ISSN 2350-1022

International Journal of Recent Research in Mathematics Computer Science and Information Technology
Vol. 8, Issue 2, pp: (4-13), Month: October 2021 – March 2022, Available at: www.paperpublications.org

MATHEMATICAL MODELING OF COVID-19 TRANSMISSION DYNAMICS WITH SOCIAL DISTANCE AS AN INTERVENTION

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Abstract: **The dynamics of coronavirus (COVID-19) infection with social distance as an intervention are studied using a mathematical model based on a system of ordinary differential equations. The next generation matrix technique is used to calculate the fundamental reproduction number. The existence of the model's steady states is established, and the model's stability is investigated. The disease free (DFE) and endemic equilibrium (EE) points are found to be locally asymptotically stable using the Routh-Hurwitz criterion and center manifold theory, respectively. The model's numerical simulation revealed that social distance has a significant impact in COVID-19 transmission decrease.**

Keywords: **Coronavirus(COVID-19), Vaccination, Reproduction number, Stability.**

1. INTRODUCTION

The coronavirus 2019 (COVID-19) is a highly contagious respiratory disease caused by the new coronavirus-2 (SARS-CoV-2). Middle East Respiratory Syndrome (MERS) and Severe Acute Respiratory Syndrome (SARS) viruses are related to this virus. COVID-19 instances were recorded in Wuhan, Hubei Province, China, in late December 2019. COVID-19 was declared a world health pandemic by the World Health Organization (WHO) on March 11th, 2020. The pandemic is wreaking havoc on the world's economy, society, and health-care systems [1]. Researchers and policymakers are still working around the clock to discover solutions and devise measures to contain the epidemic and reduce its impact on human health and the global economy.

Humans are infected with the virus through respiratory droplets from sneezing, coughing, and talking, as well as contaminated surfaces [2]. Fever, dry cough, and exhaustion are the most typical COVID-19 symptoms. Body soreness and pain, sore throat, diarrhoea, headache, loss of taste and smell, difficulty breathing, rash on skin, and discolouration of fingers and toes are some of the other symptoms.

Because there are no specialized antiviral prophylaxis or treatments available, most afflicted countries are relying on non-pharmaceutical interventions such as isolation and quarantine of sick individuals, contact tracing, and community lockdown. The goal of these interventions is to contain and mitigate the epidemic before it overwhelms the country's health-care infrastructure. The number of deaths and hospitalizations appears to have decreased as a result of these preventative efforts [3].

Pre-existing disorders like cardiovascular disease, pulmonary disease, cancer, infectious disease, and substance addiction have been proven to increase COVID-19 morbidity and death in studies. Furthermore, pre-existing variables such as environmental, demographic, and socioeconomic factors may have an impact on COVID-19 incidence rates [1, 3]. Several vaccines have been approved by the World Health Organization (WHO), and each has a different effectiveness and mode of action. Furthermore, vaccination programs in different regions may differ due to variances in execution, such as vaccine availability. [4].

Ecology, population dynamics, tumor growth (cancer), immunology, epidemiology, and other biological applications have all made extensive use of mathematical models. In [5], an SEIQR model to investigate the spread of COVID-19 is formulated and analysed. The formulated model's disease-free equilibrium point was found to be globally asymptotically stable. The existence of endemic states has been demonstrated if the fundamental reproduction number is bigger than unity. The endemic states are proven to be locally and globally asymptotically stable using the Routh-Hurwitz criterion and appropriate Lyapunov functions.

In [6], the transmission of COVID-19 is studied using a compartmental mathematical model. The authors wanted to see if there was a way to control the unique COVID-19 using non-clinical methods like lockdown, frequent handwashing, controlling the disease's side effects, wearing a face mask, and using a sanitizer. To acquire the indices of the model's parameters, a sensitivity test was performed. By inserting control variables, the authors demonstrated that the most active transmission parameters are interposed. The impact of social distancing as an intervention on the transmission dynamics of COVID-19 disease is examined in this research.

2. MODEL FORMULATION

Our model classifies population into human population (N_H) and virus population (N_V). The human population is subdivided into four epidemiological classes. Namely; Susceptible individuals (S), Exposed individuals (E), Infectious individuals (I),and Removed/Recovered individuals (R). The virus population is denoted by V as the concentration of COVID-19 virus in the environment. The model assumed that susceptible individuals are recruited at the rate Λ become infected through via contact with environment contaminated by COVID-19 virus at the rate $\frac{(1-\omega)\beta V}{\kappa+V}$, where β the contact rate for human to contaminated environment and $\omega\beta(0 \leq \omega \leq 1)$ is a reduced contact rate for human to contaminated environment as result of social distancing. κ is the concentration of COVID-19 virus in the environment that yields 50% chance of getting COVID-19 and μ is natural death rate of human population. Exposed individuals progress to Infected class at the rate σ. A fraction of Infected individuals recover at the rate ϵ and some die from COVID-19 at the rate δ. α is the natural death rate of the virus. Furthermore, each infected individual contribute averagely to the virus population at the rate η and ω i. e., $(0 \leq \omega \leq 1)$ is a reduced contribution as of social distancing.

2.1 Assumptions of the Model

The following are the assumptions of the model:

1. The population birth and death rate occur at different rates.
2. Susceptibles are infected when aerosols or droplets in the environment containing the virus are inhaled or come directly into contact with the eyes, nose, or mouth.
3. Infectious individuals recover as result of treatment.
4. There is permanent immunity upon recovery.
5. All the newly born individuals will join only susceptible class.
6. Infectious individuals shade COVID-19 virus to the environment.

ISSN 2350-1022

International Journal of Recent Research in Mathematics Computer Science and Information Technology
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2.2 Model Flow Chart and Equations

The schematic flow describing the dynamics of the model is as shown in the figure below

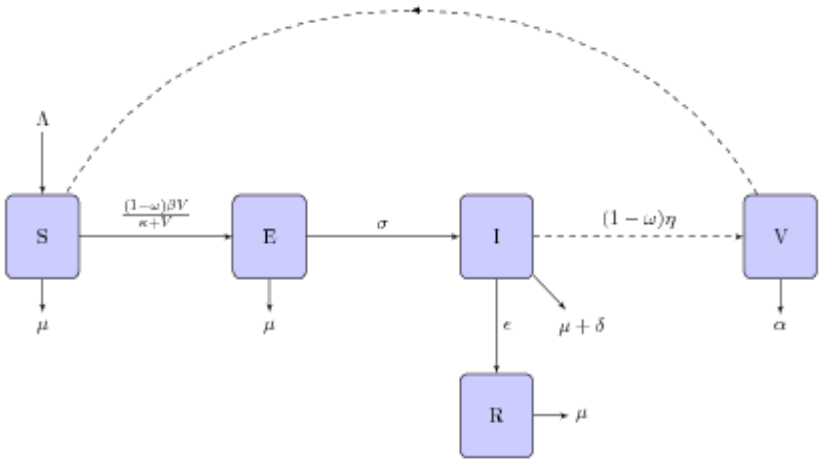

Figure 1: A schematic flow diagram

From the flow chart, Figure 1, we obtain the following differential equations of the model with $S(0) > 0$, $E(0) \geq 0$, $I(0) \geq 0$, $R(0) \geq 0$ and $V(0) \geq 0$, non-negative initial conditions.

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \frac{(1-\omega)\beta V}{\kappa+V}S - \mu S \\ \frac{dE}{dt} &= \frac{(1-\omega)\beta V}{\kappa+V}S - (\sigma+\mu)E \\ \frac{dI}{dt} &= \sigma E - (\epsilon+\delta+\mu)I \\ \frac{dR}{dt} &= \epsilon I - \mu R \\ \frac{dV}{dt} &= (1-\omega)\eta I - \alpha V \end{aligned} \tag{2.1}$$

3. ANALYSIS OF THE MODEL

Since the system (2.1) describes human population and Virus population, all the solutions of state variable with non-negative initial conditions are non-negative $\forall\ t > 0$ and they are bounded in the feasible region

$$\Gamma = \{(S,E,I,R) \in \mathbb{R}_+^4; V \in \mathbb{R}_+; S > 0; E,I,R,V \geq 0; N_H \leq \tfrac{\Lambda}{\mu}; N_V \leq \tfrac{\Lambda(1-\omega)\eta}{\mu\alpha}\}$$

Since $R(t) = N_H(t) - S(t) - I(t)$, it is enough to consider the four equations of system , our new system becomes

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \frac{(1-\omega)\beta V}{\kappa+V}S - \mu S \\ \frac{dE}{dt} &= \frac{(1-\omega)\beta V}{\kappa+V}S - (\sigma+\mu)E \\ \frac{dI}{dt} &= \sigma E - (\epsilon+\delta+\mu)I \\ \frac{dV}{dt} &= (1-\omega)\eta I - \alpha V \end{aligned} \tag{3.1}$$

3.1 Disease Free Equilibrium (DFE)

The disease free equilibrium point is given by $E^0 = (S^0, E^0, I^0, V^0) = (\frac{\Lambda}{\mu}, 0,0,0)$

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3.2 The Basic Reproduction Number

The basic reproduction number,R_0, refers to the number of secondary infections generated by a single infective individual in a completely susceptible population. We use next generation matrix, the approach by [7] to determine R_0. Using this method the basic reproduction number is given by $\rho(F_0V_0^{-1})$ (the dominant eigenvalue of $F_0V_0^{-1}$) where F_0 is the Jacobian of f_i at E^0, where f_i is the rate at which new infections appear in compartment i and V_0 is the Jacobian of v_i at E^0, where v_i is the rate of transfer of individuals into and out of compartment i. The infected population is captured in the following system of equations.

$$\begin{aligned} \frac{dE}{dt} &= \frac{(1-\omega)\beta V}{\kappa+V}S - (\sigma+\mu)E \\ \frac{dI}{dt} &= \sigma E - (\epsilon+\delta+\mu)I \\ \frac{dV}{dt} &= (1-\omega)\eta I - \alpha V \end{aligned} \tag{3.2}$$

from system (3.2) we have

$$f_i = \begin{bmatrix} \frac{(1-\omega)\beta V}{\kappa+V}S \\ 0 \\ 0 \end{bmatrix}$$

and

$$v_i = \begin{bmatrix} (\sigma+\mu)E \\ (\epsilon+\delta+\mu)I - \sigma E \\ \alpha V - (1-\omega)\eta I \end{bmatrix}.$$

Hence

$$F_0 = \begin{bmatrix} 0 & 0 & (1-\omega)\frac{\beta\Lambda}{\mu\kappa} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

and

$$V_0 = \begin{bmatrix} (\sigma+\mu) & 0 & 0 \\ -\sigma & (\epsilon+\delta+\mu) & 0 \\ 0 & -(1-\omega)\eta & \alpha \end{bmatrix}.$$

It follows that

$$F_0V_0^{-1} = \begin{bmatrix} \frac{(1-\omega)^2\beta\eta\Lambda\sigma}{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha\mu\kappa} & \frac{(1-\omega)^2\beta\eta\Lambda}{(\epsilon+\delta+\mu)\alpha\mu\kappa} & \frac{(1-\omega)\beta\Lambda}{\alpha\mu\kappa} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

Since

$$\mathcal{R}_0 = \rho(F_0V_0^{-1})$$

We have

$$\mathcal{R}_0 = \frac{(1-\omega)^2\beta\eta\Lambda\sigma}{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha\mu\kappa}$$

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3.3 Existence of the Endemic Equilibrium (EE)

This refers to a spreading point of disease in the population.

Theorem 3.1 *Let* $S^*, E^*, I^*, , V^* > 0$, *there exist a unique endemic equilibrium point* $E^* = (S^*, E^*, I^*, V^*)$ *when* $\mathcal{R}_0 > 1$.

Proof. Equating the right hand side of the system (3.1) to zero and replacing (S,E,I,V) with (S^*, E^*, I^*, V^*),we obtain

$$\begin{aligned} &\Lambda - \frac{(1-\omega)\beta V^*}{\kappa + V^*} S^* - \mu S^* = 0 \\ &\frac{(1-\omega)\beta V^*}{\kappa + V^*} S^* - (\sigma + \mu) E^* = 0 \\ &\sigma E^* - (\epsilon + \delta + \mu) I^* = 0 \\ &(1-\omega)\eta I^* - \alpha V^* = 0 \end{aligned} \tag{3.3}$$

The last three equations of system (3.3) give

$$I^* = \frac{\sigma E^*}{(\epsilon + \delta + \mu)}$$

$$V^* = \frac{(1-\omega)\eta\sigma E^*}{\alpha(\epsilon + \delta + \mu)}$$

Adding the first two equations of system (3.3) and solving for S^*, we obtain

$$S^* = \frac{\Lambda - (\sigma + \mu)E^*}{\mu}$$

Replacing S^* and V^* in the second equation of system (3.3) and solving for E^* gives

$$E^* = \frac{(1-\omega)^2\beta\eta\sigma\Lambda - (\sigma+\mu)\alpha(\epsilon+\delta+\mu)\mu\kappa}{[(1-\omega)(\sigma+\mu)\mu\eta\sigma + (1-\omega)^2\beta\eta\sigma(\sigma+\mu)]}$$

Clearly $E^* > 0$ if $\mathcal{R}_0 > 1$

3.4 Local stability of disease free equilibrium point

Now we prove the local stability of the disease-free equilibrium E^0.

Theorem 3.2 *The disease-free equilibrium (DFE) point of the system (3.1) is locally asymptotically stable if and only if* $\mathcal{R}_0 < 1$.

Proof. The Theorem 3.2 is proved using linearization method. The Jacobian matrix associated with the reduced model system (3.1) at the DFE is given as

$$J(E^0) = \begin{bmatrix} -\mu & 0 & 0 & -(1-\omega)\frac{\beta\Lambda}{\mu\kappa} \\ 0 & -(\sigma+\mu) & 0 & (1-\omega)\frac{\beta\Lambda}{\mu\kappa} \\ 0 & \sigma & -(\epsilon+\delta+\mu) & 0 \\ 0 & 0 & (1-\omega)\eta & -\alpha \end{bmatrix} \tag{3.4}$$

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The trace of the Jacobian matrix (3.4) is negative and the its determinant is given by

$$Det(J(E^0)) = \mu[(\sigma+\mu)(\epsilon+\delta+\mu)\alpha - \frac{(1-\omega)^2\eta\beta\Lambda\sigma}{\mu\kappa}]$$

Clearly, the determinant is positive if $\mathcal{R}_0 < 1$. Therefore, by Routh-Hurwitz criteria, the DFE E^0 is locally asymptotically stable.

3.5 Local stability of endemic equilibrium point

In this section we analyze the local stability of the endemic equilibrium point.

Theorem 3.3 *The endemic equilibrium E^* of system (3.1) is locally asymptotic stable for $\mathcal{R}_0$ near 1.*

Proof. Using the center manifold theory [8] as described in Theorem 4.1 of [9], we let

$$X = (x_1, x_2, x_3, x_4) = (S, E, I, V) \tag{3.5}$$

Thus, system (3.1) can be written as follows:

$$\begin{aligned} x_{1'}(t) &= f_1 = \Lambda - \frac{(1-\omega)\beta x_4}{\kappa+x_4}x_1 - \mu x_1 \\ x_{2'}(t) &= f_2 = \frac{(1-\omega)\beta x_4}{\kappa+x_4}x_1 - (\sigma+\mu)x_2 \\ x_{3'}(t) &= f_3 = \sigma x_2 - (\epsilon+\delta+\mu)x_3 \\ x_{4'}(t) &= f_4 = (1-\omega)\eta x_3 - \alpha x_4 \end{aligned} \tag{3.6}$$

Taking $\beta = \beta^*$ as the bifurcation parameter that occurs at $\mathcal{R}_0 = 1$, and solve for β^*we have

$$\beta^* = \frac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha\mu\kappa}{(1-\omega)^2\eta\Lambda\sigma}$$

The Jacobian of system (3.6) at E^0 and $\beta = \beta^*$ is given by

$$J^*(E^0) = \begin{bmatrix} -\mu & 0 & 0 & -\frac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma} \\ 0 & -(\sigma+\mu) & 0 & \frac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma} \\ 0 & \sigma & -(\epsilon+\delta+\mu) & 0 \\ 0 & 0 & (1-\omega)\eta & -\alpha \end{bmatrix} \tag{3.7}$$

The right eigenvector associated to matrix (3.7) is denoted by $w = [w_1 \quad w_2 \quad w_3 \quad w_4]^T$ and it is given by

$$\begin{bmatrix} -\mu & 0 & 0 & -\frac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma} \\ 0 & -(\sigma+\mu) & 0 & \frac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma} \\ 0 & \sigma & -(\epsilon+\delta+\mu) & 0 \\ 0 & 0 & (1-\omega)\eta & -\alpha \end{bmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$$\begin{cases} -\mu w_1 - \frac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma}w_4 = 0 \\ -(\sigma+\mu)w_2 + \frac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma}w_4 = 0 \\ \sigma w_2 - (\epsilon+\delta+\mu)w_3 = 0 \\ (1-\omega)\eta w_3 - \alpha w_4 = 0 \end{cases} \begin{cases} w_1 = -\frac{(\sigma+\mu)(\epsilon+\delta+\mu)}{\sigma\mu}w_3 \\ w_2 = \frac{(\epsilon+\delta+\mu)}{\sigma}w_3 \\ w_3 = w_3 \\ w_4 = \frac{(1-\omega)\eta}{\alpha}w_3 \end{cases}$$

ISSN 2350-1022

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The left eigenvector associated to matrix (3.7) is denoted by $v = [v_1 \quad v_2 \quad v_3 \quad v_4 \quad]^T$ and it is given by

$$\begin{bmatrix} -\mu & 0 & 0 & 0 \\ 0 & -(\sigma+\mu) & \sigma & 0 \\ 0 & 0 & -(\epsilon+\delta+\mu) & (1-\omega)\eta \\ -\dfrac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma} & \dfrac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma} & 0 & -\alpha \end{bmatrix} \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$$\begin{cases} -\mu v_1 = 0 \\ -(\sigma+\mu)v_2 + \sigma v_3 = 0 \\ -(\epsilon+\delta+\mu)v_3 + (1-\omega)\eta v_4 = 0 \\ -\frac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma} v_1 + \frac{(\sigma+\mu)(\epsilon+\delta+\mu)\alpha}{(1-\omega)\eta\sigma} v_2 - \alpha v_4 = 0 \end{cases}$$

$$\begin{cases} v_1 = 0 \\ v_2 = \dfrac{\sigma}{(\sigma+\mu)} v_3 \\ v_3 = v_3 > 0 \\ v_4 = \dfrac{(\epsilon+\delta+\mu)}{(1-\omega)\eta} v_3 \end{cases}$$

The coefficients a and b are given below

$$a = \sum_{k,i,j=1}^{4} v_k w_i w_j \left[\frac{\partial^2 f_k}{\partial x_i \partial x_j}(E^0) \right]_{\beta=\beta^*}$$

$$b = \sum_{k,i=1}^{4} v_k w_i \left[\frac{\partial^2 f_k}{\partial x_i \partial \theta}(E^0) \right]_{\beta=\beta^*}$$

For computation of a, the non-zero partial derivatives of $f = (f_1, f_2, f_3, f_4)$ at DFE E^0 and $\beta = \beta^*$ for model (3.6) are given by

$\frac{\partial^2 f_1}{\partial x_1 \partial x_4}(E^0) = \frac{\partial^2 f_1}{\partial x_4 \partial x_1}(E^0) = -\frac{(1-\omega)\beta^*}{\kappa}$, $\frac{\partial^2 f_2}{\partial x_1 \partial x_4}(E^0) = \frac{\partial^2 f_2}{\partial x_4 \partial x_1}(E^0) = \frac{(1-\omega)\beta^*}{\kappa}$

$\frac{\partial^2 f_1}{\partial x_4^2}(E^0) = \frac{2(1-\omega)\beta^*\Lambda}{\kappa\mu}$, $\frac{\partial^2 f_2}{\partial x_4^2}(E^0) = \frac{-2(1-\omega)\beta^*\Lambda}{\kappa\mu}$

Since $v_1 = 0$, it follows that

$$a = \left[v_2 w_1 w_4 \frac{\partial^2 f_2}{\partial x_1 \partial x_4}(E^0) + v_2 w_4 w_1 \frac{\partial^2 f_2}{\partial x_4 \partial x_1}(E^0) + v_2 w_4 w_4 \frac{\partial^2 f_2}{\partial x_4^2}(E^0) \right]_{\beta=\beta^*} \tag{3.8}$$

Simplifying equation (3.8), we obtain

$$a = -2\frac{\sigma}{(\sigma+\mu)} v_3 w_3^2 \left[\frac{(\sigma+\mu)(\epsilon+\delta+\mu)(1-\omega)^2\eta\beta^*}{\sigma\mu\alpha\kappa} + \frac{(1-\omega)^3\eta^2\beta^*\Lambda}{\kappa\mu\alpha^2} \right] < 0$$

For b, we have the non-zero partial derivatives of $f = (f_1, f_2, f_3, f_4)$ at DFE E^0 and $\theta = \beta = \beta^*$ for model (3.6) are given by

$\frac{\partial^2 f_1}{\partial x_4 \partial \beta}(E^0) = \frac{\partial^2 f_1}{\partial \beta \partial x_4}(E^0) = -\frac{(1-\omega)\Lambda}{\kappa\mu}$

$\frac{\partial^2 f_2}{\partial x_4 \partial \beta}(E^0) = \frac{\partial^2 f_2}{\partial \beta \partial x_4}(E^0) = \frac{(1-\omega)\Lambda}{\kappa\mu}$

Since $v_1 = 0$, it follows that

$$b = v_2 w_4 \left[\frac{\partial^2 f_2}{\partial x_4 \partial \beta}(E^0)\right]_{\beta=\beta^*}$$

$$b = \frac{(1-\omega)\sigma\eta}{(\sigma+\mu)\alpha} v_3 w_3 \frac{(1-\omega)\Lambda}{\kappa\mu} > 0$$

Therefore, by Theorem 4.1 of [9], we conclude that the endemic equilibrium E^* of system (3.1) is locally asymptotic stable for a value of the basic reproduction number $\mathcal{R}_0$ close to 1.

4. NUMERICAL SIMULATION AND DISCUSSION OF RESULTS

We carry out numerical simulations of the model (3.1). The parameter values used are presented in Table 1.

Table 1: Parameter values of the model

Parameter symbol	Value	Source
Λ	0.00018 /day	[10]
μ	4.563×10^{-5}/day	[10]
β	0.25/day	Assumed
κ	0.1	Assumed
δ	0.0018/day	Assumed
σ	$\frac{1}{5.1}$/day	Assumed
η	0.1/day	Assumed
ϵ	0.1/day	[10]
α	0.1724/day	Assumed
ω	$0 \leq \omega \leq 1$	Assumed

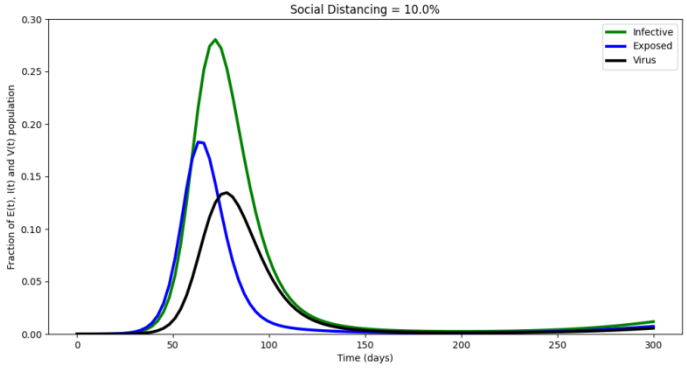

Fig. 2. Variation of E(t), I(t) and V(t) when social distancing is 10%

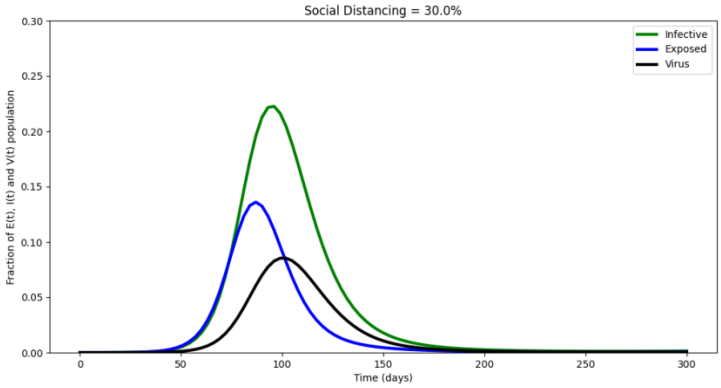

Fig. 3. Variation of E(t), I(t) and V(t) when social distancing is 30%

ISSN 2350-1022

International Journal of Recent Research in Mathematics Computer Science and Information Technology
Vol. 8, Issue 2, pp: (4-13), Month: October 2021 – March 2022, Available at: www.paperpublications.org

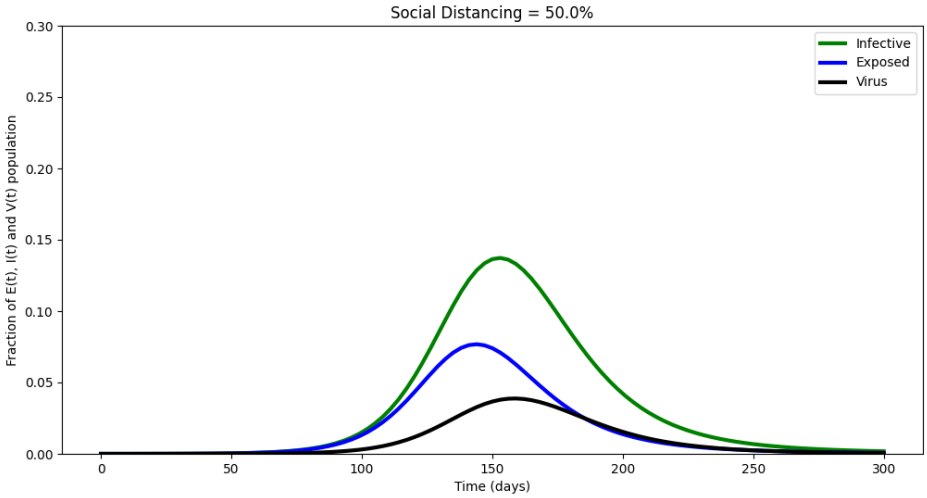

Fig. 4. Variation of E(t), I(t) and V(t) when social distancing is 50%

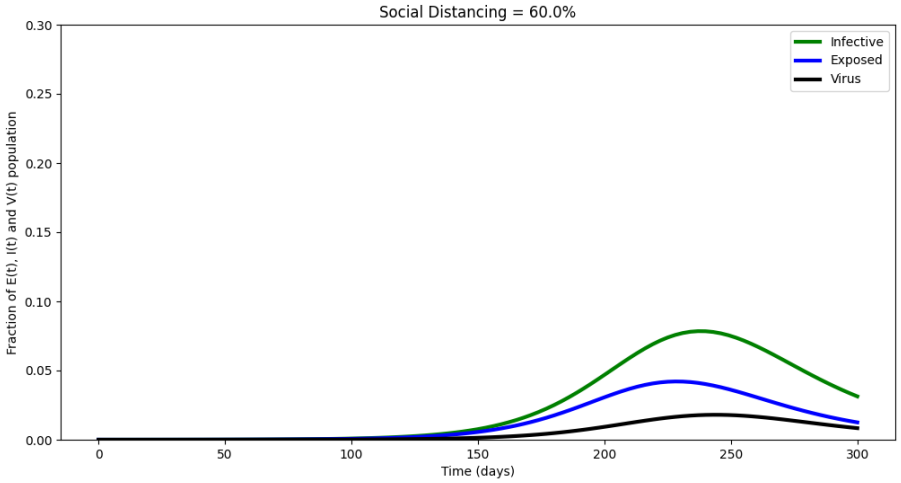

Fig. 5. Variation of E(t), I(t) and V(t) when social distancing is 60%

In Figures 2-5 the distribution of E(t), I(t) and V(t) population at different levels of social distancing is shown. From the figures, it is seen that when a constant social distancing policy is enforced, the outbreak peaks before receding. It is also seen that the curves flatten as the constant social distancing increases.

5. CONCLUSION

In this study, SEIRV model on the transmission dynamics of COVID-19 disease with social distancing as intervention is presented and analysed. First, the model well-posedness is proved. The basic reproduction number, $\mathcal{R}_0$, is computed using the next generation matrix approach. By Routh-Hurwitz criterion and center manifold theory the disease free (DFE) and the endemic equilibrium (EE) points are found to be locally asymptotically stable respectively. The stability results showed that the model solutions would always converge to the DFE whenever $\mathcal{R}_0 < 1$ which epidemiologically implies that if a few infectious individuals are introduced into a fully susceptible population, the disease would die out if there are no secondary infections produced whenever $\mathcal{R}_0 < 1$, otherwise the disease would spread. The simulations analysis shows that the infection curve flattens as social distancing increases. This implies that if social distancing protocol is adhered to, then COVID-19 infections will eventually reduce with time.

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