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A Compartmental Mathematical Model of Novel Coronavirus-19 Transmission Dynamics

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Abstract: The COVID-19 pandemic has spread quickly throughout the world, posing a serious threat to human-to-human transmission. The novel coronavirus pandemic is described quantitatively in this paper using a mathematical model of COVID-19 driven by a system of ordinary differential equations. The suggested model is used to provide predictions regarding the behavior of a COVID-19 outbreak over a shorter time frame. It is demonstrated that the system of model equations has a unique and existing solution. Furthermore, the answer is positive and bounded. Thus, it is argued that the model created and discussed in this work is both mathematically and biologically sound. A threshold parameter that controls the disease transmission is used in a qualitative analysis of the model to confirm the existence and stability of disease-free and endemic equilibrium points. Additionally, the key parameters undergo sensitivity analysis to ascertain their relative significance and potential influence on the COVID-19 virus dynamics.

Keywords: COVID-19; Disease; Sensitivity Analysis; Stability Analysis and Reproduction Number.

1 Introduction

Coronavirus-19 (COVID-19) is an infectious infection attributable to a newly located coronavirus[9]. It is a new strain that was discovered in 2019 and has not been previously identified in humans[7]. The COVID-19 is a novel Coronavirus that was first reported to the world health organization country office in China on 31 December 2019 [5]. The outbreak was declared a public health emergence of international concern on 30 January 2020. On 11 February 2020, WHO announced a name for the new Coronavirus-19 disease "COVID-19" [8]. Several studies suggest that COVID-19, including preliminary information on the COVID-19 virus may persist on the surfaces for a few hours or up-to several days.It is transmitted from person to person via respiratory droplets produced when an infected person coughs or sneezes and between people who are in close contact with one another with in about 6 feets.
Mathematical modeling helps CDC and partners respond to the COVID-19 pandemic by informing decision about pandemic planning, resource allocation and implementation of social distancing measures and other intervations. The majority of the transmission is happening through respiratory droplets that we may inhale from close contact with one another [3]. It is not certain how long the virus that causes COVID-19 survives on the surfaces, but it seems to be have like other coronaviruses.The most common symptoms of Coronavirus-19 (COVID-19) are fever, cough and shortness of breath and breathing difficulties [9]. The period within which the symptoms would appear is $2-14$ days[9]. In more severe cases infection can cause pneumonia, sever acute respiratory syndrome and even death. There is no specific treatment for the disease caused by Coronavirus-19 (COVID-19). However, many of the symptoms can be treated and therefore the treatment is based on the patient's clinical condition. The best ways that are recommended by WHO to prevent the novel coronavirus (COVID-19) are, taking vaccine of covid-19, washing hands often with soap and water, if not available use hand sanitizer, avoid touching your eyes,

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nose, or mouth with unwashed hands, avoid contact with people who are sick, stay home while you are sick and avoid close contact with others, cover your mouth/nose with a tissue or sleeve when coughing or sneezing and so on [8].

Currently, COVID-19 is of brilliant challenge to researchers, governments, and every person due to the excessive rate of infection unfold and the significant number of deaths that occurred. Chen et al developed Bats-Hosts-Reservoir -People transmission network model for simulating the potential transmission from the infection source (probably be bats) to the human infection [11]. Bats-Hosts-Reservoir network was hard to explore clearly and public worries have been focusing at the transmission from Huanan Seafood Wholesale Market (reservoir) to people, they simplified the model as Reservoir-People (RP) transmission network model[7]. The model showed that the transmission of SARS-CoV-2 was higher than the Middle East respiratory syndrome in the Middle East countries, similar to severe acute respiratory syndrome, but lower than MERS in the Republic of Korea[14]. Furthermore, Chayu Yang and Jin Wang [13] model describes the multiple transmission pathways in the infection dynamics, and emphasizes the role of the environmental reservoir in the transmission and spread of this disease. The analytical and numerical results indicate that the Coronavirus infection would remain endemic, which necessitates long-term disease prevention and intervention programs. A lot of authors developed a mathematical model to illustrate the dynamics of the disease that helped them to suggest disease control mechanism and also described the transmission dynamics of the Coronavirus infection. Li Y et al. [14] proposed a mathematical model, based on the transmission mechanism of COVID-19 in the population and the implemented prevention and control measures. Also they established the dynamic models of the six chambers, and establish the time series models based on different mathematical formulas according to the variation law of the original data. E.D.Gurmu et al [7] modify the model developed by Li Y et al[14] by adding the asymptomatic compartment. In this paper we modify the model developed by E.D.Gurmu et al [7], by adding exposed, hospitalized and death compartment.

2 Model Assumption and Formulation

Mathematical modeling methods requires translation of a biological scenario into a mathematical problem. It begins with a clear description of the processes primarily based on the modeler understanding of the system. The translation of a biological scenario into mathematical equations should be made with a specific goal or biological question in mind. Then the verbal description of the system is encoded in mathematical equations. The total number of human population at a time t, denoted by $N(t)$, is subdivided into ten compartments. Namely:

i) Protected individuals $P(t)$; are individuals who are protected against the disease over period of time at specific area not vulnerable to covid-19.
ii) Susceptible individuals $S(t)$; individuals who are vulnerable to COVID-19.
iii) Exposed individuals $E(t)$; individuals who are infected but not yet infectious.
iv) Asymptomatic individuals $A(t)$; are individuals who are infected and infectiuos but do not show a symptoms of corona virus (COVID-19).
v) Infective individual in symptomatic phase $I(t)$; individuals who are showing symptoms of corona virus (COVID-19).
vi) Quarantine individuals $Q(t)$; are individual who are infectious and compulsory quarantine due to reduce the spread of COVID-19 and get treatment.
vii) Hospitalized individuals $H(t)$; are individuals who enters a hospitals due to the hardness of COVID-19.
viii) Recovered individual $R(t)$; are individuals that recovered from COVID-19 at a time t due to treatment at quarantine class, hospitalized class and chronic class of COVID-19.
ix) Chronic individuals $C(t)$; are individuals who are in the intensive care unit (ICU) class that lead to death class of COVID-19.
x) Death class $D(t)$; are individuals who do not recovered from COVID-19 in the intensive care unit (ICU) class and died.

Then the total population at a time t denoted by $N(t)$ is given by:

$$N(t) = P(t) + S(t) + E(t) + A(t) + I(t) + Q(t) + H(t) + R(t) + C(t) + D(t).$$

Thus the model assumed that the protected individuals are generated by recruitment of individuals into the population at a constant rate of π, and decreased by natural death at a rate μ and further deceased by losing protection at a rate δ. Susceptible individuals are generated by losing protection of protected individuals at a rate δ and losing immunity of recovered individuals at a rate θ. Also susceptible individual are decreased by acquiring COVID-19 infection following effective contact with infectious individual at a rate λ, such that;

$$\lambda = \frac{\beta[I(t)+qA(t)]}{N},$$

is a force of where β is the effective contact rate (contact capable of leading to infection) and q is the transmission coefficient for the asymptomatic individuals. If $q > 1$ then the asymptomatic individuals infect the susceptible individuals more likely than the symptomatic individuals. If $q < 1$, then the infective symptomatic individuals have a good chance to infect the susceptible individuals than asymptomatic individuals and if $q = 1$,then both asymptomatic and infective symptomatic individuals have equal chance to infect the susceptible individuals. After a disease incubation period $\frac{1}{\eta}$, where η is per capita rate of becoming infections a proportion of p of the individuals in $E(t)$ may develop a symptoms of COVID-19 infection

and move to the infected compartment $I(t)$ at a rate $p\eta$ and the rest become asymptomatic individuals with COVID-19 infection with probability $(1-p)$ and moves to the asymptomatic $A(t)$ compartment at a rate $(1-p)\eta$. The population of asymptomatic individuals are generated by the fraction of exposed individuals at a rate $(1-p)\eta$ and decreased by developing a symptoms of COVID-19 at a rate ψ and quarantined at a rate γ.
The population of infective individuals are generated by the fraction of exposed individuals at a rate ηp and asymptomatic individuals by developing a symptoms of COVID-19 at a rate ψ and decreased by quarantined at a rate φ . The population of quarantine individuals are generated by isolating rate of asymptomatic and infective individuals in symptomatic phase at a rate γ and φ respectively and decreased by a rate of treatment in the quarantine class at a rate αp and failure of treatment at quarantine class at a rate $(1-p)\alpha$.
The population of hospitalized individuals are increased by the failure of treatment at the quarantine class at the rate $(1-p)\alpha$ and decreased by recovering rate at ϕ due to treatment at hospitals and failure of treatment at a rate ω in hospitals. The population of recovered individuals are generated by recovering rate at α, ϕ and ρ due to treatment at quarantine class, hospital and ICU class respectively and decreased by losing immunity at a rate θ.
The population of a chronic individuals are generated by the failure of treatment in the hospitals at a rate ω , decreased by the success of treatment at ICU class at a rate ρ and failure of treatment at ICU class at a rate τ .
The population of at the death classes are generated by the failure of treatment in the ICU class at a rate τ. All types of cells suffer natural mortality at a rate μ and all parameters in the model are non-negative .
Up on including the basic assumption the schematic diagram of the modified model can be given us below by the figure 1.

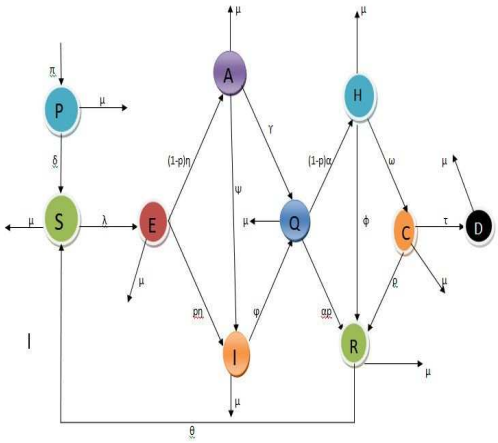

Fig. 1: Schematic Diagram of COVID-19 model

Based on the model assumptions, the notations of variables, parameters and the schematic diagram, the model equations are formulated and given as follows:

$$\begin{cases} \frac{dP}{dt} = \pi - (\mu+\delta)P, \\ \frac{dS}{dt} = \delta P + \theta R - (\mu+\lambda)S, \\ \frac{dE}{dt} = \lambda S - (\mu+\eta)E, \\ \frac{dA}{dt} = (1-p)\eta E - (\mu+\gamma+\psi)A, \\ \frac{dI}{dt} = p\eta E + \psi A - (\mu+\varphi)I, \\ \frac{dQ}{dt} = \gamma A + \varphi I - (\mu+\alpha)Q, \\ \frac{dH}{dt} = (1-p)\alpha Q - (\mu+\omega+\phi)H, \\ \frac{dR}{dt} = \alpha pQ + \phi A + \rho C - (\mu+\theta)R, \\ \frac{dC}{dt} = \omega H - (\mu+\rho+\tau)C, \\ \frac{dD}{dt} = \tau C - \mu D. \end{cases} \tag{1}$$

The non-negative initial conditions of the system of model equations (1) are denoted by $P(0)>0, S(0)>0, E(0)\geq 0, A(0)\geq 0, I(0)\geq 0, Q(0)\geq 0, H(0)\geq 0, R(0)\geq 0, C(0)\geq 0, D(0)\geq 0$. This system consists of ten first order non-linear ordinary differential equations.

3 Mathematical Analysis of the Formulated Model

3.1 Invariant Region

Theorem 1. *The total population size N of the system of model equation 1 is bounded in the invariant region Ω. That is, size of N is bounded for all t.*
proof: *We adhere to the following steps to show the positive invariance of Ω, that is all the solution of 1 that initiate in Ω remains in the region Ω,which is bounded in Ω.*
The total population is given by

$$N(t) = P(t)+S(t)+E(t)+A(t)+I(t)+Q(t)+H(t)+R(t)+C(t)+D(t).$$

The rate of change of the total population by adding all the equations considered in 1 is:

$$\frac{dN}{dt} = \pi - \mu N - \phi(A+H) \leq \pi - \mu N$$

Notice that $\frac{dN}{dt}$ is bounded above by $\pi-\mu N$ and below by 0.
Hence by using standard comparison theorem [10] it can be shown that,

$$0 \leq N(t) \leq \frac{\pi}{\mu} + (N_0 - \frac{\pi}{\mu})e^{-\mu t} \tag{2}$$

As $t \longrightarrow \infty$ in equation (2) , the population size $N \longrightarrow \frac{\pi}{\mu}$ which implies that $0 \leq N \leq \frac{\pi}{\mu}$.
Thus the feasible solution set of the equation of the model enter and remain in the the region

$$\Omega = \{(P,S,E,A,I,Q,H,R,C,D) \in \mathbb{R}_+^{10} : N \leq \frac{\pi}{\mu}\}.$$

Therefore, the basic model is well posed epidemiologically and mathematically. Hence , it is sufficient to study the dynamics of the basic model in Ω.

3.2 Existence and Positivity of solution

In order to show that the model is biologically valid, it is required to prove that the solutions of the system of differential equations (1) are both exist and positive for all time. It is done starting with proving Lemma 1.

Lemma 1.*(Existence): The solutions of the model equations (1) together with the initial conditions* $P(0) > 0, S(0) > 0, E(0) \geq 0, A(0) \geq 0, I(0) \geq 0, Q(0) \geq 0, H(0) \geq 0,$
$R(0) \geq 0, C(0) \geq 0, D(0) \geq 0$ *exist in* $\mathbb{R}_+^{10}$.

Proof: *Let the system of equation (1) be as follows:*

$$\begin{cases} f_1 = \pi - (\mu+\delta)P, \\ f_2 = \delta P + \theta R - (\mu+\lambda)S, \\ f_3 = \lambda S - (\mu+\eta)E, \\ f_4 = (1-P)\eta E - (\mu+\gamma+\psi)A, \\ f_5 = p\eta E + \psi A - (\mu+\varphi)I, \\ f_6 = \gamma A + \varphi I - (\mu+\alpha)Q, \\ f_7 = (1-p)\alpha Q - (\mu+\omega+\phi)H, \\ f_8 = \alpha pQ + \phi A + \rho C - (\mu+\theta)R, \\ f_9 = \omega H - (\mu+\rho+\tau)C, \\ f_{10} = \tau C - \mu D. \end{cases} \tag{3}$$

According to Derrick and Groosman theorem, let Ω *denote the region*
$\Omega = \{(P,S,E,A,I,Q,H,R,C,D) \in \mathbb{R}_+^{10}; N \leq \frac{\pi}{\mu}\}$.
Then equations (1) have a unique solution if $\frac{\partial f_i}{\partial x_j}, i,j = 1,2,3,4,5,6,7,8,9,10$ *are continuous and bounded in* Ω. *Here,* $x_1 = P, x_2 = S, x_3 = E, x_4 = A, x_5 = I, x_6 = Q, x_7 = H, x_8 = R, x_9 = C, x_{10} = D$. *The continuity and boundedness are verified here under:*

For f_1:	*For* f_2:
$\lvert\frac{\partial f_1}{\partial p}\rvert = \lvert-(\mu+\delta)\rvert < \infty,$	$\lvert\frac{\partial f_2}{\partial P}\rvert = \delta < \infty,$
$\lvert\frac{\partial f_1}{\partial S}\rvert = 0 < \infty,$	$\lvert\frac{\partial f_2}{\partial S}\rvert = \lvert-(\mu + \frac{\beta(I+qA)}{N})\rvert < \infty,$
$\lvert\frac{\partial f_1}{\partial E}\rvert = 0 < \infty,$	$\lvert\frac{\partial f_2}{\partial E}\rvert = 0 < \infty,$
$\lvert\frac{\partial f_1}{\partial A}\rvert = 0 < \infty,$	$\lvert\frac{\partial f_2}{\partial A}\rvert = \lvert-\frac{\beta qS}{N}\rvert < \infty,$
$\lvert\frac{\partial f_1}{\partial I}\rvert = 0 < \infty,$	$\lvert\frac{\partial f_2}{\partial I}\rvert = \lvert-\frac{\beta S}{N}\rvert < \infty,$
$\lvert\frac{\partial f_1}{\partial Q}\rvert = 0 < \infty,$	$\lvert\frac{\partial f_2}{\partial Q}\rvert = 0 < \infty,$
$\lvert\frac{\partial f_1}{\partial H}\rvert = 0 < \infty,$	$\lvert\frac{\partial f_2}{\partial H}\rvert = 0 < \infty,$
$\lvert\frac{\partial f_1}{\partial R}\rvert = 0 < \infty,$	$\lvert\frac{\partial f_2}{\partial R}\rvert = \theta < \infty,$
$\lvert\frac{\partial f_1}{\partial C}\rvert = 0 < \infty,$	$\lvert\frac{\partial f_2}{\partial C}\rvert = 0 < \infty,$
$\lvert\frac{\partial f_1}{\partial D}\rvert = 0 < \infty.$	$\lvert\frac{\partial f_2}{\partial D}\rvert = 0 < \infty.$

Similarly true for $f_3 - f_{10}$. *Thus, all the partial derivatives* $\frac{\partial f_i}{\partial x_j}, i,j = 1,2,3,4,5,6,7,8,9,10$ *exist, continuous and bounded in* Ω. *Hence, by Derrick and Groosman theorem, a solution for the model (1) exists and is unique.*

Lemma 2.*(Positivity) Solutions of the model equations (1) together with the initial conditions* $P(0) > 0, S(0) > 0, E(0) \geq 0, A(0) \geq 0, I(0) \geq 0, Q(0) \geq 0, H(0) \geq 0, R(0) \geq 0, C(0) \geq 0$ *and* $D(0) \geq 0$ *are always positive (OR) the model variables* $P(t), S(t), E(t), A(t), I(t), Q(t), H(t), R(t), C(t)$ *and* $D(t)$ *are positive for all* t *and will remain in* $\mathbb{R}_+^{10}$.

Proof*: Positivity of the model variables are shown separately for each of the model variables* $P(t), S(t), E(t), A(t), I(t), Q(t), H(t), R(t), C(t)$ *and* $D(t)$.

Positivity of P(t): *The model equation in (1) given by* $\frac{dP}{dt} = \pi - (\mu+\delta)P$ *can be expressed without loss of generality (WOLG), after eliminating the positive terms* π *which are appearing on the right hand side, as an inequality as* $\frac{dP}{dt} \geq -(\mu+\delta)P$.
Using variables separable method and on applying integration, the solution of the foregoing differentially inequality can be obtained as $P(t) \geq e^{-(\mu+\delta)t}$. *Recall that an exponential function is always non-negative irrespective of the sign of the exponent, i.e., the exponential function* $e^{-(\mu+\delta)t}$ *is a non-negative quantity. Hence, it can be concluded that* $P(t) > 0$. *Similarly, solving the system of differential equation of the model, we obtain the exponential function:*
$S(t) \geq e^{-\mu t - \frac{\beta}{N}\int(I+qA)dt}$, $E(t) \geq e^{-(\mu+\eta)t}$,
$A(t) \geq e^{-(\mu+\gamma+\psi)t}$,
$I(t) \geq e^{-(\mu+\varphi)t}$,
$Q(t) \geq e^{-(\mu+\alpha)t}$,
$H(t) \geq e^{-(\mu+\omega+\phi)t}$,
$R(t) \geq e^{-(\mu+\theta)t}$,
$C(t) \geq e^{-(\mu+\rho+\tau)t}$,
$D(t) \geq e^{-\mu t}$.
Recall that an exponential function is always non-negative irrespective of the sign of the exponent. Hence, it can be concluded that all the solutions of model equations are positive. Thus, the model variables $P(t), S(t), E(t), A(t), I(t), Q(t), H(t), R(t), C(t)$ *and* $D(t)$ *representing population sizes of various types of cells are non-negative quantities and will remain in* $\mathbb{R}_+^{10}$ *for all* t.

4 Stability Analysis of Disease-Free Equilibrium (DFE)

Disease free equilibrium points are steady state solutions where there is no disease in the population. In the absence of the disease this implies that $E(t) = A(t) = I(t) = Q(t) = H(t) = R(t) = C(t) = D(t) = 0$ and the equilibrium points require that the right hand side of the model equation set equal to zero. Thus, the disease-free equilibrium point of the model equation in (1) above is given by

$$E_0 = \{\tfrac{\pi}{\mu+\delta}, \tfrac{\delta\pi}{\mu(\mu+\delta)}, 0,0,0,0,0,0,0,0\}$$

The local stability of the DFE, E_0, of the model can be established using the basic reproduction number. The basic reproduction number is denoted by R_0 and it is defined as the expected number of people getting secondary infection among the whole susceptible population. It is computed using next-generation matrix defined in [1]. In this method R_0 is defined as the largest eigenvalue of the next generation matrix. Using the notation as in [1] for the model system (1)the associated matrices F and V for the new infectious terms and the remaining transition terms are respectively given by:

$$F_i = \begin{bmatrix} \frac{\beta(I+qA)S}{N} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

and

$$V_i = \begin{bmatrix} (\mu+\eta)E \\ -(1-p)\eta E + (\mu+\gamma+\psi)A \\ -p\eta E - \psi A + (\mu+\varphi)I \\ -\gamma A - \varphi I + (\mu+\alpha)Q \\ -(1-p)\alpha Q + (\mu+\omega+\psi)H \\ -\omega H + (\mu+\rho+\tau)C \\ -\tau C + \mu D \end{bmatrix}$$

The Jacobian of F_i and V_i at the disease free equilibrium point E_0 takes the form respectively as

$$F(E_0) = \begin{bmatrix} 0 & \frac{\beta\delta q}{\mu+\delta} & \frac{\beta\delta}{\mu+\delta} & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \end{bmatrix}$$

, and

$$V(E_0) = \begin{bmatrix} a & 0 & 0 & 0 & 0 & 0 & 0 \\ (1-p)\eta & b & 0 & 0 & 0 & 0 & 0 \\ -p\eta & -\psi & c & 0 & 0 & 0 & 0 \\ 0 & -\gamma & -\varphi & d & 0 & 0 & 0 \\ 0 & 0 & 0 & -(1-p)\alpha & e & 0 & 0 \\ 0 & 0 & 0 & 0 & -\omega & f & 0 \\ 0 & 0 & 0 & 0 & 0 & -\tau & \mu \end{bmatrix}$$

Then after some algebraic computations the product of the matrices $F(E_0)$ and $[V(E_0)]^{-1}$ can be computed as;

$$[F(E_0)][V(E_0)]^{-1} =$$

$$\begin{bmatrix} \frac{\beta\delta\eta(bp+(cq+\psi)(1-p))}{abc(\mu+\delta)} & \frac{\beta\delta(qc+\psi)}{bc(\mu+\delta)} & \frac{\beta\delta}{c(\mu+\delta)} & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \\ 0 & 0 & 0 & 0\,0\,0\,0 \end{bmatrix}$$

Now it is possible to calculate the eigenvalue to determine the basic reproduction number R_0 by taking the spectral radius of the matrix $[F(E_0)][V(E_0)]^{-1}$. Thus, the eigenvalues are computed by evaluating $det[[F(E_0)][V(E_0)]^{-1} - \lambda I] = 0$ or equivalently solving;

$$\begin{vmatrix} \frac{J}{abc(\mu+\delta)} - \lambda & \frac{\beta\delta(qc+\psi)}{bc(\mu+\delta)} & \frac{\beta\delta}{c(\mu+\delta)} & 0 & 0 & 0 & 0 \\ 0 & -\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\lambda \end{vmatrix} = 0$$

where $J = \beta\delta\eta(bp + (cq+\psi)(1-p))$. It reduces to the tenth power equation for λ as $\lambda^9[\lambda - \frac{\beta\delta\eta(bp+(cq+\psi)(1-p))}{abc(\mu+\delta)}] = 0$ giving the ten eigenvalues as $\lambda_1 = 0$, $\lambda_2 = 0$, $\lambda_3 = 0, \lambda_4 = 0, \lambda_5 = 0, \lambda_6 = 0, \lambda_7 = 0, \lambda_8 = 0, \lambda_9 = 0$, $\lambda_{10} = \frac{\beta\delta\eta(bp+(cq+\psi)(1-p))}{abc(\mu+\delta)}$.
However, the dominant eigenvalue here is $\lambda_{10} = \frac{\beta\delta\eta(bp+(cq+\psi)(1-p))}{abc(\mu+\delta)}$ and it is the spectral radius as the threshold value or the basic reproductive number. Thus, it can be concluded that the reproduction number of the model is;

$$R_0 = \frac{\beta\delta\eta(bp+(cq+\psi)(1-p))}{abc(\mu+\delta)},$$

where, $a = \mu+\eta, b = \mu+\gamma+\psi, c = \mu+\varphi, d = \mu+\alpha, e = \mu+\omega+\phi, f = \mu+\rho+\tau$

4.1 Local Stability of Disease Free Equilibrium

To find the local stability of DFE, the Jacobian of the model equations evaluated at E_0 is used. Where,

$$E_0 = \{\tfrac{\pi}{\mu+\delta}, \tfrac{\delta\pi}{\mu(\mu+\delta)}, 0,0,0,0,0,0,0,0\}.$$

Now, the stability analysis of DFE is conducted and the results are presented in the form of theorems and proofs as follows:

Theorem 2.*The DFE E_0 of the system (1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*
proof *Consider the right hand side expressions of the*

equations (1) as functions so as to find the Jacobian matrix as follows:

$$\begin{cases} \frac{dP}{dt} = \pi - (\mu+\delta)P = f_1 \\ \frac{dS}{dt} = \delta P + \theta R - (\mu+\lambda)S = f_2 \\ \frac{dE}{dt} = \lambda S - (\mu+\eta)E = f_3 \\ \frac{dA}{dt} = (1-p)\eta E - (\mu+\gamma+\psi)A = f_4 \\ \frac{dI}{dt} = p\eta E + \psi A - (\mu+\varphi)I = f_5 \\ \frac{dQ}{dt} = \gamma A + \varphi I - (\mu+\alpha)Q = f_6 \\ \frac{dH}{dt} = \frac{dA}{dt} = (1-p)\alpha Q - (\mu+\omega+\phi)H = f_7 \\ \frac{dR}{dt} = \alpha pQ + \phi A + \rho C - (\mu+\theta)R = f_8 \\ \frac{dC}{dt} = \omega H - (\mu+\rho+\tau)C = f_9 \\ \frac{dD}{dt} = \tau C - \mu D = f_{10} \end{cases} \quad (4)$$

Now, the Jacobian matrix of $(f_1,f_2,f_3,f_4,f_5,f_6,f_7,f_8,f_9,f_{10})$ *w.r.to* (P,S,E,A,I,Q,H,R,C,D) *at the disease free equilibrium* E_0 *reduces to;* $J(E_0)$=

$$\begin{bmatrix} -a & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \delta & -b & 0 & -\frac{\beta\delta q}{\mu+\delta} & -\frac{\beta\delta}{\mu+\delta} & 0 & 0 & \theta & 0 & 0 \\ 0 & 0 & -c & \frac{\beta\delta q}{\mu+\delta} & \frac{\beta\delta}{\mu+\delta} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-p)\eta & -d & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & \varphi & -e & 0 & 0 & 0 & 0 \\ 0 & 0 & k\eta & \psi & -f & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & (1-p)\alpha & -g & 0 & 0 & 0 \\ 0 & 0 & 0 & \phi & 0 & \alpha p & 0 & -h & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega & -i & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \tau & -\mu \end{bmatrix}$$

where
$a = \mu+\delta, b = \mu, c = \mu+\eta, d = \mu+\gamma+\psi, e = \mu+\varphi, f = \mu+\alpha, g = \mu+\omega+\phi, h = \mu+\theta, i = \mu+\rho+\tau.$
Now, the eigenvalues of $J(E_0)$ *are required to be found. Then, the characteristic equation* $det[J(E_0)-\lambda I] = 0$ *is simplified and found as follows:*
$= (-a-\lambda)(-b-\lambda)(-c-\lambda)(-d-\lambda)(-e-\lambda)(-f-\lambda)(-g-\lambda)(-h-\lambda)(-i-\lambda)(-\mu-\lambda) = 0$
Thus, the ten eigenvalues of the matrix are determined as

$$\lambda_1 = -a, \quad \lambda_2 = -b, \quad \lambda_3 = -c, \quad \lambda_4 = d, \quad \lambda_5 = -e,$$
$$\lambda_6 = -f, \quad \lambda_7 = -g, \quad \lambda_8 = -h, \quad \lambda_9 = -i, \quad \lambda_{10} = -\mu.$$

It can be observed that all the eigenvalues λ_1 , λ_2 ,λ_3,λ_4 ,λ_5, λ_6 , λ_7, λ_8, λ_9, λ_{10} *are absolutely negative quantities. Therefore, it is concluded that the DFE* E_0 *of the system of differential equations (1) is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

4.2 Global Stability of Disease Free Equilibrium

To investigate the global stability of the disease free equilibrium points we used technique implemented by Castillo-Chavez and Song [2]. First the model equation (1) can be re-written as

$$\frac{dX}{dt} = F(X,Z),$$
$$\frac{dZ}{dt} = G(X,Z) = G(X,0) = 0,$$

where, $X = (P,S,R)$ stands for the uninfected population and $Z = (E,A,I,Q,H,C,D)$ also stands for the infected population. The disease free equilibrium point of the model here is denoted by $U = (X^0,0)$. The point $U = (X^0,0) = \{\frac{\pi}{\mu+\delta}, \frac{\delta\pi}{\mu(\mu+\delta)}, 0,0,0,0,0,0,0,0\}$ to be globally asymptotically stable equilibrium for the model provided that $R_0 < 1$ and the following conditions must be met:
$H_1 : \frac{dX}{dt} = F(X^*,0), X^*$ is globally asymptotically stable.
$H_2 : G(X,Z) = AZ - \hat{G}(X,Z), \hat{G}(X,Z)` \geq 0$ for $(X,Z) \in \Omega$.
Where $A = D_Z G(U,0)$ is a Metzler matrix (the off diagonal elements of A are non-negative) and G is the region where the model make biologically sense. If the model (1) met the above two criteria then the following theorem holds.

Theorem 3.*The point* $U = (X^*,0)$ *is globally asymptotically stable equilibrium provided that* $R_0 < 1$ *and the condition* (H_1) *and* (H_2) *are satisfied.*
proof: *From system (1) , we get* $F(X,Y)$ *and* $G(X,Y)$*;*

$$F(X,Z) = \begin{bmatrix} \pi - (\mu+\delta)P \\ \delta P + \theta R - (\lambda+\mu)S \\ \alpha pQ + \phi A + \rho C - (\mu+\theta)R \end{bmatrix}$$

and

$$G(X,Z) = \begin{bmatrix} \frac{\beta}{N}S(I+qA) - (\mu+\eta)E \\ (1-p)\eta E - (\mu+\gamma+\psi)A \\ p\eta E + \psi A - (\mu+\varphi)I \\ \gamma A + \varphi I - (\mu+\alpha)Q \\ (1-p)\alpha Q - (\mu+\omega+\phi)H \\ \omega H - (\mu+\rho+\tau)C \\ \tau C - \mu D \end{bmatrix}$$

The compartmental model 1 stated in condition (H_1) *can be expressed in the reduced system as;*

$$\frac{dX}{dt_{Z=0}} = \begin{bmatrix} \pi - (\mu+\delta)P \\ \delta P - \mu S \\ 0 \end{bmatrix} \quad (5)$$

Analytically solving equation (5) above it is obvious that $\{\frac{\pi}{\mu+\delta}, \frac{\delta\pi}{\mu(\mu+\delta)}, 0\}$ *is the global asymptotic point. Thus,* X^* *is globally asymptotically stable for* $\frac{dX}{dt} = F(X,0)$ *and the first condition* (H_1) *holds for the system 1.*
Now for the second condition the matrices A for the model system 1 can be expressed from the equation for infected compartments in the model as;
$A =$

$$\begin{bmatrix} -a & \beta q & \beta & 0 & 0 & 0 & 0 \\ (1-p)\eta & -b & 0 & 0 & 0 & 0 & 0 \\ p\eta & \psi & -c & 0 & 0 & 0 & 0 \\ 0 & \gamma & \varphi & -d & 0 & 0 & 0 \\ 0 & 0 & 0 & (1-p)\alpha & -e & 0 & 0 \\ 0 & 0 & 0 & 0 & \omega & -f & 0 \\ 0 & 0 & 0 & 0 & 0 & \tau & -\mu, \end{bmatrix}$$

where $a=(\mu+\eta), b=(\mu+\gamma+\psi), c=(\mu+\varphi), d=(\mu+\alpha), e=(\mu+\omega+\phi), f=(\mu+\rho+\tau)$ *and the matrix* $\hat{G}(X,Z)$ *can be written as,* $\hat{G}(X,Z)=AZ-G(X,Z)$, *which is:*

$$\hat{G}(X,Z)=\begin{bmatrix}\hat{G}_1(X,Z)\\ \hat{G}_2(X,Z)\\ \hat{G}_3(X,Z)\\ \hat{G}_4(X,Z)\\ \hat{G}_5(X,Z)\\ \hat{G}_6(X,Z)\\ \hat{G}_7(X,Z)\end{bmatrix}=\begin{bmatrix}\beta(I+qA)(1-\frac{S}{N})\\ 0\\ 0\\ 0\\ 0\\ 0\\ 0\end{bmatrix}$$

Then the matrix A is a M- matrix since all its off diagonal elements are non-negative and $\hat{G}(X,Z)\geq 0$ *in the region* Ω *as* $S(t)\leq N(t)$ *for in which condition* (H_2) *holds.*
Since the two conditions (H_1) *and* (H_2) *holds, the disease free steady state* E_0 *of the model 1 is globally asymptotically stable in the region* Ω *for* $R_0<1$.

4.3 Endemic Equilibrium

The endemic equilibrium point $E_1=\{P^*,S^*,E^*,A^*,I^*,Q^*,H^*,R^*,C^*,D^*,\}$ is a steady state solution where the disease persists in the population. The endemic equilibrium point is obtained by setting rates of changes of variables with respect to time in model equations (1) to zero. That is, setting $\frac{dP}{dt}=\frac{dS}{dt}=\frac{dE}{dt}=\frac{dA}{dt}=\frac{dI}{dt}=\frac{dQ}{dt}=\frac{dH}{dt}=\frac{dR}{dt}=\frac{dC}{dt}=\frac{dD}{dt}=0$ the model equations take the form as solved for state variables interms of the parameters after some algebraic operation and obtain the following;

$$\begin{cases}P^*=\frac{\pi}{a}\\ S^*=\frac{cdefghi\delta\pi}{k}\\ E^*=\frac{defghi\lambda^*\delta\pi}{k}\\ A^*=\frac{efghi\lambda^*\delta\pi\eta(1-p)}{k}\\ I^*=\frac{pfghi\lambda^*\delta\pi\eta(k(d-1)+1)}{k}\\ Q^*=\frac{ghi\lambda^*\delta\pi[\eta(1-p)(\gamma e+\varphi)+dp\varphi\eta]}{k}\\ H^*=\frac{hi\alpha\eta\lambda^*\delta\pi(1-p)[(1-p)(e\gamma+\varphi)+dk\eta\varphi]}{k}\\ R^*=\frac{cdefghi\lambda^*\delta\pi(\mu+\lambda^*)}{k}-\frac{\delta\pi}{\alpha\theta}\\ C^*=\frac{h\alpha\omega\eta\lambda^*\delta\pi(1-p)[(1-p)(\gamma e+\varphi)+dp\varphi]}{k}\\ D^*=\frac{h\lambda^*\alpha\omega\tau\delta\pi\eta[(1-p)(\gamma e+\varphi)+dp\varphi]}{k}\end{cases}\quad(6)$$

where, $k=(cdefgi\alpha(\mu+\lambda^*)-\lambda^*\theta\alpha\eta[[\alpha kgi+\rho\alpha\omega(1-p)][(1-p)(\gamma e+\varphi)+dp\varphi]+efgi\phi(1-p)])$, and
$a=\mu+\delta, b=\mu+\lambda^*, c=\mu+\eta, d=\mu+\gamma+\psi, e=\mu+\varphi, f=\mu+\alpha, g=\mu+\omega+\phi, h=\mu+\theta, i=\mu+\rho+\tau$ and $\lambda^*=\frac{\beta(I^*+qA^*)}{N^*}$

4.3.1 Stability Analysis of Endemic Equilibrium

In the presence of the infectious disease, the model populations have a unique endemic steady state E_1. To find the local stability of E_1, the Jacobian of the model equations evaluated at E_1 is used. Now, the stability analysis of E_1 is conducted and the results are presented in the form of theorems and proofs as follows:

Theorem 4.*The endemic equilibrium point* E_1 *of the system (1) is locally asymptotically stable if* $R_1>1$ *and unstable if* $R_1<1$.
proof: *Consider the right hand side expressions of the equations (1) as functions so as to find the Jacobian matrix as follows:*
$J(E_1)=$

$$\begin{bmatrix}-a & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\ \delta & -k_3 & 0 & \frac{-\beta qS^*}{N^*} & \frac{-\beta S^*}{N^*} & 0 & 0 & \theta & 0 & 0\\ 0 & \lambda^* & -c & \frac{\beta qS^*}{N^*} & \frac{-\beta S^*}{N^*} & & 0 & & 0 & \\ 0 & 0 & k_4 & -d & 0 & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & \gamma & \varphi & -e & 0 & 0 & 0 & 0\\ 0 & 0 & k\eta & \psi & -f & 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & k_5 & -g & 0 & 0 & 0\\ 0 & 0 & 0 & \phi & 0 & \alpha p & 0 & -h & 0 & 0\\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega & -i & 0\\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \tau & -\mu\end{bmatrix}$$

where $k_3=(\mu+\lambda^*), k_4=(1-p)\eta, k_5=(1-p)\alpha$. *Now, the eigenvalues of* $J(E_1)$ *are required to be found. The characteristic equation* $det[J(E_1)-\lambda I]=0$ *is expanded and simplified as follows:*
$(-a-\lambda)(-(\mu+\lambda^*)-\lambda)(-c-\lambda)(-d-\lambda)(-e-\lambda)(-f-\lambda)(-g-\lambda)(-h-\lambda)(-i-\lambda)(-\mu-\lambda)=0$
Thus the eigenvalues of the endemic equilibrium points are:
$\lambda_1=-\mu-\delta,$
$\lambda_2=-[\mu+\frac{\beta}{N^*}(I^*+qA^*)],$
$\lambda_3=-\mu-\eta,$
$\lambda_4=-\mu-\gamma-\psi,$
$\lambda_5=-\mu-\varphi,$
$\lambda_6=-\mu-\alpha,$
$\lambda_7=-\mu-\omega-\phi,$
$\lambda_8=-\mu-\theta,$
$\lambda_9=-\mu-\rho-\tau,$
$\lambda_{10}=-\mu$. *where*

$$A^*=\frac{efghi\lambda^*\delta\pi\eta(1-p)}{k}$$
$$I^*=\frac{pfghi\lambda^*\delta\pi\eta(k(d-1)+1)}{k}$$

It can be observed that all the eigenvalues λ_1 , λ_2 ,λ_3,λ_4 ,λ_5, λ_6 , λ_7, λ_8, λ_9, λ_{10} *are absolutely negative quantities. Therefore, it is concluded that the endemic equilibrium point* E_1 *of the system of differential equations (1) is locally asymptotically stable if* $R_0>1$ *and unstable if* $R_0<1$.

Theorem 5. *The endemic equilibrium point of the model equation (1) is globally asymptotically stable if $R_0 > 1$.*

Proof *To show the result we define the following Lyapunov function as follows:*

$L(P^*,S^*,E^*,A^*,I^*,Q^*,H^*,R^*,C^*,D^*) = [P - P^* - P^*ln(\frac{P}{P^*})] + [S - S^* - S^*ln(\frac{S}{S^*})] + [E - E^* - E^*ln(\frac{E}{E^*})] + [A - A^* - A^*ln(\frac{A}{A^*})] + [I - I^* - I^*ln(\frac{I}{I^*})]$

By taking the derivative of L with respect to t:

$\frac{dL}{dt} = (1 - \frac{p^*}{p})\frac{dp}{dt} + (1 - \frac{S^*}{S})\frac{dS}{dt} + (1 - \frac{E^*}{E})\frac{dE}{dt} + (1 - \frac{A^*}{A})\frac{dA}{dt} + (1 - \frac{I^*}{I})\frac{dI}{dt} + (1 - \frac{Q^*}{Q})\frac{dQ}{dt} + (1 - \frac{H^*}{H})\frac{dH}{dt} + (1 - \frac{R^*}{R})\frac{dR}{dt} + (1 - \frac{C^*}{C})\frac{dC}{dt} + (1 - \frac{D^*}{D})\frac{dD}{dt}.$

$= (1 - \frac{p^*}{p})[\pi - (\mu + \delta)P] + (1 - \frac{S^*}{S})[\delta P + \theta R - (\mu + \lambda)S] + (1 - \frac{E^*}{E})[\lambda S - (\mu + \eta)E] + (1 - \frac{A^*}{A})[(1 - p)\eta E - (\mu + \gamma + \psi)A] + (1 - \frac{I^*}{I})[p\eta E + \psi A - (\mu + \varphi)I] + (1 - \frac{Q^*}{Q})[\gamma A + \varphi I - (\mu + \alpha)Q] + (1 - \frac{H^*}{H})[(1 - p)\alpha Q - (\mu + \omega + \phi)H] + (1 - \frac{R^*}{R})[\alpha pQ + \phi A + \rho C - (\mu + \theta)R] + (1 - \frac{C^*}{C})[\omega H - (\mu + \rho + \tau)C] + (1 - \frac{D^*}{D})[\tau C - \mu D]$

$= (1 - \frac{p^*}{p})(\pi - aP) + (1 - \frac{S^*}{S})(\delta P + \theta R - bS) + (1 - \frac{E^*}{E})(\lambda S - cE) + (1 - \frac{A^*}{A})((1 - p)\eta E - dA) + (1 - \frac{I^*}{I})(p\eta E + \psi A - eI) + (1 - \frac{Q^*}{Q})(\gamma A + \varphi I - fQ) + (1 - \frac{H^*}{H})((1 - p)\alpha Q - gH) + (1 - \frac{R^*}{R})(\alpha pQ + \phi A + \rho\rho C - hR) + (1 - \frac{C^*}{C})(\omega H - iC) + (1 - \frac{D^*}{D})(\tau C - \mu D)$

After some simplification and rearrangement we obtain;

$\frac{dL}{dt} = \pi + \delta P + \theta R + \lambda S + \eta E + dA + \varphi I + \alpha Q + (\rho + \tau)C + \omega H + aP^* + bS^* + cE^* + dA^* + eI^* + fQ^* + gH^* + \mu D^* - [aP + bS + cE + dA + eI + fQ + gH + hR + iC + \mu D + (\delta P + \theta R)\frac{S^*}{S} + \lambda S\frac{E^*}{E} + (1 - p)\eta E\frac{A^*}{A} + (p\eta E + \psi A)\frac{I^*}{I} + \gamma A\frac{Q^*}{Q} + (1 - p)\alpha Q\frac{H^*}{H} + (\alpha pQ + \phi A + \rho C)\frac{R^*}{R} + \omega H\frac{C^*}{C} + \tau C\frac{D^*}{D}].$

$= (\pi + \delta P + \theta R + \lambda S + \eta E + dA + \varphi I + \alpha Q + (\rho + \tau)C + \omega H - [aP + bS + cE + dA + eI + fQ + gH + hR + iC + \mu D]) + aP^* + bS^* + cE^* + dA^* + eI^* + fQ^* + gH^* + \mu D^* - [(\delta P + \theta R)\frac{S^*}{S} + \lambda S\frac{E^*}{E} + (1 - p)\eta E\frac{A^*}{A} + (p\eta E + \psi A)\frac{I^*}{I} + \gamma A\frac{Q^*}{Q} + (1 - p)\alpha Q\frac{H^*}{H} + (\alpha pQ + \phi A + \rho C)\frac{R^*}{R} + \omega H\frac{C^*}{C} + \tau C\frac{D^*}{D}].$

$= \mu N + aP^* + bS^* + cE^* + dA^* + eI^* + fQ^* + gH^* + \mu D^* - [(\delta P + \theta R)\frac{S^*}{S} + \lambda S\frac{E^*}{E} + (1 - p)\eta E\frac{A^*}{A} + (p\eta E + \psi A)\frac{I^*}{I} + \gamma A\frac{Q^*}{Q} + (1 - p)\alpha Q\frac{H^*}{H} + (\alpha pQ + \phi A + \rho C)\frac{R^*}{R} + \omega H\frac{C^*}{C} + \tau C\frac{D^*}{D}].$

$\frac{dL}{dt} = M - K.$

where, $M = \mu N + aP^* + bS^* + cE^* + dA^* + eI^* + fQ^* + gH^* + \mu D^*$ *and*

$K = [(\delta P + \theta R)\frac{S^*}{S} + \lambda S\frac{E^*}{E} + (1 - p)\eta E\frac{A^*}{A} + (p\eta E + \psi A)\frac{I^*}{I} + \gamma A\frac{Q^*}{Q} + (1 - p)\alpha Q\frac{H^*}{H} + (\alpha pQ + \phi A + \rho C)\frac{R^*}{R} + \omega H\frac{C^*}{C} + \tau C\frac{D^*}{D}]$

Now, $\frac{dL}{dt} = M - K < 0$ *if* $M < K$.

Thus if $M < K$ *then* $\frac{dL}{dt} < 0$, *nothing that* $\frac{dL}{dt} = 0$ *if and only if* $P = P^*, P = P^*,$ $S = S^*, E = E^*, A = A^*, I = I^*, Q = Q^*, H = H^*, R = R^*, C = C^*, D = D^*$.

Therefore the largest compact invariant set in $\{(P^*,S^*,E^*,A^*,I^*,Q^*,R^*,C^*,D^*) \in \Omega; \frac{dL}{dt} = 0\}$ *is a singleton* E_1 *is the endemic equilibrium point of the system (1). By LaSalle's invariant principle [15], it implies that* E_1 *is globally asymptotically stable in* Ω *if* $M < k$ *and* $R_0 > 1$.

4.4 Sensitivity Analysis

Sensitivity analysis allows us to assess the impact that changes in a certain parameter will have on the model and it can help someone to determine which parameters are the key drivers of a model's results. To investigate which parameters have high impact on the R_0, we apply the approach presented in [18]. The main goal of this section is to perform sensitivity analysis of COVID-19 transmission model to the parameters describing in it, i.e. to determine the amount that the entire model changes when each parameter is altered. For instance, the normalized forward sensitivity index on R_0 , which depends differentially on a parameter P, as defined in [16] as;

$$\Upsilon_P^{R_0} = \frac{\partial R_0}{\partial P} \times \frac{P}{R_0}. \tag{7}$$

The explicit expression of R_0 is given by

$$R_0 = \frac{\beta\delta\eta(bp+(cq+\psi)(1-p))}{abc(\mu+\delta)} = \frac{\beta\delta\eta[(\mu+\gamma+\psi)p+(q(\mu+\varphi)+\psi)(1-p)]}{(\mu+\eta)(\mu+\gamma+\psi)(\mu+\varphi)(\mu+\delta)}.$$

where, $a = \mu + \eta, b = \mu + \gamma + \psi, c = \mu + \varphi$.

Since R_0 depends only on nine parameters, we derive an analytical expression for its sensitivity to each parameters using the normalized forward sensitivity index as in [18] by taking the values of the parameters from table 2 in 7 and computed as follows:

$\Upsilon_\beta^{(R_0)} = \frac{\partial R_0}{\partial \beta} \times \frac{\beta}{R_0} = 1,$

$\Upsilon_\eta^{(R_0)} = \frac{\partial R_0}{\partial \eta} \times \frac{\eta}{R_0} = \frac{\mu}{\mu+\eta}$,

$\Upsilon_\psi^{(R_0)} = \frac{\partial R_0}{\partial \psi} \times \frac{\psi}{R_0} = \frac{(1-p)[\mu+\gamma+2\psi+q(\mu+\varphi)]}{\mu(p+q(1-p)-p^2)+(\eta q+\psi)(1-p)} \times \psi,$

$\Upsilon_\varphi^{(R_0)} = \frac{\partial R_0}{\partial \varphi} \times \frac{\varphi}{R_0} = \frac{q(1-p)(\mu+\varphi)-[(\mu+\gamma+\psi)p+(q(\mu+\varphi)+\psi)(1-p)]}{(\mu+\varphi)(\mu+\gamma+\psi)p+(q(\mu+\varphi)+\psi)(1-p)} \times \varphi,$

$\Upsilon_\delta^{(R_0)} = \frac{\partial R_0}{\partial \delta} \times \frac{\delta}{R_0} = \frac{\mu}{\mu+\delta},$

$\Upsilon_\gamma^{(R_0)} = \frac{\partial R_0}{\partial \gamma} \times \frac{\gamma}{R_0} = \frac{-(q(\mu+\varphi)+\psi)(1-p)\gamma}{(\mu+\gamma+\psi)[(\mu+\gamma+\psi)p+(q(\mu+\varphi)+\psi)(1-p)]},$

$\Upsilon_p^{(R_0)} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0} = \frac{[\mu+\gamma-q(\mu+\varphi)]p}{(\mu+\gamma+\psi)p+(q(\mu+\varphi)+\psi)(1-p)},$

$\Upsilon_q^{(R_0)} = \frac{\partial R_0}{\partial q} \times \frac{q}{R_0} = \frac{(\mu+\varphi)(1-p)}{(\mu+\gamma+\psi)p+(q(\mu+\varphi)+\psi)(1-p)}.$

$\Upsilon_\mu^{(R_0)} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0}$

$$= \frac{[(p+q(1-p))(\mu+\eta)(\mu+\gamma+\psi)(\mu+\varphi)(\mu+\delta)]\times\mu}{(\mu+\eta)(\mu+\gamma+\psi)(\mu+\varphi)(\mu+\delta)[(\mu+\gamma+\psi)p+(q(\mu+\varphi)+\psi)(1-p)]}$$

$$- \frac{D}{(\mu+\eta)(\mu+\gamma+\psi)(\mu+\varphi)(\mu+\delta)[(\mu+\gamma+\psi)p+(q(\mu+\varphi)+\psi)(1-p)]},$$

where, $D = [(\mu+\gamma+\psi)p+(q(\mu+\varphi)+\psi)(1-p)][(\mu+\gamma+\psi)(\mu+\varphi)(\mu+\delta)+(\mu+\eta)(\mu+\varphi)(\mu+\delta)+(\mu+\eta)(\mu+\gamma+\psi)(\mu+\delta)+(\mu+\eta)(\mu+\gamma+\psi)(\mu+\varphi)]\times\mu$

Table 1: Table of Sensitivity indices

Parameter Symbol	Sensitivity indices
β	1
ψ	0.416
q	0.394
η	0.241
δ	0.026
φ	-0.054
γ	-0.057
p	-0.063
μ	-0.464

Those parameters which have a positive indices 1 i.e. β, ψ, q, η and δ show that they have great impact on expanding the virus transmission in the community if their values are increasing as the result the basic reproduction number increases as their values increase, it means that the average number of secondary cases of infection increases in the community. Also, those parameters in which their sensitivity indices are negative i.e. φ, γ, p and μ have an influence of minimizing the burden of the disease in the community as their values increase while the others are left constant. And also, as their values increase, the basic reproduction number decreases, which leads to minimizing then endemicity of the disease in the community.

5 Numerical Simulation

In this section, numerical simulation study of model equations [1] are carried out using the software *MATLABR*2015*b* with *ODE*45 solver. To conduct the study, a set of physically meaningful values are assigned to the model parameters. These values are either taken from literature review or assumed on the basis of reality. Using the parameter values given in Table 1 and the initial conditions $P(0) = 19000, S(0) = 700, E(0) = 600, A(0) = 500, I(0) = 400, Q(0) = 280, H(0) = 8000, R(0) = 500, C(0) = 1000, D(0) = 300$ in the model equations [1] a simulation study is conducted and the results are given in the following table below.

Figure (2) shows that protected individuals decreases due to loss of protection at a rate δ and more number of protected individuals join susceptible class and converges to disease free equilibrium points. Like wise figure (3)

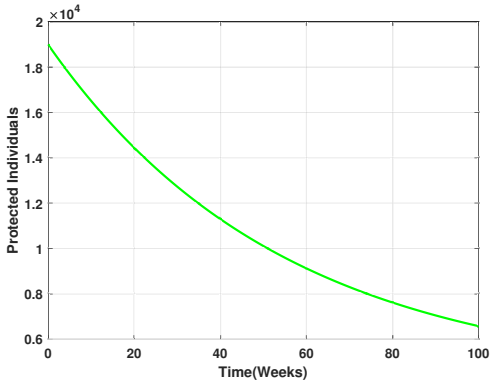

Fig. 2: Dynamics of protected individuals

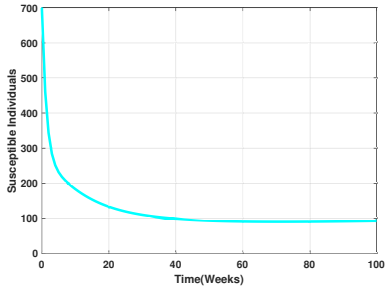

Fig. 3: Dynamics of Susceptible Individuals

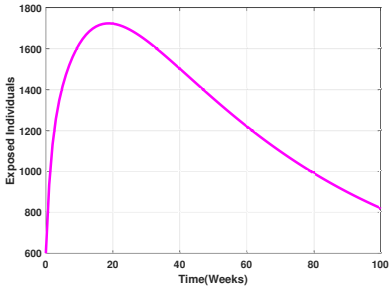

Fig. 4: Dynamics of Exposed Individuals

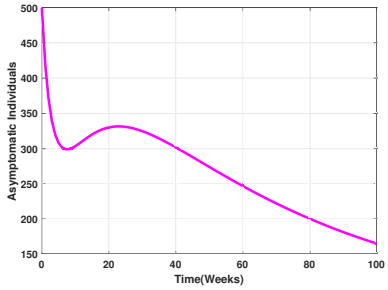

Fig. 5: Dynamics of Asymptomatic Individuals

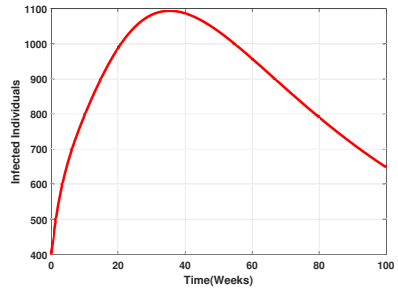

Fig. 6: Dynamics of Infected Individuals

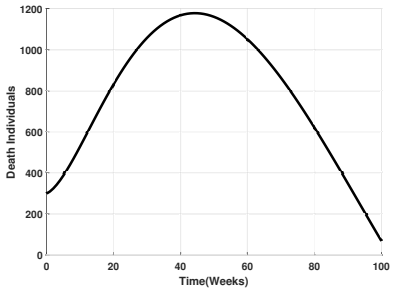

Fig. 10: Dynamics of Death Individuals

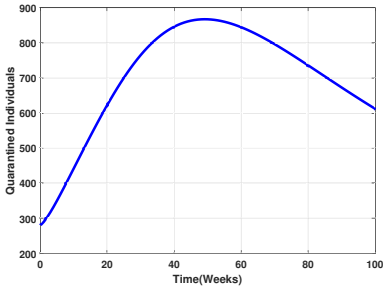

Fig. 7: Dynamics of Quarantined Individuals

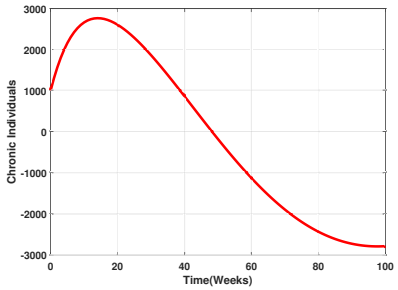

Fig. 11: Dynamics of Chronic Individuals

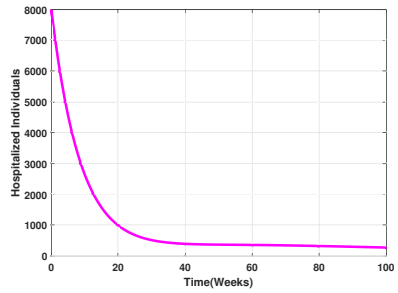

Fig. 8: Dynamics of Hospitalized Individuals

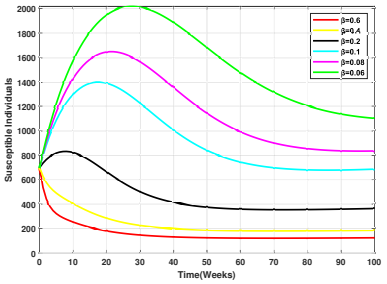

Fig. 12: Effect of varying effective contact rate on susceptible individuals

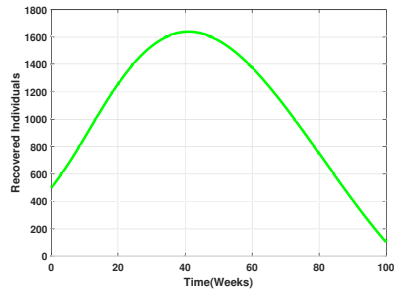

Fig. 9: Dynamics of Recovered Individuals

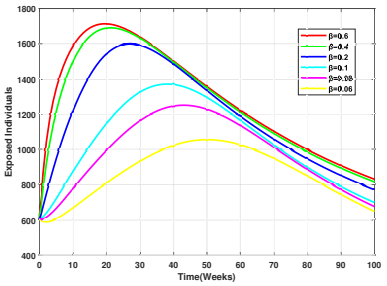

Fig. 13: Effect of varying effective contact rate on Exposed individuals

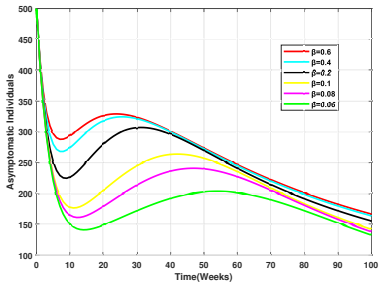

Fig. 14: Effect of varying effective contact rate on asymptomatic individuals

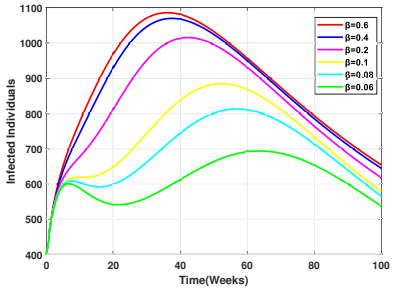

Fig. 15: Effect of varying effective contact rate on infected individuals

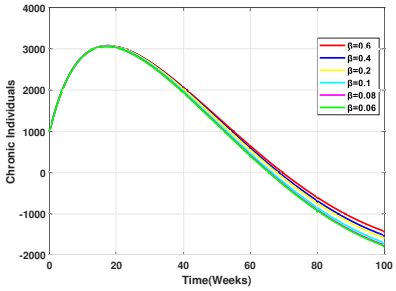

Fig. 16: Effect of varying effective contact rate on chronic individuals

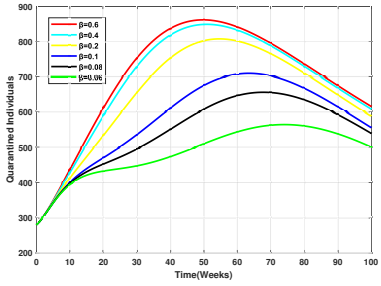

Fig. 17: Effect of varying effective contact rate on quarantined individuals

Table 2: Parameter values used in numerical simulations

Parameter	Value	Reference
π	125	Assumed
μ	0.0200	[7]
β	0.6000	Assumed
φ	0.0640	[7]
α	0.0360	Assumed
γ	0.0100	[7]
η	0.0630	Assumed
ρ	0.0150	Assumed
δ	0.0456	Assumed
ϕ	0.0500	Assumed
τ	0.0400	Assumed
ϕ	0.0500	Assumed
ψ	0.2300	Assumed
θ	0.0023	[7]
p	0.2000	Assumed
q	1.001	Assumed
ω	0.0450	Assumed

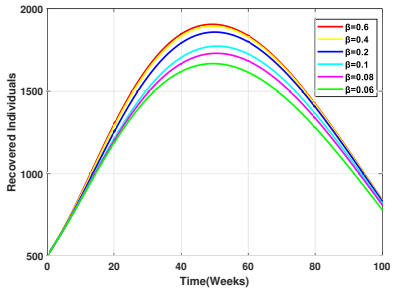

Fig. 18: Effect of varying effective contact rate on recovered individuals

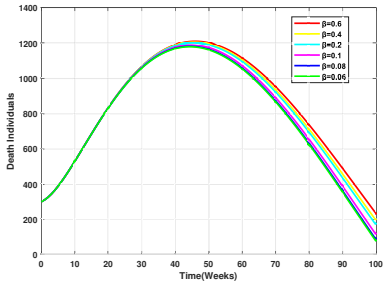

Fig. 19: Effect of varying effective contact rate on death individuals

shows that susceptible individuals decrease due to effective contact with infectious individuals and join infectious class.

Figure (4) shows that exposed individuals increase firstly as a result of some susceptible individual joins exposed

class because of effective contact with infectious individuals and decrease due to it join the asymptomatic and infected classes. Likewise figure (5) and (6) shows that asymptomatic and infected individuals decrease due to it join quarantine class. Similarly, figure (7) shows that quarantined individuals decrease due it recovers from COVID-19 and join hospital as result of the hardness of the disease in the quarantined class. Moreover figure (8) shows that hospitalized individuals decrease due to loss of immunity and recovered from COVID-19 and decrease due to it enters the intensive care unity (ICU) class as a result of the hardness of COVID-19 and figure (9) shows that recovered individuals decrease due to it loss immunity and join susceptible class. Also figure (11) shows that chronic individuals increase firstly as a consequence some hospitalized individuals joins the intensive care unity(ICU) and decrease due to it recover from COVID-19 and join recovered class and died by the disease and enters the death class and similarly figure(10) shows that death individuals increase firstly as a result of some chronic individuals dies in the ICU class and join the death class and decrease due to death. Finally figure (12), (13), (14) (15), (16), (17), (18) and (19) shows that effective contact rate has an effect on reducing COVID-19 among the community. When the contact rate is increasing the disease transmission in the community is increasing and when the contact rate is decreasing the disease transmission in the community is decreasing. That is why reducing contact remains among the reasons for reducing COVID-19.

6 Conclusion and Recommendation

This study examines a mathematical model of COVID-19 transmission dynamics. Furthermore, the existence, positivity, and boundedness of the formulated model have been shown, indicating that it is biologically meaningful and mathematically well posed. Specifically, the model's stability analyses were studied utilizing the fundamental reproduction number. In addition, the solution to the model equation is numerically enhanced, and the model's sensitivity analysis is performed to identify which parameter has the most impact on disease transmission. Numerical simulations reveal that each parameter in the model has an effect on the model variables, and when the contact rate increases, the transmission increases in the community, while when the contact rate decreases, disease transmission decreases. Although combating COVID-19 infection remains a worldwide issue, the findings of this study suggest that the government implement education programs emphasizing the necessity of voluntary and routine COVID-19 screening. There is also a need to increase the number of hospitals that deal with COVID-19 infection and screen more people who have it. Furthermore, future research should explore including optimal control against COVID-19 transmission dynamics in the model.

Data Availability

The data included in this paper are available without any restriction.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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