

Numerical Simulation and Sensitivity Analysis of COVID-19 Transmission Involves Virus in the Environment

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Abstract

This paper is aimed to develop a new COVID-19 mathematical model involving viruses in the environment. In this mathematical model, the human population is divided into five subpopulations: susceptible, exposed, infected, hospitalized, and cured individuals. In addition, the model also contains the virus population in the environment. Infection in the model occurs due to interactions between susceptible individual subpopulations and infected individuals and hospitalizations, as well as the spread of the virus in the environment. Based on the results of dynamic analysis, this model has two equilibrium points, the disease-free and endemic equilibrium points. The disease-free equilibrium point always exists, and both equilibrium points are locally asymptotically stable if they meet the Routh-Hurwitz criteria. Model sensitivity analysis was carried out on model parameters that affect the basic reproduction number with the most sensitive parameters are the natural death rate, the recruitment rate, the transmission rate of the virus in the environment, the virus clearance rate, and the rate of wearing PPE (Personal Protective Equipment), as well as the parameter that does not affect the basic reproduction number that is the rate of leaving the recovered population. Numerical simulations performed show results in accordance with the analysis, also from the simulations can be concluded that the increase (or decrease) of the transmission rate of the virus in an environment that has a higher sensitivity index has more significant influences on the basic reproduction number and the number of infected population than the transmission rate of hospitalized individuals.

Keywords: Basic Reproduction Number, Dynamics Analysis, Epidemic Models of COVID-19, Local Stability Analysis, Sensitivity Analysis.

INTRODUCTION

Coronavirus is an infectious disease called acute respiratory syndrome by the International Virus Taxonomy community. In general, the coronavirus is known as COVID-19. This disease is transmitted through direct contact such as touch, body fluids, or air when sneezing or coughing [1]. Researchers are still tracking the spread of COVID-19 in all parts of the world. To describe the dynamics of the spread of the virus in a population, a mathematical model is needed so that the solutions can be obtained in handling the spread of the virus.

Mathematical modeling is a field of mathematics that describes real-world problems in mathematical statements. The COVID-19 model is an epidemic model where the model is used to describe the infectious disease COVID-19. The COVID-19 epidemic model has been studied by several researchers.

Researchers [2-5] developed a model of three SIR subpopulations. Then Victor [6] developed this model by adding a subpopulation of exposed individuals (E) and the assumption that subpopulations of individuals who recovered could be susceptible again (R). Zeb *et al.* [7] also developed the previous model researched [2,4] by adding a subpopulation of exposed individuals (E) and isolated individual subpopulations (Q) to the model.

Naik *et al.* [8] modeled the effect of COVID-19 transmission on contaminated environments in India using seven subpopulations, namely susceptible (S), exposed (E), asymptomatic infected (A), infected with symptoms (I), confirmed (C), hospitalized (H), and recovered (R) with the virus population in the environment (V). The intended population is a combined population that interacts homogeneously where each individual interacts directly, without isolation or quarantine, and without closing access to a particular area.

Masandawa *et al.* [9] presented a COVID-19 model consisting of five subpopulations namely, susceptible (S), exposed (E), infected (I), hospitalized (H), and recovered (R). This model assumes that individuals who have

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recovered have the possibility to become vulnerable again.

This paper will constructed a mathematical model for COVID-19 by modifying Masandawa et al. [9] model by adding the virus compartment in the environment as in Naik et al. [8]. It is done to determine the virus's behavior in the environment with the constructed model. Then the dynamic analysis of the model is applied by determining the equilibrium point and analyzing its stability. In the final part, a numerical simulation is carried out to describe the results of dynamic analysis using the fourth-order Runge-Kutta method.

MATERIALS AND METHOD

Model Formulation

In this paper, the COVID-19 epidemic model [9] is the main object of the study. The model construction is done by modifying the model of Masandawa et al. [9] by adding the virus compartment, such as in the model of Naik et al. [8].

Determination of the Equilibrium Points

The first step to be done in dynamics analysis is to determine the equilibrium points. The equilibrium points of the model are obtained when the population rate of the system is unchanged or equal to zero. From this condition, the existence properties of equilibrium points are also obtained.

Stability of the Equilibrium Points

The local stability of equilibrium points is analyzed by linearizing the model using the Taylor series. The linearization is done to change the nonlinear model into its linear form.

The approximation used is in the form of a Jacobian matrix. From there, we can determine the eigenvalues or roots of the characteristics equation. The local stability determination is obtained from the absolute of its eigenvalue argument.

Sensitivity Analysis

The sensitivity analysis is done to determine the parameter that mainly affects the spread of the disease. In this stage, the study calculates the sensitivity index of each model parameter that correlates with the basic reproduction number, \mathcal{R}_0 .

Numerical Simulation

The numerical simulation is used to verify the analytical results and illustrate the model's behavior. The approach used is the fourth-order Runge-Kutta method using MATLAB software. In this stage, it is crucial to determine the parameters that match the condition of existences and stability of equilibrium points. The interpretation results of numerical simulations are done as the last step in this stage.

RESULTS AND DISCUSSION

The COVID-19 Model Construction

This section will give a mathematical model of the COVID-19 epidemic involving viruses in the environment. The epidemic model used as a reference is the Masandawa et al. [9] epidemic model involving viruses in the environment, as referred to by Naik et al. [8]. The compartment diagram for the COVID-19 epidemic model involving viruses in the environment is shown in Figure 1.

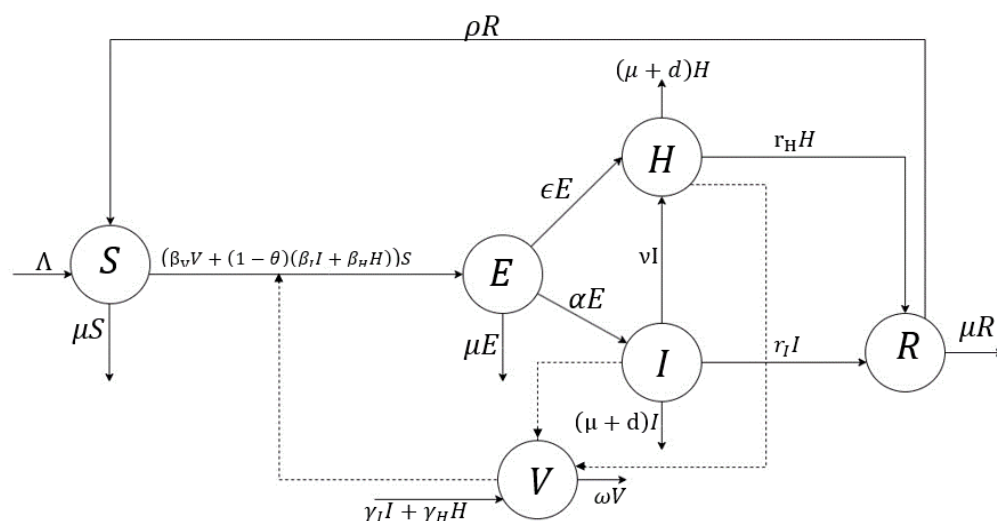


Figure 1. The COVID-19 epidemic model involving viruses in the environment

The assumptions used in the mathematical model are as follows: 1) individuals who have recovered will be susceptible again, 2) the virus population in the environment (V) is spread by infected (I) and hospitalized individuals (H), 3) susceptible individuals will be infected with the virus by interacting with infected and hospitalized individuals as well as interactions with viruses in the environment, 4) interactions between susceptible and hospitalized individuals occur when susceptible individuals are in the hospital, and 5) there are significant influences in the use of personal protective equipment (PPE) to prevent the spread of the virus.

The COVID-19 epidemic model involving virus in the environment consists of five groups of human subpopulations; susceptible (S), exposed (E), infected (I), hospitalized (H), and recovered (R) subpopulations, also the virus population in the environment (V). The COVID-19 epidemic model involving virus in the environment is given by the following equation. The parameter description can be seen in Table 1.

$$\begin{aligned}\frac{dS}{dt} &= \Lambda + \rho R - (\mu + \beta_V V + (1 - \theta)(\beta_I I + \beta_H H))S \\ \frac{dE}{dt} &= (\beta_V V + (1 - \theta)(\beta_I I + \beta_H H))S - (\alpha + \epsilon + \mu)E \\ \frac{dI}{dt} &= \alpha E - (r_I + \nu + d + \mu)I \\ \frac{dH}{dt} &= \epsilon E + \nu I - (r_H + d + \mu)H \\ \frac{dR}{dt} &= r_I I + r_H H - (\rho + \mu)R \\ \frac{dV}{dt} &= \gamma_I I + \gamma_H H - \omega V\end{aligned}\quad (1)$$

and

$$N = S + E + I + H + R$$

Table 1. Description of all parameters

Parameter	Description
Λ	Recruitment rate of susceptible
μ	Natural death rate
ρ	Rate of leaving the recovered population
β_V	virus transmission rate in the environment
β_I	Transmission rate of infected individuals
β_H	Transmission rate of hospitalized individuals
θ	The rate of wearing PPE
α	'Exposed individuals become infected' rate
ϵ	'Exposed individuals are hospitalized' rate
ν	'Infected individuals are hospitalized' rate
r_I	Recovery rate of infected individuals
r_H	Recovery rate of hospitalized individuals
d	The disease induced death rate
γ_I	Virus released rate via infected individuals
γ_H	Virus release rate via the hospitalized individuals
ω	The virus clearance rate

Equilibrium points

The system equilibrium points are obtained when $\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dH}{dt} = \frac{dR}{dt} = \frac{dV}{dt} = 0$, such that

$$\begin{aligned}\Lambda + \rho R - (\mu + \beta_V V + (1 - \theta)(\beta_I I + \beta_H H))S &= 0, \\ (\beta_V V + (1 - \theta)(\beta_I I + \beta_H H))S - k_1 E &= 0, \\ \alpha E - k_2 I &= 0, \\ \epsilon E + \nu I - k_3 H &= 0, \\ r_I I + r_H H - k_4 R &= 0, \\ \gamma_I I + \gamma_H H - \omega V &= 0,\end{aligned}\quad (2)$$

where

$$\begin{aligned}k_1 &= \alpha + \epsilon + \mu, \\ k_2 &= r_I + \nu + d + \mu, \\ k_3 &= r_H + d + \mu, \\ k_4 &= \rho + \mu.\end{aligned}$$

Disease-free equilibrium

$$Y_0 = (S_0, E_0, I_0, H_0, R_0, V_0) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right),$$

With assumptions $S \neq 0, E \neq 0, I \neq 0, H \neq 0, R \neq 0$, and $V \neq 0$, we get the endemic equilibrium

$Y^* = (S^*, E^*, I^*, H^*, R^*, V^*)$ where

$$\begin{aligned}S^* &= \frac{\Lambda + a_3 E^*}{\mu + (a_4 + a_5) E^*} \\ I^* &= \frac{\alpha E^*}{k_2} \\ H^* &= \frac{E^*}{k_3} \left(\epsilon + \frac{\alpha \nu}{k_2} \right) \\ R^* &= \frac{E^*}{k_4} \left(\frac{\alpha r_I}{k_2} + \frac{r_H}{k_3} \left(\epsilon + \frac{\alpha \nu}{k_2} \right) \right) \\ V^* &= \frac{E^*}{\omega} \left(\frac{\alpha \gamma_I}{k_2} + \frac{\gamma_H}{k_3} \left(\epsilon + \frac{\alpha \nu}{k_2} \right) \right)\end{aligned}\quad (3)$$

and

$$\begin{aligned}a_1 &= \frac{\alpha}{k_2}, a_2 = \frac{1}{k_3} \left(\epsilon + \frac{\alpha \nu}{k_2} \right), \\ a_3 &= \frac{\rho}{k_4} (a_1 r_I + a_2 r_H), \\ a_4 &= \frac{\beta_V}{\omega} (\gamma_I a_1 + \gamma_H a_2), \\ a_5 &= (1 - \theta)(\beta_I a_1 + \beta_H a_2).\end{aligned}$$

Endemic equilibrium Y^* exists when

$$k_1 \mu > \Lambda(a_4 + a_5) \text{ and } a_3 > k_1$$

or

$$k_1 \mu < \Lambda(a_4 + a_5) \text{ and } a_3 < k_1.$$

Basic reproduction number

The next generation matrix method was used to obtain the basic reproduction number (\mathcal{R}_0). The components forming the next generation matrix consist of the infected population group, i.e

$$\begin{aligned}\frac{dE}{dt} &= (\beta_V V + (1 - \theta)(\beta_I I + \beta_H H))S - k_1 E, \\ \frac{dI}{dt} &= \alpha E - k_2 I,\end{aligned}\quad (4)$$

$$\begin{aligned}\frac{dH}{dt} &= \epsilon E + \nu I - k_3 H, \\ \frac{dV}{dt} &= \gamma_I I + \gamma_H H - \omega V.\end{aligned}$$

to differ new infection, so (4) is changed to

$$\frac{dx_i}{dt} = \mathcal{F}_i(x) - \mathcal{G}_i(x),$$

Where \mathcal{F}_i is the new infection rate, \mathcal{G}_i is the transfer of infection between subpopulations and x are the infected subpopulations such that $x_1 = E, x_2 = I, x_3 = H, x_4 = V$.

$$\mathcal{F}_i = \begin{pmatrix} (\beta_V V + (1-\theta)(\beta_I I + \beta_H H))S \\ 0 \\ 0 \\ 0 \end{pmatrix},$$

$$\mathcal{G}_i = \begin{pmatrix} k_1 E \\ k_2 I - \alpha E \\ k_3 H - \epsilon E - \nu I \\ \omega V - \gamma_I I - \gamma_H H \end{pmatrix}.$$

The spectral radius of the next generation matrix $\mathcal{K} = \mathcal{F}\mathcal{G}^{-1}$ as the basic reproduction number \mathcal{R}_0 and $Y_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$, where,

$$\mathcal{F} = \left(\frac{\partial f_i(x_i)}{\partial x_i}\right) = \begin{pmatrix} 0 & \frac{\beta_I \Lambda}{\mu}(1-\theta) & \frac{\beta_H \Lambda}{\mu}(1-\theta) & \frac{\beta_V \Lambda}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$\mathcal{G} = \left(\frac{\partial g_i(x_i)}{\partial x_i}\right) = \begin{pmatrix} k_1 & 0 & 0 & 0 \\ -\alpha & k_2 & 0 & 0 \\ -\epsilon & -\nu & k_3 & 0 \\ 0 & -\gamma_I & -\gamma_H & \omega \end{pmatrix}.$$

After computing the eigen values of the matrix $\mathcal{K} = \mathcal{F}\mathcal{G}^{-1}$, we have the expression of \mathcal{R}_0 ,

$$\begin{aligned}\mathcal{R}_0 &= \frac{\alpha \beta_I \Lambda (1-\theta)}{\mu k_1 k_2} + \frac{\beta_H \Lambda (1-\theta)(\alpha \nu - \epsilon k_2)}{\mu k_1 k_2 k_3} \\ &+ \frac{\beta_V \Lambda (\alpha \gamma_I k_3 + \alpha \gamma_H \nu + \gamma_H \epsilon k_2)}{\mu k_1 k_2 k_3 \omega} \\ &+ \frac{\omega k_3 \alpha \beta_I \Lambda (1-\theta) + \omega \beta_H \Lambda (1-\theta)(\alpha \nu - \epsilon k_2)}{\mu k_1 k_2 k_3 \omega} \\ &+ \frac{\beta_V \Lambda (\alpha \gamma_I k_3 + \alpha \gamma_H \nu + \gamma_H \epsilon k_2)}{\mu k_1 k_2 k_3 \omega}\end{aligned}$$

$$\mathcal{R}_0 = \mathcal{R}_0^{isp} + \mathcal{R}_0^{ive},$$

where

$$\begin{aligned}\mathcal{R}_0^{isp} &= \frac{\omega k_3 \alpha \beta_I \Lambda (1-\theta) + \omega \beta_H \Lambda (1-\theta)(\alpha \nu - \epsilon k_2)}{\mu k_1 k_2 k_3 \omega}, \\ \mathcal{R}_0^{ive} &= \frac{\beta_V \Lambda (\alpha \gamma_I k_3 + \alpha \gamma_H \nu + \gamma_H \epsilon k_2)}{\mu k_1 k_2 k_3 \omega}.\end{aligned}$$

Here, \mathcal{R}_0^{isp} indicates the average number of secondary infections generated by a single infected individual introduced to susceptible populations directly during their life cycle. \mathcal{R}_0^{ive} indicates the average number of secondary infections generated by the virus released into the environment during its life cycle.

Local stability analysis

The general Jacobian matrix associated to system (1) is given by

$$J = \begin{pmatrix} -(\mu + \beta_V V + (1-\theta)(\beta_I I + \beta_H H)) & 0 & -(1-\theta)\beta_I S & -(1-\theta)\beta_H S & \rho & -\beta_V \\ \beta_V V + (1-\theta)(\beta_I I + \beta_H H) & -k_1 & (1-\theta)\beta_I S & (1-\theta)\beta_H S & 0 & \beta_V \\ 0 & \alpha & -k_2 & 0 & 0 & 0 \\ 0 & \epsilon & \nu & -k_3 & 0 & 0 \\ 0 & 0 & \gamma_I & \gamma_H & -k_4 & 0 \\ 0 & 0 & \gamma_I & \gamma_H & 0 & -\omega \end{pmatrix}.$$

We will prove the local stability of equilibrium points following theorems with proofs.

Theorem 1. The disease-free equilibrium point Y_0 of the proposed COVID-19 epidemic model (1) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable otherwise.

Proof. Substitute the point Y_0 to the general Jacobian matrix J that yields

$$J(Y_0) = \begin{pmatrix} -\mu & 0 & \frac{-\beta_I \Lambda (1-\theta)}{\mu} & \frac{-\beta_H \Lambda (1-\theta)}{\mu} & \rho & -\beta_V \\ 0 & -k_1 & \frac{\beta_I \Lambda (1-\theta)}{\mu} & \frac{\beta_H \Lambda (1-\theta)}{\mu} & 0 & \beta_V \\ 0 & \alpha & -k_2 & 0 & 0 & 0 \\ 0 & \epsilon & \nu & -k_3 & 0 & 0 \\ 0 & 0 & \gamma_I & \gamma_H & -k_4 & 0 \\ 0 & 0 & \gamma_I & \gamma_H & 0 & -\omega \end{pmatrix}$$

as per the Routh-Hurwitz criterion, for $\mathcal{R}_0 < 1$, the disease-free equilibrium point Y_0 of the proposed model (1) is locally asymptotically stable if all eigenvalues $\lambda_i, i = 1, 2, \dots, 6$ of the matrix $J(Y_0)$ are negative numbers or have negative real parts. We can evaluate these eigenvalues from the following characteristics polynomial

$$|J(Y_0) - \lambda \vec{I}| = 0 \quad (5)$$

where \vec{I} is an identity matrix of order six and λ is the eigenvalues. By using cofactor expansion method, we get $\lambda_1 = -\mu < 0$ and $\lambda_2 = -k_4 < 0$ and the fourth-order Routh-Hurwitz matrix such that, we get the characteristic polynomial of the form

$$P^{Y_0}(\lambda) = \lambda^4 + b_1 \lambda^3 + b_2 \lambda^2 + b_3 \lambda + b_4 = 0$$

where b_1, b_2, b_3 , and b_4 is given in the Box 1.

Therefore, the stability when $\mathcal{R}_0 < 1$ follows the Routh-Hurwitz criterion for fourth-order polynomials, the disease-free equilibrium point is asymptotically stable if and only if

- $b_1 > 0$ and $b_4 > 0$,
- $h_1 = b_1 b_2 - b_3 > 0$,
- $h_2 = b_3(b_1 b_2 - b_3) - b_1^2 b_4 > 0$.

Theorem 2. The endemic equilibrium point Y^* of the proposed COVID-19 epidemic model (1) is locally asymptotically stable if $\mathcal{R}_0 > 1$ and unstable otherwise.

Proof. Substitute the point Y^* to the general Jacobian matrix J that yields

$$J(Y^*) = \begin{pmatrix} -(\mu + n_1) & 0 & -n_2 & -n_3 & \rho & -\beta_V \\ n_1 & -k_1 & n_2 & n_3 & 0 & \beta_V \\ 0 & \alpha & -k_2 & 0 & 0 & 0 \\ 0 & \epsilon & \nu & -k_3 & 0 & 0 \\ 0 & 0 & r_I & r_H & -k_4 & 0 \\ 0 & 0 & \gamma_I & \gamma_H & 0 & -\omega \end{pmatrix},$$

where

$$\begin{aligned} n_1 &= \beta_V V^* + (1 - \theta)(\beta_I I^* + \beta_H H^*), \\ n_2 &= (1 - \theta)\beta_I S^*, \\ n_3 &= (1 - \theta)\beta_H S^*. \end{aligned}$$

as per the Routh-Hurwitz criterion, for $\mathcal{R}_0 > 1$, the endemic equilibrium Y^* of the proposed model (1) is locally asymptotically stable if all eigenvalues $\lambda_i, i = 1, 2, \dots, 6$ of the matrix $J(Y^*)$ are negative numbers or have negative real parts. We can evaluate these eigenvalues from the following characteristics polynomial

$$|J(Y^*) - \lambda \vec{I}| = 0 \quad (6)$$

where \vec{I} is an identity matrix of order six and λ is the eigenvalues. Therefore, we get the characteristic polynomial of the form

$$P^{Y^*}(\lambda) = \lambda^6 + b_1 \lambda^5 + b_2 \lambda^4 + b_3 \lambda^3 + b_4 \lambda^2 + b_5 \lambda + b_6 = 0$$

where b_1, b_2, b_3, b_4, b_5 , and b_6 is given in Box 1.

Therefore, the stability when $\mathcal{R}_0 > 1$ follows the Routh-Hurwitz criterion for sixth-order polynomials, the endemic equilibrium point is asymptotically stable if and only if

- i) $b_1 > 0$ and $b_6 > 0$,
- ii) $h_3 = b_1 b_2 - b_3 > 0$,
- iii) $h_4 = b_3(b_1 b_2 - b_3) - b_1(b_1 b_4 - b_5) > 0$,
- iv) $h_5 = b_4(b_3(b_1 b_2 - b_3) - b_1(b_1 b_4 - b_5)) - b_5(b_2(b_1 b_2 - b_3) - (b_1 b_4 - b_5)) + b_6 b_1(b_1 b_2 - b_3) > 0$,
- v) $h_6 = b_5(b_4(b_3(b_1 b_2 - b_3) - b_1(b_1 b_4 - b_5)) - b_5(b_2(b_1 b_2 - b_3) - (b_1 b_4 - b_5)) + 2b_6 b_1(b_1 b_2 - b_3)) - b_6(b_3(b_3(b_1 b_2 - b_3) - b_1(b_1 b_4 - b_5)) - b_1^2 b_2 b_3) > 0$.

Sensitivity analysis

Sensitivity analysis can help analyze the parameters that influence the spread of disease. The normalized forward sensitivity index of a variable \mathcal{R}_0 , depends on a parameter p , that is defined in Chitnis *et al.* [10].

$$\Omega_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0}, \quad (7)$$

where p is one of the parameters whose sensitivity on \mathcal{R}_0 is sought. This index implies that the higher the value, the more sensitive \mathcal{R}_0 is to the parameter. The positive (or negative) of the index indicates the increases (or decreases) of \mathcal{R}_0 as p increase. Based on the parameter values given in Table 2, we get $\mathcal{R}_0 = 6.238762263$. The sensitivity index of the parameters to \mathcal{R}_0 given in Table 3.

Table 2. Parameter values and sources

Parameters	Value	Sources
Λ	40	[9]
β_I	0.55	[8]
β_H	0.05	Estimated
β_V	0.3	[8]
θ	0.61	[9]
α	0.08	[9]
ϵ	0.37	[9]
ν	0.08	[9]
r_I	0.2	[9]
r_H	0.65	[9]
γ_I	0.1	[8]
γ_H	0.05	Estimated
d	0.011	Estimated
μ	0.5	Estimated
ω	0.172	[8]
ρ	0.05	Estimated

Table 3. Sensitivity index to \mathcal{R}_0

Parameters	Sensitivity index to \mathcal{R}_0
μ	-2.07027958
Λ	1
β_V	0.621457651
ω	-0.621457651
θ	-0.592079059
α	0.45676089
γ_H	0.383349503
β_I	0.2928254
r_H	-0.26261257
γ_I	0.238108148
r_I	-0.136781648
β_H	0.085716949
ϵ	0.0695549
ν	-0.044674791
d	-0.011967203

For our proposed COVID-19 epidemic model (1), we follow the analysis done by Chitnis *et al.* [10]. A positive sensitivity index indicates an increase in the parameter leads to an increase of \mathcal{R}_0 , while a negative sensitivity index indicates an increase in the parameter leads to a decrease of \mathcal{R}_0 . Among these parameters, $\Lambda, \beta_V, \beta_I, \gamma_H, \gamma_I, \alpha, \epsilon$, and β_H have the positive index and $\mu, \omega, \theta, r_H, r_I, \nu$, and d have the negative index. Thus, the sensitivity analysis results show μ, Λ, β_V , and ω are the most influential parameters for proposed model (1).

In Table 4 and Table 5, we show the influences of the increase (or decrease) of parameters to \mathcal{R}_0 . The positive sensitivity index given in Table 4 shows that with an increase (or decrease) of one parameter, the \mathcal{R}_0 will get an addition (or reduction) and the endemic rate will increase (or decrease). The negative sensitivity index given in Table 5 shows that with an increase (or decrease) of one parameter, the \mathcal{R}_0 will get a reduction (or addition) and the endemic rate will decrease (or increase).

Disease-free equilibrium	Endemic equilibrium
$b_1 = \omega + k_1 + k_2 + k_3$ $b_2 = \omega(k_1 + k_2 + k_3) + k_1(k_2 + k_3) + k_2k_3$ $- \frac{\Lambda}{\mu}(1 - \theta)(\alpha\beta_I + \epsilon\beta_H)$ $b_3 = k_1k_2k_3 + \omega(k_1(k_2 + k_3) + k_2k_3)$ $- \frac{\Lambda}{\mu}(1 - \theta)(\alpha(v\beta_H + (\omega + k_3)\beta_I)$ $+ \epsilon\beta_H(\omega + k_2)) - \beta_V(\alpha\gamma_I + \epsilon\gamma_H)$ $b_4 = \omega k_1 k_2 k_3 - \frac{\Lambda\omega}{\mu}(1 - \theta)(\alpha(v\beta_H + k_3\beta_I) + \epsilon k_2\beta_H)$ $- \alpha\beta_V(\gamma_H(v + k_2) + \gamma_I k_3)$	$b_4 = \mu(\omega(k_1(k_2 + k_3 + k_4) + k_2(k_3 + k_4) + k_3k_4)$ $+ k_1k_2(k_3 + k_4) + k_3k_4(k_1 + k_2))$ $+ \omega(k_1(k_2(k_3 + k_4) + k_3k_4)$ $+ n_1(k_1(k_2 + k_3 + k_4) + k_2(k_3 + k_4)$ $+ k_3k_4))$ $+ n_1(k_1k_2(k_3 + k_4)$ $+ k_3k_4(k_1 + k_2)) + k_1k_2k_3k_4$ $- \alpha(\mu(n_2(\omega + k_3 + k_4) + n_3v$ $+ \beta_V\gamma_I) + v(n_3(\omega + k_4) + \beta_V\gamma_H)$ $+ \omega n_2(k_3 + k_4))$ $- \epsilon(\mu(n_3(\omega + k_2 + k_4) + \beta_V\gamma_H)$ $+ \omega n_3(k_2 + k_4) + \beta_V\gamma_H(k_2 + k_4)$ $+ n_3k_2k_4 + \tilde{n}n_1r_H),$ $b_5 = \mu\omega(k_1k_2(k_3 + k_4) + k_3k_4(k_1 + k_2))$ $+ (\mu + \omega + n_1)k_1k_2k_3k_4$ $+ \omega n_1(k_1k_2(k_3 + k_4)$ $+ k_3k_4(k_1 + k_2))$ $- \alpha(\mu(v(n_3(\omega + k_4) + \beta_V\gamma_H)$ $+ (\omega n_2 + \beta_V\gamma_I)(k_3 + k_4) + n_2k_3k_4)$ $+ v(k_4(n_3\omega + \beta_V\gamma_H) + \rho n_1r_H)$ $+ \omega(\rho n_1r_I + n_2k_3k_4)$ $+ k_3(\rho n_1r_I + \beta_V\gamma_I k_4))$ $- \epsilon(\mu((\omega n_3 + \beta_V\gamma_H)(k_2 + k_4)$ $+ n_3k_2k_4) + \omega(\rho n_1r_H + n_3k_3k_4)$ $+ k_2(\rho n_1r_H + \beta_V\gamma_H k_4)),$ $b_6 = \omega(k_1k_2k_3k_4(\mu + n_1))$ $- \alpha(\mu(k_4(v(\omega n_3 + \beta_V\gamma_H)$ $+ k_3(\omega n_2 + \beta_V\gamma_I)))$ $+ \omega\rho n_1(vr_H + k_3r_I))$ $- \epsilon(k_2(\mu k_4(\omega n_3 + \beta_V\gamma_H)$ $+ \omega\rho n_1r_H)).$
Endemic equilibrium	
$b_1 = \mu + \omega + n_1 + k_1 + k_2 + k_3 + k_4,$ $b_2 = \mu(\omega + k_1 + k_2 + k_3 + k_4)$ $+ \omega(n_1 + k_1 + k_2 + k_3 + k_4)$ $+ k_1(k_2 + k_3 + k_4) + k_2(k_3 + k_4)$ $+ k_3k_4 + n_1(k_1 + k_2 + k_3 + k_4)$ $- \alpha n_2 - \epsilon n_3,$ $b_3 = \mu(\omega(k_1 + k_2 + k_3 + k_4) + k_1(k_2 + k_3 + k_4)$ $+ k_2(k_3 + k_4) + k_3k_4)$ $+ \omega(n_1(k_1 + k_2 + k_3 + k_4)$ $+ k_1(k_2 + k_3 + k_4) + k_2(k_3 + k_4)$ $+ k_3k_4) + k_1(k_2(k_3 + k_4))$ $+ k_3k_4(k_1 + k_2)$ $+ n_1(k_1(k_2 + k_3 + k_4)$ $+ k_2(k_3 + k_4) + k_3k_4)$ $- \alpha(n_2(\mu + \omega + k_3 + k_4) + v n_3$ $+ \beta_V\gamma_I)$ $- \epsilon(n_3(\mu + \omega + k_2 + k_4) + \beta_V\gamma_H),$	

Box 1

Numerical simulations

This section aids in predicting the stability of the model (1) numerically using the fourth-order Runge-Kutta method with the help of MATLAB software. The numerical simulations are used to verify the analytical results.

Table 4. Influences of the positive sensitivity index parameters to \mathcal{R}_0

No.	Parameter (p)	\mathcal{R}_0 value	
		p + 10%	p - 10%
1.	Λ	6.862638485	5.614886035
2	β_V	6.626474917	5.851049609
3.	α	6.521344877	5.951379936
4.	γ_H	6.477924903	5.999599620
5.	β_I	6.421449069	6.056075458
6.	γ_I	6.387312276	6.090212250
7.	β_H	6.292239030	6.185285497
8.	ϵ	6.280529198	6.193610053

Table 5. Influences of the negative sensitivity index parameters to \mathcal{R}_0

No.	Parameter (p)	\mathcal{R}_0 value	
		p + 10%	p - 10%
1.	μ	5.110513732	7.738847295
2.	ω	5.886296211	6.6695541
3.	θ	5.869378215	6.608146312
4.	r_H	6.083610862	6.412316656
5.	r_I	6.155531881	6.326310693
6.	v	6.211169788	6.266918568
7.	d	6.231305433	6.246237566

Simulation on disease-free equilibrium points

The parameters used for this simulation is from Table 2 except $d = 0.5, \mu = 0.8, \omega = 1.72$, and $\beta_V = 0.03$ with $\mathcal{R}_0 = 0.5942745861 < 1$. The disease-free equilibrium point $Y_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right) = (50, 0, 0, 0, 0)$.

Local stability analysis based on Routh-Hurwitz criterion where

- i) $b_1 = 6.5 > 0$ and $b_4 = 2.7539565 > 0$,
- ii) $h_1 = 81.832575 > 0$,
- iii) $h_2 = 897.8537228 > 0$.

where $b_2 = 14.49635$, and $b_3 = 12.3937$.

These show that Routh-Hurwitz criterion is fulfilled and the characteristics polynomial have negative real parts.

Figure 2 shows that using initial condition $NA = (100,60,20,20,10,50)$, the susceptible subpopulation ends at equilibrium point 50, and the other subpopulations end at equilibrium points. It means that the numerical simulation approve of the analytical results.

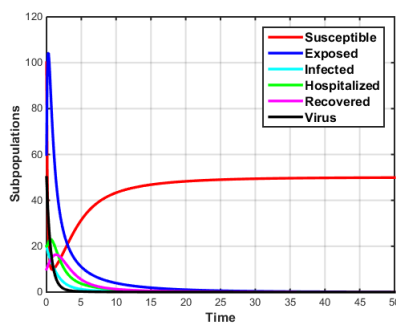


Figure 2. Numerical simulation when $R_0 < 1$

Simulation on endemic equilibrium points

The parameters used for this simulation is from Table 2 with $R_0 = 6.238762263 > 1$. Y^* exists where $k_1\mu < \Lambda(a_4 + a_5) = 0.475 < 2.963412073$ and $a_3 < k_1 = 0.02108240255 < 0.95$. Based on those parameters we get the endemic equilibrium

$$Y^* = (S^*, E^*, I^*, H^*, R^*, V^*) \\ = (12.82305634, 36.15872058, 3.657013458, 11.77544159, 15.24625405, 5.549264101).$$

Local stability analysis based Routh-Hurwitz criterion where

- i) $b_1 = 6.802829728 > 0$ and $b_6 = 0.24046706 > 0$,
- ii) $h_3 = 91.79853862 > 0$,
- iii) $h_4 = 1209.367776 > 0$,
- iv) $h_5 = 8872.761805 > 0$,
- v) $h_6 = 93025.31869 > 0$.

where $b_2 = 16.17011194$, $b_3 = 18.20397962$, $b_4 = 18.20397962$, and $b_5 = 2.705938479$.

These show that Routh-Hurwitz criterion is fulfilled and the characteristics polynomial have negative real parts. Figure 3 shows that using initial condition $NA = (100,60,20,20,10,50)$, all of the subpopulations end at the equilibrium point Y^* . It means that the numerical simulation approves the analytical results.

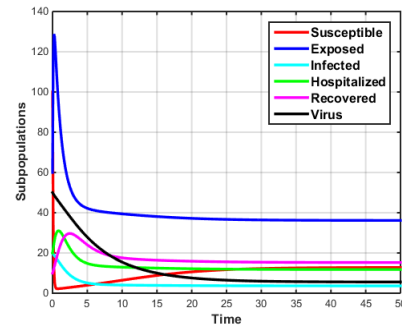


Figure 3. Numerical simulation when $R_0 > 1$

Numerical simulations of the influences of the changed parameters to E and I

From the sensitivity analysis, we got parameters that have influences on R_0 . Here, we give numerical simulations for the high sensitivity index and the low sensitivity index to see which one has the greatest influence on the population.

It can be seen that in Figure 4 and Figure 5, when β_V value is increased, R_0 will increase from 6.238762263 to 6.626474917 and the subpopulations of E and I also increase. When β_V value is decreased then R_0 will decrease to 5.851049609 and the subpopulations of E and I also decrease.

In Figure 6 and Figure 7, when β_H value is increased, R_0 will increase from 6.238762263 to 6.292239030 and the subpopulations of E and I also increase. When β_V value is decreased then R_0 will decrease to 6.185285497 and the subpopulations of E and I also decrease.

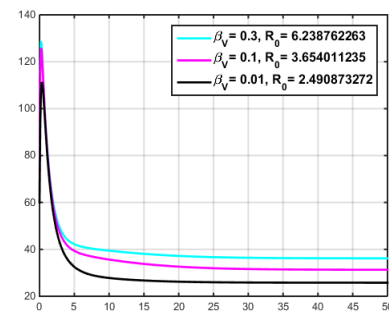


Figure 4. Influence of β_V to E

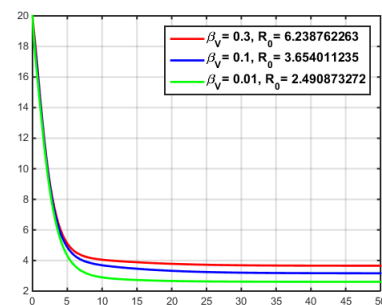


Figure 5. Influence of β_V to I

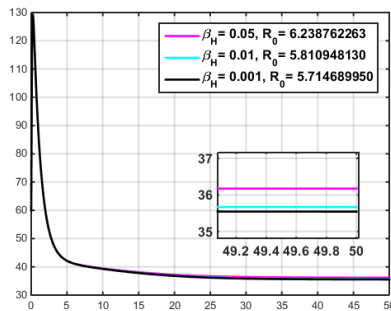


Figure 6. Influence of β_H to E

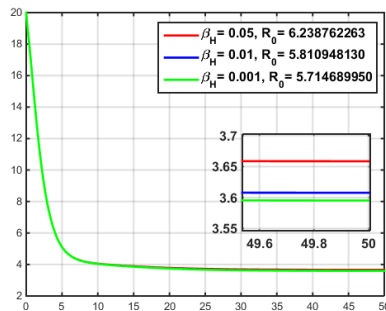


Figure 7. Influence of β_H to I

From the description above, we can conclude as follows. β_V , as a parameter with higher sensitivity index, have a more significant influences to the increase (or decrease) of \mathcal{R}_0 for each change of parameter value than β_H that has lower sensitivity index.

CONCLUSION

In this paper, we have proposed and investigated a mathematical model of the COVID-19 epidemic involving viruses in the environment. This model has been used to describe the transmission in the dynamics of the infection and affirms the role of the virus in the environment to the spread of COVID-19 disease. The model is numerically simulated to aid the analytical results that have been done. We have proposed a detailed analysis of the model, including the derivation of equilibrium points, disease-free and endemic, and the basic reproduction number \mathcal{R}_0 . The local stability analysis of the model fulfilled the Routh-Hurwitz criterion and the sensitivity analysis was also done to know the parameters that have more influence on \mathcal{R}_0 . The numerical simulations are used to aid and approve the analytical results and prove the sensitivity analysis of the model's parameters. The simulations show that β_V has more influences on \mathcal{R}_0 for it has a more sensitivity index than β_H .

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