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A Mathematical Model of COVID-19 Infection Transmission Dynamics

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Abstract
A compartmental nonlinear deterministic epidemic model of coronavirus infection 2019 (COVID-19) transmission dynamics incorporating social distancing; face masks use and hospitalization is formulated. The disease-free equilibrium state was obtained and at this disease-free equilibrium state, the basic reproduction number was computed using the next generator matrix operator. It was shown, using linearization method and Routh-Hurwitz stability criterion, that the disease-free equilibrium state is locally asymptotically stable whenever the basic reproduction number is less than unity. The sensitivity analysis of R_0 with respect to the model parameters was carried out using the normalized forward sensitivity indices. The results of the sensitivity index of R_0 shows that the most sensitive parameter is the infection transmission probability which was also taken as the social distancing parameter. Numerical simulations show that, the use of single intervention strategy is beneficial in reducing COVID-19 disease burden. It is further shown that effective combination of social distancing, use of face masks in public and isolation (hospitalization) of infected individuals will lead to a great decrease in COVID-19 infection burden.

Keywords: Coronavirus, Social Distancing, Basic reproduction number, Routh-Hurwitz criterion, Sensitivity indices

1. Introduction

Coronavirus disease 2019 (COVID-19) recognized as pandemic on 11th March, 2020 by the World Health Organisation (WHO) was first identified December, 2019 in Wuhan, the capital of Hubei, China (Gao, 2020; WHO, 2020). The COVID-19 pandemic is considered as the biggest global threat worldwide because of thousands of confirmed infections, accompanied by thousands deaths over the world. Apart from the dramatic loss of human life worldwide, it also presents an unprecedented challenge to public health, food systems and the world of work. The economic and social disruption caused by the pandemic is devastating as tens of millions of people are at risk of falling into extreme poverty. Millions of enterprises face an existential threat. Informal economy workers were particularly vulnerable because the majority lacks social protection and access to quality health care and have lost access to productive assets. Without the means of earning an income during lockdowns, many are unable to feed themselves and their families. For most, no income means no food, or at best, less food and less nutritious food. Border closures, trade restrictions and confinement measures prevented farmers from accessing markets among other things (WHO, 2021). COVID-19 is an infectious disease caused the SARS-COV-2 virus. The virus's outer layers are covered with spike proteins that surround them like a crown. Beneath these

spikes are a layer of membrane. This membrane can be disrupted by detergents and alcohols, which is why soap and water, and alcohol hand sanitizer gels are effective against the virus. Inside the membrane is the virus' genetic material-its genome. Whereas, the genomes of some viruses like chickenpox and smallpox are made of DNA (Deoxyribonucleic Acid) like humans, those of coronaviruses are made of the closely related RNA (Ribonucleic Acid). RNA viruses have small genomes which are subject to constant change. These changes, called mutations, can cause a new variant, or strain, of the virus to form and help the virus adapt to and infect new host species (Lauren, 2020). The time from exposure of COVID-19 to the moment when symptoms begin is on average 5-6 days and can range from 1-14 days. Most people infected with the virus will experience mild to moderate respiratory illness and recover without requiring special treatment. However, some will become seriously ill and require medical attention.(Vince, 2020). The virus that causes COVID-19 spreads easily among people. When people with COVID-19 breathe out or cough, they expel tiny droplets that contains the virus. These droplets can enter the mouth or nose of someone without the virus, causing an infection to occur. The most common way that this virus spreads is through close contact (about 2 meters or 6 feet). These droplets can be inhaled by the person nearby. The disease is most contagious when a person's symptoms are at their peak. However, it is possible for someone without the symptoms to spread the virus. Studies have shown that 10% of infections are from people exhibiting no symptoms. In some situations, one can be exposed to small droplets or aerosols that stay in the air for several minutes (called airborne transmission). It can also spread if a person touches a surface or object with the virus on it and then touches his or her mouth, nose or eyes (Liang, 2020). Various mathematical models have been used to model the spread of infectious diseases (Ivorra et al, 2020). Due to the exponential spread of COVID-19 around the world, the need to develop a mathematical model to study the dynamics of the transmission and the spread of this infectious diseases became necessary. Several modeling studies have been done to analyze the spread of COVID-19 pandemic, ranging from stochastic modeling to mathematical modeling. Daniel (2020) developed a mathematical model to study the current outbreak of COVID-19 in Nigeria with nonlinear forces of infection. The model studied the impact of the environmental reservoir in the transmission and spread of this disease to humans. Numerical simulation indicated that Nigeria's cumulative number of confirmed cases would reach 55,000 individuals in December 2020 if no mitigation strategies are adopted. Okuonghae and Omame (2020) examined the impact of various non-pharmaceutical control measures (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 use numerical simulations to show the effect of control measures, specifically the common social distancing, use of face mask and case detection (via contact tracing and subsequent testing) on the dynamics of COVID-19. Iboi et al (2020) developed a mathematical model for understanding the transmission dynamics and control of COVID-19 in Nigeria. Their model, which was parameterized using COVID-19 data published in Nigeria Centre for Disease Control (NCDC), was used to assess the community-wide impact of various control and mitigation strategies in the entire Nigeria nation. From the results, they stated that, for the worst-case scenario where social

distancing, lockdown and other community transmission reduction measures are not implemented, Nigeria would have recorded a devastatingly high COVID-19 mortality by April 2021. Ivorra et al (2020) developed a mathematical model for the spread of the coronavirus disease 2019 (COVID-19) by taking into account the known unique characteristics of the diseases, as the existence of undetected infectious cases and the different sanitary and infectiousness conditions of hospitalised people. The results of different scenarios show how the different values of the percentage of detected cases would have changed the global magnitude of COVID-19 in China. Tiwari (2020) used a susceptible-infectious-quarantined-recovered model to estimate the parameters that can be used to quantify the temporal evolution of COVID-19 in India. The model has limited application according to the author because it be used at the state or district level to check the temporal evolution of pandemic. Weighted parameters was not added to the SIQR model to overcome the assumption of equal weight of infection to each one in the population. Suleiman et al (2020) employed two statistical regression models such as linear and polynomial models in order to estimate the case fatality rate (CFR) based on the early phase of COVID-19 outbreak in Nigeria. The results from the linear model estimated that the CFR was 3.11% (95% CI: 2.59-.80%) with R^2 value of 90% and p-value of < 0.0001. Cano et al (2020) used stochastic simulations to evaluate COVID-19 modelling taken into consideration the effect of social distancing. Results from their initial simulations aligned well with the updated data in UK and South Africa on how the pandemic has evolved the five months following the lockdown. They further opined that the models are, not fully accurate, but exact enough to be used as a guideline to the evolution of the disease in both high and middle income countries. Zeb et al (2020) developed a mathematical model to present the dynamical behavior of COVID-19 infection by incorporating isolation class. Their findings show that human to human contact is the potential cause of outbreaks of COVID-19. Al-Hussein and Tahir (2020) considered a generalized SEIR model to simulate the spread of COVID-19 disease and forecast the future behavior of the outbreak. From their findings, the expected cumulative number of the quarantined cases and exposed and infectious cases were presented. Okolo and Onoja (2021) developed a mathematical model for COVID-19 transmission to investigate the role of social distancing and isolation. Their numerical results show that whilst COVID-19 can spark a major epidemic in the absence, it can be controlled with optimum enforcement of rules.

This paper is organized as follows: The model is formulated in section 2. The local and global analysis of the model is explored in section 3. Sensitivity analysis of the basic reproduction number is carried out in section 4. Numerical simulations and results is presented in section 5. Discussion of results is carried out in section 6 while concluding remarks are made in section 7.

2. The Model Formulation

The model is developed by splitting the total population at a time t denoted by N(t) into mutually exclusive compartment of susceptible individuals $S(t)$, exposed individuals $E(t)$, infectious individuals $I(t)$, recovered individuals $R(t)$ and hospitalized individuals H(t), so that:

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

The variables and parameters used in the model are defined in Table 1 and Table 2 respectively.

Table 1. Description of model variables.

Variables	Description
$S(t)$	The number of susceptible humans at time t
$E(t)$	The number of exposed (latent) humans at time t
$I(t)$	The number of infectious humans at time t
$R(t)$	The number of recovered humans at time t
$H(t)$	The number of hospitalized humans at time t

Table 2. Description of model parameters.

Parameter	Description
β	Effective community contact rate (a measure of social distancing effectiveness).
ε	Masks Compliance.
γ	Progression rate from latent to infectious class.
δ	Hospitalization rate for infectious individuals.
α	Efficacy of face mask to prevent the acquisition of infective compartment.
$\varphi_1 \varphi_2$	The recovery rate for individuals in Infective and Hospitalization Compartment.
κ_1 κ_2	The disease induced mortality rate.

From the above definition of variables (Table 1) and parameters (Table 2), the interactions and flow in the different compartments are as depicted in the schematic flow diagram (Figure 1).

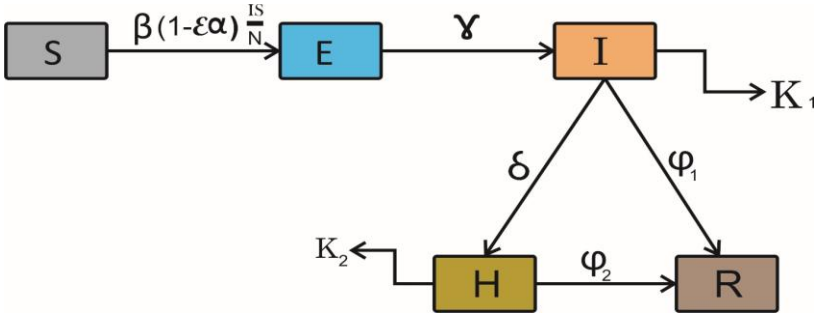

Figure 1: schematic diagram of the model

2.1. The Model Equations

The above assumptions and derivatives lead to the following system of ordinary differential equations:

$$\frac{ds}{dt} = -\beta(1-\varepsilon\alpha)\frac{I}{N}s \quad (2)$$

$$\frac{dE}{dt} = \beta(1-\varepsilon\alpha)\frac{I}{N}s - \gamma E \quad (3)$$

$$\frac{dI}{dt} = \gamma E - (\delta + K_1 + \varphi_1)I \quad (4)$$

$$\frac{dH}{dt} = \delta I - (K_2 + \varphi_2)H \quad (5)$$

$$\frac{dR}{dt} = \varphi_1 I + \varphi_2 H \quad (6)$$

3. Model Analysis

3.1 Basic Properties of the model equations

The existence, the positivity and the boundedness of the solution of equations (2) – (6) need to be proved to ensure that the model has a mathematical and biological meaning. All model variables and parameters are assumed to be non-negative for $t \geq 0$ since the model monitor changes in the population.

Equations (2) – (6) can be written as follows:

$$dY = P(Y(t)) \quad (7)$$

where

$$Y(t) = \begin{pmatrix} S(t) \\ E(t) \\ I(t) \\ H(t) \\ R(t) \end{pmatrix} \tag{8}$$

And P is a C^1 function mapping $\mathbb{R}^5$ into itself, define by

$$P(Y) = \begin{pmatrix} P_1(S,E,I,H,R) \\ P_2(S,E,I,H,R) \\ P_3(S,E,I,H,R) \\ P_4(S,E,I,H,R) \\ P_5(S,E,I,H,R) \end{pmatrix} = \begin{pmatrix} -\beta(1-\varepsilon\alpha)\frac{I}{N}s \\ \beta(1-\varepsilon\alpha)\frac{I}{N}s \; - \gamma E \\ \gamma E - (\delta + K_1 + \varphi_1)I \\ \delta I - (K_2 + \varphi_2)H \\ \varphi_1 I + \varphi_2 H \end{pmatrix} \tag{9}$$

By the fundamental theory of functional differential equations, equations (2) – (6) has a unique solution $(S(t), E(t), I(t), H(t), R(t))$ with respect to the initial data Y_0 such that
$S(t) \geq 0, E(t) \geq 0, I(t) \geq 0, H(t) \geq 0, R(t) \geq 0$
If we put $S + E + I + H + R \leq N$, then the following theorem hold.

Theorem 1
The following biological feasible region of equations (2) – (6) $B = (S, E, I, H, R) \in \mathbb{R}^5_+; (S + E + I + H + R \leq N)$ is positively invariant and attracting.

Theorem 2
Let $t_0 > 0$ and the initial conditions satisfy
$S(t_0) \geq 0, E(t_0) \geq 0, I(t_0) \geq 0, H(t_0) \geq 0, R(t_0) \geq 0$, then the solution
$S(t), E(t), I(t), H(t), R(t)$ of equations (2) – (6) are positive for all $t \geq 0$.

Proof
From equation (2), we have

$$S(t) = S(0)e^{\frac{-\beta(1-\varepsilon\alpha)}{N}\int_0^t I(u)du} \tag{10}$$

Hence $S(t)$ is nonnegative for all $t \geq 0$.
From equations (3) – (5), we set

$$E(t) = \left[E(0) + \frac{-\beta(1-\varepsilon\alpha)}{N}\int_0^t e^{\gamma u}S(u)I(u)du\right]e^{-\gamma t} \tag{11}$$

$$I(t) = \left[I(0) + \gamma\int_0^t e^{u(\delta + K_1 + \varphi_1)}E(u)du\right]e^{-(\delta + K_1 + \varphi_1)t} \tag{12}$$

$$H(t) = \left[H(0) + \delta\int_0^t e^{u(K_2 + \varphi_2)}I(u)\right]e^{-(K_2 + \varphi_2)t} \tag{13}$$

Therefore, $E(t), I(t)$ and $H(t)$ are all nonnegative for all $t \geq 0$.
From equation (6) we can easily deduce the positivity of $R(t)$ for all $t \geq 0$.

Hence the positivity of solution has been proved,

3.3: The Model Equations in terms of Proportion

For prevalence of the disease, it is necessary to rewrite the model in terms of proportion of the susceptible, exposed, infected, hospitalized and recovered compartments.

Adding equations (2) – (6) gives the rate of change of the total population as

$$\frac{\mathrm{dN}}{\mathrm{dt}} = -\mathrm{K}_1\mathrm{I} - K_2\mathrm{H} \tag{14}$$

The proportion for each class are defined as follows

$\mathrm{s} = \frac{\mathrm{S}}{N}$, $\mathrm{e} = \frac{\mathrm{E}}{N}$, $i = \frac{\mathrm{I}}{N}$, $\mathrm{h} = \frac{H}{N}$, $\mathrm{r} = \frac{R}{N}$

so that $\mathrm{s} + \mathrm{e} + i + \mathrm{h} + \mathrm{r} = 1$ for all $\mathrm{t} \geq 0$

$$\mathrm{s} = 1 - (\mathrm{e} + i + \mathrm{h} + \mathrm{r}) \tag{15}$$

Hence, the system (2) – (6) expressed in terms of proportion is given below

$$\frac{ds}{dt} = -\beta(1 - \varepsilon\alpha)si \tag{16}$$

$$\frac{de}{dt} = \beta(1 - \varepsilon\alpha)si - \gamma e \tag{17}$$

$$\frac{di}{dt} = \gamma e - (\delta + K_1 + \varphi_1)i \tag{18}$$

$$\frac{dh}{dt} = \delta i - (K_2 + \varphi_2)h \tag{19}$$

$$\frac{dr}{dt} = \varphi_1 i + \varphi_2 h \tag{20}$$

3.4: Equilibrium state of the model

The model given by the equations above has a unique Disease Free Equilibrium DFE (in the absence of infection) it is obtained by setting the right hand side of the equations to zero and solving the nonlinear systems.

Note at the disease free equilibrium,

$\frac{\mathrm{ds}}{\mathrm{dt}} = \frac{\mathrm{de}}{\mathrm{dt}} = \frac{\mathrm{d}i}{\mathrm{dt}} = \frac{\mathrm{dh}}{\mathrm{dt}} = \frac{\mathrm{dr}}{\mathrm{dt}} = 0$. It is enough to consider the first four equations, equating them to zero.

$$\frac{\mathrm{ds}}{\mathrm{dt}} = -\beta(1 - \varepsilon\alpha)si = 0$$

$\frac{\mathrm{de}}{\mathrm{dt}} = \beta(1-\varepsilon\alpha)si\ -\gamma e\ = 0$

$\frac{\mathrm{di}}{\mathrm{dt}} = \gamma e - (\ \delta +\ K_1 +\ \varphi_1)i = 0$

$\frac{\mathrm{dh}}{\mathrm{dt}} = \delta i - (K_2 +\ \varphi_2)h\ = 0$

Hence,

$-\beta(1-\varepsilon\alpha)si = 0$ (21)

$\beta(1-\varepsilon\alpha)si\ -\gamma e\ = 0$ (22)

$\gamma e - (\ \delta +\ K_1 +\ \varphi_1)i = 0$ (23)

$\delta i - (K_2 +\ \varphi_2)h = 0$ (24)

From equation (23)

e = $\frac{((\ \delta + K_1 + \varphi_1)i)}{\gamma}$

Also, from equation (24), we have

h = $\frac{\delta i}{(K_2 + \varphi_2)}$

Substituting the value of e in equation (22), we have that

$\beta(1-\varepsilon\alpha)si\ \ -\ \gamma\frac{((\ \delta + K_1 + \varphi_1)i)}{\gamma} = 0$

$\Longrightarrow [\beta(1-\varepsilon\alpha)\mathrm{s} -\ (\ \delta +\ K_1 +\ \varphi_1)]\ i\ = 0$

Therefore i = 0 or $\beta(1-\varepsilon\alpha)\mathrm{s} -\ (\ \delta +\ K_1 +\ \varphi_1) = 0$

Hence i = 0 or s = $\frac{(\delta + K_1 + \varphi_1)}{\beta(1-\varepsilon\alpha)}$

For i = 0, substituting the value of i in e and h, we see that e = 0 and h =0 also r = 0

But from equation (15)

s = 1- (e + i + h + r),

hence

s = 1

Thus, the DFE is given by

$\mathcal{E}_0$ = (s, e, i, h, r) = (1, 0, 0, 0, 0) (25)

3.5: Basic Reproduction Number R_0

We will study the local stability of the disease free equilibrium state, $\mathcal{E}_0$, exploring the basic reproduction number R_0. The basic reproduction, R_0, measures the average number of new infection generated by a single infected individual in a completely susceptible population. For the recipe on computation of basic reproduction number using the next generation operator method, see Van den Driessche and Watmough (2002), Castillo-Chavez *et al* (2002), Diekmann *et al* (1990) and Heffernan (2005). Let the non-negative matrix, F, of new infection terms and the M-matrix, V, of transfer terms associated with the model (16) – (20) are given respectively, by

$$F = \begin{pmatrix} 0 & \beta(1-\varepsilon\alpha)s \\ 0 & 0 \end{pmatrix} \quad (26)$$

and

$$V = \begin{pmatrix} \gamma & 0 \\ -\gamma & \delta + K_1 + \varphi_1 \end{pmatrix} \quad (27)$$

Now

$$V^{-1} = \frac{1}{\gamma(\delta+K_1+\varphi_1)} \begin{pmatrix} \delta + K_1 + \varphi_1 & 0 \\ \gamma & \gamma \end{pmatrix} = \begin{pmatrix} \frac{1}{\gamma} & 0 \\ \frac{1}{\delta+K_1+\varphi_1} & \frac{1}{\delta+K_1+\varphi_1} \end{pmatrix} \quad (28)$$

So that

$$FV^{-1} = \begin{pmatrix} \frac{\beta(1-\varepsilon\alpha)}{\delta+K_1+\varphi_1} & \frac{\beta(1-\varepsilon\alpha)}{\delta+K_1+\varphi_1} \\ 0 & 0 \end{pmatrix} \quad (29)$$

The dominant eigenvalue of $FV^{-1} = \begin{pmatrix} \frac{\beta(1-\varepsilon\alpha)}{\delta+K_1+\varphi_1} & \frac{\beta(1-\varepsilon\alpha)}{\delta+K_1+\varphi_1} \\ 0 & 0 \end{pmatrix}$ is given by

$$\lambda = \frac{\beta(1-\varepsilon\alpha)}{\delta+K_1+\varphi_1} \quad (30)$$

and therefore, the basic reproduction number of the system (1) – (5), denoted by R_0, is given by

$$R_0 = \frac{\beta(1-\varepsilon\alpha)}{\delta+K_1+\varphi_1} \quad (31)$$

3.6: Local Stability of Disease Free Equilibrium (DFE) State

We investigate the local stability of the disease free (DFE) state by evaluating the associated Jacobian of equations (1) – (5) at the DFE state. The Jacobian matrix J for the system (1) – (5), evaluated at the disease-free equilibrium, $\mathbf{E_0}$ is given by

$$J = \begin{bmatrix} 0 & 0 & -\beta(1-\varepsilon\alpha) & 0 & 0 \\ 0 & \gamma & \beta(1-\varepsilon\alpha) & 0 & 0 \\ 0 & -\gamma & -(\delta+\kappa_1+\varphi_1) & 0 & 0 \\ 0 & 0 & \delta & -(\kappa_2+\varphi_2) & 0 \\ 0 & 0 & \varphi_1 & \varphi_2 & 0 \end{bmatrix} \quad (32)$$

Theorem 3: The DFEs of the model (1) – (5), given by $\mathbf{E_0}$, is locally asymptotically stable (LAS) if $R_0 < 1$ and $\mathbf{E_0}$ is unstable if $R_0 > 1$.

Proof

It suffices to show that all the eigenvalues of the characteristic equation of the Jacobian matrix $J_1(\mathbf{E_0})$, have negative real parts.

The eigenvalues are given by

$(0-\lambda)[0-\lambda][-((K_2+\varphi_2)] - \lambda][(-\gamma-\lambda)-(\delta+K_1+\varphi_1)-\lambda] - \gamma\beta(1-\varepsilon\alpha)] = 0$

$(0-\lambda)[0-\lambda][\gamma(\delta+K_1+\varphi_1)+\gamma\lambda+(\delta+K_1+\varphi_1)\lambda+\lambda^2] - \gamma\beta(1-\varepsilon\alpha)] = 0$

$(0-\lambda)[0-\lambda][\lambda^2+(\gamma+(\delta+K_1+\varphi_1)]\lambda+\gamma(\delta+K_1+\varphi_1) - \gamma\beta(1-\varepsilon\alpha)] = 0$

Here

$\lambda = 0$ twice, $\lambda = -(K_2+\varphi_2)$ (33)

and the root of the polynomial

$P(\lambda) = \lambda^2 + A\lambda + B$ (34)

Where the coefficients are given by

$A = (\gamma+(\delta+K_1+\varphi_1) > 0$

$B = \gamma(\delta+K_1+\varphi_1) - \gamma\beta(1-\varepsilon\alpha)$

$= \gamma(\delta+K_1+\varphi_1)[1-\frac{\beta(1-\varepsilon\alpha)}{\delta+K_1+\varphi_1}]$

$= \gamma(\delta+K_1+\varphi_1)[1-Ro]$ (35)

For $R_0 < 1$ we have $A > 0$ and $B > 0$ and thus following Routh-Hurwitz stability criterion (Hurwitz, 1964) for the polynomial $P(\lambda)$, the state $\mathcal{E}_0$ is locally asymptotically stable whenever $R_0 < 1$.

4. Sensitivity Analysis of R_0 With Respect to the Control Parameters

Sensitive analysis is commonly used to determine the robustness of model predictions to parameter values. It is used to discover parameters that have high impact on the threshold R_0, and should be targeted by intervention strategies. More accurately, sensitivity indices' allow us to measure the relative changes in a variable when a parameter changes. We carried out sensitivity analysis on the basis of the control parameters, $\alpha, \delta, \varphi_1, \varepsilon, \kappa_1$ and β, by the normalized forward sensitivity indices (Chitnus etal, 2006; Wu et al, 2013) using the following formula:

$$\Lambda_{\nu}^{R_0} = \left(\frac{\partial R_0}{\partial \nu}\right)\left(\frac{\nu}{R_0}\right)$$

where ν denotes the model parameter. The sensitivity index of R_0, with respect to each model parameter is given in Table 3.

Table 3: Sensitivity indices of R_0

Parameter	Sensitivity indices
β	1
ε	-0.0475
α	-0.0475
δ	-0.1391
K_1	-0.0834
φ_1	-0.7775

5. Numerical Simulations

Various numerical simulations are carried out to illustrate the theoretical results in this paper. The parameter values for the simulation, unless otherwise stated is given in Table 4.

Table 4: Base line parameter values for the equations (16) – (20)

Parameter	Values	Source
β	0.4383 days-1	Iboi et al (2020)

$\boldsymbol{\varepsilon}$	0.1days-1	Ngonghala et al (2020)
$\boldsymbol{\alpha}$	0.5days-1	Ngonghala et al (2020)
$\boldsymbol{\gamma}$	$\frac{\mathbf{1}}{\mathbf{5.2}}$ days-1	Rothana and Byraraddy (2020)
$\varphi_1\varphi_2$	0.13978 days-1	Tang et al (2020)
$\boldsymbol{\delta}$	0.025 days-1	Furguson et al (2020)
$K_1(K_2)$	0.015,0.103 days-1	Furguson et al (2020)

The numerical results are shown in Figure 2 – Figure 6

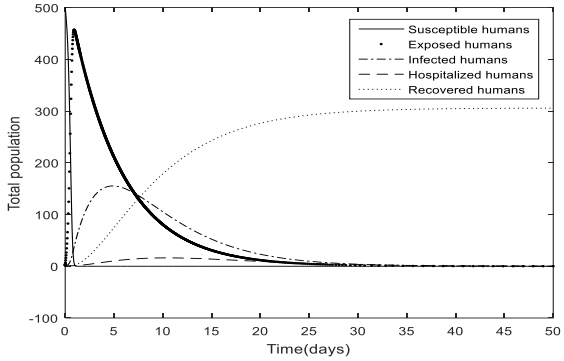

Figure 2. The proportion of Susceptible, Exposed, Infected, Hospitalized and Recovered Human as a function of time.

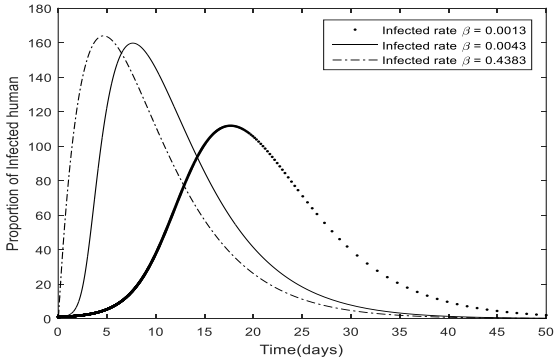

Figure 3: Proportion of infected humans as a function of time varying infection transmission rate$\beta = 0.0013, \mathrm{R}_0 = 0.0083990$; for $\beta = 0.0043$, $\mathrm{R}_0 = 2.8317$; $\beta = 0.4383$, $\mathrm{R}_0 = 2.8317$. All other parameters used are as given in Table 4.

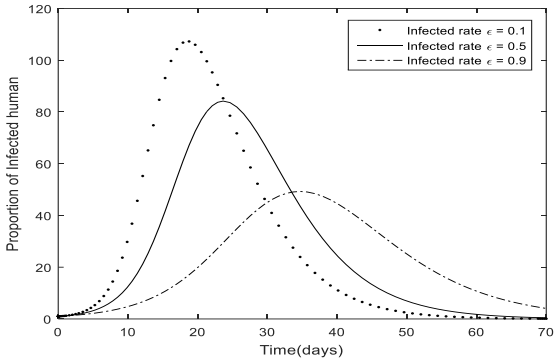

Figure 4: Effect of face mask (mask compliance) without isolation of infected humans. R_0= 0.00798 for ε = 0.1, R_0 = 0.00630 for ε = 0.5 and R_0= 0.00462 ε =0.9

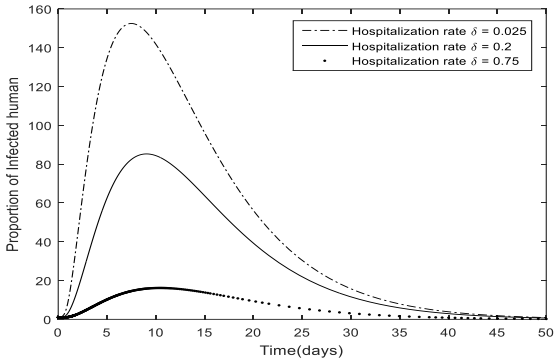

Figure 5: Effect of Hospitalization of infected humans. R_0 = 2.3167 for δ = 0.025, R_0 = 1.17380 for δ = 0.2 and R_0 = 0.46023 for δ = 0.75

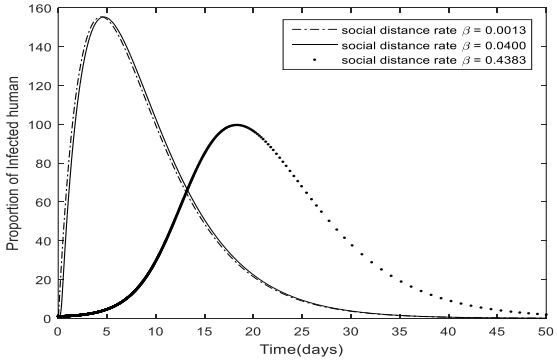

Figure 6: Effect of social distancing. R_0 =5.2773 for β = 0.0013, R_0 = 5.2630 for β =0.0400 and R_0 = 2.96815 for β = 0.4383

6. Discussion of Results

This section is broadly divided into two parts, namely, the qualitative analysis and the numerical analysis. The qualitative analysis of the proposed model shows that the solution of the model exists, is bounded and positively invariant. We computed the basic reproduction number, R_0, using the next generation method given by $R_0 = \frac{\beta(1-\varepsilon\alpha)}{\delta + K_1 + \varphi_1}$ as a threshold in the of COVID-19 infection both for predicting its outbreak and for evaluating its control strategies. The result from the stability analysis of the disease-free equilibrium (DFE) state is shown to be locally asymptotically stable whenever R_0, is less than unity. The implication of this result is that the COVID-19 pandemic can be controlled if the initial sizes of the infected component of the populations of the model are in the basin of attraction of the DFE. The sensitivity analysis of R_0 with respect to the model parameters was carried out using the normalized forward sensitivity indices. Sensitivity analysis is commonly used to determine the robustness of model predictions to parameter values. It is used to discover parameter that have high impact on the threshold R_0, and should be targeted by intervention strategies. The results of the sensitivity index of R_0 is given in Table 3 and it shows that the most sensitive parameter is the infection transmission probability, β, which was also taken as the social distancing parameter. It is followed by the recovery rate φ_1, hospitalization δ, disease induced mortality rate K_1, face mask use and compliance ε, α . The parameter, β has positive index, 1 as shown in Table 3 reveals that by decreasing or increasing infection parameter will decrease or increase the basic reproduction number, R_0. Thus effort should be geared towards decreasing the community infection transmission by observing strict adherence to social distancing.

The parameter with negative sensitivity indices, $-0.7775; -0.1391; -0.0834; -0.0475$ as shown in Table 3 have influence of reducing the disease burden in the population as their values increases. Various numerical simulations of the model are carried out to assess the effectiveness of various control strategies using the baseline parameter values in Table 4 and depicted in Figure 2 – 6. The simulation in Figure 2 shows the solution for the proportion of population of Susceptible, exposed, infected, hospitalized and recovered individuals. It further reveals that as the infected individuals decreased as a result of hospitalization, more people are recovering. As more and more people recover by the day, the infection can be brought under control. Figure 3 shows increasing prevalence of Covid-19 infection for increasing infection transmission rates (β=0.0013, 0.004, 0.4383) in the absence of face mask compliance and hospitalization of infected humans. Hence, for an effective preventive strategy, effort should be geared at reducing the infection transmission rate. Thus the infection transmission rate plays a significant role as a preventive strategy. In Figure 3, we vary the use of face mask without hospitalization α(0.03, 0.3, 0.8). We discover that by increasing the use of facemask, it reduces the number of infected individuals in the population, with basic reproductive number $R_0 < 1$ in each case, shows convergence of the solution profile to the disease free equilibrium (DFE). In Figure 4, the effect of using face mask in public is depicted using the baseline parameters in Table 4 at various levels of face masks compliance and efficacy

$\varepsilon(0.1, 0.5. 0.9)$. The figure also show a decrease in the proportion of infected individuals with increasing face mask efficacy and compliance. The effect of hospitalization of infected individuals on the dynamics of COVID-19 is simulated in Figure 5 using the baseline parameters in Table 4 by varying the control parameter values, $\delta(0.025, 0.2. 0.75)$. The Figure shows a decrease in the basic reproduction number with increasing hospitalization (home isolation and hospital isolation). Thus hospitalization of infected humans overall can reduce the risk of future COVID-19 spread. Figure 6 also shows the simulation for various effectiveness of social distancing control measures, $\beta(0.4383, 0.0013, 0.004)$ using the baseline parameters in Table 4. It further reveals that increasing the social distance measure can significantly decreases cases of COVID-19 infection.

7. Conclusion

A compartmental nonlinear deterministic epidemic model for COVID-19 transmission dynamics is formulated. This model incorporated non-pharmaceutical measures such as social distancing; face masks use and hospitalization as control strategies. The qualitative analysis of the proposed model shows that the solution of the model exists, is bounded and positively invariant. Local stability analysis for the disease free equilibrium state is given and $R_0 < 1$, ensures the local asymptotical stability of the infection-free equilibrium. The sensitivity analysis of R_0 with respect to the model parameters was carried out using the normalized forward sensitivity indices. The results of the sensitivity index of R_0 is given in Table 3 and it shows that the most sensitive parameter is the infection transmission probability, β, which was also taken as the social distancing parameter. Various numerical simulations of the model are carried out to assess the effectiveness of various control strategies using the baseline parameter values in Table 4 and depicted in Figure 2 – Figure 6. Numerical simulations of the model show that the infection transmission rate constitutes an essential key to preventive strategies. Further simulations shows that the social distancing should be complemented with the use of face masks in the public and deliberate effort at hospitalization (home or hospital isolation) to achieve realistic and effective control of COVID-19 spread.

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