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Mathematical Modeling of the Transmission Dynamics of Covid-19 with Quarantine and Hospitality Treatment

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Abstract: ***This study presents a COVID-19 epidemic disease model that has been tailored to fit the specific circumstances of world. The Nigerian population has been partitioned into seven subpopulations in this model system. These subpopulations include the Susceptible class, Exposed class, Symptomatic Infected class, Asymptomatically Infected class, Quarantined individuals, Hospitalised individuals, and Recovered individuals. The model was augmented with control measures parameters, specifically those related to hospitalisation and quarantine. The disease-free equilibrium and endemic equilibrium points were derived. The determination of the basic reproduction number was achieved through the utilisation of the novel generation matrix. Additionally, an analysis of the local and global stability was conducted, revealing that the system is both locally and globally asymptotically stable at the aforementioned point of R_0<1 for the DFE. We did numerical simulations using (Maple 17) software. The results showed the importance of the control measures and social distancing through graph.***
Keyword: Mathematical Modeling, Transmission Dynamics, COVID-19, Quarantine and Hospitality

I. INTRODUCTION

A novel coronavirus known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2; formerly 2019-nCoV), which was first discovered during an outbreak of respiratory illness cases in Wuhan City, Hubei Province, China, is known as coronavirus disease 2019 (COVID-19). Since December 2019, there have been a large number of unexplained cases of pneumonia in Wuhan, China, with cough, dyspnoea, fatigue, and fever as the primary symptoms (Adedayo *et al.* 2022).

Infection with the new coronavirus pandemic, COVID-19, is characterised by respiratory symptoms, fever, coughing, shortness of breath, and dyspnoea. The new coronavirus SARS-CoV-2 is a novel strain of coronaviruses that have not yet been discovered in humans. In more severe cases, this illness may result in mortality, renal failure, severe acute respiratory syndrome, pneumonia, and other complications (WHO, 2019).

Chinese health officials in Wuhan City reported the first incidence of the new coronavirus severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in December 2019. The Middle East respiratory syndrome and severe acute respiratory syndrome are two diseases that can be brought on by the coronavirus family of viruses (Mayo, 2020). The COVID-19 infection has a wide clinical spectrum, ranging from minimal symptoms to severe pneumonia. In one study, 40–50% of COVID-19 patients did not exhibit any symptoms (Verity *et al.*, 2020). Other patients experienced fever, body aches, nausea, or diarrhoea typically 2 to 14 days after virus exposure (CDC, 2020). Only 14% of all infections during COVID-19's initial phase in China (10–23 January 2020) were confirmed. On February 6, 2020, there were a total of 31,161 confirmed cases, including 636 fatalities, on the Chinese mainland; 22,112 confirmed cases, including 618 fatalities; and 11,618 confirmed cases, including 478 fatalities; in Hubei province. Numerous interventions and the spread of COVID-19 have had a tremendously detrimental effect on people's daily lives and societal norms. Different levels of closures and traffic restrictions have been implemented in cities throughout China's Hubei Province (Chan *et al.*2020).

In China, Europe, America, and Africa, the coronavirus disease 2019 (COVID-19) has caused high morbidity and mortality rates, resulting in unprecedented public health crises around the globe. The World Health Organisation (WHO) classified COVID-19 a global pandemic on March 11, 2020.

A new coronavirus known as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the cause of COVID-19. After SARS-CoV in 2002 and the Middle East respiratory syndrome coronavirus (MERSCoV) in 2012, SARS-CoV-2 is regarded as the third zoonotic human coronavirus to emerge in the twenty-first century.

Nigeria, a West African nation of around 207 million people, reported its first COVID-19 case on February 27, 2020 (NCDC, 2020), and as of March 30, 2020 (22:00 WAT), 131 people had contracted the virus. Eight recoveries in all and two fatalities. The state of Lagos in Nigeria has the most infected residents, followed by Abuja, the nation's capital. Currently, the country's major cities are under lockdown (Mbah F, 2020), and entry flights from nations with more than 1000 cases are prohibited (Mbah F, 2020).

In actuality, there are a lot of pressing concerns regarding COVID-19's proliferation. How many people will contract the illness tomorrow?

When will the infection rate reach its turning point? How many people will contract the disease during the busiest time? Can the COVID-19 be adequately controlled by current interventions? What mathematical tools are at our disposal to help us resolve these issues? Since the COVID-19 is a brand-new coronavirus that was only identified in December 2019, there is still a lack of information about the outbreak, and medical interventions like clinical trials are still in a challenging exploratory phase (Chan *et al.*, 2020).

There are still issues to be resolved regarding the effectiveness of the current emergency response, how to allocate medical resources more scientifically in the future, and other issues because it has been challenging to directly apply epidemic data to mathematical models that are already in use. One of the infectious viruses in the globe is COVID-19. The World Health Organisation (WHO) estimates that there are at least 26 million COVID-19 infections worldwide and at least one million deaths, 30% of which occur in Africa, are recorded every few months. Understanding how a virus spreads, how to prevent it from happening, and how to forecast when an outbreak will occur are all crucial. In developing nations, the spread of several vector-borne viruses has also increased due to population expansion. The majority of COVID-19 mathematical modelling took asymptomatic infected and interacting peoples into account when predicting how the virus would spread, however in this research, tracing and partial Recovery will be taken into account.

II. MODEL FORMATION

in this research paper, we shall study the transmission mechanism of COVID-19 using a deterministic compartmental model. In order to formulate the model mathematically, In this model, the total of human population was denoted in $N(t)$ and divided into seven classes: susceptible individuals $S(t)$, exposed individuals $E(t)$, asymptotically infected individuals $A(t)$, symptomatic infected individuals $I(t)$, quarantined individuals $Q(t)$, hospitality individual $H(t)$ and individuals that have recovered/remove from COVID-19 $R(t)$. Therefore, the total population was stated in $N(t) = S(t) + E(t) + I(t) + A(t) + Q(t) + H(t) + R(t)$. Recruitment rate of natural human natality and mortality is given the parameter π and μ sequentially. Susceptible individuals (S) and symptomatic infected individuals (I) in respective of β each states the contact rate between susceptible individuals (S) and individual groups of I. The parameter θ and σ in respective order is the proportion of asymptotically infected individuals and quarantined exposed individuals, while parameter σ states movement rate of exposed individuals to the quarantined individuals. Parameter ϕ and θ each represent transmission rate after incubation period and status change to I and A class. Quarantined individuals can be transferred to the class of infected individuals with symptoms at the rate of φ. Parameter υ states movement rate of quarantined to the hospitality individuals Parameter τ, α, ρ each states the recovery rate of infected individuals without symptoms, hospitality individuals, and infected individuals with symptoms to be transferred to recovered individuals class I. Further, death rate of COVID-19 in I, H class is represented in δ.

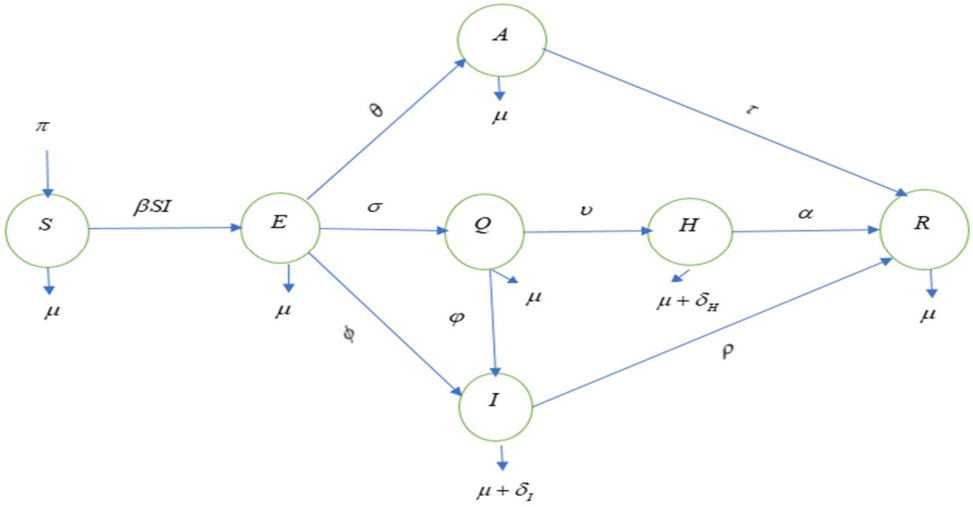

Figure 1: Schematic Diagram of the Model

Table 1: Definition of Model Variables and Parameters

Variables	Definition
(S)	The population of susceptible individuals at a given time (t)
(E)	The population of Exposed individuals at a given time (t)
(A)	The population of infectious asymptomatic individuals at a given time (t)
(I)	The population of infectious symptomatic individuals at a given time (t)
(Q)	The population of quarantined individuals at a given time (t)
(H)	The population of hospitality individuals at a given time (t)
(R)	The population of recovered individuals at a given time (t)

Parameters	Definition
π	Recruitment rate of individuals into the population (Birth and immigration)
β	Rate of contact between susceptible individuals and symptomatic infected individuals
μ	Natural mortality rate
δ_I	Mortality rate due to infection
δ_H	Mortality rate due to hospitality
α	Recovery rate of hospitality individuals and transferred to recovery class
τ	Recovery rate of asymptomatically infected individuals and transferred to recovery class
ρ	Recovery rate of symptomatic infected individuals and transferred to recovery class
υ	Hospitality rate of quarantined individuals and transferred to hospitality class

σ	Movement rate of exposed individuals to quarantined individuals
φ	Movement rate of quarantined individuals to symptomatic infected individuals
ϕ	Transmission rate after incubation period and transferred to symptomatic infected class
θ	Transmission rate after incubation period and transferred to asymptomatic infected class

A. Model Equations

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \beta SI - \mu S \\
\frac{dE}{dt} &= \beta SI - (\theta + \sigma + \phi + \mu)E \\
\frac{dI}{dt} &= \phi E + \gamma Q - (\mu + \rho + \delta_I)I \\
\frac{dA}{dt} &= \theta E - (\tau + \mu)A \\
\frac{dQ}{dt} &= \sigma E - (\gamma + \upsilon + \mu)Q \\
\frac{dH}{dt} &= \upsilon Q - (\alpha + \delta_H + \mu)H \\
\frac{dR}{dt} &= \rho I + \tau A + \alpha H - \mu R
\end{aligned}\right\} \quad (1)$$

III. MODEL ANALYSIS

We provide comprehensive qualitative analysis of the model equation in this section.

A. The Positive Invariant Region

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dA}{dt} + \frac{dQ}{dt} + \frac{dH}{dt} + \frac{dR}{dt} \quad (2)$$

$$\frac{dN}{dt} = \pi - \mu(S + E + I + A + Q + H + R) - (\delta_I I + \delta_H H) \quad (3)$$

The positive invariant region can be obtained by using the following theorem as applied by (Adedayo *et al.*, 2022).

Theorem 1:

The solutions of the system (1) are feasible for $t > 0$ if they enter the invariant region D.

Proof:

Let $D = (S, E, I, A, Q, H, R) \in \Re_+^7$ be any solution of the system (1) with non-zero initial conditions.

Assuming there are no disease-induced deaths, equation (4) now becomes; $\frac{dN}{dt} \le \Lambda - \mu(S + E + I + A + Q + H + R)$ (4)

But $N(t) = S + E + I + A + Q + H + R$ in equation (1)

$$\frac{dN}{dt} \le \pi - \mu N(t) \quad (5)$$

Applying the initial conditions $t = 0, N(0) = N_0$

$$N(t)e^{\mu t} \leq \frac{\pi}{\mu}e^{\mu t} + c \quad (6)$$

$$= N_0 e^{\mu(0)} \leq \frac{\pi}{\mu}e^{\mu(0)} + c \quad (7)$$

$$N_0 \leq \frac{\pi}{\mu} + c \Rightarrow N_0 - \frac{\pi}{\mu} \leq c \quad (8)$$

$$N - \frac{\pi}{\mu} \leq (N_0 - \frac{\pi}{\mu})e^{-\mu t} \quad (9)$$

$$N(t) \leq \frac{\pi}{\mu} + (N_0 - \frac{\pi}{\mu})e^{-\mu t} \quad (10)$$

At $t \to \infty$ in equation (10) the human population $N(t)$ approaches $\kappa = \frac{\pi}{\mu}$ (that is $N \to k = \frac{\pi}{\mu}$) the parameter $\kappa = \frac{\pi}{\mu}$ is called the carrying capacity.

Therefore, all feasible solution of the human population of the Model is in the region

$$\Omega = \{(S,E,I,A,Q,H,R) \in \Re^7 : S \succ 0, E \succ 0, I \succ 0, A \succ 0, Q \succ 0, H \succ 0, R \succ 0, N \leq \frac{\Lambda}{\mu}\} \quad (11)$$

B. Positivity of the Solutions

Theorem 2:

Let the initial data be $\{S(0) > 0, (E(0), I(0), A(0), Q(0), H(0), R(0) \geq 0)\} \in D$.

Then the solution set $\{S, E, I, A, Q, H, R\}(t)$ of the system of equations (1) is positive for all $t > 0$

Proof:

From the first equation of (1), we have:

$$\frac{dS}{dt} = \pi - \beta SI - \mu S \geq -\mu S \quad (12)$$

$$\Rightarrow \frac{dS}{dt} \geq -\mu S \quad (13)$$

Where $K = e^c$, using the initial condition $t = 0 \Rightarrow S(0) \geq K$

Therefore $S(t) \geq S(0)e^{-\mu t} > 0$ (14)

From equation (1) we have;

$$\frac{dE}{dt} = \beta SI - (\theta + \sigma + \phi + \mu)E \geq -(\theta + \sigma + \phi + \mu)E \quad (15)$$

$$\Rightarrow \frac{dE}{dt} \geq -(\theta + \sigma + \phi + \mu)E \quad (16)$$

Where $K = e^c$, using the initial condition $t = 0 \Rightarrow E(0) \geq K$

Therefore $E(t) \geq E(0)e^{-(\theta+\sigma+\phi+\mu)t} > 0$ (17)

Similarly, it can be verified that the rest of the equations are positive for all $t > 0$, since $e^{\omega} > 0 \forall \omega \in \Re$

C. Disease Free Equilibrium of the Model

The disease-free equilibrium of the model (1) is obtained by setting $\frac{dS}{dt}=\frac{dE}{dt}=\frac{dI}{dt}=\frac{dA}{dt}=\frac{dQ}{dt}=\frac{dH}{dt}=\frac{dR}{dt}=0$ (18)

In this case there is no disease: $E=I=A=Q=H=R=0$. Hence, the DFE of our equation is given by:

$$E_0=\left(S^*,E^*,I^*,A^*,Q^*,H^*,R^*\right)=\left(\frac{\pi}{\mu},0,0,0,0,0,0\right) \quad (19)$$

It's important to note that in the absence of COVID-19 infection, the Human population is bounded by birth and natural mortality rates only i.e.

$$S\approx N\to\frac{\pi}{\mu} \quad (20)$$

D. The Basic Reproduction Number $\left(R_0\right)$

The basic reproduction number $\left(R_0\right)$ as the average number of secondary infections produced by individuals that are infectious during his or her entire period of infectiousness. R_0 determines if a disease will persist or will die out in a community. If $R_0<1$ it indicates that infectious individual will cause less than one secondary infection and hence the disease will not remain, then when $R_0>1$ the disease will take over the population. In a more complicated epidemic, the R_0 can be calculated by using the next generation operator approach by (van den Driessche & Watmough, 2002).

From the system (1) we define f_i and v_i

$$F_i=\begin{bmatrix}\beta IS\\0\\0\\\sigma E\\\upsilon Q\end{bmatrix} \quad \text{and } V_i=\begin{bmatrix}(\theta+\sigma+\phi+\mu)E\\-\phi E-\gamma Q+\left(\rho+\mu+\delta_I\right)I\\-\theta E+(\tau+\mu)A\\(\gamma+\upsilon+\mu)Q\\(\alpha+\delta_H+\mu)H\end{bmatrix} \quad (21)$$

The characteristic polynomial $\left|FV^{-1}-\Psi I\right|$ is given as;

$$FV^{-1}=\begin{bmatrix}\frac{\beta\pi\phi}{\mu B_{11}B_{22}}-\psi & \frac{\beta\pi}{\mu B_{22}} & 0 & \frac{\beta\pi\gamma}{\mu B_{44}B_{22}} & 0\\0 & -\psi & 0 & 0 & 0\\0 & 0 & -\psi & 0 & 0\\\frac{\sigma}{B_{11}} & 0 & 0 & -\psi & 0\\0 & 0 & 0 & \frac{\beta\upsilon}{B_{44}} & -\psi\end{bmatrix} \quad (22)$$

$\therefore$ Evaluating the characteristic polynomial equation $\left|FV^{-1}-\Psi I\right|=0$

Since it's an upper triangular matrix the determinant can be gotten by multiplying the leading diagonal.

$$-\Psi^4\left(\frac{\beta\pi\phi}{\mu B_{11}B_{22}}-\Psi\right)=0 \quad (23)$$

We have;

$$\Psi_1 = \Psi_2 = \Psi_3 = \psi_4 = 0 \text{ and } \Psi_5 = \frac{\beta\pi\phi}{\mu B_{11} B_{22}} \tag{24}$$

Since R_0 is the most positive value among the Eigen values we have;

$$R_0 = \frac{\beta\pi\phi}{\mu B_{11} B_{22}} \tag{25}$$

E. Local Stability Analysis of Disease Free Equilibrium State.

Theorem 3: The disease-free equilibrium, E^* of (25) is locally asymptotically stable (LAS) in D if $R_0 < 1$

Proof: We shall use Jacobean stability technique to carry out the local stability analysis of the disease disease-free equilibrium.

Jacobean matrix of the system of equations at disease-free equilibrium is:

Where $K_1 = (\theta + \sigma + \phi + \mu), K_2 = (\rho + \mu + \delta_I), K_3 = (\tau + \mu), K_4 = (\gamma + \upsilon + \mu)$ and $K_5 = (\alpha + \delta_H + \mu)$

$$J\left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0, 0\right)$$

$$J(E) = \begin{bmatrix} -\mu & 0 & -\beta S^* & 0 & 0 & 0 & 0 \\ 0 & -K_1 & \beta S^* & 0 & 0 & 0 & 0 \\ 0 & \phi & -K_2 & 0 & \gamma & 0 & 0 \\ 0 & \theta & 0 & -K_3 & 0 & 0 & 0 \\ 0 & \sigma & 0 & 0 & -K_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & \upsilon & -K_5 & 0 \\ 0 & 0 & \rho & \tau & 0 & \alpha & -\mu \end{bmatrix} \tag{24}$$

From the leading diagonals we have

$$\left.\begin{aligned} \Psi_1 &= -\mu \\ \Psi_2 &= -(\varepsilon + \mu) \\ \Psi_3 &= -(\delta_2 + \mu + \tau_2) \\ \Psi_4 &= -\mu \\ \Psi_5 &= -(\mu + \gamma + \phi)(1 - R_0) \\ \Psi_6 &= -\left(\frac{(\delta_1 + \mu + \tau_1 + \omega)(1 - (\mathrm{R}_0 + R_{00})) - R_0}{(1 - R_0)}\right) \end{aligned}\right\} \tag{25}$$

Hence, DFE is Locally Asymptotically Stable (LAS) if $R_0 < 1$. The epidemiology implication of the theorem is that Covid-19 can be eliminated (control) from the population when $R_0 < 1$, if the initial size of the sub-populations are in the basin of attraction of the DFE.

F. Existence of Endemic Equilibrium

The equilibrium state in the presence of infection is known as the Endemic equilibrium. Let $B_0 = (S^{**}, E^{**}, I^{**}, A^{**}, Q^{**}, H^{**}, R^{**})$ be the Endemic equilibrium points.

To solve for the Endemic equilibrium points $E \neq 0$

$$\pi - \beta S^{**} I^{**} - \mu S^{**} = 0 \quad (26)$$

$$\beta S^{**} I^{**} - (\theta + \sigma + \phi + \mu) E^{**} = 0 \quad (27)$$

$$\phi E^{**} + \gamma Q^{**} - (\rho + \mu + \delta_I) I^{**} = 0 \quad (28)$$

$$\theta E^{**} - (\tau + \mu) A^{**} = 0 \quad (29)$$

$$\sigma E^{**} - (\gamma + \upsilon + \mu) Q^{**} = 0 \quad (30)$$

$$\upsilon Q^{**} - (\alpha + \delta_H + \mu) H^{**} = 0 \quad (31)$$

$$\rho I^{**} + \tau A^{**} + \alpha H^{**} - \mu R^{**} = 0 \quad (32)$$

where

$$S^{**}\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right) = (\gamma + \upsilon + \mu)(\rho + \mu + \delta_I)(\theta + \sigma + \phi + \mu) \quad (33)$$

$$S^{**} = \frac{(\gamma + \upsilon + \mu)(\rho + \mu + \delta_I)(\theta + \sigma + \phi + \mu)}{\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right)} \quad (34)$$

Adding equation (26) and (27) and

$$\pi - \mu S^{**} - (\theta + \sigma + \phi + \mu) E^{**} = 0 \quad (35)$$

Putting equation (34) in equation (35) we have

$$E^{**} = \frac{\pi\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right) - \mu(\gamma + \upsilon + \mu)(\rho + \mu + \delta_I)(\theta + \sigma + \phi + \mu)}{\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right)(\theta + \sigma + \phi + \mu)} \quad (36)$$

Putting equation (36) in equation (29) we have

$$A^{**} = \frac{\theta\left(\pi\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right) - \mu(\gamma + \upsilon + \mu)(\rho + \mu + \delta_I)(\theta + \sigma + \phi + \mu)\right)}{\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right)(\theta + \sigma + \phi + \mu)(\tau + \mu)} \quad (37)$$

putting equation (34) into equation (30) we have

$$Q^{**} = \frac{\sigma\left(\pi\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right) - \mu(\gamma + \upsilon + \mu)(\rho + \mu + \delta_I)(\theta + \sigma + \phi + \mu)\right)}{\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right)(\theta + \sigma + \phi + \mu)(\gamma + \upsilon + \mu)} \quad (38)$$

putting equation (36) and (38) into equation (28) we have

$$I^{**} = \frac{\begin{array}{l}(\gamma + \upsilon + \mu)\phi\pi\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right) - \mu(\gamma + \upsilon + \mu)(\rho + \mu + \delta_I)(\theta + \sigma + \phi + \mu) + \\ \gamma\sigma\left(\pi\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right) - \mu(\gamma + \upsilon + \mu)(\rho + \mu + \delta_I)(\theta + \sigma + \phi + \mu)\right)\end{array}}{\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right)(\theta + \sigma + \phi + \mu)(\gamma + \upsilon + \mu)(\mu + \rho + \delta_I)} \quad (39)$$

putting equation (38) into equation (31) we have

$$H^{**} = \frac{\upsilon\sigma\left(\pi\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right) - \mu(\gamma + \upsilon + \mu)(\rho + \mu + \delta_I)(\theta + \sigma + \phi + \mu)\right)}{\left(\beta\phi(\gamma + \upsilon + \mu) + \beta\gamma\sigma\right)(\theta + \sigma + \phi + \mu)(\gamma + \upsilon + \mu)(\alpha + \delta_H + \mu)} \quad (40)$$

let

$$\left.\begin{array}{l} I^{**} = D_1 \\ A^{**} = D_2 \\ H^{**} = D_3 \end{array}\right\} \quad (41)$$

and putting equation (41) into equation (42) we have

$$R^{**} = \frac{\rho D_1 + \tau D_2 + \alpha D_3}{\mu} \tag{42}$$

Hence, the endemic equilibrium points of our model equation in terms of forces of infection are given as;

$$\begin{pmatrix} S^{**} \\ E^{**} \\ I^{**} \\ A^{**} \\ Q^{**} \\ H^{**} \\ R^{**} \end{pmatrix} = \begin{pmatrix} \frac{(\gamma+\upsilon+\mu)(\rho+\mu+\delta_I)(\theta+\sigma+\phi+\mu)}{(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)} \\ \frac{\pi(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)-\mu(\gamma+\upsilon+\mu)(\rho+\mu+\delta_I)(\theta+\sigma+\phi+\mu)}{(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)(\theta+\sigma+\phi+\mu)} \\ \frac{(\gamma+\upsilon+\mu)\phi\pi(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)-\mu(\gamma+\upsilon+\mu)(\rho+\mu+\delta_I)(\theta+\sigma+\phi+\mu)+\gamma\sigma(\pi(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)-\mu(\gamma+\upsilon+\mu)(\rho+\mu+\delta_I)(\theta+\sigma+\phi+\mu))}{(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)(\theta+\sigma+\phi+\mu)(\gamma+\upsilon+\mu)(\mu+\rho+\delta_I)} \\ \frac{\theta(\pi(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)-\mu(\gamma+\upsilon+\mu)(\rho+\mu+\delta_I)(\theta+\sigma+\phi+\mu))}{(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)(\theta+\sigma+\phi+\mu)(\tau+\mu)} \\ \frac{\sigma(\pi(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)-\mu(\gamma+\upsilon+\mu)(\rho+\mu+\delta_I)(\theta+\sigma+\phi+\mu))}{(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)(\theta+\sigma+\phi+\mu)(\gamma+\upsilon+\mu)} \\ \frac{\upsilon\sigma(\pi(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)-\mu(\gamma+\upsilon+\mu)(\rho+\mu+\delta_I)(\theta+\sigma+\phi+\mu))}{(\beta\phi(\gamma+\upsilon+\mu)+\beta\gamma\sigma)(\theta+\sigma+\phi+\mu)(\gamma+\upsilon+\mu)(\alpha+\delta_H+\mu)} \\ \frac{\rho D_1+\tau D_2+\alpha D_3}{\mu} \end{pmatrix} \tag{43}$$

IV. NUMERICAL SIMULATIONS

It is difficult to get a reliable data; we estimated the parameter value based on the available data from the Nigeria Centre for Disease Control (NCDC) and reliable literature. The estimates are clearly explained in the following sub-sections as shown in Table 1.

Table 1 Shows Initial Conditions for Each Plot and Parameters Value (NCDC, 2020).

Parameters and State Variables	Value	Source
$N(t)$	211,660,928	Worldometer, (2021)
$S(t)$	196,763,667	Adedayo *et al.*, (2022)
$E(t)$	14,549,903	Adedayo *et al.*, (2022)
$Q(t)$	104855	Assumed
$I(t)$	69904	Adedayo *et al.*, (2022)
$H(t)$	7552	NCDC 3, 2020
$A(t)$	12300	Assumed
$R(t)$	165047	NCDC 3, 2020
β	0.000002	Assumed
μ	0.0357	Assumed

$\backslash\delta_I$	0.000178	Assumed
δ_H	0.000211	Assumed
θ	0.5	Assumed
γ	0.05	Adedayo *et al.*, (2022)
ρ	0.2	Assumed
Λ	0.02537	Assumed
ϕ	0.01	Adedayo *et al.*, (2022)
α	0.2	Assumed
υ	0.0015	Assumed
τ	4	Assumed
σ	0.002	Assumed
φ	0.02	Assumed

V. GRAPHICAL REPRESENTATION OF SOLUTIONS OF THE MODEL EQUATIONS

The graphical representations are from the semi-analytical solutions of the model equations. They were plotted using MAPLE software.

Figures 1 to 7 are graphical solution of the model varying different parameter of the model.

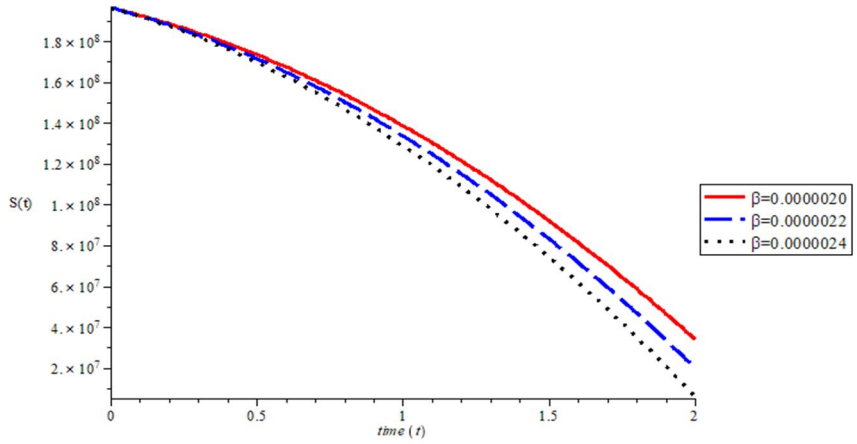

Figure 1: Graph of Susceptible Individuals Against Time for Different Contact Rate.

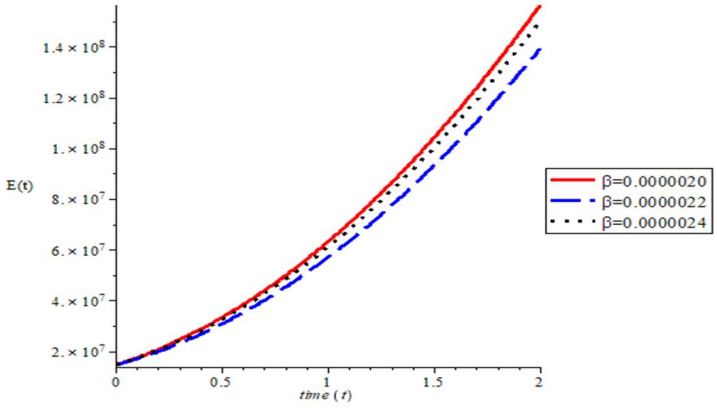

Figure 2: Graph of Exposed Individuals Against Time for Different Contact Rate.

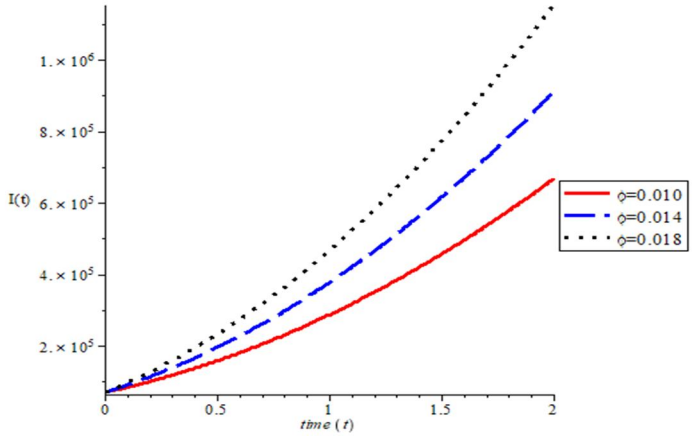

Figure 3: Graph of Infectious Symptomatic Against Time for Different Transmission rate after incubation period and transferred to symptomatic infected class.

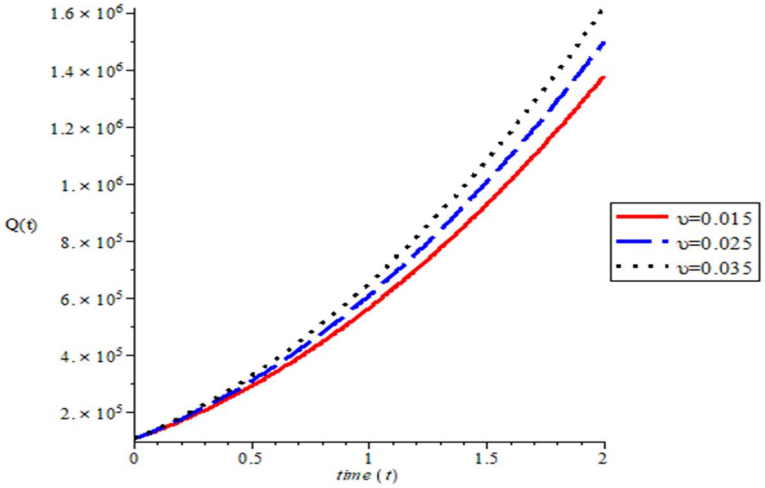

Figure 4: Graph of Quarantined Individuals Against Time at Different Hospitality rate of quarantined individuals and transferred to hospitality class.

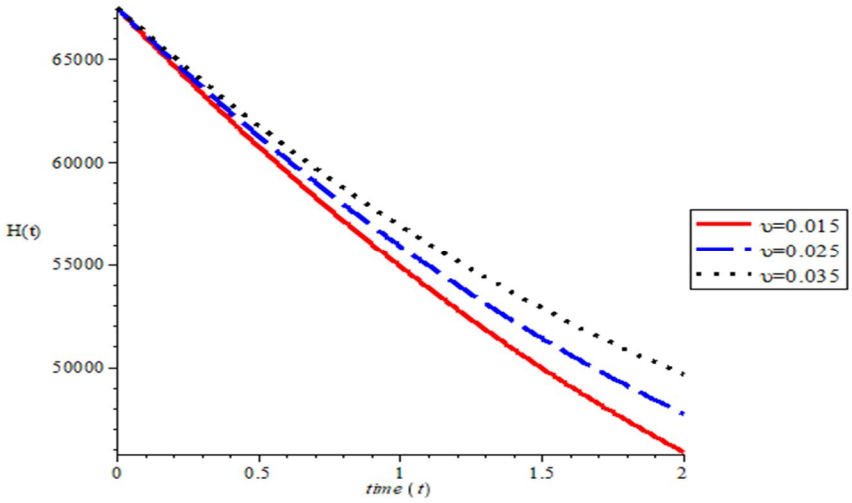

Figure 5: Graph of Hospitality Individuals Against Time at Different Hospitality rate of quarantined individuals and transferred to hospitality class in Humans.

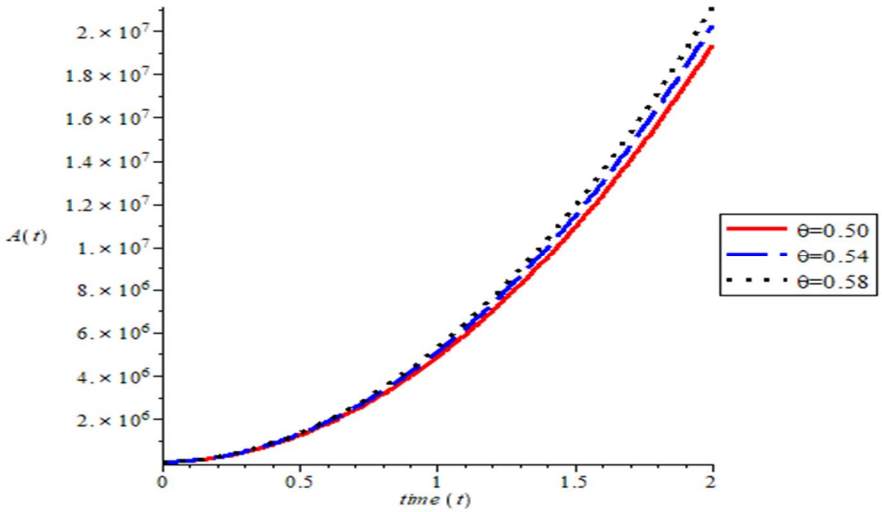

Figure 6: Graph of Infectious Asymptomatic Individuals Against Time at Different Transmission rate after incubation period and transferred to asymptomatic infected class.

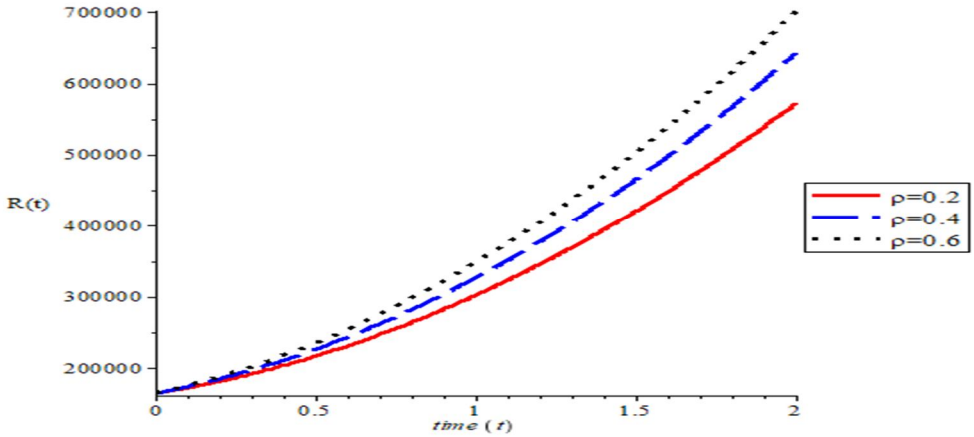

Figure 7: Graph of Recovered Individuals Against Time at Different Recovery rate of symptomatic infected individuals and transferred to recovery class.

VI. DISCUSSION OF RESULTS

Figure 1: is the graph of Susceptible Individuals Against Time for Different Contact Rate. It is observed that the population susceptible individual decreases as the rate of the Contact Rate increases.

Figure 2: is the graph of Exposed Individuals Against Time for Different Contact Rate. It is observed that the population of Exposed individuals increases as the Contact Rate increases.

Figure 3: is the graph of Infectious Symptomatic Against Time for Different Transmission rate after incubation period and transferred to symptomatic infected class. It is observed that the population Infectious Symptomatic individuals increase as the transmission rate after incubation period and transferred to symptomatic infected class increase.

Figure 4: is the graph of Quarantined Individuals Against Time at Different Hospitality rate of quarantined individuals and transferred to hospitality class. It is observed that the population of the quarantined individuals increases as the hospitality rate of quarantined individuals and transferred to hospitality class increases.

Figure 5: is the graph of Hospitality Individuals Against Time at Different Hospitality rate of quarantined individuals and transferred to hospitality class in Humans. It is observed that the population of the hospitality individuals decreases as the hospitality rate of quarantined individuals and transferred to hospitality class in humans increases.

Figure 6: is the graph of Infectious Asymptomatic Individuals Against Time at Different Transmission rate after incubation period and transferred to asymptomatic infected class. It is observed that the population of the infectious asymptomatic individuals increases as the transmission rate after incubation period and transferred to asymptomatic infected class rates increases.

Figure 7: is the graph of Recovered Individuals Against Time at Different Recovery rate of symptomatic infected individuals and transferred to recovery class. It is observed that the population of the recovery individuals increases as the recovery rate of symptomatic infected individuals and transferred to recovery class rates increases.

VII. CONCLUSION AND RECOMMENDATIONS

In this research paper, a mathematical model for the transmission dynamic of COVID-19 with quarantine and hospitality treatment in Nigeria was developed and analyzed in this study. The Disease Free State (DFE) was analyzed for stability and it revealed that it is stable. The Reproduction number was analyzed and the result shows the stability of the disease, which implies that the disease would be wiped out if vaccination is used as a control parameter.

COVID-19 eradication needs systematic thinking, effective hospital isolation, and effective COVID-19 drug and vaccination. The desired eradication deadline based on our models can determine the demand of the three weapons against COVID-19 virus. It is recommended that **the** model shows that the spread of COVID-19 infection depends largely on the contact rate, hence the NCDC and Hospitals should emphasize on the improvement in early detection of COVID-19 infection cases by developing strategies to improve the rate of testing so that transmission rate can be minimized. The Government should improve on sensitization measures in order to make individuals fully aware of the virus potency and also encourage them to be generally available for testing, infectious individuals should be isolated and treated immediately. And It is recommended that the government strictly enforces the use of hand sanitizers, nose masks and adherence to social distancing. This would enable the reduction to the exposure of the populace to the virus and turn reduces the overcrowding of the isolation centres and Hospitals.

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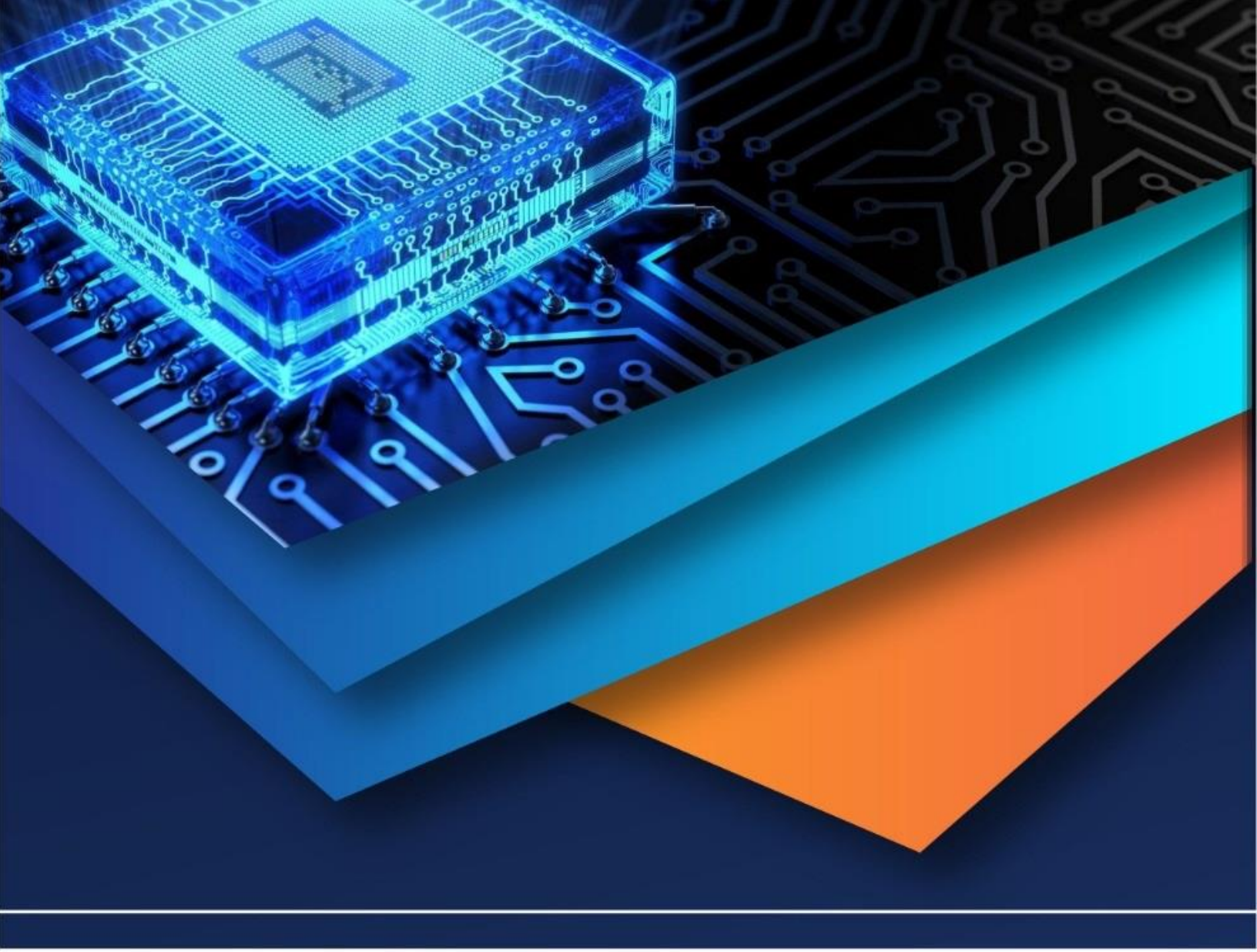

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