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The Impact of Vaccine on the Dynamical Spread of Covid-19 Pandemic

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Abstract: The present study was undertaken to investigate the impact of effective COVID-19 vaccines in controlling or eradicating the disease. The proposed model was shown to be well-posed by establishing the biologically feasible regions. The qualitative analyses of the model showed that there exist both disease-free and endemic equilibria, which are globally asymptotically stable if the threshold value $R_0 < 1$ and $R_0 > 1$ respectively, and unstable otherwise.

Sensitivity analysis and numerical simulations were carried out, and the results showed that the rates of recruitment and vaccination are very sensitive to the reproduction number; and the contact rates of the exposed individuals and environmental virus increase the infection in the population while increased rates of vaccination and recovery slow down the infection.

It was therefore concluded that the influx of people into the population be reduced by placing restrictions on immigration, in order to eliminate the negative effect of the recruitment rate. Also, very effective COVID-19 vaccines and antiviral cures should be developed as these will respectively provide protection against the disease and increase recovery of any infected individual.

Index Terms **– SARS-CoV-2, COVID-19, Vaccination, Saturated Incidence, Reproduction number, Sensitivity, Stability**

I. INTRODUCTION

The novel Severe Acute Respiratory Coronavirus 2 (SAR-CoV-2) that causes coronavirus disease (COVID-19) has spread rapidly since emerging in late 2019 leading World Health Organization (WHO) to declare the disease a global Pandemic. It is likely that the virus has a zoonotic origin as it is believed to have originated from bats [1], based on evidence it is spread from person to person through droplet expelled by an infected person, contact with contaminated surfaces [2] and faces of infected persons [3] which may also contaminate the environment. The incubation period ranges from 1-14 days, symptoms which could be mild, moderate or severe depending on individuals underlying medical conditions could develop 4-6 days after exposure. [4, 5] and asymptomatic and symptomatic transmission have been reported [6]. Symptoms include fever, coughing, shortness of breath, tiredness, aches, runny nose, sore throat, headaches, diarrhea, vomiting and some may experience loss of smell or taste [7].There is currently no specific antiviral treatment approved to cure COVID-19, but treatment focuses on managing symptoms as the virus runs its course.

The implementation of non-pharmaceutical intervention such as case isolation, the closure of schools and universities, banning of mass gatherings or public event, mostly recently wide scale social distancing including local, national and worldwide lockdown, as much as the adoption of proven public health measures including testing, isolation of cases and wider social distancing to prevent onward transmission are critical in curbing the impact of the pandemic [8, 9]. If individuals who have recovered eventually become exposed to the virus again it is likely to be endemic. Therefore the development of effective vaccine will may free the entire globe from the threat of COVID-19 [10].

Ferguson *et al* also reported that while assessing the potential role of a number of public health measures aimed at reducing contact rates in the population as a consequence also reducing transmission of the virus, that in the absence of COVID-19 vaccine, the effectiveness of any one of the intervention in isolation is likely to be limited requiring multiple interventions to be combined to have a substantial impact on transmission. In a case where vaccines are available population immunity builds up through the epidemic, leading to an eventual rapid decline in case numbers and transmission dropping to low levels. Thus many infectious diseases can be prevented by vaccination of the susceptible population [11].

Additionally, Vaccines help immune system to recognize and fight pathogens such as bacteria or virus that can cause disease. Vaccines help prepare a person‘s body to fight potential future exposure to these pathogens by getting their immune system ready. It is well known that the higher the basic reproduction number the higher the proportion of the population will have to be vaccinated over time to achieve herd immunity [12]. Herd immunity is a form of immunity that occurs when the vaccination of a significant proportion of a population provides a measure of protection for individuals who have not developed immunity [13]. Immunity can be acquired naturally after an individual has successfully recovered from an infection, in some cases through maternal antibody in a new born baby and can also be induced through vaccine. Furthermore, some infections confers recovered persons with short or long immunity against re-infection [14], in

the case of COVID-19 whether the infection confers permanent or temporal immunity in recovered patients is not certain [10, 15]. Immunity acquired from vaccines requires boosting after some period of time, as vaccine effectiveness wanes with time [16].

Many attempts have been made to develop realistic mathematical models to investigate the transmission dynamics of COVID-19 and to predict the spread over the time [17, 18, 19]. As a result various control measures have also been developed to ensure that those that are most vulnerable are protected [20, 21, 22]. As treatment and effective vaccine are currently under study, the proposed model is formulated to determine the ideal vaccine coverage needed to control or eradicate the SARS-COV-2 and to analyze the effect of having an effective vaccines in the control or eradication of the disease. The rest of this work is organized as follows: we give the formulation of the model and a full description of the model's variables and parameters in Section 2; Section 3 provides the model's qualitative analyses, including a domain where the model is biologically feasible and mathematically wellposed, the existence of equilibria, a derivation of the basic reproduction number and stability analysis of the equilibria. We perform sensitivity analysis and numerical simulations of the model with graphical illustrations in Section 4. And in Section 5, the discussion and concluding remark were given.

II. MODIFICATION/MODEL FORMULATION

Yang and Wang [19] investigated the current outbreak of COVID-19 taking into account the role of environment in the transmission of virus. The model is represented with the following differential equation:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta_E(E)SE - \beta_I(I)SI - \beta_V(V)SV - \mu S \\
\frac{dE}{dt} &= \beta_E(E)SE + \beta_I(I)SI + \beta_V(V)SV - (\alpha+\mu)E \\
\frac{dI}{dt} &= \alpha E - (w+\gamma+\mu)I \\
\frac{dR}{dt} &= \gamma I - \mu R \\
\frac{dV}{dt} &= \xi_1 E + \xi_2 I - \sigma V.
\end{aligned} \tag{2.1}$$

Modifying the Yang and Wang model in [19] to include a vaccination class and changing the bilinear incidence to saturated incidence rate, together with the following additional assumptions:

i. Once exposed to COVID-19 individual can transmit infection;
ii. Recovered individuals can become susceptible (i.e. re-infection can occur);
iii. Vaccinated individuals cannot be infected unless the vaccine wanes;
iv. Based on the fact that the soil samples of the Huanan sea food wholesale market where the coronavirus broke out tested positive for the virus, Virus population, *P*, in the environment, is considered,

the proposed model becomes:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{\beta_E SE}{1+\alpha_1 E} - \frac{\beta_I SI}{1+\alpha_2 I} - \frac{\beta_P SP}{1+\alpha_3 P} + \tau R + \theta V - (c+\mu)S \\
\frac{dE}{dt} &= \frac{\beta_E SE}{1+\alpha_1 E} + \frac{\beta_I SI}{1+\alpha_2 I} + \frac{\beta_P SP}{1+\alpha_3 P} - (\alpha+\mu)E \\
\frac{dI}{dt} &= \alpha E - (w+\gamma+\mu)I \\
\frac{dR}{dt} &= \gamma I - (\tau+\mu)R \\
\frac{dV}{dt} &= cS - (\theta+\mu)V \\
\frac{dP}{dt} &= \xi_1 E + \xi_2 I - \sigma P,
\end{aligned} \tag{2.2}$$

with the initial conditions $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, R(0) \geq 0, V(0) \geq 0, P(0) \geq 0.$ (2.3)

And the model parameters are defined as follows:

***Table* 1:** *Description of the Model's Variables and Parameters*

Variables and Parameters	Description
S	The susceptible class
E	The exposed class
I	The infected class
R	The recovered class
V	The vaccinated class
P	The coronavirus concentration in the environmental reservoir
Λ	Recruitment rate of susceptible individuals (by birth or immigration)
β_E	Transmission rate between exposed and susceptible
β_I	Transmission rate between infected and susceptible
β_V	Transmission rate of indirect environment to susceptible
$\alpha_k, k = 1,2,3$	The saturation factor that measures the inhibitory effect
α	The period between exposure and the onset of symptoms
μ	The natural mortality rate of individuals in all the classes
w	The disease induced death
c	The rate of vaccination
θ	Tate at which vaccinated individuals become susceptible
γ	The rate of recovery
τ	The rate at which recovered individual become susceptible
ξ_1	The rate of the exposed individual contributing the coronavirus to the environmental reservoir
ξ_2	The rate of the infected individual contributing the coronavirus to the environment reservoir
σ	The rate of removal of the coronavirus from the environment

III. Model Analysis

3.1 Positivity and Boundedness of Solutions

It is vital to show that the solution of the model (2.2) with non-negative initial conditions are non-negative at any time, $t \geq 0$.

***Lemma* 1:** The solutions $S(t), E(t), I(t), R(t), V(t), P(t)$ of the model (2.2) are non-negative for all $t \geq 0$, with nonnegative initial conditions.

Proof: We have

$$\begin{cases} \left.\frac{dS}{dt}\right|_{\eta(s)} = \Lambda + \tau R + \theta V \geq 0 \\ \left.\frac{dE}{dt}\right|_{\eta(E)} = \frac{\beta_P SP}{1+\alpha_3 P} \geq 0 \\ \left.\frac{dI}{dt}\right|_{\eta(I)} = \alpha E \geq 0 \\ \left.\frac{dR}{dt}\right|_{\eta(R)} = \gamma I \geq 0 \\ \left.\frac{dV}{dt}\right|_{\eta(V)} = cS \geq 0 \\ \left.\frac{dP}{dt}\right|_{\eta(P)} = \xi_1 E + \xi_2 I \geq 0 \end{cases} \tag{3.1}$$

Therefore, $\eta(D) = \left\{ D(t) = 0 \text{ and } S, E, I, R, V, P \in \mathbb{R}_+^6 \right\}$, and $D(t) \in S, E, I, R, V, P$. Thus, based on Lemma 2 in [23], any solution of the system (2.2) is such that $S(t), E(t), I(t), R(t), V(t), P(t) \in \mathbb{R}_+^6$ for all $t \geq 0$.

***Theorem* 1:** Let $S(t), E(t), I(t), R(t), P(t), V(t)$ be the solutions of the system (2.2) with initial conditions (2.3). Let the compact set $\Omega_H = \left\{ S, E, I, R, V, P \in \mathbb{R}_+^5 \,|\, 0 \le S(t) + E(t) + I(t) + R(t) + V(t) \le N(t) \le \frac{\Lambda}{\mu} \right\}$ and $\Omega_P = \left\{ P \in \mathbb{R}_+ \,|\, 0 \le P \le \frac{\xi_1 + \xi_2}{\sigma} \right\}$. Define

$$\Omega = \left\{ S, E, I, R, V, P \in \mathbb{R}_+^6 \,|\, 0 \le N(t) \le \frac{\Lambda}{\mu},\ 0 \le P \le \frac{\xi_1 + \xi_2}{\sigma} \right\}.$$

Then the region Ω is positively invariant for the model (2.2) with the initial conditions (2.3) in $\mathbb{R}_+^6$.

***Proof*:** The system (2.2) is split into two parts the human population $S(t), E(t), I(t), R(t), V(t)$ and the pathogen population, $P(t)$.

For the human population:

$$\frac{dN(t)}{dt} = \frac{dS(t)}{dt} + \frac{dE(t)}{dt} + \frac{dI(t)}{dt} + \frac{dR(t)}{dt} + \frac{dV(t)}{dt}$$
$$= \Lambda - \mu N(t) - wI$$
$$\frac{dN(t)}{dt} \le \Lambda - \mu N(t). \qquad (3.2)$$

Solving by separating the variable and integrating yields

$$N(t) \le \frac{\Lambda}{\mu} - Ae^{-\mu t}. \qquad (3.4)$$

When $t = 0$, $N(0) = N_0 \le \frac{\Lambda}{\mu} + Ae^{-\mu t}$

$$N(t) \le \frac{\Lambda}{\mu} - \frac{(\Lambda - N_0\mu)}{\mu} e^{-\mu t}$$
$$0 \le N(t) \le \frac{\Lambda}{\mu}, \quad \text{for } t \to \infty. \qquad (3.5)$$

For this reason the biologically feasible region for human population is defined

$$\Omega_H = \left\{ S, E, I, R, V \in \mathbb{R}_+^5, 0 \le N(t) \le \frac{\Lambda}{\mu} \right\} \qquad (3.6)$$

For the pathogen population:

$$0 \le P \le \frac{\xi_1 + \xi_2}{\sigma} \qquad (3.7)$$

Thus the feasible region of the pathogen is defined as

$$\Omega_P = \left\{ 0 \le P \le \frac{\xi_1 + \xi_2}{\sigma} \right\} \qquad (3.8)$$

From Lemma 1, $\Omega = \Omega_H \times \Omega_P$.

Therefore the biologically feasible region $\Omega = \left\{ S, E, I, R, V, P \in \mathbb{R}_+^6 \,|\, 0 \le N(t) \le \frac{\Lambda}{\mu},\ 0 \le P \le \frac{\xi_1 + \xi_2}{\sigma} \right\}$ is positively invariant for the system (2.2). Hence, the solutions and the initial conditions of the system (2.2) are considered inside the region $\Omega = \left\{ S, E, I, R, V, P \in \mathbb{R}_+^6 \,|\, 0 \le N(t) \le \frac{\Lambda}{\mu},\ 0 \le P \le \frac{\xi_1 + \xi_2}{\sigma} \right\}$.

3.2 Equilibrium points of the model

In the absence of infection the Disease free equilibrium point (DFE) is given by

$$E^0 = (S,0,0,0,V,0) = \left(\frac{\Lambda(\theta + \mu)}{\mu(c + \theta + \mu)}, 0,0,0,0 \frac{\Lambda c}{\mu(c + \theta + \mu)} \right) \qquad (3.9)$$

The Endemic Equilibrium Point (EEP) satisfies

$$\Lambda - \frac{\beta_E S^* E^*}{1+\alpha_1 E} - \frac{\beta_I S^* I^*}{1+\alpha_2 I} - \frac{\beta_P S^* P^*}{1+\alpha_3 P} + \pi R^* + \theta V^* - (c+\mu)S^* = 0$$

$$\frac{\beta_E S^* E^*}{1+\alpha_1 E} + \frac{\beta_I S^* I^*}{1+\alpha_2 I} + \frac{\beta_P S^* P^*}{1+\alpha_3 P} - (\alpha+\mu)E^* = 0$$

$$\alpha E^* - (w+\gamma+\mu)I^* = 0 \tag{3.10}$$

$$\gamma I^* - (\tau+\mu)R^* = 0$$

$$cS^* - (\theta+\mu)V^* = 0$$

$$\xi_1 E^* + \xi_2 I^* - \sigma P^* = 0$$

By simple calculation,

$$E^* = \frac{(w+\gamma+\mu)I^*}{\alpha} \tag{3.11}$$

$$R = \frac{\gamma I^*}{(\tau+\mu)} \tag{3.12}$$

$$V = \frac{c(\Lambda K_4\alpha + (\tau\gamma\alpha - (\alpha+\mu))I^*)}{\mu\alpha K_6 K_4} \tag{3.13}$$

$$P = \frac{(\xi_1 K_3 + \xi_2\alpha)I^*}{\sigma\alpha} \tag{3.14}$$

Adding the first and second equations of (3.10) gives

$$S^* = \frac{K_5(\Lambda K_4\alpha + (\tau\gamma\alpha - K_2K_3K_4)I^*)}{\mu\alpha K_6 K_4}, \tag{3.15}$$

where $K_1 = (c+\mu), K_2 = (\alpha+\mu), K_3 = (w+\gamma+\mu), K_4 = (\tau+\mu), K_5 = (\theta+\mu), K_6 = (c+\theta+\mu).$

With (3.11), (3.12) and (3.14) substituted in the second equation in (3.10) to have:

$$\left(\frac{K_5(\Lambda K_4\alpha + (\tau\gamma\alpha - K_2K_3K_4)I^*)}{\mu\alpha K_6 K_4}\right)\left(\frac{\beta_E K_3}{\alpha+\alpha_1 K_3 I} + \frac{\beta_I}{1+\alpha_2 I} + \frac{\beta_V(\xi_1 K_3+\xi_2\alpha)}{\sigma\alpha+\alpha_3(\xi_1 K_3+\xi_2\alpha)I}\right) - \frac{K_2K_3}{\alpha}\Bigg)I^* = 0 \tag{3.16}$$

This implies that $I^* = 0$ or

$$\frac{K_5(\Lambda K_4\alpha + (\tau\gamma\alpha - K_2K_3K_4)I^*)}{\mu\alpha K_6 K_4}\left(\frac{\beta_E K_3}{\alpha+\alpha_1 K_3 I} + \frac{\beta_I}{1+\alpha_2 I} + \frac{\beta_V(\xi_1 K_3+\xi_2\alpha)}{\sigma\alpha+\alpha_3(\xi_1 K_3+\xi_2\alpha)I}\right) - \frac{K_2K_3}{\alpha} = 0$$

For endemic case $I^* \neq 0$. So, following the approach of [24, 25], the equation becomes

$$\frac{K_5(\Lambda K_4\alpha + (\tau\gamma\alpha - K_2K_3K_4)I^*)}{\mu\alpha K_6 K_4} = \frac{K_2K_3}{\alpha}\Bigg/\left(\frac{\beta_E K_3}{\alpha+\alpha_1 K_3 I} + \frac{\beta_I}{1+\alpha_2 I} + \frac{\beta_V(\xi_1 K_3+\xi_2\alpha)}{\sigma\alpha+\alpha_3(\xi_1 K_3+\xi_2\alpha)I}\right)$$

Assuming that $g_1(I^*) = g_2(I^*)$, where

$$g_1(I^*) = \frac{K_5(\Lambda K_4\alpha + (\tau\gamma\alpha - K_2K_3K_4)I^*)}{\mu\alpha K_6 K_4},$$

$$g_2(I^*) = \frac{K_2K_3}{\alpha}\Bigg/\left(\frac{\beta_E K_3}{\alpha+\alpha_1 K_3 I} + \frac{\beta_I}{1+\alpha_2 I} + \frac{\beta_V(\xi_1 K_3+\xi_2\alpha)}{\sigma\alpha+\alpha_3(\xi_1 K_3+\xi_2\alpha)I}\right).$$

To determine the uniqueness of I^*, it is noted that $I = I^*$.

If $I = 0$, then $g_1(I) = \frac{K_5\Lambda}{\mu K_6} = S_0$ while $g_2(I) = \frac{K_2K_3\sigma}{(\beta_E K_3\sigma + \beta_I\sigma\alpha + \beta_V(\xi_1 K_3+\xi_2\alpha))}$.

If $I^* > 0$, then $g_1(I^*) < 0$ while $g_2(I^*) > 0$.

Thus $g_2(I^*)$ is an increasing function for $I^* \geq 0$, so the control reproduction number is given to be

$$R_0 = \frac{g_1(0)}{g_2(0)} = \frac{S_0(\beta_E K_3\sigma + \beta_I\sigma\alpha + \beta_V(\xi_1 K_3+\xi_2\alpha))}{K_2K_3\sigma}. \tag{3.17}$$

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Hence there is one and only one intersection between the curves of $g_1(I)$ and $g_2(I)$; that is , there is a unique solution I^* to the equation $g_1(I^*) = g_2(I^*)$. Consequently, (S^*, E^*, R^*, V^*, P^*) are uniquely determined by I^*.

3.3 Local Stability of the Disease Free Equilibrium Point

***Theorem* 2:** If $R_0 < 1$, then the disease-free equilibrium is locally asymptotically stable. Otherwise, it is unstable.

***Proof*:** To investigate the local stability of the disease-free equilibrium, a Jacobian/variational matrix is constructed as follows:

$$J(E_0) = \begin{pmatrix} -K_1 & -\beta_E S_0 & -\beta_I S_0 & \tau & \theta & -\beta_P S_0 \\ 0 & \beta_E S_0 - K_2 & \beta_I S_0 & 0 & 0 & \beta_P S_0 \\ 0 & \alpha & -K_3 & 0 & 0 & 0 \\ 0 & 0 & \gamma & -K_4 & 0 & 0 \\ c & 0 & 0 & 0 & -K_5 & 0 \\ 0 & \xi_1 & \xi_2 & 0 & 0 & -\sigma \end{pmatrix} \tag{3.18}$$

where $K_1 = c+\mu, K_2 = \alpha+\mu, K_3 = w+\gamma+\mu, K_4 = \tau+\mu, K_5 = \theta+\mu$

The characteristic equation of $J(E_0)$ is given by

$$(\lambda + K_4)(y_1\lambda^2 + y_2\lambda + y_3)(b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4) = 0 \tag{3.19}$$

where

$$y_1 = 1, y_2 = (c+\theta+2\mu), y_3 = (c+\theta+\mu)\mu$$

$$b_1 = 1, b_2 = w + c + \theta + \alpha + 2\mu - \beta_E S_0$$

$$b_3 = \sigma(\alpha+\mu) + (\alpha+\mu+\sigma)(w+\gamma+\mu) - S_0(\beta_E(w+\gamma+\mu+\sigma) + \alpha\beta_I + \xi_1\beta_p)$$

$$b_4 = \sigma(w+\gamma+\alpha+2\mu)(1-R_0).$$

The value of $\lambda_1 = -(\tau+\mu)$, by the Routh-Hurwitz criteria if $y_2 > 0$, $y_3 > 0$ then $y_2 y_3 > 0$ for the quadratic equation and if $b_2 > 0$, $b_3 > 0$, $b_4 > 0$ then $b_2 b_3 - b_4 > 0$ for the cubic equation the roots will be negative or have negative real parts. It follows that all the eigenvalues $J(E_0)$ have negative real parts. This implies that the disease free equilibrium is locally asymptotically stable if $R_0 < 1$.

Also from $b_4 = \sigma(w+\gamma+\alpha+2\mu)(1-R_0) > 0$,

$$(1-R_0) > 0$$

$$R_0 < 1$$

Hence the disease free equilibrium point E_0 is locally asymptotically stable if $R_0 < 1$.

3.4 Global Stability of the Disease Free Equilibrium Point

The global asymptotic stability (GAS) of the disease free equilibrium (DFE) state of the model is proved using the theorem by Castillo-Chavez *et al* [26]. The model is written as:

$$\frac{dW}{dt} = Y(W, Z) \tag{3.20}$$

$$\frac{dZ}{dt} = G(W, Z), G(W^*, 0) = 0, \tag{3.21}$$

where $W = (S, V, R) \in \mathbb{R}^3_+$ denotes the number of uninfected individual

$Z = (E, I, P) \in \mathbb{R}^3_+$ denotes the number of infected individual including latent, infected and the pathogen in the environment.

The DFE of the system is denoted $E_0 = (W^* 0)$.

The conditions below must be met to guarantee the global stability

$H_1: \frac{dW}{dt} = Y(W,0), W^*$ is globally asymptotically stable (GAS)

$H_2: \frac{dZ}{dt} = G(W, Z) = AZ - \hat{G}(W, Z),\ \hat{G}(W, Z) \geq 0$ for $(W, Z) \in \Omega$,

where $A = D_z G(W^*, 0)$ is an M-matrix (the off diagonal elements of A are non-negative) and Ω is the region where the model makes biological sense. If the system (2.2) satisfies the two conditions then the following theorem holds.

***Theorem* 3:** The equilibrium point $E_0 = (W^* 0)$ of the system (2.2) is globally asymptotically stable provided $R_0 < 1$ and the conditions H_1 and H_2 are satisfied.

Proof: Breaking the system into subsystems $W=(S,V)$ and $Z=(E,I,P)$ to have two vector valued functions

$$Y(W,Z)=\begin{pmatrix}\Lambda-\frac{\beta_E(E)SE}{1+\alpha_1E}-\frac{\beta_I(I)SI}{1+\alpha_2I}-\frac{\beta_V(P)SP}{1+\alpha_3P}+\tau R+\theta V-(c+\mu)S\\ \gamma I-(\tau+\mu)R\\ cS-(\theta+\mu)V\end{pmatrix} \tag{3.22}$$

$$G(W,Z)=\begin{pmatrix}\frac{\beta_E(E)SE}{1+\alpha_1E}+\frac{\beta_I(I)SI}{1+\alpha_2I}+\frac{\beta_V(P)SP}{1+\alpha_3P}-(\alpha+\mu)E\\ \alpha E-(w+\gamma+\mu)I\\ \xi_1E+\xi_2I-\sigma P\end{pmatrix} \tag{3.23}$$

Considering the reduced system $\frac{dW}{dt}=Y(W,0)$ from condition H_1

$$Y(W,0)=\begin{pmatrix}\Lambda-\theta V-(c+\mu)S\\ -(\tau+\mu)R\\ cS-(\theta+\mu)V\end{pmatrix} \tag{3.24}$$

$W^*=(S^*,0,V^*)=\left(\frac{\Lambda(\theta+\mu)}{\mu(c+\theta+\mu)},0,\frac{\Lambda c}{\mu(c+\theta+\mu)}\right)$ is a GAS equilibrium point for the reduced system (3.24) to show this, the second equation in (3.24) is solved to obtain $R(t)=R_0\,e^{-(\tau+\mu)t}$; it approaches zero as $t\to\infty$.

Similarly the solution of the last equation gives $V=\frac{c\Lambda}{\mu(c+\theta+\mu)}+\left(V_0-\frac{c\Lambda}{\mu(c+\theta+\mu)}\right)e^{-(\theta+\mu)t}$ which approaches $\frac{c\Lambda}{\mu(c+\theta+\mu)}$ as $t\to\infty$ and the first equation yields $S=\frac{\Lambda(\theta+\mu)}{\mu(c+\theta+\mu)}+\left(S_0-\frac{\Lambda(\theta+\mu)}{\mu(c+\theta+\mu)}\right)e^{-(c+\mu)t}\ )\to\frac{\Lambda(\theta+\mu)}{\mu(c+\theta+\mu)}$ as $t\to\infty$.

The asymptotic dynamics is dependent of the initial condition in Ω. Thus the convergence of the solution of the reduced system in (3.24) is global in Ω. To compute $\frac{dZ}{dt}=G(W,Z)=AZ-\hat{G}(W,Z),$ and show $\hat{G}(W,Z)\geq 0$

$$A=D_zG(W^*,0)=\begin{pmatrix}\beta_ES_0-(\alpha+\mu) & \beta_IS_0 & \beta_pS_0\\ \alpha & -(w+\gamma+\mu) & 0\\ \xi_1 & \xi_2 & -\sigma\end{pmatrix} \tag{3.25}$$

$$\hat{G}(W,Z)=\begin{pmatrix}\beta_E\left(S_0-\frac{S}{1+\alpha_1E}\right)+\beta_I\left(S_0-\frac{S}{1+\alpha_2I}\right)+\beta_I\left(S_0-\frac{S}{1+\alpha_3P}\right)\\ 0\\ 0\end{pmatrix}. \tag{3.26}$$

Since $S_0>\frac{S}{1+\alpha_1E}, S_0>\frac{S}{1+\alpha_2I}, S_0>\frac{S}{1+\alpha_3P}$ it is clear that $\hat{G}(W,Z)\geq 0$.

Hence DFE is globally asymptotically stable when $R_0<1$.

3.5 Local Stability of the Endemic Equilibrium Point

***Theorem* 4:** If for each $i=0,1,2\ldots5$, $A_i>0$, where A_i are defined constants, then the endemic equilibrium is locally asymptotically stable. Otherwise, it is unstable.

Proof: To investigate the local stability of the endemic equilibrium, a Jacobian/variational matrix is constructed as follows:

$$J(E_*)=\begin{pmatrix}-(K_1+J_1) & -J_2 & -J_3 & \tau & \theta & -J_4\\ J_1 & J_2-K_2 & J_3 & 0 & 0 & J_4\\ 0 & \alpha & -K_3 & 0 & 0 & 0\\ 0 & 0 & \gamma & -K_4 & 0 & 0\\ c & 0 & 0 & 0 & -K_5 & 0\\ 0 & \xi_1 & \xi_2 & 0 & 0 & -\sigma\end{pmatrix}, \tag{3.27}$$

where

$$J_1=\frac{\beta_E E}{1+\alpha_1 E}+\frac{\beta_I I}{1+\alpha_2 I}+\frac{\beta_P P}{1+\alpha_3 P}, J_2=\frac{\beta_E SE}{1+\alpha_1 E}-\frac{\beta_E SE\alpha_1}{(1+\alpha_1 E)^2}, J_3=\frac{\beta_I SI}{1+\alpha_2 I}-\frac{\beta_I SI\alpha_2}{(1+\alpha_2 I)^2}, J_4=\frac{\beta_P SP}{1+\alpha_3 P}-\frac{\beta_P SP\alpha_3}{(1+\alpha_3 P)^2}.$$

The characteristic equation associated with $J(E_*)$ is

$$\lambda^6+A_5\lambda^5+A_4\lambda^4+A_3\lambda^3+A_2\lambda^2+A_1\lambda+A_0=0, \tag{3.28}$$

where

$$A_5=\sigma+K_5+K_4+K_3+K_2-J_2+K_1+J_1$$

$$A_4=\left(K_2+K_4+K_5+\sigma+J_1-J_2+K_1\right)K_3+\left(K_2+K_5+\sigma+J_1-J_2+K_1\right)K_4+\left(K_2+\sigma+J_1-J_2+K_1\right)K_5+\left(K_2+J_1-J_2+K_1\right)\sigma+\left(K_2-J_2\right)K_1-c\theta+J_1K_2-J_4\xi_1-\alpha J_3$$

$$A_3=\left(\left(K_2+K_5+\sigma+J_1-J_2+K_1\right)K_3+\left(K_2+\sigma+J_1-J_2+K_1\right)K_5+\left(K_2+J_1-J_2+K_1\right)\sigma+\left(K_2-J_2\right)K_1-c\theta+J_1K_2-J_4\xi_1-\alpha J_3\right)K_4+\left(\left(K_2+\sigma+J_1-J_2+K_1\right)K_5+\left(K_2+J_1-J_2+K_1\right)\sigma+\left(K_2-J_2\right)K_1-c\theta+J_1K_2-J_4\xi_1\right)K_3+\left(\left(K_2+J_1-J_2+K_1\right)\sigma+\left(K_2-J_2\right)K_1+J_1K_2-J_4\xi_1-\alpha J_3\right)K_5+\left(\left(K_2-J_2\right)K_1-c\theta+J_1K_2-\alpha J_3\right)\sigma+\left(-\alpha J_3-J_4\xi_1\right)K_1-c\theta K_2+c\theta J_2-\alpha J_4\xi_2$$

$$A_2=\left(\left(\left(K_2+\sigma+J_1-J_2+K_1\right)K_5+\left(K_2+J_1-J_2+K_1\right)\sigma+\left(K_2-J_2\right)K_1-c\theta+J_1K_2-J_4\xi_1\right)K_3+\left(\left(K_2+J_1-J_2+K_1\right)\sigma+\left(K_2-J_2\right)K_1+J_1K_2-J_4\xi_1-\alpha J_3\right)K_5+\left(\left(K_2-J_2\right)K_1-c\theta+J_1K_2-\alpha J_3\right)\sigma+\left(-\alpha J_3-J_4\xi_1\right)K_1-c\theta K_2+c\theta J_2-\alpha J_4\xi_2\right)K_4+\left(\left(\left(K_2+J_1-J_2+K_1\right)\sigma+\left(K_2-J_2\right)K_1+J_1K_2-J_4\xi_1\right)K_5+\left(\left(K_2-J_2\right)K_1-c\theta+J_1K_2\right)\sigma-J_4K_1\xi_1-c\theta\left(K_2-J_2\right)\right)K_3+\left(\left(\left(K_2-J_2\right)K_1+J_1K_2-\alpha J_3\right)\sigma+\left(-\alpha J_3-J_4\xi_1\right)K_1-\alpha J_4\xi_2\right)K_5+\left(-\alpha J_3K_1-c\theta\left(K_2-J_2\right)\right)\sigma-\alpha J_4K_1\xi_2-\gamma\alpha J_1\tau+c\theta\left(\alpha J_3+J_4\xi_1\right)$$

$$A_1=\left(\left(\left(\left(K_2+J_1-J_2+K_1\right)K_5+\left(K_2-J_2\right)K_1-c\theta+J_1K_2\right)K_3+\left(\left(K_2-J_2\right)K_1+J_1K_2-\alpha J_3\right)K_5-\alpha J_3K_1-c\theta\left(K_2-J_2\right)\right)\sigma+\left(\left(\left(K_2-J_2\right)K_1-J_4\xi_1+J_1K_2\right)K_5-J_4K_1\xi_1-c\theta\left(K_2-J_2\right)\right)K_3+\left(\left(-\alpha J_3-J_4\xi_1\right)K_1-\alpha J_4\xi_2\right)K_5-\alpha J_4K_1\xi_2+c\theta\left(\alpha J_3+J_4\xi_1\right)\right)K_4+\left(\left(\left(\left(K_2-J_2\right)K_1+J_1K_2\right)K_5-c\theta\left(K_2-J_2\right)\right)K_3+\alpha\left(c\theta J_3-\gamma\tau J_1-J_3K_1K_5\right)\right)\sigma+J_4\xi_1\left(c\theta-K_1K_5\right)K_3+\alpha\left(\left(-\gamma\tau J_1-J_4K_1\xi_2\right)K_5+cJ_4\xi_2\theta\right)$$

Now, from the polynomial equation (3.28), if the constants A_i (i = 0,1,2…5) are all positive, then by Descartes' rule of sign, there is no sign change in the polynomial equation (3.28). This implies that all the roots will be negative or complex having negative real parts. Hence, the endemic equilibrium is locally asymptotically stable.

3.6 Global Stability of the Endemic Equilibrium Point

Theorem 5: The endemic equilibrium E_* of the system (2.2) is globally asymptotically stable in Ω, if $R_0>1$.

Proof: when $R_0>1$ and there exist endemic equilibrium points for the system of equation in (2.2), considering the Lyapunov function of the Goh-Volterra type [27].

$$F=S-S^{**}-S^{**}\ln\frac{S}{S^{**}}+E-E^{**}-E^{**}\ln\frac{E}{E^{**}}+A\left(I-I^{**}-I^{**}\ln\frac{I}{I^{**}}\right)+B\left(P-P^{**}-P^{**}\ln\frac{P}{P^{**}}\right) \tag{3.29}$$

Taking the time derivative of (3.27)

$$\frac{dF}{dt}=\left(\frac{dS}{dt}-\frac{S^{**}}{S}\frac{dS}{dt}\right)+\left(\frac{dE}{dt}-\frac{E^{**}}{E}\frac{dE}{dt}\right)+A\left(\frac{dI}{dt}-\frac{I^{**}}{I}\frac{dI}{dt}\right)+B\left(\frac{dP}{dt}-\frac{P^{**}}{P}\frac{dP}{dt}\right) \tag{3.30}$$

Let $\lambda=\frac{\beta_E E}{1+\alpha_1 E}+\frac{\beta_I I}{1+\alpha_2 I}+\frac{\beta_P P}{1+\alpha_3 P}$; now having $\tilde{\lambda}=\tilde{\beta}_E SE+\tilde{\beta}_I SI+\tilde{\beta}_P SP$

Substituting the derivative of (E, I, P) in equation (2.2) into (3.30)

$$\frac{dF}{dt} = \Lambda + \tau R + \theta V - S(\tilde{\beta}_E E + \tilde{\beta}_I I + \tilde{\beta}_P P) - K_1 S - \frac{S^{**}}{S}(\Lambda + \tau R + \theta V) + S^{**}(\tilde{\beta}_E E + \tilde{\beta}_I I + \tilde{\beta}_P P) +$$
$$K_1 S^{**} + S(\tilde{\beta}_E E + \tilde{\beta}_I I + \tilde{\beta}_P P) - K_2 E - \frac{E^{**}}{E} S(\tilde{\beta}_E E + \tilde{\beta}_I I + \tilde{\beta}_P P) + K_2 E^{**} + \quad (3.31)$$
$$A\left(\alpha E - K_3 I - \frac{I^{**}\alpha E}{I} + K_3 I^{**}\right) + B\left(\frac{(\xi_1 K_3 + \xi_2 \alpha)I}{\alpha} - \sigma P - \frac{P^{**}(\xi_1 K_3 + \xi_2 \alpha)I}{P\alpha} + \sigma P^{**}\right)$$

At steady state the first equation of system (2.2) is

$$\Lambda + \tau R + \theta V = S^{**}(\tilde{\beta}_E E^{**} + \tilde{\beta}_I I^{**} + \tilde{\beta}_P P^{**}) + K_1 S^{**} \quad (3.32)$$

Substituting (3.30) into (3.29) and simplifying to have

$$\frac{dF}{dt} = S^{**}(\tilde{\beta}_E E^{**} + \tilde{\beta}_I I^{**} + \tilde{\beta}_P P^{**}) + K_1 S^{**} - K_1 S - \frac{S^{**2}}{S}(\tilde{\beta}_E E^{**} + \tilde{\beta}_I I^{**} + \tilde{\beta}_P P^{**}) - K_1 S^{**2}$$
$$- S(\tilde{\beta}_E E + \tilde{\beta}_I I + \tilde{\beta}_P P) - K_1 S + S^{**}(\tilde{\beta}_E E + \tilde{\beta}_I I + \tilde{\beta}_P P) + K_1 S^{**} - K_2 E - \frac{E^{**}}{E} S(\tilde{\beta}_E E + \tilde{\beta}_I I + \tilde{\beta}_P P) \quad (3.33)$$
$$+ K_2 E^{**} + A\left(\alpha E - K_3 I - \frac{I^{**}\alpha E}{I} + K_3 I^{**}\right) + B\left(\frac{(\xi_1 K_3 + \xi_2 \alpha)I}{\alpha} - \sigma P - \frac{P^{**}(\xi_1 K_3 + \xi_2 \alpha)I}{P\alpha} + \sigma P^{**}\right)$$

Separating the infected terms without the double star to have

$$S^{**}(\tilde{\beta}_E E + \tilde{\beta}_I I + \tilde{\beta}_P P) - K_2 E^{**} + A\alpha E - AK_3 I + B\frac{(\xi_1 K_3 + \xi_2 \alpha)I}{\alpha} - B\sigma P = 0 \quad (3.34)$$

Obtaining the expression for A and B also the expression at steady states to have

$$A = \frac{S^{**}\tilde{\beta}_P}{\sigma},\ B = \frac{S^{**}\tilde{\beta}_I I}{\sigma K_3} + \frac{S^{**}\tilde{\beta}_P(\xi_1 K_3 + \xi_2 \alpha)}{\sigma K_3},\ K_2 = \frac{S^{**}(\tilde{\beta}_E E^{**} + \tilde{\beta}_I I^{**} + \tilde{\beta}_P P^{**})}{E^{**}},$$
$$\alpha = \frac{K_3 I^{**}}{E^{**}},\ \frac{\sigma P^{**}}{E^{**}} = \frac{(\xi_1 K_3 + \xi_2 \alpha)}{K_3} \quad (3.35)$$

With the expression in (3.34) substituted into the remaining expressions in (3.33), after some algebraic simplification gives

$$\frac{dF}{dt} = S^{**}(\tilde{\beta}_E E^{**} + \tilde{\beta}_I I^{**} + \tilde{\beta}_P P^{**}) + K_1 S^{**} - K_1 S - \frac{S^{**2}}{S}(\tilde{\beta}_E E^{**} + \tilde{\beta}_I I^{**} + \tilde{\beta}_P P^{**}) -$$
$$K_1 S^{**2} + K_1 S^{**} - \frac{E^{**}}{E} S(\tilde{\beta}_E E + \tilde{\beta}_I I + \tilde{\beta}_P P) + S^{**}(\tilde{\beta}_E E^{**} + \tilde{\beta}_I I^{**} + \tilde{\beta}_P P^{**}) + \quad (3.36)$$
$$\left(\frac{S^{**}\tilde{\beta}_I I}{K_3} + \frac{S^{**}\tilde{\beta}_P \sigma P^{**}}{\sigma E^{**} K_3}\right)\left(-\frac{I^{**}K_3 I^{**}E}{IE^{**}} + K_3 I^{**}\right) + \frac{S^{**}\tilde{\beta}_P}{\sigma}\left(-\frac{P^{**}\sigma P^{**}}{P\alpha} + \sigma P^{**}\right)$$

Factorizing the expression (3.36)

$$\frac{dF}{dt} = K_1 S^{**}\left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}}\right) + \tilde{\beta}_E S^{**} E^{**}\left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}}\right)$$
$$+ \tilde{\beta}_I S^{**} I^{**}\left(3 - \frac{S^{**}}{S} - \frac{SIE^{**}}{S^{**} I^{**} E} - \frac{I^{**}E}{IE^{**}}\right) + \tilde{\beta}_P S^{**} P^{**}\left(3 - \frac{S^{**}}{S} - \frac{SIE^{**}}{S^{**} I^{**} E} - \frac{I^{**}E}{IE^{**}}\right) \quad (3.37)$$

Since the arithmetic mean exceeds the geometric mean, it implies that

$$K_1 S^{**}\left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}}\right) \le 0,\ \tilde{\beta}_E S^{**} E^{**}\left(2 - \frac{S^{**}}{S} - \frac{S}{S^{**}}\right) \le 0$$
$$\tilde{\beta}_I S^{**} I^{**}\left(3 - \frac{S^{**}}{S} - \frac{SIE^{**}}{S^{**} I^{**} E} - \frac{I^{**}E}{IE^{**}}\right) \le 0,\ \tilde{\beta}_P S^{**} P^{**}\left(3 - \frac{S^{**}}{S} - \frac{SIE^{**}}{S^{**} I^{**} E} - \frac{I^{**}E}{IE^{**}}\right) \le 0$$

Thus $\frac{dF}{dt} \le 0$ for $R_0 > 1$, by LaSalle's invariance principle [28], E_* is globally asymptotically stable in Ω since $\frac{dF}{dt} \le 0$.

IV. Sensitivity Analysis and Numerical Simulations

4.1 Sensitivity Analysis of R_0

Sensitivity Analysis is a crucial notion in epidemiology that determines the strength of each parameter in the transmission of diseases. It is used to determine the responsiveness of model prediction to parameter values. It is used to determine the parameters, which have high impact on the R_0 and which intervention strategies should target. Following the approach of [22, 29], the normalized forward sensitivity index of R_0 that depends differentially on a parameter p is defined as:

$$\chi_p^{R_0} = \frac{\partial R_0}{\partial p} \cdot \frac{p}{R_0}. \quad (3.38)$$

Given this explicit formula for R_0, we can easily derive an analytical expression for the sensitivity of R_0 with respect to each parameter that comprises it. For example, the sensitivity index of R_0 with respect to the rate of vaccination, c, is obtained as:

$$\chi_c^{R_0} = \frac{\partial R_0}{\partial c}.\frac{c}{R_0} = -0.7999518430.$$

Similarly, the obtained values for the sensitivity index of R_0 with respect to other parameters, for the given base line parameter values are given in Table 2 below.

***Table* 2:** *Sensitivity Indices of R_0*

Parameters	Baseline Values	Sensitivity Indices
Λ	273.23 day^{-1}	+ 1.0000000000
β_E	3.11 x 10^{-8} person^{-1} day^{-1}	+ 0.4707090842
β_I	0.62 x 10^{-8} person^{-1} day^{-1}	+ 0.1748486199
β_V	1.01 x 10^{-8} person^{-1} day^{-1}	+ 0.3544422960
α	$^1/_7$ day^{-1}	– 0.8222819698
μ	3.01 x 10^{-5} day^{-1}	– 0.9997873333
w	0.01 day^{-1}	– 0.0231769544
c	0.04 day^{-1}	– 0.7999518430
θ	0.01 day^{-1}	+ 0.7997111279
γ	$^1/_{15}$ day^{-1}	– 0.1545130296
ξ_1	2.3 day^{-1}	+ 0.3515939558
ξ_2	0.1 day^{-1}	+ 0.0028483404
σ	1.0 day^{-1}	– 0.3544422960

From the above index table, it was revealed that the most sensitive parameter is the rates of recruitment (Λ). Other parameters like rate of vaccination coverage (c) and rate of vaccine waning (θ), among others, are also sensitive to the reproduction number. As a way of illustration, $\chi_\Lambda^{R_0} = +1.00000$ means that increasing (or decreasing) Λ by 10% increases (or decreases) R_0 by 10%; while $\chi_c^{R_0} = -0.79995$ means that increasing (or decreasing) c by 10% decreases (or increases) R_0 by 7.9995%. The interpretation of the sensitivity indices of other parameters follows as of that of Λ and c.

4.2 Numerical Simulations and Results

The numerical simulation for the COVID-19 model was carried out by Maple 18.0 software using direct substitution method to show solution of the model equation, the global stability of the equilibria and the effects of various transmission parameters and the rates of vaccination and recovery.

***Table* 3:** *Parameter Values Used in the Model*

Parameters	Baseline Values	Sources
Λ	273.23 day^{-1}	[19]
β_E	3.11 x 10^{-8} person^{-1} day^{-1}	[19]
β_I	0.62 x 10^{-8} person^{-1} day^{-1}	[19]
β_V	1.01 x 10^{-8} person^{-1} day^{-1}	[19]
$\alpha_k, k = 1,2,3$	[0.585 x 10^{-4}, 1.426 x 10^{-4}]	[19]
α	$^1/_7$ day^{-1}	[19]
μ	3.01 x 10^{-5} day^{-1}	[19]
w	0.01 day^{-1}	[19]
c	0.04 day^{-1}	Assumed
θ	0.01 day^{-1}	Assumed
γ	$^1/_{15}$ day^{-1}	[30]
τ	0.03 day^{-1}	[22]
ξ_1	2.3 day^{-1}	[19]
ξ_2	0.1 day^{-1}	Assumed
σ	1.0 day^{-1}	[19]

The parameter values used are given in the Table 3, with initial conditions: $S(0)=500,\ V(0)=150,\ E(0)=200,\ I(0)=100,\ R(0)=50,$ and $P(0)=1000.$ The results of the numerical simulations are given in Figures 1 – 14 to illustrate the system's behaviour for different values of the COVID-19 model's parameters.

V. Discussion of Results and Conclusion

5.1 Discussion of Results

The plots in Fig. 1 and Fig.2 illustrate the global stability of the disease-free and endemic equilibria respectively, and they agree with the results of the global stability analyses given in Theorem 3 and 5. These imply that irrespective of the initial value of the infective, the disease can be controlled or wiped out from the population when $R_0 < 1$, since from Fig. 1, the solutions converge at the disease-free equilibrium points. However, whenever $R_0 > 1$, then all solutions converge to the endemic equilibrium points, rather than zero. Thus the disease will persist in the population until when measures are taken to lower the reproduction number below unity. The reproduction number of the model is $R_0 = 0.189$ in the presence of vaccination and symptoms management (which increases the rate of recovery). When these controls are absent, then the contact rates increase and the reproduction number becomes $R_0 = 3.589$. These values of R_0 are comparable to the ones obtained in [17, 19, 22].

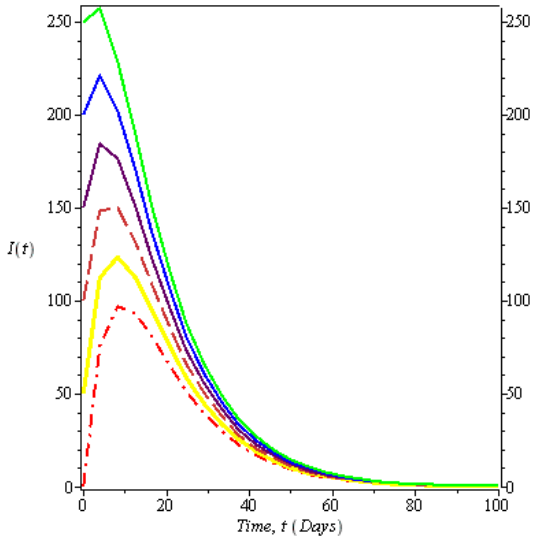

Fig. 1: *Plot of the global stability of the disease-free equilibrium with various initial conditions*

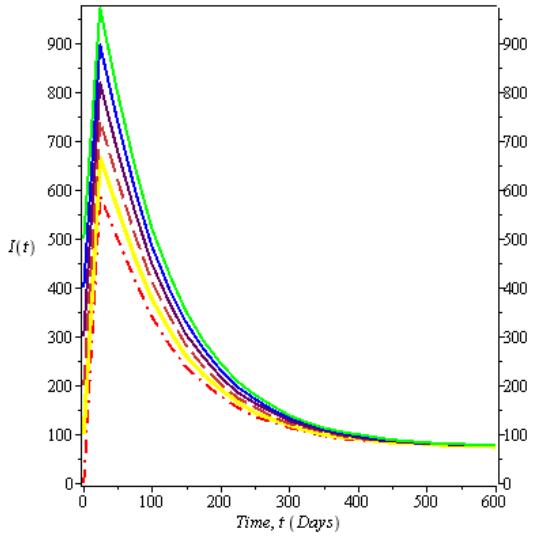

Fig. 2: *Plot of the global stability of the endemic equilibrium with various initial conditions*

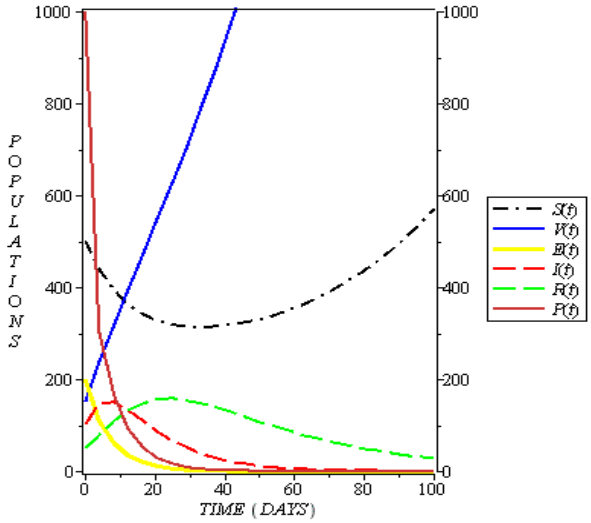

Fig. 3: *Plot of the populations with time at the given model's parameters*

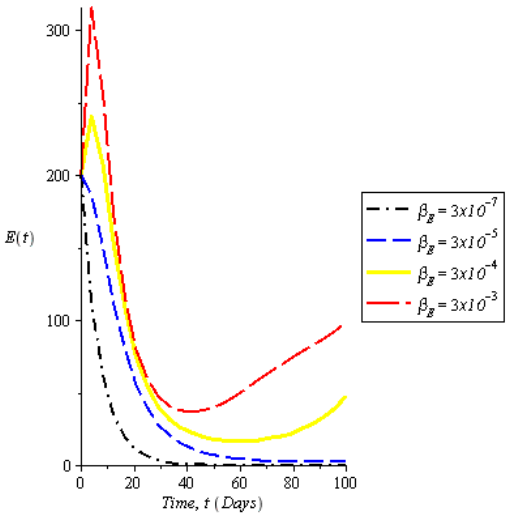

Fig. 4: *Plot of the effect of exposed contact rate, β_E, on the exposed population*

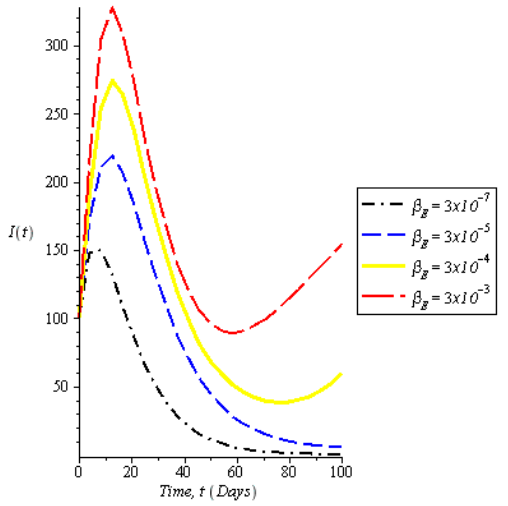

Fig. 5: *Plot of the effect of exposed contact rate, β_E, on the infected population*

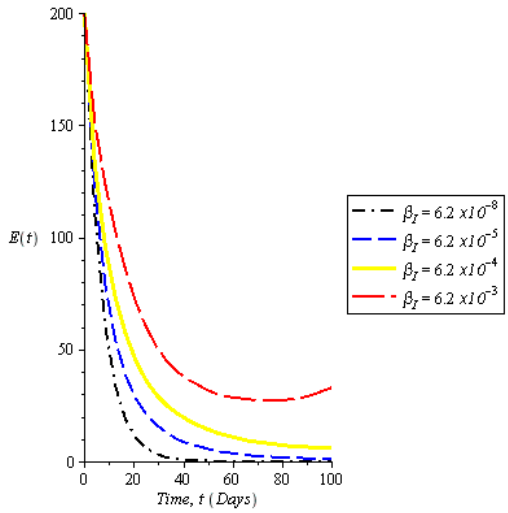

Fig. 6: *Plot of the effect of infected contact rate, β_I, on the exposed population*

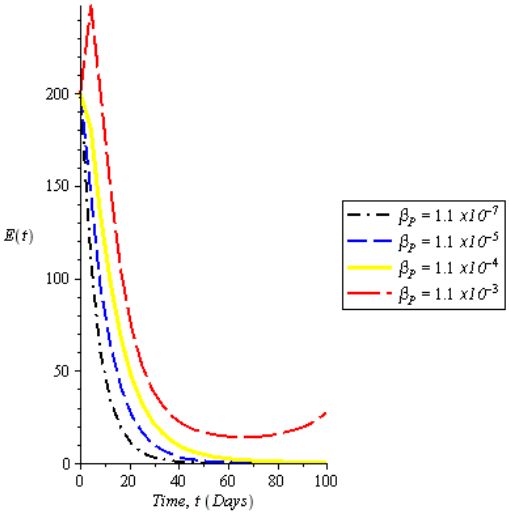

Fig. 7: *Plot of the effect of environmental virus contact rate, β_P, on the exposed population*

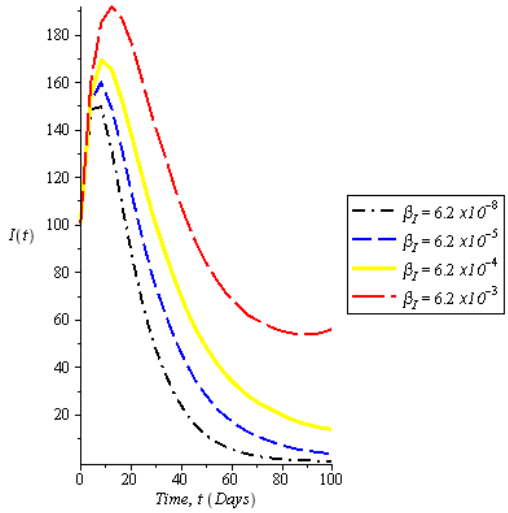

Fig. 8: *Plot of the effect of infected contact rate, β_I, on the infected population*

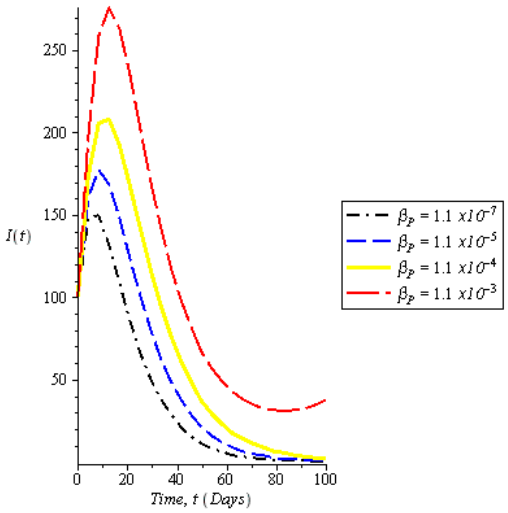

Fig. 9: *Plot of the effect of environmental virus contact rate, β_P, on the infected population*

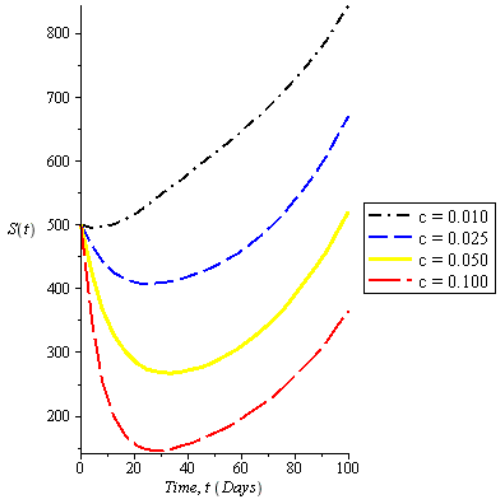

Fig. 10: *Plot of the effect of vaccination rate, c, on the susceptible population*

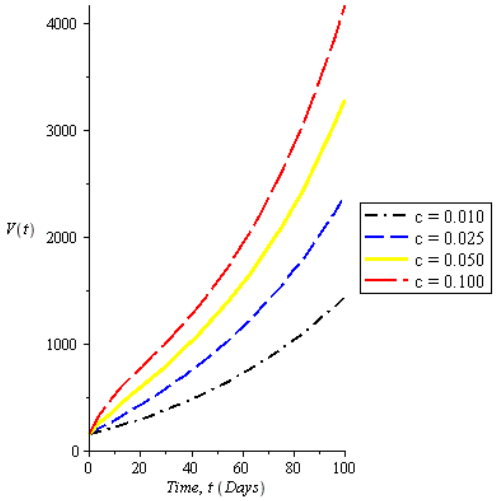

Fig. 11: *Plot of the effect of vaccination rate, c, on the vaccinated population*

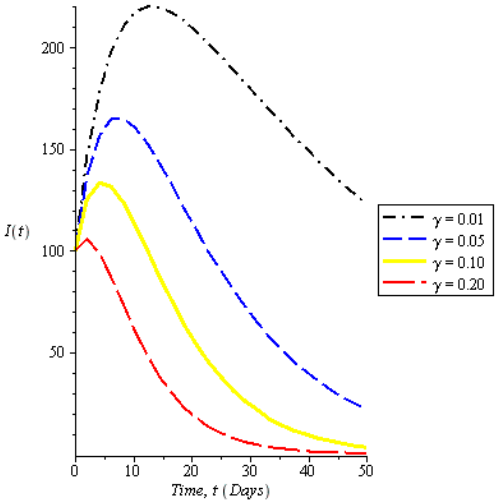

Fig. 12: *Plot of the effect of recovery rate, γ, on the infected population*

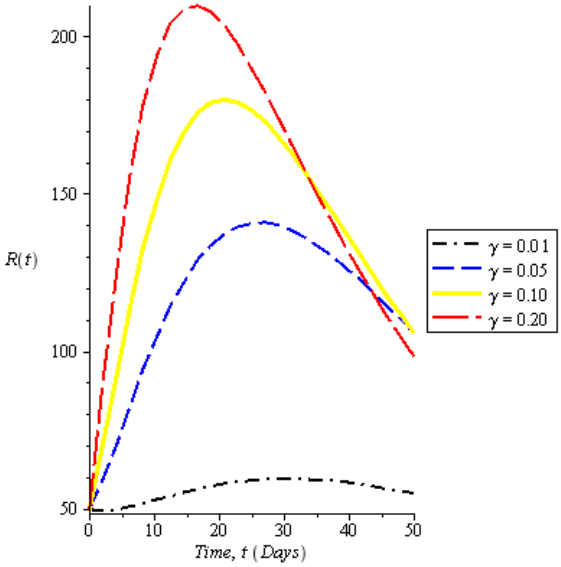

Fig. 13: *Plot of the effect of recovery rate, γ, on the recovered population*

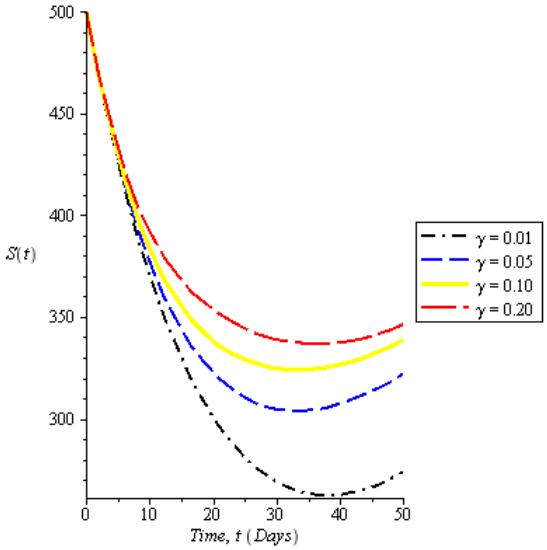

Fig. 14: *Plot of the effect of recovery rate, γ, on the susceptible population*

From Fig.3, which is the plot of all the populations against time for the given parameter values in Table 3. This plot indicates that the susceptible population declines as a result of the infection, but later shoots up. This shooting up is attributed to the manifestation of the vaccine effectiveness, which consequently increases the vaccinated population. The exposed, infected and the virus populations all reduce, while the recovered population increases initially, but later drops, due to loss in the acquired immunity.

Fig. 4 – Fig. 9 are plots showing the effects of the various transmission rates on the dynamics of COVID-19. Fig. 4 and Fig. 5 show that increasing the rate of contact with the exposed individuals (β_E) increases both the exposed and the infected populations. Similarly, Fig. 6 and Fig. 7 show that increasing the rate of contact with the infected individuals (β_I) increases the exposed and the infected populations, while increase in the rate of contact with the virus reservoir in the environment (β_P) also increases both the exposed and the infected populations as shown in Fig. 8 and Fig. 9. Comparisons between these plots in Fig. 4, Fig. 6, Fig. 7 and Fig. 5, Fig. 8, Fig. 9 show that contact with exposed individuals (β_E) results to more infections more than the contacts with the other two infectious populations (i.e. the infected and virus reservoir populations). This is due to the fact that the exposed individuals do not show symptoms, and as such may not be aware of their infection status, thereby multiplying the viral load in the environment and at the same time infecting the susceptible individuals. Contacts with the virus in the environment (β_P) also contribute to the increase of infection in the population, and hence, the need to take measures to clear the viruses from the environment, so as to bring down the curve of the infection in the population.

The effect of the rate of vaccination (c) was investigated and the results shown by the plots in Fig. 10 and Fig. 11. These plots depict that increasing the rate of vaccination (c) declines the susceptible population and increases the vaccinated population. A direct outcome of this is that lesser people will be prone to the disease as more people are being protected against it via vaccination, and so, there will be a great reduction in the infection.

Furthermore, we investigated the effect of the rate of recovery (γ) on the infection dynamics, the results of which are given by the plots in Fig. 12 – Fig. 14. Fig. 12 and Fig. 13, it was shown that increase in the rate of recovery (γ) decreases the infected population, while increasing the recovered population. However, it was noticed that the recovered population later declined. This is as a result of the effect of the immunity loss rate (τ), since COVID-19 is not known to confer permanent immunity. This implies that recovered individuals eventually lose their immunity and become susceptible again to the infection. This scenario is accounted for by the increase observed in the susceptible population when the rate of recovery increases, as depicted by the plot in Fig. 14.

5.2 Conclusion

In this research paper, we formulated and analysed an epidemic model for COVID-19 with saturated incidence rates. Intervention strategy focuses on vaccination and supportive treatment. The model is shown to be epidemiologically feasible and mathematically well-posed by establishing the region where the solutions set is nonnegative. The existence and stability of both disease-free and endemic equilibria were obtained, and these are dependent on a threshold value, called the basic reproduction number, R_0. If this value $R_0 < 1$, the disease, is under control, but if $R_0 > 1$, then COVID-19 will persist in the population.

We performed a sensitivity analysis on R_0 and the result showed that the most sensitive parameter is the rates of recruitment (Λ). Other parameters like rate of vaccination coverage (c) and rate of vaccine waning (θ), among others, are also sensitive to the reproduction number. Therefore, intervention strategies should be targeted towards these parameters, among others, so that the spread of the disease would be reduced. The negative effect of the recruitment rate (Λ) can be reversed by reducing the influx of people into the population. This can be achieved if restriction is placed on immigration. Movement of people from COVID-19 endemic places should be disallowed. And if at all such people will be allowed entry permit for one reason or the other, they should be quarantined for a minimum period of 14 days.

The numerical simulations performed showed that parameters like rates of contact with exposed individuals (β_E), contact with environmental virus (β_P), vaccination (c) and recovery (γ) have some effects on the dynamical spread of the disease. We therefore recommend that the contribution of the exposed individuals to infection be controlled by conducting mass testing for everyone irrespective of whether or not they show symptoms; and the use of quality face masks should be generally encouraged. This (together with frequent use of hand and surface sanitizers, and good personal and environmental hygiene) will also prevent contracting the viruses from the environment and surfaces, and as such the rate at which virus reservoirs contribute to COVID-19 will be reduced. Furthermore, very effective COVID-19 vaccines and antiviral cures should be developed as these will respectively provide protection against the disease and increase recovery of any infected individual, as obtained from the analyses of the model in the present work.

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