + \mu) \, I_c, \\
\frac{dI_m}{dt} &= \sigma_m \, E_d - (\rho_m + \phi + \mu) \, I_m, \\
\frac{dI_a}{dt} &= \sigma_a \, E_d - (\rho_a + \mu) \, I_a, \\
\frac{dR}{dt} &= \rho_c \, E_d + \rho_m \, I_m + \rho_a \, I_a - \mu \, R, \\
\frac{dV}{dt} &= \nu \, S - (\alpha_s \, E + \mu) \, V.
\end{align*}
\]
with the positive initial conditions:

\[ S(0) = S_0 \geq 0, \quad E(0) = E_0 \geq 0, \quad E_a(0) \geq 0, \quad E_c(0) = E_{c0} \geq 0, \quad I_a(0) \geq 0, \quad I_m(0) = I_{m0} \geq 0, \quad I_s(0) \geq 0, \quad R(0) = R_0 \geq 0, \quad V(0) = V_0 \geq 0. \] (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 \((S(t), E(t), E_a(t), E_c(t), I_a(t), I_m(t), I_s(t), R(t), V(t))\) of the system (1) remains nonnegative for all \(t \geq 0\).

**Proof.** Assume that \(S(0) \geq 0, E(0) \geq 0, E_a(0) \geq 0, E_c(0) \geq 0, I_a(0) \geq 0, I_m(0) \geq 0, I_s(0) \geq 0, R(0) \geq 0, V(0) \geq 0\). 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

\[
S(t) = \exp \left( - \int_0^t \tau(u) du \right) \left( \theta N \int_0^t \exp \left( \int_0^u \tau(v) dv \right) du + S(0) \right).
\]

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

\[
\frac{dE(t)}{dt} = (\alpha_S S + \alpha_V V) E - (\beta_d + \beta_c + \mu) E
\]

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

\[
\frac{dV(t)}{dt} = \nu S - (\alpha_v E + \mu) V \geq - (\alpha_v E + \mu) V \text{ (since } S \geq 0\).
\]

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

\[
E_a(t) \geq E_a(0) \exp \left( - \int_0^t (\sigma_c + \sigma_m + \sigma_a + \mu) du \right) > 0,
\]

\[
E_c(t) \geq E_c(0) \exp \left( - \int_0^t (\gamma + \mu) du \right) > 0,
\]

\[
I_a(t) \geq I_a(0) \exp \left( - \int_0^t (\rho_c + \alpha + \mu) du \right) > 0,
\]

\[
I_m(t) \geq I_m(0) \exp \left( - \int_0^t (\rho_m + \phi + \mu) du \right) > 0,
\]

\[
R(t) \geq R(0) \exp \left( - \int_0^t \mu du \right) > 0.
\]

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

**Theorem 2.** Under the initial conditions (2), the solution \((S(t), E(t), E_a(t), E_c(t), I_a(t), I_m(t), I_s(t), R(t), V(t))\) 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

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dE_d}{dt} + \frac{dE_c}{dt} + \frac{dI_c}{dt} + \frac{dI_m}{dt} + \frac{dI_a}{dt} + \frac{dR}{dt} + \frac{dV}{dt}$$

$$= \theta N - \mu N - \delta I_c$$

$$\Rightarrow \frac{dN}{dt} = 0 \text{ since } \theta N = \mu N + \delta I_c \text{ by the first assumption.}$$

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

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

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

Proof. Let $E^0 = (S^0, E^0, E_d^0, E_c^0, I_c^0, I_m^0, I_a^0, R^0, V^0)$ be an equilibrium point of the model (1), that is

$$\theta N - (\alpha_x E^0 + \nu + \mu) S^0 = 0,$$

$$(\alpha_x S^0 + \alpha_c V^0) E^0 - (\beta_d + \beta_c + \mu) E^0 = 0,$$

$$\beta_d E^0 + \gamma E_c^0 - (\sigma_c + \sigma_m + \sigma_a + \mu) E_c^0 = 0,$$

$$\beta_c E^0 - (\gamma + \mu) E^0 = 0,$$

$$\sigma_c E_d^0 + \phi I_m^0 - (\rho_c + \delta + \mu) I_m^0 = 0,$$

$$\sigma_m E_d^0 - (\rho_m + \phi + \mu) I_m^0 = 0,$$

$$\sigma_a E_d^0 - (\rho_a + \mu) I_a^0 = 0,$$

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

$$\nu S^0 - (\alpha_x E^0 + \mu) V^0 = 0.$$  

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, Exposed and tested, Exposed, Exposed and contact traced, Critical and severe infected, Moderate and mild infected, and Asymptomatic infected compartments.

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

$$F = \begin{bmatrix} (\alpha_x S + \alpha_c V) E & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \end{bmatrix}$$

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

The Jacobian of $F$ and $V$ at $E^0$ are $F$ and $V$, respectively.

$$F = \begin{bmatrix} \frac{\theta N (\mu x + \nu x)}{(\mu + \nu) \mu} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \end{bmatrix} \text{ and }$$

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

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

$$FV^{-1} = \begin{bmatrix} \frac{\theta N (\mu x + \nu x)}{\mu (\mu + \nu) \mu} (\beta_d + \beta_c + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \end{bmatrix}.$$
The basic reproduction number is the dominant eigenvalue of $FV^{-1}$ which is $R_0 = \rho \left(FV^{-1}\right)$. Thus,

$$R_0 = \frac{\theta N \left(\mu \alpha_s + \nu \alpha_v\right)}{\mu \left(\mu + \nu\right) \left(\beta_d + \beta_e + \mu\right)}.$$  

### 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 $E^* = (S^*, E^*, E_d^*, E_c^*, I_c^*, I_m^*, I_a^*, R^*, V^*)$ 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 $E^* = (S^*, E^*, E_d^*, E_c^*, I_c^*, I_m^*, I_a^*, R^*, V^*)$ be an endemic equilibrium point of the model (1), that is

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

Suppose that $E^* = (S^*, E^*, E_d^*, E_c^*, I_c^*, I_m^*, I_a^*, R^*, V^*)$ 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).

$$S^* = \frac{\theta N}{\alpha_s E^* + \nu + \mu}.$$

We solve for $E^*_c$ from the fourth equation.

$$E^*_c = \frac{\beta_c E^*}{\gamma + \mu}.$$

We substitute $E^*_c$ to the third equation and solve for $E^*_d$.

$$\begin{align*}
\beta_d E^* + \gamma E^*_c - (\sigma_c + \sigma_m + \sigma_a + \mu) E^*_d &= 0, \\
\beta_d E^* + \gamma \frac{\beta_c E^*}{\gamma + \mu} - (\sigma_c + \sigma_m + \sigma_a + \mu) E^*_d &= 0, \\
\Rightarrow E^*_d &= \frac{\left(\beta_c + \beta_d \gamma + \beta_e \mu\right) E^*}{(\gamma + \mu) \left(\sigma_c + \sigma_m + \sigma_a + \mu\right)},
\end{align*}$$

We substitute $E^*_d$ to the sixth equation and solve for $I^*_m$.

$$\begin{align*}
\sigma_m E^*_d - (\rho_m + \phi + \mu) I^*_m &= 0, \\
\sigma_m \frac{\left(\beta_c + \beta_d \gamma + \beta_e \mu\right) E^*}{(\gamma + \mu) \left(\sigma_c + \sigma_m + \sigma_a + \mu\right)} - (\rho_m + \phi + \mu) I^*_m &= 0, \\
\Rightarrow I^*_m &= \frac{\sigma_m \left(\beta_d + \beta_e \mu\right) E^*}{(\gamma + \mu) \left(\sigma_c + \sigma_m + \sigma_a + \mu\right)}.
\end{align*}$$

We substitute $E^*_d$ to the seventh equation and solve for $I^*_a$.

$$\begin{align*}
\sigma_a E^*_d - (\rho_a + \mu) I^*_a &= 0, \\
\sigma_a \frac{\left(\beta_d + \beta_e \mu\right) E^*}{(\gamma + \mu) \left(\sigma_c + \sigma_m + \sigma_a + \mu\right)} - (\rho_a + \mu) I^*_a &= 0, \\
\Rightarrow I^*_a &= \frac{\sigma_a \left(\beta_d + \beta_e \mu\right) E^*}{(\gamma + \mu) \left(\sigma_c + \sigma_m + \sigma_a + \mu\right)}.
\end{align*}$$

We substitute $E^*_d$ and $I^*_m$ to the fifth equation and solve for $I^*_c$.

$$\sigma_c E^*_d + \phi I^*_m - (\rho_c + \delta + \mu) I^*_c = 0.$$

After the substitution and quick algebraic manipulation, we get

$$I^*_c = \frac{\left(\beta_c + \beta_d \gamma + \beta_e \mu\right) \left(\mu \sigma_s + \phi \sigma_m + \phi \sigma_a + \rho_m \sigma_a\right) E^*}{(\gamma + \mu) \left(\sigma_c + \sigma_m + \sigma_a + \mu\right) \left(\rho_c + \delta + \mu\right)}.$$

We substitute $I^*_c$, $I^*_m$, and $I^*_a$ to the eighth equation and solve for $R^*$.

$$\rho_c I^*_c + \rho_m I^*_m + \rho_a I^*_a - \rho R^* = 0.$$

After the substitution and quick algebraic manipulation, we get

$$R^* = \frac{\left(\beta_c + \beta_d \gamma + \beta_e \mu\right) \left(\mu \sigma_s + \phi \sigma_m + \phi \sigma_a + \rho_m \sigma_a\right) E^*}{(\gamma + \mu) \left(\sigma_c + \sigma_m + \sigma_a + \mu\right) \left(\rho_c + \delta + \mu\right)}.$$

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

$$\begin{align*}
(\alpha_s S^* + \alpha_v V^*) E^* - (\beta_d + \beta_e + \mu) E^* &= 0, \\
\Rightarrow E^* &= \frac{\nu S^* - (\alpha_v E^* + \mu) V^*}{(\alpha_s E^* + \nu + \mu)}.
\end{align*}$$

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

$$\begin{align*}
(\alpha_s S^* + \alpha_v V^*) E^* - (\beta_d + \beta_e + \mu) E^* &= 0, \\
\Rightarrow E^* &= \frac{\nu S^* - (\alpha_v E^* + \mu) V^*}{(\alpha_s E^* + \nu + \mu)}.
\end{align*}$$

After the substitution and lengthy algebraic manipulation, we get

$$\begin{align*}
E^* &= \frac{\rho (\alpha_s E^* + \nu + \mu)}{\alpha_s \theta} \left(\frac{\rho \alpha_s \theta}{\alpha_s \theta} - (\alpha_v E^* + \mu) V^* = 0
\Rightarrow V^* &= \frac{\nu S^* - (\alpha_v E^* + \mu) V^*}{(\alpha_s E^* + \nu + \mu)}.
\end{align*}$$

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, \( E^0 = (S^0, 0, 0, 0, 0, 0, V^0) \) is locally asymptotically stable if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

Proof. The Jacobian of the system (1) is given by
\[
J = \begin{bmatrix}
-a_S S & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & a_{d,2} & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & a_{s,3,3} & \gamma & 0 & 0 & 0 \\
0 & 0 & 0 & a_{c,4,4} & \phi & 0 & 0 \\
0 & 0 & 0 & 0 & a_{m,5,5} & \phi & 0 \\
0 & 0 & 0 & 0 & 0 & a_{m,7,7} & 0 \\
\nu & -V & 0 & 0 & 0 & 0 & -\mu \\
\end{bmatrix},
\]
where
\[
a_{1,1} = -\alpha_S S - \nu, \quad a_{2,2} = \alpha_S S + V\alpha_v - \beta_c - \beta_d - \mu, \\
a_{3,3} = -\sigma_c - \sigma_m - \sigma_a - \mu, \quad a_{4,4} = -\gamma - \mu, \\
a_{5,5} = -\rho_m - \delta - \mu, \\
a_{6,6} = -\rho_m - \delta - \mu, \\
a_{7,7} = -\rho_a - \mu, \quad \text{and} \quad a_{9,9} = -\alpha_S E - \mu.
\]

The Jacobian matrix (4) evaluated at the disease-free equilibrium is of the form
\[
J(E^0) = \begin{bmatrix}
a_{1,1} & -\alpha_S S^0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & a_{d,2} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & a_{s,3,3} & \gamma & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & a_{c,4,4} & \phi & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & a_{m,5,5} & \phi & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & a_{m,7,7} & 0 & 0 & 0 \\
\nu & -V & 0 & 0 & 0 & 0 & -\mu & 0 & 0 \\
\end{bmatrix},
\]
where
\[
a_{1,1} = -\mu - \nu, \quad a_{2,2} = \alpha_S S^0 + \alpha_v V^0 - \beta_c - \beta_d - \mu, \\
a_{3,3} = -\sigma_c - \sigma_m - \sigma_a - \mu, \quad a_{4,4} = -\gamma - \mu, \\
a_{5,5} = -\rho_m - \delta - \mu, \\
a_{6,6} = -\rho_m - \delta - \mu, \\
a_{7,7} = -\rho_a - \mu, \quad \text{and} \quad a_{9,9} = -\alpha_S E - \mu.
\]

Theorem 6. The disease-endemic equilibrium, \( E^* = (S^*, E^*, E_v, E_r, I^*, I_m, I_a, R^*, V^*) \) 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
\[
J(E^*) = \begin{bmatrix}
a_{1,1} & -\alpha_S S^* & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & a_{d,2} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & a_{s,3,3} & \gamma & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & a_{c,4,4} & \phi & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & a_{m,5,5} & \phi & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & a_{m,7,7} & 0 & 0 & 0 \\
\nu & -V & 0 & 0 & 0 & 0 & -\mu & 0 & 0 \\
\end{bmatrix},
\]
where
\[
a_{1,1} = \alpha_S S^* + \alpha_v V^* - \beta_c - \beta_d - \mu, \\
a_{2,2} = \alpha_S S^* + \alpha_v V^* - \beta_c - \beta_d - \mu, \\
a_{3,3} = -\sigma_c - \sigma_m - \sigma_a - \mu, \\
a_{4,4} = -\gamma - \mu, \\
a_{5,5} = -\rho_m - \delta - \mu, \\
a_{6,6} = -\rho_m - \delta - \mu, \\
a_{7,7} = -\rho_a - \mu, \quad \text{and} \quad a_{9,9} = -\alpha_S E - \mu.
\]
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

$$
j_1(\mathcal{E}^*) = \begin{bmatrix}
    a_{1,1} & -a_sS^* & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
    0 & a_{2,2} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
    \beta_d & a_{3,3} & \gamma & 0 & 0 & 0 & 0 & 0 & 0 \\
    0 & \sigma_c & 0 & a_{5,5} & \phi & 0 & 0 & 0 & 0 \\
    0 & 0 & \sigma_m & 0 & 0 & a_{6,6} & 0 & 0 & 0 \\
    0 & 0 & 0 & \sigma_d & 0 & 0 & 0 & a_{7,7} & 0 \\
\end{bmatrix},$
$$

where $a_{1,1} = -\mu - \nu$, $a_{2,2} = \alpha_sS^* + \alpha_eV^* - \beta_c - \beta_d - \mu$, $a_{3,3} = -\sigma_c - \sigma_m - \sigma_a - \mu$, $a_{4,4} = -\gamma - \mu$, $a_{5,5} = -\rho_c - \delta - \mu$, $a_{6,6} = -\rho_m - \phi - \mu$, 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 $-(\mu + \nu)$, $-\left((\rho_c + \delta + \mu)\right)$, and $-\left((\rho_a + \mu)\right)$, 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.

$$
j_2(\mathcal{E}^*) = \begin{bmatrix}
    b_{1,1} & 0 & 0 & 0 & 0 \\
    \beta_d & b_{2,2} & \gamma & -\mu & 0 \\
    \beta_c & 0 & 0 & 0 & -\mu \\
    \sigma_m & 0 & 0 & 0 & b_{3,3} \\
\end{bmatrix},$
$$

where $b_{1,1} = \alpha_sS^* + \alpha_eV^* - \beta_c - \beta_d - \mu$, $b_{2,2} = -\sigma_c - \sigma_m - \sigma_a - \mu$, 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 $J_3(\mathcal{E}^*)$, 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.

$$
j_3(\mathcal{E}^*) = \begin{bmatrix}
    c_{1,1} & 0 & 0 & 0 \\
    \beta_d & c_{2,2} & \gamma & -\mu \\
    \beta_c & 0 & 0 & 0 \\
\end{bmatrix},$
$$

where $c_{1,1} = \alpha_sS^* + \alpha_eV^* - \beta_c - \beta_d - \mu$ 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 $-(\gamma + \mu)$. 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.

$$
j_4(\mathcal{E}^*) = \begin{bmatrix}
    \alpha_sS^* + \alpha_eV^* - \beta_c - \beta_d - \mu & 0 \\
    \beta_c & -\gamma - \mu \\
\end{bmatrix}.$$

In a similar way, the 2nd column of $J_4(\mathcal{E}^*)$ contains only the diagonal element which form the negative eigenvalue $-(\gamma + \mu)$. The remaining eigenvalue is $\alpha_sS^* + \alpha_eV^* - \beta_c - \beta_d - \mu$. Now, we can see that $\nu = 9.97 \times 10^{-4}$.

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 $\nu$ and contact tracing rate $\beta_c$ to the basic reproduction number $R_0$.

Table 2. Parameter values for the model simulation

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\alpha_s$</td>
<td>$2.02 \times 10^{-9}$</td>
<td>[4]</td>
</tr>
<tr>
<td>$\alpha_e$</td>
<td>$4.05 \times 10^{-10}$</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\rho_c$</td>
<td>$1.23 \times 10^{-1}$</td>
<td>[4]</td>
</tr>
<tr>
<td>$\rho_m$</td>
<td>$1.23 \times 10^{-1}$</td>
<td>[4]</td>
</tr>
<tr>
<td>$\rho_a$</td>
<td>$1.23 \times 10^{-1}$</td>
<td>[4]</td>
</tr>
<tr>
<td>$\theta$</td>
<td>$2.93 \times 10^{-5}$</td>
<td>[17]</td>
</tr>
<tr>
<td>$\mu$</td>
<td>$1.56 \times 10^{-5}$</td>
<td>[17]</td>
</tr>
<tr>
<td>$\nu$</td>
<td>$9.97 \times 10^{-4}$</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\sigma_c$</td>
<td>$5.00 \times 10^{-2}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\sigma_m$</td>
<td>$1.60 \times 10^{-3}$</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\sigma_a$</td>
<td>$1.29 \times 10^{-1}$</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>$4.33 \times 10^{-3}$</td>
<td>Estimated</td>
</tr>
<tr>
<td>$\beta_c$</td>
<td>$2.00 \times 10^{-1}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\beta_d$</td>
<td>$4.78 \times 10^{-4}$</td>
<td>Assumed</td>
</tr>
<tr>
<td>$\beta_e$</td>
<td>$4.32 \times 10^{-2}$</td>
<td>Assumed</td>
</tr>
</tbody>
</table>

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.

5.2. Impact of $\nu$ on $R_0$

Figure 3 illustrates the impact of the vaccination rate $\nu$ on the basic reproduction number $R_0$. According to Figure 3, as the vaccination rate $\nu$ increases, the basic reproduction number $R_0$ decreases. Therefore, the government should aim to increase
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 \( R_0(\nu) = \frac{\theta N (\mu \alpha_s + \nu \alpha_v)}{\mu (\beta_d + \beta_c + \mu)} \) 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 \( R_0'(\nu) = \frac{\theta N \mu (\nu - \alpha_s)}{\mu (\beta_d + \beta_c + \mu) - \theta N \alpha_v} < 0 \) 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 \( \nu > \frac{\theta N \mu \alpha_s - \mu^2 (\beta_d + \beta_c + \mu)}{\mu (\beta_d + \beta_c + \mu) - \theta N \alpha_v} > 0 \). Otherwise, the spread of the disease will persist.

**Proof.** Let \( \nu > \nu_0 \) where \( \nu_0 = \frac{\theta N \mu \alpha_s - \mu^2 (\beta_d + \beta_c + \mu)}{\mu (\beta_d + \beta_c + \mu) - \theta N \alpha_v} > 0 \).

By Theorem 7, \( R_0(\nu) \) is decreasing for all \( \nu \in [0, 1] \). It follows that

\[
R_0(\nu) < R_0(\nu_0)
\]

\[
= \frac{\theta N (\mu \alpha_s + \frac{\theta N \mu \alpha_s - \mu^2 (\beta_d + \beta_c + \mu)}{\mu (\beta_d + \beta_c + \mu) - \theta N \alpha_v}) \alpha_v}{\mu (\mu + \frac{\theta N \mu \alpha_s - \mu^2 (\beta_d + \beta_c + \mu)}{\mu (\beta_d + \beta_c + \mu) - \theta N \alpha_v}) (\beta_d + \beta_c + \mu)}
\]

\[
= 1.
\]

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

The disease is likely to persist in the population based on the given parameter values. However, if the parameter \( \nu \) 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 \( \beta_c \) to \( R_0 \)

Figure 4 shows the impact of the contact tracing rate \( \beta_c \) to the basic reproduction number \( R_0 \).

![Figure 4](image)

Based on Figure 4, as the contact tracing rate \( \beta_c \) 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 \( R_0(\beta_c) = \frac{\theta N (\mu \alpha_s + \nu \alpha_v)}{\mu (\beta_d + \beta_c + \mu)} \) 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 \( \nu = -\beta_d + \mu < 0 \). It follows that this function is continuous and differentiable for all \( \beta_c \in [0, 1] \).

Observe that \( R_0'(\beta_c) = \frac{\theta N (\mu \alpha_s + \nu \alpha_v)}{\mu (\beta_d + \beta_c + \mu)} < 0 \) since \( \alpha_s < \alpha_v \).

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 \( \beta_c > \frac{\theta N (\mu \alpha_s + \nu \alpha_v)}{\mu (\beta_d + \beta_c + \mu)} \). Otherwise, the spread of the disease will persist.

**Proof.** Let \( \beta_c > \beta^0_c \) where \( \beta^0_c = \frac{\theta N (\mu \alpha_s + \nu \alpha_v)}{\mu (\beta_d + \mu)} < \beta_d + \mu \).

By Theorem 9, \( R_0(\beta_c) \) is decreasing for all \( \beta_c \in [0, 1] \). It follows that

\[
R_0(\beta_c) < R_0(\beta^0_c)
\]

\[
= \frac{\frac{\theta N (\mu \alpha_s + \nu \alpha_v)}{\mu (\beta_d + \mu + \nu)} - (\beta_d + \mu)}{\mu (\beta_d + \mu + \nu)}
\]

\[
= 1.
\]

This means that the disease will eventually die out as long as \( \beta_c > \frac{\theta N (\mu \alpha_s + \nu \alpha_v)}{\mu (\beta_d + \mu + \nu)} - (\beta_d + \mu) \). The spread of the disease will persist if \( \beta_c < \frac{\theta N (\mu \alpha_s + \nu \alpha_v)}{\mu (\beta_d + \mu + \nu)} - (\beta_d + \mu) \).
If the contact tracing rate $\beta_c$ is greater than $0.08847694$, the disease outbreak is likely to be controlled and eventually eliminated. On the other hand, if $\beta_c$ 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