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RESEARCH ARTICLE

Dynamics of COVID-19 Incorporating Preventive Measures and Treatment

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Abstract:
The surge of Coronavirus disease (COVID-19) was felt all over the world greatly after it was declared a pandemic in the year 2020. After 3 years in 2023, the disease passed the pandemic phase and entered an endemic phase. But that didn't reduce the global threat of the disease as the disease continues to still claim lives daily. In this work, we examined the dynamics of the coronavirus disease from a mathematical view using a deterministic SEI_AI_SQVRI_PL_P model. This consists of investigating the disease-free and endemic equilibria, basic reproduction number and stability. The local stability of the disease-free equilibrium was determined by solving the Jacobian matrix of the system of differential equations while the basic reproduction number was calculated using the next generation matrix method. Numerical simulations to determine the active factor(s) in the transmission, preventive and possible elimination of the disease were carried out using a computational software called Maple. It was revealed that over time when all modalities are out into place the rate of recovery increases and as the rate of the pathogen virus death increases, the pathogen virus gradually fades from the environment.

Keywords: Coronavirus, basic reproduction number, stability, Jacobian matrix, pandemic

1. Introduction

Throughout history, there has been occurrences of various diseases with each one's level varying. One of such diseases is Coronavirus also known as COVID-19 which is caused by a novel virus severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). In December 2019, the first cases of the disease were reported in Wuhan, China. By January 2020, the disease had spread to other countries, and the World Health Organization (WHO) declared the COVID-19 outbreak as a global health emergency. It was declared as a pandemic by WHO on March 11, 2020 [1]. It is believed to have originated from a seafood and wet animal market in which the first victims contacted the disease. Scientists gave the virus its name due to research showing genetic similarities in bat coronaviruses. The virus spreads mainly between people who are in close contact with each other, for example a conversational distance. The virus can spread from an infected person's mouth or nose in small liquid particles when they cough, sneeze, speak, sing or breathe.

Table 1.1: Description of Parameters

Parameters	Biological significance	Values
b	birth rate	0.00018
μ	natural death rate	0.1724
α_1	proportion of interaction with an infectious environment	0.1
α_2	proportion of interaction with an infectious individual	0.1
β_1	rate of transmission from S to E due to contact with P	0.00414
β_2	rate of transmission from S to E due to contact with I_A and/or I_S	0.0115
φ	progression rate from E back to S due to robust immune system	0.0051
δ	proportion of symptomatic infectious people	0.05
ω	progression rate from E to either I_A or I_S	0.09
σ	disease induced death rate	0.0018
γ_S	rate of recovery of the symptomatic population	0.05
γ_Q	rate of recovery of the quarantined population	0.1
γ_A	rate of recovery of the asymptomatic population	0.0714
η_S	rate of virus spread to the environment by symptomatic infectious individuals	0.1
η_A	rate of virus spread to the environment by asymptomatic infectious individuals	0.05
λ	rate of transmission from R to S due to recovery from the disease	$\frac{1}{14}$
c	vaccine effectiveness	0.5
τ_5	progression rate from symptomatic infected class to quarantined class	0.04
τ_2	progression rate from asymptomatic infected class to quarantined class	0.04
τ_6	progression rate from symptomatic infected class to vaccinated class	0.02
μ_P	natural death rate of the pathogen	0.1724
τ_3	progression rate from asymptomatic infected class to vaccinated class	0.02
m	progression rate from Q to either V or R	0.3
σ_1	rate of treated humans	0.0018
σ_2	rate of recovered humans	0.0018
d_v	positive 12-month periodic continuous function	0.02
β	positive 12-month periodic continuous function	0.01

Another person can contract the virus when infectious particles that pass through the air are inhaled at short range (this is often called short-range airborne transmission) or if infectious particles come into direct contact with the eyes, nose or mouth (droplet transmission). It can also be spread in poorly ventilated and/or crowded indoor settings, where people spend longer periods of time (this is often called long-range airborne transmission). People may also become infected when touching their eyes, nose or mouth after touching surfaces or objects that have been contaminated by the virus [2]. The severity of COVID-19 symptoms can range from very mild to severe. Some people may have only a few symptoms while some may have no symptoms at all, but can still spread it. This is called

asymptomatic transmission. Some people may experience worsened symptoms such as pneumonia or respiratory failure [3].

Several researchers have worked on the transmission dynamics of Covid-19 using various numerical techniques. The dynamics of local outbreaks of COVID-19 by developing a SEIQR type deterministic model which uses a system of ordinary differential equations was analysed by [4]. From the data gotten from the outbreak in Hubei they were able to predict the trajectory of daily cases, daily deaths, and other features of the Hubei outbreak. Through numerical experiments they observed the effects of quarantine, social distancing, and COVID-19 testing on the dynamics of the outbreak. Din and Algehyne (2021) developed a Covid-19 SIR model containing three classes; Susceptible $S(t)$, Infected $I(t)$, and Recovered $R(t)$ with the convex incidence rate. The disease-free and endemic equilibrium were calculated for the model as well as the basic reproduction number. Also, the Global Stability was calculated using the Lyapunov Function construction, while the Local Stability was determined using the Jacobian matrix, while [5] worked on the impact of various non-pharmaceutical control measures both government and personal on the population dynamics of the novel coronavirus disease 2019 (COVID-19) in Lagos, Nigeria, using an appropriately formulated mathematical model. They used numerical stimulations to show the effect of control measures on the dynamics of Covid while [6] developed a mathematical model to understand the transmission dynamics and control of Covid-19 in Nigeria, one of the epicenters of Covid-19 in Africa. The epidemiological implication of the result showed that the pandemic can be effectively controlled or even eliminated in Nigeria if the control strategies implemented can bring and maintain the epidemiological threshold (R_o) to a value less than unity. It was however shown that Covid-19 can be effectively controlled using social distancing measures provided its effectiveness level is at least moderate.

Also, in considering the grave implications of the continuous spread of coronavirus disease, A SEIHRD epidemic model which consisted of the Susceptible, Exposed, Infected, Hospitalized, Recovered and Deceased individuals was formulated [7], to gain insight into the disease transmission dynamics with impacts of proposing control measures. The model captured the impact of undetected infectious individuals and detected hospitalized individuals with saturated treatment on the spread, death and recovery of Covid-19 patients in Nigeria. Results obtained suggested that decreasing the transmission rate for infective alone is not sufficient to eradicate the disease because of the presence of backward bifurcation, and recommended that Nigerians must also adhere strictly to COVID-19 protocols in mitigating the spread and demise of the coronavirus disease. A SEAIQR model to examine the transmission mechanism of COVID-19 among humans was developed by [8]. The population was distributed into Susceptible, Exposed, Asymptomatic Infected,Symptomatic Infected, Quarantined and Recovered humans respectively. The existence and stability of disease-free equilibrium were established. Results showed that the effectiveness of control measures (reducing contact rate and usage of face mask) when being applied. It is noticed that the best option is to observe social distance against the use of a mask. The effective approach is the compliance with both control measure which are social distancing and usage of mask. It was recommended that there should be educational campaigns on the impact of embracing social distancing, wearing a mask, need to be vaccinated as well as the enforcement and sanctions for non-compliance with the control measures. Other relevant works by researchers on Covid can be found in [9],[10],[11],[12],[13] and [14].

Despite the contributions of the aforementioned authours and several other ones, research on COVID-19 transmission dynamics, incorporating preventive measures and treatment, is essential to address research gaps regarding the effectiveness of interventions, long-term implications on public health and healthcare systems, vaccine efficacy, and the adaptation of healthcare systems, informing evidence-based policies to mitigate the spread and minimize its impact on communities.

2. Mathematical/ Problem Formulation

In this current study, we modified the work of [15] to include transmission dynamics, severity of the virus on human population, quarantine, vaccination, and disease induced death rate. We shall also make use of available data to suggest and predict the dynamics of the disease in the future. To study the spread rate of the disease among humans and the environment, we developed a model in which the total human population at time t, denoted by $N_h(t)$, is split into a mutually exclusive sub-populations of susceptible humans ($S(t)$), Exposed humans $E(t)$, Quarantine on exposed humans $Q(t)$, Vaccinated humans $V(t)$ asymptomatic infectious humans $I_A(t)$, symptomatic infectious humans ($I_S(t)$), and recovered humans ($R(t)$). The virus pathogen class denoted $P(t)$ is divided into two classes viz: the latent class $L_P(t)$ and infectious class $I_P(t)$ class and it is assumed to have interaction between exposed, and infected classes.

Thus, we have total human population $N_h(t)$ and pathogen population P(t) defined respectively:

$$\begin{aligned} N_h(t) &= S(t) + E(t) + Q(t) + V(t) + I_S(t) + I_A(t) + R(t) \\ P(t) &= L_{\mathrm{P}}(t) + I_P(t) \end{aligned} \tag{2.1}$$

The human population is born into susceptible population at a rate b. The terms $\beta_1 S(I_P + L_P)$ and $\beta_2 S(I_A + I_S)$ describe the rate at which susceptible individuals $S(t)$ gets infected by pathogens in the environment $L_P(t)$ and $I_P(t)$ respectively and from infectious human $I_A(t)$ and $I_S(t)$ respectively. Health experts and governments have been advising people, during this outbreak, to minimize contact with infectious individuals through social distancing. But it is extremely difficult if not impossible to identify infectious individuals except those who are symptomatic. Therefore, as proposed by [15], we assume to have a new infection respectively in the form

$$\frac{\beta_1 S(I_P + L_P)}{1 + \alpha_1 I_P} \text{and} \frac{\beta_2 S\,(I_A + I_S)}{1 + \alpha_2 I_P}$$

Where the interaction proportion α_1 and α_2 denotes reciprocal of the frequency with which susceptible individuals gets infected with Covid-19 from the environment and from infectious individuals, respectively.

Assumptions: Taking all the sub-classes enumerated above into consideration, we assume the following:

1. We assume that those in the $Q(t)$ and $V(t)$ are completely isolated and do not come in contact with the general population.
2. The migration rate to the community increases the total population.
3. Recovered population could still become susceptible.
4. Exposed persons could either be symptomatic or asymptomatic after exposure.
5. Either symptomatic or asymptomatic could be vaccinated or quarantine.
6. Both susceptible and exposed persons could also be vaccinated or quarantine.
7. Exposed, asymptomatic, symptomatic, quarantine, could recovered fully due to natural immunity.
8. When the natural immunity wane, the recovery becomes partial recovery.
9. Symptomatic or asymptomatic humans could recover or die due to the disease, every other person in the system could die a natural death.

Owing to the above assumption, the compartmental block diagram represented below, shows the interaction within the community.

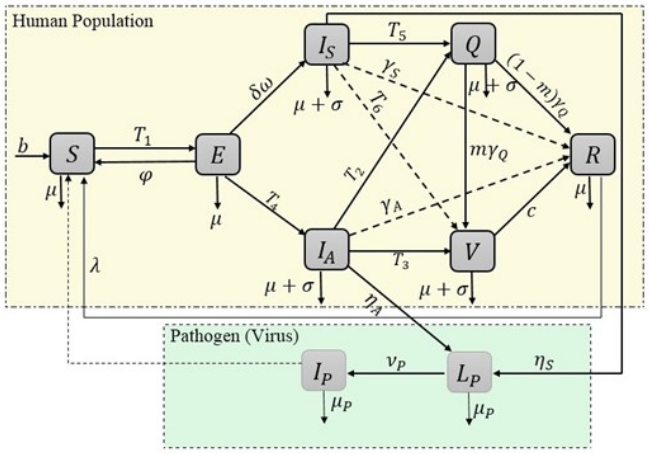

Figure 2.1: Model Compartmental Diagram

Mathematically, these interactions are described by a system of ordinary differential equations as shown below:

$$\frac{\mathrm{dS}}{\mathrm{dt}} = b + \lambda R + \varphi E - \left(\frac{\beta_1 S\left(I_P + L_p\right)}{1+\alpha_1 I_P} + \frac{\beta_2 S\left(I_A + I_S\right)}{1+\alpha_2 I_P}\right) - \mu S, \tag{2.2}$$

$$\frac{dE}{dt} = \left(\frac{\beta_1 S\left(I_P + L_p\right)}{1+\alpha_1 I_P} + \frac{\beta_2 S\left(I_A + I_S\right)}{1+\alpha_2 I_P}\right) - \varphi E - \mu E - \delta\omega E - (1-\delta)\omega E, \tag{2.3}$$

$$\frac{dI_A}{\mathrm{dt}} = (1-\delta)\omega E - \tau_3 I_A - \gamma_A I_A - \tau_2 I_A - (\mu+\sigma) I_A - \eta_A I_A, \tag{2.4}$$

$$\frac{dI_S}{\mathrm{dt}} = \delta\omega E - \tau_6 I_S - \tau_5 I_S - (\mu+\sigma) I_S - \eta_S I_S - \gamma_S I_S, \tag{2.5}$$

$$\frac{dQ}{dt} = \tau_5 I_S + \tau_2 I_A - m\gamma_Q Q - (\mu+\sigma) Q - (1-m)\gamma_Q Q, \tag{2.6}$$

$$\frac{dV}{dt} = m\gamma_Q Q + \tau_6 I_S + \tau_3 I_A - cV - (\mu+\sigma) V, \tag{2.7}$$

$$\frac{\mathrm{dR}}{\mathrm{dt}} = cV + \gamma_A I_A + \gamma_S I_S + (1-m)\gamma_Q Q - \lambda R - \mu \tag{2.8}$$

$$\frac{dL_P}{\mathrm{dt}} = \eta_S I_S + \eta_A I_A - \frac{H(t)}{N_h(t)} L_P - \left(d_v \beta + \mu_P\right) L_p \tag{2.9}$$

$$\frac{dI_P}{\mathrm{dt}} = \frac{H(t)}{N_h(t)} L_P - \left(d_v \beta + \mu_P\right) I_p \tag{2.10}$$

where, $H(t)$ and $N_h(t)$ are respectively define by;

$$H(t) = I_h(t) + \sigma_1 T_h(t) + \sigma_2 R_h(t) \quad \text{and}$$

$$N_h(t) = S(t) + E(t) + Q(t) + V(t) + I_S(t) + I_A(t) + R(t)$$

In Figure 2.1, we have the following denotations with the biological significance defined in the table of parameters, Table 1.1.

$$T_1 = \frac{\beta_1 S\left(I_P + L_p\right)}{1+\alpha_1 I_P} + \frac{\beta_2 S\left(I_A + I_S\right)}{1+\alpha_2 I_P}, T_4 = (1-\delta)\omega E,\ T_2 = \tau_2,\ T_3 = \tau_3,\ T_5 = \tau_5,\ T_6 = \tau_6$$

3. Method of Solution

3.1. Equilibrium States

Equilibrium points in epidemiology refer to stable states in disease dynamics where the number of individuals infected with a disease remains constant over time. These points represent a balance between the factors driving disease transmission (such as contact rates and infectiousness) and those reducing transmission (such as immunity or intervention measures). Equilibrium points can occur at different levels of disease prevalence, including no infection (disease-free equilibrium) or a stable level of infection (endemic equilibrium). We examine both the Disease-Free Equilibrium Point (DFEP) and Endemic Equilibrium point in a bid to assess the effectiveness of interventions in controlling infectious diseases.

3.1.1. The Disease-Free Equilibrium Point (DFEP)

This represents the average size of each of the compartments when the entire population is free from the infection. It is denoted by $\mathbb{E}_0$. We obtain $\mathbb{E}_0$ by equating the right-hand side of the model equations to zero and solving the resulting algebraic system of equations. Since we are considering the disease-free equilibrium point, we put $I_A = I_S = L_P = I_P = 0$, which implies that $E = Q = V = R = 0$. We then have:

$$S = \frac{b}{\mu}$$

Therefore $\mathbb{E}_0 = \left(\frac{b}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0\right)$

3.1.2. The Endemic Equilibrium Point (EEP)

The endemic equilibrium point is the average size of each of the model compartments, when the disease has become part of the human population. The model admits an endemic equilibrium $\mathbb{E}_e = (S,\ E,\ I_A,\ I_S, Q,\ V,\ R,\ L_P,\ I_P)_e$ when $I_A > 0,\ I_S > 0,\ L_P > 0,\ I_P > 0$. $\mathbb{E}_e$ is obtained by equating the right-hand side of the model equations to zero and solving the corresponding system. Thus, we obtain the following result:

$$E_e = (S, E, I_A, I_S, Q, V, R, L_P, I_P) = \left(\frac{b}{\mu}, 0, 0, 0, 0, 0, 0, 0, 0\right),$$
$$\left(S_0 + k_6 I_S,\ \frac{k_2}{\delta\omega} I_S,\ \frac{(1-\delta)k_2}{k_1\delta} I_S, -\frac{n_1 \mp \sqrt{n_1^2 - 4n_0 n_2}}{2n_2}, k_3 I_S, k_4 I_S, k_5 I_S\ ,\right.$$
$$\left.\frac{k_8 S_0 + k_8\left(k_6 + \frac{k_2}{\delta\omega} + k_7\right) I_S}{S_0 + k_9 I_S}, T_1 \frac{\left(S_0 + k_6 + \frac{k_2}{\delta\omega} + k_7\right) I_S}{\left(S_0 + \left(k_6 + \frac{k_2}{\delta\omega} + k_7\right) I_S\right)(S_0 + k_9 I_S)}\right)$$

where

$$k_3 = \frac{\delta k_1 \tau_5 + \tau_2(1-\delta)k_2}{\delta k_1(\mu+\sigma+\gamma_Q)},\ k_4 = \frac{\delta k_1(m\gamma_Q k_3 + \tau_6) + \tau_3(1-\delta)k_2}{\delta k_1(c+\mu+\sigma)},$$

$$k_5 = \frac{ck_4 + \frac{\gamma_A(1-\delta)k_2}{\delta k_1} + \gamma_S + (1-m)\gamma_Q k_3}{\lambda+\mu},\ k_6 = \frac{\delta\omega\lambda k_5 - (\mu+\omega)k_2}{\mu\delta\omega},$$

$$k_7 = \frac{(1-\delta)k_2}{\delta k_1} + 1 + \sigma_1(k_3+k_4) + \sigma_2 k_5,\ k_8 = \frac{\delta k_1\eta_S + \eta_A(1-\delta)k_2}{\delta k_1},$$

$$k_9 = \left(k_6 + \frac{k_2}{\delta\omega} + k_7\right)(d_v\beta + \mu_P + k_7), T_1 = \frac{k_7 k_8}{d_v\beta+\mu_P}, n_0 = s_0^2\beta_1 T_1 - \frac{s_0\beta_2 k_2(1-\delta)}{\delta k_1},$$

$$n_1 = s_0 k_7\beta_1 T_1 + s_0\beta_1 T_1\frac{k_2}{\delta\omega} + s_0\beta_1 T_1 k_6 + \frac{s_0\beta_2 k_2(1-\delta)}{\delta k_1} + \frac{\beta_2 k_2(1-\delta)\left(k_6 + \frac{k_2}{\delta\omega} + k_7\right)s_0}{\delta k_1}$$
$$- \frac{\left(\left(k_6 + \frac{k_2}{\delta\omega} + k_7\right)s_0 + s_0\alpha_1 T_1\right)(\varphi+\mu+\omega)k_2}{\delta\omega},$$

$$n_2 = k_7\beta_1 T_1 k_6 + \frac{\beta_2 k_2(1-\delta)\left(k_6 + \frac{k_2}{\delta\omega} + k_7\right)k_9}{\delta k_1}$$
$$- \frac{\left(\left(k_6 + \frac{k_2}{\delta\omega} + k_7\right)k_9 + \alpha_1 T_1 k_7\right)(\varphi+\mu+\omega)k_2}{\delta\omega};$$

Hence, there exist three (3) equilibrium points:

1. the first correspond to the Disease Free Equilibrium
2. the second correspond to
$$I_S = -\frac{n_1 - \sqrt{n_1^2 - 4n_0 n_2}}{2n_2} \tag{3.11}$$
owing to positivity of parameters, is a feasible endemic equilibrium point
3. the third correspond to
$$I_S = -\frac{n_1 + \sqrt{n_1^2 - 4n_0 n_2}}{2n_2} \tag{3.12}$$
gives a negative values, which is not feasible

3.2. The Basic Reproduction Number

The basic reproduction number is the average number of secondary infections caused by a single infectious individual in an entirely susceptible population during his/her infective period. The next generation matrix approach is used to obtain R_0. Here we shall consider the classes that have infections excluding the quarantined and vaccinated classes. These two classes are excluded as a result of being the treatment classes because it is assumed that they no longer have the disease after receiving treatment. The latent class of the pathogen is also considered because it introduces the disease into the pathogen class and is also the incubation period for the virus. Suppose X is the set of the infectious classes, then we can write:

$X(t) = (E,\ I_A,\ I_S,\ L_P,\ I_P)$ and obtain that $X^{'}(t) = \mathcal{F}(t) - \mathcal{V}(t)$, where:

$$\mathcal{F}(t) = \begin{pmatrix} \frac{\beta_1 S(I_P+L_p)}{1+\alpha_1 I_P} + \frac{\beta_2 S(I_A+I_S)}{1+\alpha_2 I_P} \\ 0 \\ 0 \\ \eta_A I_A + \eta_S I_S \\ 0 \end{pmatrix}$$

and

$$\mathcal{V}(t) = \begin{pmatrix} -(\varphi+\mu+\omega)E \\ (1-\delta)\omega E - (\tau_3+\gamma_A+\tau_2+\mu+\sigma+\eta_A)I_A \\ \delta\omega E - (\tau_6+\tau_5+\mu+\sigma+\eta_S+\gamma_S)I_S \\ -(\frac{H(t)}{N_h(t)} - (d_v\beta+\mu_P))L_p \\ \frac{H(t)}{N_h(t)}L_P - (d_v\beta+\mu_P)\,I_p \end{pmatrix}$$

Evaluating the derivatives of F and V at the disease-free equilibrium point obtained above, yields $F\mathcal{V}^{-1}$ as seen below:

$$F\mathcal{V}^{-1} = \begin{pmatrix} \frac{\beta_2 b(-1+\delta)\omega}{\mu de} - \frac{\beta_2 b\delta\omega}{\mu df} & -\frac{\beta_2 b}{\mu e} & -\frac{\beta_2 b}{\mu f} & -\frac{\beta_1 b}{\mu a} & -\frac{\beta_1 b}{\mu a} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\eta_A(-1+\delta)\omega}{\mathrm{de}} - \frac{\eta_S\delta\omega}{\mathrm{df}} & -\frac{\eta_A}{e} & -\frac{\eta_S}{f} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

where,

$$d = (\varphi+\mu+\omega),\ e = (\tau_3+\gamma_A+\tau_2+\mu+\sigma+\eta_A),$$
$$f = (\tau_6+\tau_5+\mu+\sigma+\eta_S+\gamma_S)\,,\ a = \beta d_v + \mu_P.$$

By solving the dominant eigenvalue of the next generation matrix $F\mathcal{V}^{-1}$, we get the basic reproduction number to be

$$R_0 = \frac{\beta_2 b(-1+\delta)\omega}{\mu\,\mathrm{de}} - \frac{\beta_2 b\delta\omega}{\mu\mathrm{df}} = \frac{f\beta_2 b(-1+\delta)\omega - \mathrm{e}\beta_2 b\delta\omega}{\mu\mathrm{def}}$$

Therefore, the basic reproduction number of the given system of equations denoted by R_0 is:

$$R_0 = \frac{(\tau_6+\tau_5+\mu+\sigma+\eta_S+\gamma_S)\beta_2 b(-1+\delta)\omega - (\tau_3+\gamma_A+\tau_2+\mu+\sigma+\eta_A)\beta_2 b\delta\omega}{\mu(\varphi+\mu+\omega)(\tau_3+\gamma_A+\tau_2+\mu+\sigma+\eta_A)(\tau_6+\tau_5+\mu+\sigma+\eta_S+\gamma_S)}$$

3.3. Local Stability of the Disease-free Equilibrium

We shall use the Jacobian matrix $J(\mathbb{E}_0)$ in establishing the local stability of the disease-free equilibrium. The Jacobian matrix which is evaluated at the disease-free equilibrium, is given by

Theorem

The disease-free equilibrium (DFE) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof

For local stability, the Jacobian matrix with respect to the model equations is given by:

$$\begin{vmatrix} -\mu-\Lambda & \varphi & -\frac{\beta_2 b}{\mu} & -\frac{\beta_2 b}{\mu} & 0 & 0 & \lambda & -\frac{\beta_1 b}{\mu} & -\frac{\beta_1 b}{\mu} \\ 0 & -d-\Lambda & \frac{\beta_2 b}{\mu} & \frac{\beta_2 b}{\mu} & 0 & 0 & 0 & \frac{\beta_1 b}{\mu} & \frac{\beta_1 b}{\mu} \\ 0 & (1-\delta)\omega & -e-\Lambda & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta\omega & 0 & -f-\Lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_2 & \tau_5 & -g-\Lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_3 & \tau_6 & m\gamma_Q & -h-\Lambda & 0 & 0 & 0 \\ 0 & 0 & \gamma_A & \gamma_S & (1-m)\gamma_Q & c & -n-\Lambda & 0 & 0 \\ 0 & 0 & \eta_A & \eta_S & 0 & 0 & 0 & -a-\Lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -a-\Lambda \end{vmatrix}$$

where

$$d = (\varphi + \mu + \omega),\ e = (\tau_3 + \gamma_A + \tau_2 + \mu + \sigma + \eta_A)\,,\ f = (\tau_6 + \tau_5 + \mu + \sigma + \eta_S + \gamma_S)\,,$$

$$g = (\mu + \sigma + \gamma_Q)\,,\ h = (c + \mu + \sigma),\ n = (\lambda + \mu) \text{ and } a = (\beta d_v + \mu_P)$$

The characteristics equation is

$$(\mu + \Lambda)\left[(\varphi + \mu + \omega + \Lambda)\ \left[(\tau_3 + \gamma_A + \tau_2 + \mu + \sigma + \eta_A + \Lambda)\ \left[(\tau_6 + \tau_5 + \mu + \sigma + \eta_S + \gamma_S + \Lambda)\right.\right.\right.$$

$$\left.\left.\left.\left[(\mu + \sigma + \gamma_Q + \Lambda)\left[(c + \mu + \sigma + \Lambda)\left[-\quad (\lambda + \mu + \Lambda)\,(\beta d_v + \mu_P + \Lambda)^2\right]\right]\right]\right]\right] +$$

$$\frac{\beta_2 b}{\mu}\left[(1-\delta)\omega\ \left[(\tau_6 + \tau_5 + \mu + \sigma + \eta_S + \gamma_S + \Lambda)\ \left[(\mu + \sigma + \gamma_Q + \Lambda)\ \left[(c + \mu + \sigma + \Lambda)\right.\right.\right.\right.$$

$$\left.\left.\left.\left.\left[(\lambda + \mu + \Lambda)\quad (\beta d_v + \mu_P + \Lambda)^2\right]\right]\right]\right]\right] - \frac{\beta_2 b}{\mu}\ \left[(\tau_3 + \gamma_A + \tau_2 + \mu + \sigma + \eta_A + \Lambda)\left[\delta\omega\right.\right.$$

$$\left.\left.\left[(\mu + \sigma + \gamma_Q + \Lambda)\ \left[(c + \mu + \sigma + \Lambda)\quad -\quad (\lambda + \mu + \Lambda)\,(\beta d_v + \mu_P + \Lambda)^2\right]\right]\right]\right]\right] = 0$$

Where:

$$\Lambda_1 = -\mu, \quad \Lambda_2 = -\mu - \sigma - \gamma_Q, \quad \Lambda_3 = -c - \mu - \sigma, \quad \Lambda_4 = -\lambda - \mu$$

The quadratic $\Lambda^2 + (\beta d_v)^2 + {\mu_P}^2 + 2\beta d_v \mu_P + 2\mu_P \beta d_v + 2\Lambda\beta d_v$ has all terms positive and thus it's roots must all be negative. Hence, Λ_5 and $\Lambda_6\ < 0$

$$\Lambda_7 = -\tau_3 - \gamma_A - \tau_2 - \mu - \sigma - \eta_A$$

$$\Lambda_8 = -\tau_6 - \tau_5 - \mu - \sigma - \eta_S - \gamma_S$$

From $(\varphi + \mu + \omega + \Lambda)\,(\tau_3 + \gamma_A + \tau_2 + \mu + \sigma + \eta_A + \Lambda)\,(\tau_6 + \tau_5 + \mu + \sigma + \eta_S + \gamma_S + \Lambda)\ = 0$, when expanded and solved it satisfies the Routh-Hurwitz criterion governing the polynomials of order 3. Hence from the above the disease-free equilibrium is locally asymptotically stable. This completes the proof

3.4. Stability of the Endemic Equilibrium Point

Corollary 1: (Corollary of Gershgorin Circle Theorem)

Let A be an $n\ \times\ n$ matrix with real entries. If the diagonal elements a_{ii} of A satisfy
$a_{\text{ii}} < -r_i$
where

$$r_i = \sum_{j=1, j\neq i}^{n} |a_{ij}|$$

for $i = 1, \ldots, n$, then the eigenvalues of A are negative or have negative real parts.

Theorem

The endemic equilibrium is locally asymptotically stable if $R_0 > 1$.

Proof

The Jacobian matrix with respect to the system at the endemic equilibrium is given by:

$$\begin{bmatrix} -B-K-\mu & \varphi & -W & -W & 0 & 0 & \lambda & -X & U \\ B+K & d & W & W & 0 & 0 & 0 & X & -U \\ 0 & (1-\delta)\omega & e & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta\omega & 0 & f & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_2 & \tau_5 & g & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_3 & \tau_6 & m\gamma_Q & h & 0 & 0 & 0 \\ 0 & 0 & \gamma_A & \gamma_S & (1-m)\gamma_Q & c & n & 0 & 0 \\ Y & Y & \eta_A - C + Y & \eta_S - C + Y & -\alpha_1 C + Y & -\alpha_1 C + Y & -\alpha_2 C + Y & U_1 & 0 \\ -Y & -Y & C - Y & C - Y & 0 & 0 & 0 & \frac{H}{N_h} & -\beta d_v - \mu_P \end{bmatrix}$$

in the above,

$$H = I_h + \sigma_1 T_h + \sigma_2 R_h,\ B = \frac{\beta_1 \left(I_{Pe} + L_{p_e}\right)}{(1+\alpha_1 I_{Pe})}, J = \frac{\beta_1 \left(I_{Pe} + L_{p_e}\right)}{(1+\alpha_1 I_{Pe})^2}, K = \frac{\beta_2 \left(I_{Ae} + I_{Se}\right)}{(1+\alpha_2 I_{Pe})},$$

$$M = \frac{\beta_2 \left(I_{Ae} + I_{Se}\right)}{(1+\alpha_2 I_{Pe})^2},\ W = \frac{\beta_2 S_e}{1+\alpha_2 I_{Pe}}, N_h = S_e + E_e + I_{Ae} + I_{Se} + Q_e + V_e + R_e,$$

$$X = \frac{\beta_1 S_e}{1+\alpha_1 I_{Pe}}, Y = \frac{\left[I_{Ae} + I_{Se} + \sigma_1 \left(Q_e + V_e\right) + \sigma_2 R_e\right] L_{p_e}}{\left(S_e + E_e + I_{Ae} + I_{Se} + Q_e + V_e + R_e\right)^2},\ d = -\varphi - \mu - \omega, h = -c - \mu - \sigma,$$

$$C = \frac{L_{p_e}}{\left(S_e + E_e + I_{Ae} + I_{Se} + Q_e + V_e + R_e\right)},\ e = -\tau_3 - \gamma_A - \tau_2 - \ - \sigma - \eta_A,\ n = -\lambda - \mu,$$

$$f = -\ \tau_6 - \tau_5 - \ - \sigma - \eta_S - \gamma_S,\ g = - - \sigma - \gamma_Q\ ,\ U_1 = \frac{H}{N_h} - \beta d_v - \mu_P,\ U = -X + JS + MS$$

The corollary indicates that if the diagonal elements are smaller than the sum of the absolute values of the off-diagonal elements in the same row, then the eigenvalues will have negative real parts.

$$\begin{aligned} -B - K - \mu &< -\left(\varphi + 2W + \lambda + 2X + JS + MS_e\right) \\ d &< -\left(B + K + 2W + 2X + \mathrm{JS} + MS_e\right) \\ e &< -\left((1+\delta)\omega\right) \\ f &< -\left(\delta\omega\right) \\ g &< -\left(\tau_2 + \tau_5\right) \\ h &< -\left(\tau_3 + \tau_6 + m\gamma_Q\right) \\ n &< -\left(\gamma_A + \gamma_S + (1+m)\gamma_Q + c\right) \\ \frac{H}{N_h} - \beta d_v - \mu_P &< -\left(4Y + \eta_A + 2C + \eta_S\right) \\ -\beta d_v - \mu_P &< -\left(4Y + 2C + \frac{H}{N_h}\right) \end{aligned}$$

From the above, the diagonal elements are smaller than the sum of the absolute values of the off-diagonal elements in the same row, hence the eigenvalues will have negative real parts which indicates that the endemic equilibrium is stable. The implication of the above is that, it indicates a level of resilience in the system, where it can withstand small disturbances and maintain a relatively stable disease prevalence over time.

3.5. Sensitivity Analysis of Parameters on Basic Reproduction Number

In this section, we analyze the sensitivity of the parameters of the basic reproduction number (R_0). We employ the approach used by [16] to compute the sensitivity of the parameters of R_0. The sensi-

tivity of a parameter, say μ, of R_0 is defined as

$$\xi_{\mu}^{R_0} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0}. \tag{3.13}$$

The sensitivity indices of the parameters are presented as follows:

Table 3.2: Sensitivity Analysis on Basic Reproduction R_0

Parameter	Sensitivity	Evaluation at base values		
$\xi_{b}^{R_0}$	1	1.0000	>	0
$\xi_{\beta_2}^{R_0}$	$\frac{-\delta\ a_1+\delta\ a_2+\ a_1}{a_2}$	1.0000	>	0
$\xi_{\omega}^{R_0}$	$\frac{a_3-\omega}{a_3}$	0.7371	>	0
$\xi_{\tau_6}^{R_0}$	$\frac{a_2\delta\tau_6(\phi++\omega)}{a_1(\ a_1(\delta-1)-\ a_2\delta)a_3}$	-0.0024	<	0
$\xi_{\delta}^{R_0}$	$\frac{(\ a_1-\ a_2)\delta}{a_1(\delta-1)-\ a_2\delta}$	-0.0037	<	0
$\xi_{\tau_5}^{R_0}$	$-\frac{\delta\tau_5}{a_1}$	-0.0048	<	0
$\xi_{\sigma}^{R_0}$	$-\frac{\left((\delta-1)a_1^2-\delta\ a_2^2\right)\sigma}{(\ a_1(\delta-1)-\ a_2\delta)a_1\ a_2}$	-0.0050	<	0
$\xi_{\gamma_S}^{R_0}$	$\frac{a_2\delta\gamma_S}{(\ a_1(\delta-1)-\ a_2\delta)a_1}$	-0.0060	<	0
$\xi_{\eta_S}^{R_0}$	$\frac{a_2\delta\eta_S}{(\ a_1(\delta-1)-\ a_2\delta)a_1}$	-0.0121	<	0
$\xi_{\tau_3}^{R_0}$	$-\frac{\tau_3(\delta-1)a_1}{a_2(\ a_1(\delta-1)-\ a_2\delta)}$	-0.0536	<	0
$\xi_{\tau_2}^{R_0}$	$-\frac{\tau_2(\delta-1)a_1}{a_2(\ a_1(\delta-1)-\ a_2\delta)}$	-0.1073	<	0
$\xi_{\eta_A}^{R_0}$	$-\frac{a_1(\delta-1)\eta_A}{(\ a_1(\delta-1)-\ a_2\delta)a_2}$	-0.1341	<	0
$\xi_{\gamma_A}^{R_0}$	$-\frac{a_1(\delta-1)\gamma_A}{(\ a_1(\delta-1)-\ a_2\delta)a_2}$	-0.1915	<	0
$\xi_{\phi}^{R_0}$	$-\frac{\phi}{\phi++\omega}$	-0.2336	<	0
$\xi_{\mu}^{R_0}$	$\frac{-(\delta-1)((+\ a_3)a_2+\ a_3)\ a_1^2+\delta\ a_2^2\ (+\ a_3)a_1+\delta\ a_2^2\ a_3}{a_3\ a_1\ a_2(\ a_1(\delta-1)-\ a_2\delta)}$	-1.9866	<	0

$$a_1 = \tau_6 + \tau_5 + \mu + \sigma + \eta_S + \gamma_S, \tag{3.14}$$
$$a_2 = \tau_3 + \gamma_A + \tau_2 + \mu + \sigma + \eta_A, \tag{3.15}$$
$$a_3 = \phi + \mu + \omega. \tag{3.16}$$

The analysis revealed that the positively sensitive parameters of the basic reproduction number, R_0, are the recruitment rate (b) into the susceptible class, the probability (β_2) that each contact is effective enough to cause infection, and the progression rate (ω) of exposed individuals to either asymptomatic or symptomatic class. Thus, reducing the number of susceptible individuals, reducing or eliminating contact with contaminated environment, effectively restricting infected humans from adding to the pathogen population, and ensuring that exposed individuals remain protected can greatly lower the value of the basic reproduction number (R_0) and thereby increasing the stability of the disease-free equilibrium. on the other hand, increasing the values of the positively sensitive parameters has the effect of increasing the value of the basic reproduction number (R_0), which is not a desired condition. Amongst the negatively sensitive parameters are the death rate (μ), the rate of recoveries $\gamma_{S,A}$, progression rate to quarantine class due to treatment and compliance to hygienic regulations $\tau_{2,3,5,6}$, the disease induced death rate (δ). Increasing the values of these negatively sensitive parameters, reduces the value of the basic reproduction number (R_0), which is the desired condition.

In general, sensitivity parameters are assessed as positive or negative based on our model's context and for the specific parameters under consideration. A positive sensitivity parameter indicates a direct correlation between an input parameter and the model output, meaning that an increase in the parameter value results in a corresponding increase in the model output, and vice versa, implying a positive influence on the system. Conversely, a negative sensitivity parameter suggests an inverse relationship between the input parameter and the model output, where an increase in the parameter value leads to a decrease in the model output and vice versa, indicating a negative impact on the system. These understanding of the sign and magnitude of sensitivity parameters is crucial for comprehending how individual factors affect model behavior and guiding decision-making processes.

4. Numerical Simulations

4.1. Method of Solution

The 'dsolve' command in computer algebra systems, such as those like Maple or Mathematica, provides a method for finding numerical solutions to ordinary differential equations (ODEs) or systems of ODEs. When used with the 'numeric' or 'type=numeric' option, it computes a numerical solution. This command is versatile and can handle both initial value problems (IVPs) and boundary value problems (BVPs), as well as initial differential algebraic problems. The maple software was used in this case and a function within the 'plots' package, 'odeplot' was used to plot the result while another function 'plots[display]' also in the 'plots' package was used to display the graph in two dimensional plane. The result of the above procedure is discussed below

4.2. Discussion of Result

Figure 1 and 2 shows the effect of the disease-induced death rate on the quarantined and vaccinated population. When the disease-induced death rate is high, it will lead to a decrease in the population size. On the other hand, if the disease-induced death rate is low, it will lead to an increase in the population size. A high disease-induced death rate can actually have a counterintuitive effect on transmission rate because when people are dying from the disease, there are fewer people available to become infected and spread the disease. Also, if the rate of recovery is high, then even if the death rate is high, the population may still be able to rebound. From Figure 3 and 10, we can see the effect of the progression rate from the asymptomatic infected class to both the quarantined and vaccinated classes respectively. If the rate of progression from the asymptomatic class to the symptomatic class is high then there's going to be an increase in the quarantined or vaccinated population. From Figure 4 and 5, we can see the effect of the progression rate from the symptomatic infected class to both the quarantined and vaccinated classes respectively. If the progression rate from the symptomatic class is high, a large number of people will need to be quarantined or treated which could cause the quarantined and vaccinated populations to increase. There is also a case whereby the high progression rate would cause a large number of people to become symptomatic but the high recovery rate would cause many of them to recover quickly. This could mean that the total number of people in the quarantined and vaccinated populations remains relatively constant.

From Figure 6, the effect of rate of recovery of the quarantined population on the quarantined class is displayed. For the quarantined population, a high rate of recovery means that more people are being released from quarantine which frees up resources for other public health issues. From Figure 7, we can see the effect of vaccine effectiveness on the vaccinated population. Vaccine effectiveness refers to how well the vaccine protects people from the disease. A high vaccine effectiveness means that more people in the population are protected from the disease and therefore, the number of people who need to be vaccinated is lower. On the other hand, a low vaccine effectiveness means that more people need to be vaccinated to achieve the same level of protection. From Figure 8 and 9 we can see the effect of the natural death rate on both the quarantined and vaccinated population. For

the quarantined class, a high natural death rate means that people are more likely to die from causes other than the disease being studied This can free up resources that would otherwise be used to treat the disease. For the vaccinated class, a high natural death rate means that there is less pressure to vaccinate people since they are likely to die from other causes. It is important to note that the natural death rate affects the overall life expectancy of the population. If the natural death rate is high, it means that people are likely to die at a younger age, which can affect the number of people who are eligible for vaccination. It can also affect the number of people who are likely to be hospitalized or require long-term care.

Conclusion

In this paper, we modified a SEIR model to include transmission dynamics, severity of the virus on human population, quarantine, vaccination and disease induced death rate. We determined the existence and local stability of the disease-free equilibrium along with the existence of the endemic equilibrium.

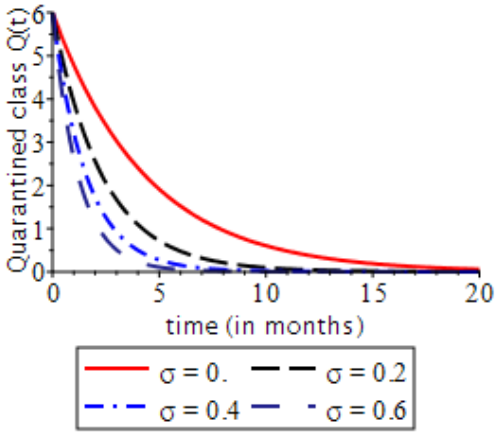

Figure 4.2: Effect of disease-induced death rate on quarantined class

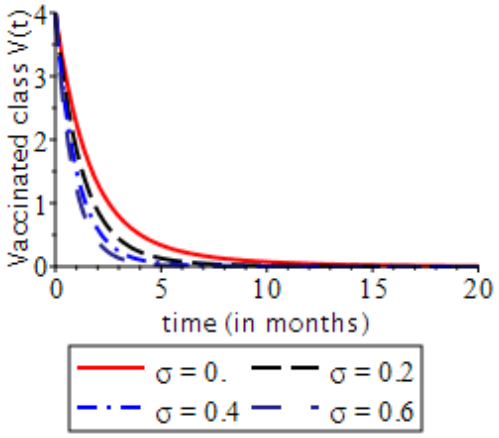

Figure 4.3: Effect of disease-induced death rate on vaccinated class

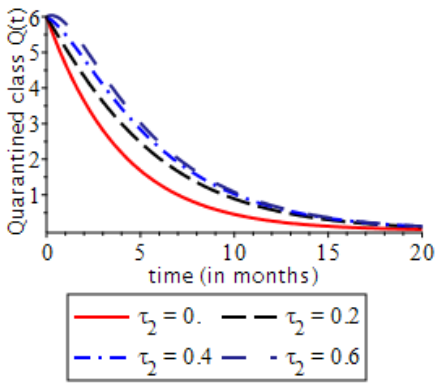

Figure 4.4: Effect of progression rate from asymptomatic infected class to quarantined class on quarantined humans

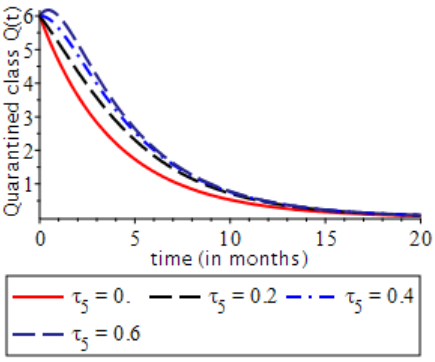

Figure 4.5: Effect of progression rate from symptomatic infected class to quarantined class on quarantined class

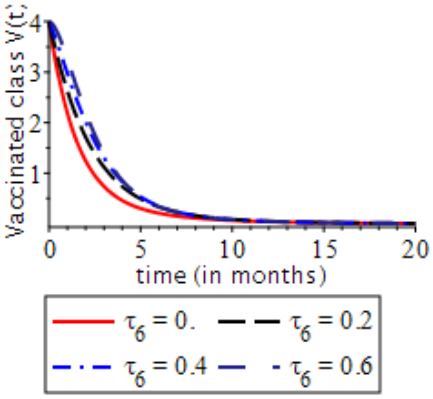

Figure 4.6: Effect of progression rate from symptomatic infected class to vaccinated class on vaccinated class

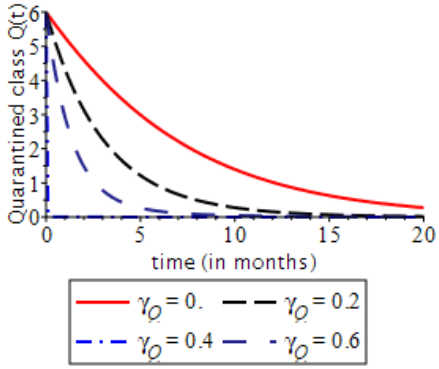

Figure 4.7: Effect of rate of recovery of the quarantined population on quarantined class

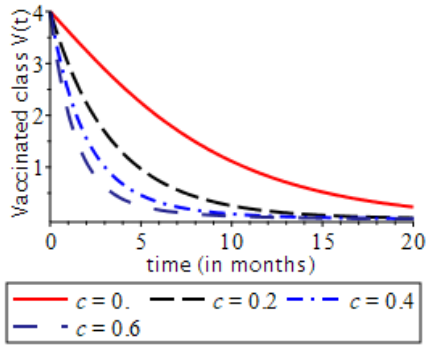

Figure 4.8: Effect of vaccine effectiveness on vaccinated class

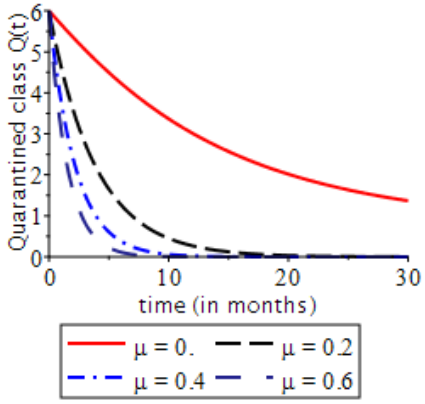

Figure 4.9: Effect of the natural death rate on the quarantined humans

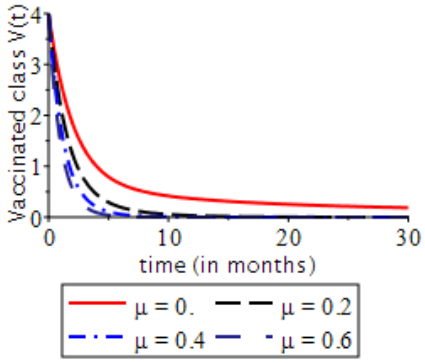

Figure 4.10: Effect of the natural death rate on the vaccinated humans

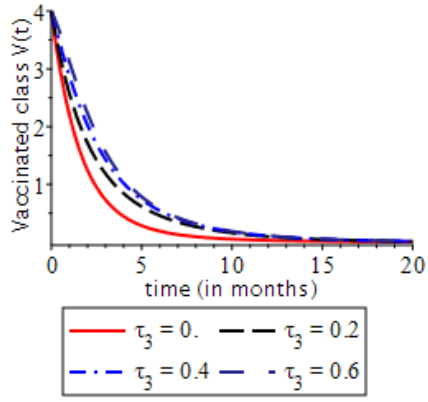

Figure 4.11: Effect of the progression rate from asymptomatic infected class to vaccination class on the vaccinated humans

Then, the basic reproduction number was computed using the next generation matrix. From the model we can see how all the various factors considered can help reduce its tenacity along with the various preventive and treatment procedures being put together to help curtail the spread of the disease.

We explored comprehensively, the sensitivity analysis on Basic Reproduction Number (BRN)– R_0. From the results, conditions that can mitigate and even eradicate the disease were uncovered. Vaccination helps to protect people from getting sick which can reduce the strain on the health care systems and prevent a large number of deaths. Quarantine on the other hand, helps to stop the spread of the disease which can prevent an epidemic or even a pandemic. It is recommended that other researchers who intend to research on this field can consider other aspects of the population including the economy, the government, the culture and the health care system and how they interact with each other.

From the discussion so far, the following are deduced:

1. Incorporating preventive measures and treatment into models helps understand their impact on disease transmission dynamics, aiding in predicting the pandemic's trajectory and assessing control strategies' effectiveness.
2. The study informs policymakers about the effectiveness of preventive measures like mask-wearing and vaccination, aiding in decision-making for implementing and adjusting public health measures.
3. Understanding transmission dynamics helps healthcare systems allocate resources efficiently, prepare for surges, and plan the distribution of vaccines and treatments.
4. The study evaluates interventions' impact on key parameters, such as R_0 and ξ^{R_0}, crucial for assessing their effectiveness in controlling virus spread across different populations.
5. Insights from this study inform clear public health messaging to promote adherence to preventive measures and vaccination, building trust and compliance.

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Author's contributions

All authors worked together to produce the results and read and approved the final manuscript.

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