



# Controlling the Dynamical Spread of Coronavirus Disease (COVID-19) in a Population

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## ABSTRACT

In the paper, a model governed by a system of ordinary differential equations was considered; the whole population was divided into Susceptible individuals (S), Exposed individuals (E), Infected individuals (I), Quarantined individuals (Q) and Recovered individuals (R). The well-posedness of the model was investigated by the theory of positivity and boundedness. Analytically, the equilibrium solutions were examined. A key threshold which measures the potential spread of the Coronavirus in the population is derived using the next generation method. Bifurcation analysis and global stability of the model were carried out using centre manifold theory and Lyapunov functions respectively. The effects of some parameters such as Progression rate of exposed class to infectious class, Effective contact rate, Modification parameter, Quarantine rate of infectious class, Recovery rate of infectious class and Recovery rate of quarantined class on  $R_0$  were explored through sensitivity analysis. Numerical simulations were carried out to support the theoretical results, to reduce the burden of COVID 19 disease in the population the following parameters  $\tau_1, \beta, \theta, \eta_1, \eta_2, r, \tau_2$  and  $\tau_3$  play a significant in the spread of it in the population.

**Keywords:** reproduction number, bifurcation analysis, Lyapunov functions.

## 1 INTRODUCTION

The recent outbreak of the deadly and highly infectious COVID-19 disease caused by SARS-CoV-2 in Wuhan and other cities in China in 2019 has become a global pandemic as declared by World Health Organization (WHO) in the first quarter of 2020 [3]. The incubation period was estimated to be 14 days (the time between the successive onset of symptoms in a chain of transmission) [53]. According to worldometer [4], the total number of cases in the world today 18th June 2020 is 8,468,941 with 3,577,610 (42.2%) in active case, 4,439,532 (52.4%) recovered and 451,979 (5.3%) death of COVID 19.

The traditional model for respiratory disease transmission assumes the existence of the infection via infectious droplets (generally 5–10  $\mu\text{m}$ ) which have a short lifespan in the air and attack the upper respiratory tract, or finer aerosols, and stays in the air for many hours [20], with ongoing uncertainties in the comparative standing of these modes (and in the conceptual model itself [21]) for SARS-CoV-2 transmission [21, 23]. The WHO [22] has stated that SARS-CoV-2 spreads primarily via coarse respiratory droplets and contact routes. An experimental study [24] using a nebulizer found SARS-CoV-2 to remain viable in

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aerosols ( $<5 \mu\text{m}$ ) for three hours (the study duration), but the clinical relevance of this setup is in question [22]. One out of three symptomatic COVID-19 patients caused widespread environmental contamination in [25], including of air exhaust outlets, though the air itself tested negative.

The transmission dynamics of infectious diseases has been well-studied and researched in mathematics and usually referred to as mathematical epidemiology. Mathematical models have played a major role in increasing understanding, control and intervention of the underlying mechanisms which influence the spread of diseases [5,6,7], which has been a strong tool in providing deeper and better understanding on the transmission dynamics and burden of the current COVID-19 pandemic, having a greater impact on the development of public health policy, intervention and control. Majority of the mathematical models of the COVID-19 pandemic can broadly be divided into:- population-based models, SIR (Kermack-McKendrick) - type models, driven by (potentially stochastic) differential equations [26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36], or agent-based models [37, 38, 39, 40, 41], in which individuals typically interact on a network structure and exchange infection in a stochastic manner.

The recent outbreak of the deadly and highly infectious COVID-19 disease has attracted the attention of many researchers who have studied and discussed the nature of the virus, its transmission dynamics and the basic reproduction number of the disease, possible controls, see eg. [8,9,10,11,12,13]. Recently, Elsevier and Springer have made open access to several literature for interested researchers [14,15]. An SEIR mathematical model for the transmission dynamics of COVID-19 disease with data fitting, parameter estimations and sensitivity analysis was studied in [9] also a deterministic model for COVID-19 that captures the effect of delay diagnosis on the disease transmission was presented, see [16]. In [17], the authors study a statistical analysis of COVID-19 disease data to estimate time-delay adjusted risk for death from this deadly virus in Wuhan, as well as for China excluding Wuhan. The study by [17] reported that effective social distancing and movement restrictions practices can help in reducing the disease transmission. A real-time forecast phenomenological model has also been formulated to examine the spread pattern of COVID-19 infectious disease, see e.g., [18]. In addition, an SEIR-type compartmental modelling concept applied to design a data-driven epidemic model that incorporates governmental actions and individual behavioural reactions for the COVID-19 disease outbreak in Wuhan was studied by [19].

Moore and Okyere [56] consider an optimal control COVID-19 transmission model that was used to assess the impact of some control measures that can lead to the reduction of exposed and infectious individuals in the population. Three control strategies were used; personal protection, treatment with early diagnosis, treatment with delay diagnosis and spraying of virus in the environment are the time-dependent control functions. Also, Eikenberry et al. [54] develop a compartmental model for assessing the community wide impact of mask use by the general, asymptomatic public, a portion of which may be asymptotically infectious. Their results suggest use of face masks by the public is potentially of high value in curtailing community transmission and the burden of the pandemic. In addition, Usaini et al. [55] formulate and analyzed deterministic mathematical model for the transmission dynamics of Middle East Respiratory Syndrome Coronavirus (MERS-CoV). The impact of quarantine and isolation are accessed via threshold analysis approach, while the impact of immigration on the disease prevalence was discussed. From

there findings it was discovered that MERS-CoV can be controlled by quick isolation or monitoring close contacts and quarantining of suspected latent immigrants.

This article aimed at examining the impact of immigration on the dynamics spread of COVID-19, the nature of the stability and to check the conditions to put in place to force the basic reproduction number below unity in community. In Section 2, a new mathematical model was been formulate for COVID-19 with five compartments. The stability analysis of the model was presented in Section 3. In Section 4, the sensitivity analysis was presented for the autonomous model. Finally, discussion and conclusion were reported in Section 5 and 6 respectively.

## 2. MATHEMATICAL MODEL

The study uses five (5) compartmental deterministic mathematical model of the S, E, I, J, R to have better understanding of the dynamical spread of COVID 19 diseases in the population. The population size  $N(t)$  is sub-divided into sub-classes of individuals who are Susceptible  $S(t)$ , Exposed  $E(t)$ , Infectious  $I(t)$ , Quarantined  $Q(t)$ , and Recovered  $R(t)$ ,

Where

$$N(t) = S(t) + E(t) + I(t) + Q(t) + R(t) \quad (1)$$

**Susceptible (S):** Susceptible individual is a member of a population who is at risk of becoming infected by a disease, COVID 19 diseases. The population of susceptible individuals increases by the recruitment of active individuals at the rate  $\pi$ . The population decreased by natural death at a rate  $\mu$ , also by force of infection  $\lambda$ .

$$\frac{dS}{dt} = \pi - \lambda S(t) - \mu S(t) \quad (2)$$

**Exposed (E):** Exposed / Latent individual is a member of a population who is infected individual and show no clinical symptoms of the disease but such fellow is infectious i.e capable of transmitting the diseases. The population of latent individuals increases through interaction with infected individual at the rate  $\lambda$ . The population of latent class diminished by the progression rate of infected individual to infectious class  $I$ , at the rate  $\tau_1$ , quarantine at the rate  $\theta$  and natural death at a rate  $\mu$ .

$$\frac{dE}{dt} = \lambda S(t) - (\tau_1 + \theta + \mu) E(t) \quad (3)$$

**Infectious (I):** Infectious individual is a member of a population who is infected with clinical symptoms of the disease and capable of transmitting the disease, COVID 19 in the population. The population of infectious individuals increases through the progression rate of infectious individuals  $I$  from latent at the rate  $\tau_1$ . The population is decreased by quarantine rate, recovery rate of infectious, natural death and disease induced death ( $r$ ), ( $\tau_2$ ), ( $\mu$ ), and ( $\delta_1$ ) respectively.

$$\frac{dI}{dt} = \tau_1 E(t) - (r + \tau_2 + \delta_1 + \mu) I(t) \quad (4)$$

**Quarantine (Q):** Quarantine individual is a member of a population who has contact with infected individual and show mild or no clinical symptoms and they are under watch. The population of quarantine individuals increases through the rate of quarantine rate of latent individual and infectious individual at the rate  $\theta$  and  $r$  respectively. The population

decreases by treatment rate of quarantined individual ( $\tau_3$ ), natural death ( $\mu$ ) and disease induced death ( $\delta_Q$ ).

$$\frac{dQ}{dt} = \theta E(t) + r I(t) - (\tau_3 + \delta_Q + \mu)Q(t) \quad (5)$$

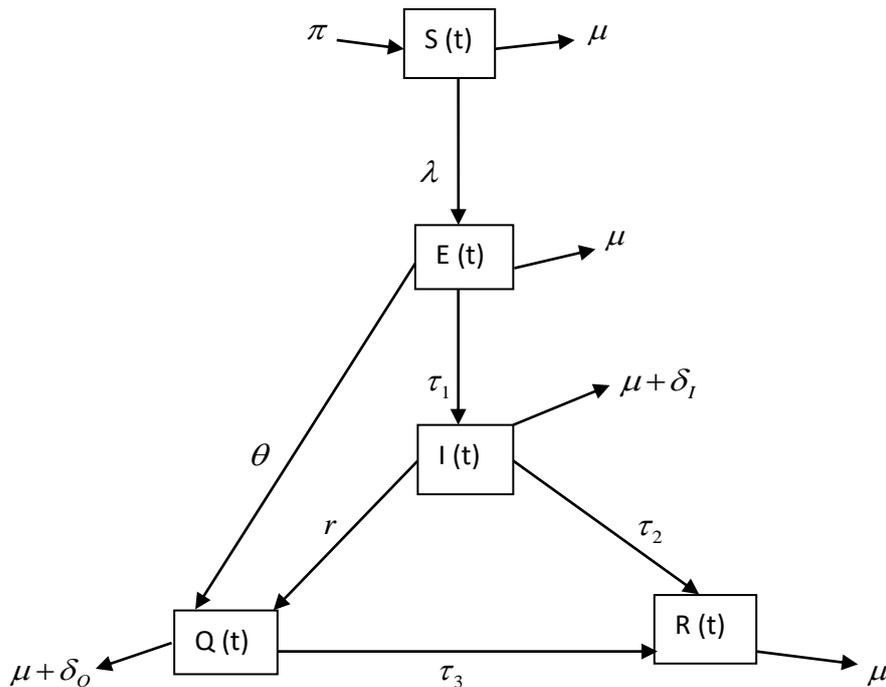
**Recovered (R):** Recovered individual is a member of a population who recovered from the disease. The population of recovered individual is increased by the treatment of infectious individual at a rate ( $\tau_2$ ) and treatment of quarantine individual at a rate ( $\tau_3$ ), this population later decreased by natural death at the rate ( $\mu$ ).

$$\frac{dR}{dt} = \tau_2 I(t) + \tau_3 Q(t) - \mu R(t) \quad (6)$$

The dynamics of the model is depicted by system (7), figure 1, the variable are listed in the Table 1 and the parameter are defined in the Table 2.

### Model Equation

$$\left. \begin{aligned} \frac{dS}{dt} &= \pi - \lambda S(t) - \mu S(t) \\ \frac{dE}{dt} &= \lambda S(t) - (\tau_1 + \theta + \mu) E(t) \\ \frac{dI}{dt} &= \tau_1 E(t) - (r + \tau_2 + \delta_I + \mu) I(t) \\ \frac{dQ}{dt} &= \theta E(t) + r I(t) - (\tau_3 + \delta_Q + \mu) Q(t) \\ \frac{dR}{dt} &= \tau_2 I_d(t) + \tau_3 I_u(t) - \mu R(t) \end{aligned} \right\} \quad (7)$$



**Figure 1. Flow Chat**

where

$$\lambda = \frac{\beta(\eta_1 L + \eta_2 Q + I)}{N} \quad (8)$$

**Table 1. Description of Variables**

Variables	Definitions
$S$	Susceptible individuals
$E$	Exposed individual
$I$	Infectious individual
$Q$	Quarantined individual
$R$	Recovered individual

**Table 2. Description of parameters**

Parameters	Definitions
$\tau_1$	Progression rate of exposed class to infectious class
$\tau_2$	Recovery rate of infectious class
$\tau_3$	Recovery rate of quarantined class
$r$	Quarantine rate of infectious class
$\pi$	Recruitment rate
$\mu$	Natural death rate
$\eta_1$	Modification parameter for exposed class
$\eta_2$	Modification parameter for quarantined class
$\delta_i$	Induced mortality rate
$\beta$	Effective contact rate
$N$	Total population
$\lambda$	Force of infection
$\theta$	Quarantine rate of exposed class

## 2.1 Basic Property

### 2.1.1 Positivity and boundedness of solutions

Since model (7) monitors human population, all the parameters are non-negative. Therefore, it is needful to show that all the state variables are also non-negative for all time  $t > 0$ .

**Theorem 1**

The state variables,  $S(t)$ ;  $L(t)$ ;  $I(t)$ ;  $Q(t)$ ; and  $R(t)$ , of the autonomous version of the COVID 19 disease of model (7), with the non-negative initial data, remain non-negative for all  $t > 0$ .

**Proof**

One can see from the first equation of (7) that

$$\frac{dS}{dt} \geq -(\lambda + \mu)S(t), \quad (9)$$

so that,

$$\frac{d}{dt} (S(t) \exp(\mu t + \int_0^t \lambda(\varpi) d\varpi)) \geq 0, \quad (10)$$

it follows that

$$S(t) \geq S(0) \exp(-(\mu t + \int_0^t \lambda(\varpi) d\varpi)) > 0. \quad (11)$$

It can be shown, using similar approach, that other state variables,  $E(t)$ ;  $I(t)$ ;  $Q(t)$ ; and  $R(t)$ , are non-negative for all  $t > 0$ .

Next, consider the biologically feasible region, define by  $\Gamma \subset R_+^5$

Where:

$$\Gamma = \left\{ (S, E, I, Q, R) \in R_+^5 : N \leq \frac{\pi}{\mu} \right\} \quad (12)$$

It can be shown that  $\Gamma$  is positively invariant.

**Theorem 2**

The region  $\Gamma$  is positively invariant with respect to the model (7)

**Proof:**

The rate of change of the total population is given by

$$\frac{dN}{dt} = \pi - (S + E + I + Q + R)\mu - (I + Q)\delta, \quad (13)$$

it results into;

$$N(t) = N(0) \exp(-\mu t) + \frac{\pi}{\mu} (1 - \exp(-\mu t)), \quad (14)$$

which follows that  $N(t) \rightarrow \frac{\pi}{\mu}$  as  $t \rightarrow \infty$ , in particular,  $N(t) \leq \frac{\pi}{\mu}$  if  $N(0) \leq \frac{\pi}{\mu}$ . Hence, it

suffices to consider the dynamics of the model in  $\Gamma$ . In this region, the COVID 19 model can be considered as being mathematically well-posed [42].

**3.0 Stability Property****3.1 Disease Free Equilibrium (DFE)**

Disease free means when there is disease in the population, i.e,  $E = I = Q = 0$ . At equilibrium points, all other compartment are set to be zero;

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dQ}{dt} = \frac{dR}{dt} = 0 \quad (15)$$

Let  $E_0$  denotes the disease free equilibrium. Thus; the model in (7) has disease free equilibrium given by

$$E_0 = (S, E, I, Q, R) = \left( \frac{\pi}{\mu}, 0, 0, 0, 0 \right) \quad (16)$$

### 3.2 Endemic Equilibrium

The endemic equilibrium of the model (7) is given below;

$$\left. \begin{aligned} S^* &= \frac{\pi}{\mu R_0} \\ E^* &= \frac{\pi(R_0 - 1)}{d_1 R_0} \\ I_u^* &= \frac{\tau_1 \pi(R_0 - 1)}{K_1 K_3 R_0} \\ I_d^* &= \frac{(K_1 K_3 + K_3 \tau_1 + r \tau_1) \pi(R_0 - 1)}{K_1 K_2 K_3 R_0} \\ R^* &= \frac{(\tau_2 (K_1 K_3 + K_3 \tau_1) + \tau_1 (r \tau_2 + d_2 \tau_3)) \pi(R_0 - 1)}{K_1 K_2 K_3 \mu R_0} \end{aligned} \right\} \quad (17)$$

Where

$$\begin{aligned} K_1 &= \tau_1 + \theta + \mu \\ K_2 &= r + \tau_2 + \delta_I + \mu \\ K_3 &= \tau_3 + \delta_Q + \mu \end{aligned} \quad (18)$$

### 3.3 Basic Reproduction Number ( $R_0$ )

Using next generation matrix [43],[44] the non-negative matrix  $F$  (new infection terms) and non-singular matrix  $V$  (other transferring terms) of the model are given, respectively by;

$$F = \begin{pmatrix} \frac{\beta(\eta_1 E + I + \eta_2 Q)S}{N} \\ 0 \\ 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} K_1 E \\ K_2 I - \tau_1 E \\ K_3 Q - \theta E - r I \end{pmatrix} \quad (19)$$

After taking partial derivatives of  $F$  and  $V$ , we have:

$$F = \begin{pmatrix} \eta_1 \beta & \beta & \eta_2 \beta \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} K_1 & 0 & 0 \\ -\tau_1 & K_2 & 0 \\ -\theta & -r & K_3 \end{pmatrix} \quad (20)$$

Thus;

$$R_0 = \frac{\beta(r \eta_2 \tau_1 + \theta \eta_2 K_2 + K_2 \eta_1 K_3 + K_3 \tau_1)}{K_1 K_2 K_3} \quad (21)$$

The threshold quantity  $R_0$  is the basic reproduction number of the model system (7) for COVID 19 infection. It is the average number of new secondary infections generated by a single infected individual in his or her infectious period. [45].

### 3.4 Local Stability

**Theorem 3:** The disease free equilibrium of the modeled in equation (7) is locally asymptotically stable (LAS) if  $R_0 < 1$  and unstable if  $R_0 > 1$ .

**Proof:**

To determine the local stability of  $E_0$ , the following Jacobian matrix is computed corresponding to equilibrium point  $E_0$ .

Considering the stability of the disease free equilibrium at the critical point  $\left(\frac{\pi}{\mu}, 0, 0, 0, 0\right)$ .

$$J = \begin{pmatrix} -\mu & -\eta_1 \beta & -\beta & -\beta \eta_2 & 0 \\ 0 & -K_1 + \eta_1 \beta & \beta & \beta \eta_2 & 0 \\ 0 & \tau_1 & -K_2 & 0 & 0 \\ 0 & \theta & r & -K_3 & 0 \\ 0 & 0 & \tau_2 & \tau_3 & -\mu \end{pmatrix} \quad (22)$$

A necessary and sufficient condition for local asymptotic stability is for the real part of the eigenvalue to be in the negative half plane [44]. Thus, it can show that  $J(E_0)$  given by (22) has eigenvalues all have a negative real part.

To this purpose, it is obvious from (22) that  $-\mu$  (twice) are the two of the five eigenvalues of  $J(E_0)$  since the first and fifth columns contain only the diagonal terms. Hence, the other three eigenvalues can be obtained from the sub-matrix of 3 by 3 matrix,  $J^*(E_0)$  given by

$$J^* = \begin{pmatrix} -K_1 + \eta_1 \beta & \beta & \beta \eta_2 \\ \tau_1 & -K_2 & 0 \\ \theta & r & -K_3 \end{pmatrix} \quad (23)$$

In what follows, the characteristic equation of  $J^*(E_0)$  is of the form  $|J^* - \lambda| = 0$  is given by:

$$J^* = \begin{pmatrix} -K_1 + \eta_1 \beta - \lambda & \beta & \beta \eta_2 \\ \tau_1 & -K_2 - \lambda & 0 \\ \theta & r & -K_3 - \lambda \end{pmatrix} \quad (24)$$

Simplifying matrix (24), can be written as:

$$B_3 \lambda^3 + B_2 \lambda^2 + B_1 \lambda + B_0 = 0 \quad (25)$$

And

$$B_0 = K_1 K_2 K_3 - \tau_1 r \beta \eta_2 - \theta \beta K_2 \eta_2 - \beta \eta_1 K_3 K_2 - \beta K_3 \tau_1$$

It is easy to see that  $B_0$  can be written in terms of  $R_0$  as:

$$B_0 = [1 - R_0] \quad (26)$$

If in (26)  $R_0 < 1$ , then  $B_0 > 0$ . Since the coefficients  $B_i$ ,  $i = 1, 2, 3$  and the Hurwitz matrices of the polynomial (25) are positive, using Routh-Hurwitz criterion (see, [32, 40, 46]), all the eigenvalues of (25) have negative real parts. Therefore, the disease free equilibrium,  $E_0$ , is stable. Otherwise, whenever  $R_0 > 1$  then  $B_0 < 0$ . By Descartes' rule of signs [47], there exists one eigenvalue with positive real part. Hence,  $E_0$  is unstable for  $R_0 > 1$ .

**The implication of Theorem 3** is that reduction or elimination of COVID 19 diseases governed by model (7) can be eliminated from the population whenever an influx by infected individual is small such that  $R_0 < 1$ .

### 3.5 Global Stability

**Theorem 4:** The disease free-equilibrium of the system in (7) is globally asymptotically stable (GAS) whenever  $R_0 < 1$  and unstable if  $R_0 > 1$ .

**Proof:**

Consider the linear Lyapunov function  $V: \Gamma \rightarrow R_0$  defined by

$$V = A_1 E(t) + A_2 I(t) + A_3 Q(t) \quad (27)$$

where  $A_1 = \frac{\tau_1 K_3 + r \tau_1}{K_1 K_3}$ ,  $A_2 = 1$  and  $A_3 = \frac{r}{K_3}$ . The time derivative of (27) along the solution path of the system (7) is given by

$$V' = \left[ \frac{\tau_1 K_3 + r \tau_1}{K_1 K_3} \right] (\lambda S(t) - K_1 E(t)) + (\tau_1 E(t) - K_2 I(t)) + \frac{r}{K_3} (\theta E(t) + r I - K_3 Q(t)) \quad (28)$$

$$V' = \frac{\tau_1 \lambda}{K_1} S(t) + \frac{K_2 \theta \lambda}{K_1 r} S(t) + \frac{K_2 K_3 \eta_1 \lambda}{K_1 \eta_2 r} S(t) + \frac{K_3 \tau_1 \lambda}{K_1 \eta_2 r} S(t) - K_2 I(t)$$

$$V' = \frac{\tau_1 \beta r \eta_2}{K_1 K_3} I(t) + \frac{\eta_2 K_2 \beta \theta}{K_1 K_3} I(t) + \frac{K_2 \eta_1 \beta}{K_1} I(t) + \frac{\tau_1 \beta}{K_1} I(t) - K_2 I(t)$$

$$V' = \left[ \frac{\beta (\eta_2 (r \tau_1 + \theta K_2) + K_3 (K_2 \eta_1 + \tau_1))}{K_1 K_3} - K_2 \right] I(t)$$

$$V' = K_2 [R_0 - 1] I(t) \quad (29)$$

Thus,  $V' \leq 0$  if  $R_0 \leq 0$  with  $V' = 0$  if and only if  $I = 0$ . This shows that as  $t \rightarrow \infty$ , then  $(S(t),$

$E(t), I(t), Q(t), R(t)) \rightarrow \left( \frac{\pi}{\mu}, 0, 0, 0, 0 \right)$ . It follows that the largest compact invariant set in  $\{$

$(S(t), E(t), I(t), Q(t), R(T)) \in \Gamma : V' = 0$  is the singleton  $E_0$ . Therefore by LaSalle's Invariance Principle [48], the DFE given by  $E_0$  is GAS in  $\Gamma$  if  $R_0 \leq 0$ .

**The implication of Theorem 4** is that reduction or elimination of COVID 19 disease is independent of the initial sizes of the sick people in the population. Hence, COVID 19 disease can be eliminated if the associated reproduction number is less than unity.

### 3.6 Bifurcation Analysis

Bifurcation analysis is used to explore how the asymptotic stability of disease-free equilibrium is exchanged for asymptotic stability of endemic equilibrium of model (7) as the threshold quantity,  $R_0$ , cross the unity. In other words, to investigate the bifurcation at  $R_0 = 1$ , using a center manifold theory of bifurcation analysis described by [42], used in some literatures like [49], [50], [51],[52], [53].

Choosing  $\beta$  as the bifurcation parameter, then at  $R_0 = 1$ .

$$R_0 = \frac{\beta(r\eta_2\tau_1 + \theta\eta_2K_2 + K_2\eta_1K_3 + K_3\tau_1)}{K_1K_2K_3} = 1 \quad (30)$$

then,

$$\beta^* = \frac{K_1K_2K_3}{r\eta_2\tau_1 + \theta\eta_2K_2 + K_2\eta_1K_3 + K_3\tau_1} \quad (31)$$

So that the disease-free equilibrium,  $D_0$ , is locally stable when  $\beta < \beta^*$ , and is unstable when  $\beta > \beta^*$ , this,  $\beta^*$ , is bifurcation value.

The linearized matrix of the system (7) around the disease-free equilibrium  $E_0$  and evaluated at  $\beta^*$  is given by;

Then,

$$J(E_0, \beta^*) = \begin{pmatrix} -\mu & -\eta_1\beta & -\beta & -\beta\eta_2 & 0 \\ 0 & -K_1 + \eta_1\beta & \beta & \beta\eta_2 & 0 \\ 0 & \tau_1 & -K_2 & 0 & 0 \\ 0 & \theta & r & -K_3 & 0 \\ 0 & 0 & \tau_2 & \tau_3 & -\mu \end{pmatrix} \quad (32)$$

The eigenvalues  $(\lambda)$ , of  $J(E_0, \beta^*)$  given by (32) are the roots of the characteristic equation of the form:

$$(\lambda + \mu)^2 P(\lambda) = 0 \quad (33)$$

Where  $P(\lambda)$  is a polynomial of degree three whose roots are real and negative except one zero eigenvalue.

#### 3.6.1 Determination of right eigen-vector and left eigen-vector

The right eigenvector,  $w = (w_1, w_2, w_3, w_4, w_5)^T$ , associated with this simple zero eigenvalue can be obtained from  $J(D_0, \beta^*)w = 0$ . Furthermore, the left eigenvector,

$v = (v_1, v_2, v_3, v_4, v_5)$ , corresponding to the simple zero eigenvalue of (32) is obtained from  $v J(D_0, \beta^*) = 0$

### 3.6.2 Computation of bifurcation coefficient

The direction of the bifurcation at  $R_0 = 1$  is determined by the signs of bifurcation coefficient “a” and “b”, obtained from the above partial derivatives, given, respecting, by

$$a = \frac{DK_1[AC+BK_1K_2K_3\theta]}{CY^2\pi} v_2 w_2^2 \quad (34)$$

Similarly,

$$b = \frac{K_2\tau_1(1+\theta)-\theta Y}{K_2K_3} + \frac{K_1K_2\theta\tau_1(1+\theta)-K_1\theta^2Y}{K_2Y-K_1K_2K_3\theta} v_2 w_2 \quad (35)$$

Where:

$$\begin{aligned} A &= K_1Y - K_1K_2\tau_1 \\ B &= K_1K_2\tau_1 - K_1Y \\ C &= K_1K_2K_3\theta - K_2Y \\ Y &= \theta K_3\tau_1 + \theta K_1K_2 + \tau_1(K_2 + \theta r) \end{aligned} \quad (36)$$

By numerical evaluation, using value of parameter in Table 3, it was found that  $a < 0$  and  $b > 0$ , which follows from the theorem of [42] that the model (7) exhibits a supercritical (forward) bifurcation and the endemic equilibrium  $E^*$  is locally asymptotically stable.

**Table 3. Parameters Value and Source**

Parameters	Value	Baseline	Source
$\tau_1$	0.9 – 0.4	0.6	[10]
$\tau_2$	0.9 – 0.4	0.7	[17]
$\tau_3$	0.9 – 0.4	0.7	[13]
$r$	0.2 – 0	0.05	[15]
$\pi$	1 – 0.2	0.9	[41, 32]
$\mu$	0.2 – 0	0.1	[12,25]
$\eta_1$	0.8 – 0.4	0.5	Assumed
$\eta_2$	0.9 – 0.2	0.6	[21]
$\delta$	0.2 – 0	0.01	Assumed
$\beta$	0.9 – 0.2	0.7	[36]
$\theta$	0.4 – 0.1	0.2	[30]

## 4 Sensitivity Analysis

To determine how changes in parameters affect the transmission and spread of the disease, a sensitivity analysis of model (7) is carried out in the sense of [50],[52]. This was done to examines changing effects of the model parameters with respect to basic reproduction number,  $R_0$ , of the model (7).

**Definition 1.** The normalized forward-sensitivity index of a variable,  $v$ , depends differentiable on a parameter,  $p$ , is defined as:

$$Y_p^v = \frac{\partial v}{\partial p} \times \frac{p}{v} \quad (37)$$

In particular, sensitivity indices of the basic reproduction number,  $R_0$ , with respect to the model parameter. The following results were obtained using the parameter value in Table 3:

**Table 4 Sensitivity indices with the Parameters**

Parameter	Sign
$\beta$	Positive
$\theta$	Negative
$\eta_1$	Positive
$\eta_2$	Positive
$\tau_1$	Positive
$\tau_2$	Negative
$\tau_3$	Negative
$r$	Negative

The positive sign of S.I of  $R_0$  to the model parameters shows that an increase (or decrease) in the value of each of the parameter in this case will lead to an increases (or decrease) in  $R_0$  of the model (7) and asymptotically results into persistence (or elimination) of the disease in the community . On the contrary, the negative sign of  $R_0$  to the model parameters indicates that an increase (or decrease) in the value of each of the parameter in this case leads to a corresponding decrease (or increases) on  $R_0$  of the model (7). Hence, with sensitivity analysis, one can get insight on the appropriate intervention strategies to prevent and control the spread of the disease described by model (7).

**Table 5 Sensitivity value with the Parameters**

Parameter	Sign
$\beta$	+ 1
$\theta$	- 0.6361563518
$\eta_1$	+ 0.3159609121
$\eta_2$	+ 0.08905979791
$r$	- 0.06219721649
$\tau_1$	+ 0.03054932245
$\tau_3$	- 0.02654932245
$\tau_3$	- 0.02118264309

The most sensitive parameter is  $\beta$  follow by  $\theta$  and the least sensitive parameter is  $\varepsilon$  . All these eight parameters play an important role in the dynamical spread of the COVID 19 disease in the population. The effect of some of them will be graphically illustrated below.

## 5. RESULTS AND DISCUSSION

In this study, five (5) deterministic epidemiological model of (S, E, I, Q, R) are presented to gain insight into the dynamical spread of COVID 19 disease. Positivity of solution shows that, the model presented is mathematically and epidemiologically well posed. Local and global stability of the model shows that, disease-free equilibrium is asymptotically

stable whenever the threshold quantity ‘ $R_0$ ’ is less than unity and otherwise endemic when it is greater than unity. The sensitivity analysis reveals that eight (8) parameters plays an important role in the dynamical spread of COVID 19 disease according to the model (7), the parameters are  $\tau_1, \beta, \theta, r, \tau_2, \eta_1, \eta_2$  and  $\tau_3$ . Four (4) were positive and four (4) were negative as it can be seen in Table 4 and Table 5, increasing those with positive index will result in the higher spread of the disease in the population, so effects must be made to keep it loss while increasing those with negative index will result in the reducing the spread of the disease in the population, so effects must be made to raise it up. The bifurcation analysis was a forward which shows that the disease can be control if all effect is put in place to force  $R_0$  to be less than one.

## 6. CONCLUSION

In conclusion, reduction or elimination of COVID 19 diseases governed by model (2) can be eliminated from the population whenever an influx by infected individual is small such that  $R_0 < 1$ , also reduction or elimination of COVID 19 disease is independent of the initial sizes of the sick people in the population. Hence, COVID 19 disease can be eliminated if the associated reproduction number is less than unity. The bifurcation analysis was a forward which shows that the disease can be control if all effect is put in place to force  $R_0$  to be less than one. The sensitivity analysis reveals that four (4) were positive, which are  $\tau_1, \beta, \eta_1$  and  $\eta_2$ ; increasing these one will result in the more spread of the disease in the population, all hand must be on deck to keep it loss. Four (4) were negative are  $\theta, r, \tau_2$  and  $\tau_3$ ; increasing those with negative index will result in the reducing the spread of the disease in the population, so effects must be made to raise it up.

## 7. Competing Interests

The author declared that no conflict of interest exists in this publication.

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