

ANALYSIS OF TRANSMISSION DYNAMICS AND MITIGATION SUCCESS OF COVID-19 IN NIGERIA: AN INSIGHT FROM A MATHEMATICAL MODEL

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Abstract

The first case of COVID-19 was confirmed in Nigeria on February 27 2020. The government of Nigeria took drastic steps in the form of enforcement of lockdown and social distancing order which necessitated closure of schools, worship centres, markets, offices, leisure spots and businesses to curtail the spread of the virus which have resulted in low confirmed cases and mortality with a case fatality ratio of 2% as at September 3 2020. While experts had predicted doom for Africa in the wake of COVID-19 pandemic, the ability of the Nigerian government to contain the pandemic in the first six months of emergence remained the biggest surprise. Therefore, a mathematical model was designed to analyse effectiveness of government mitigation

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measures in minimising the spread and mortality of COVID-19 in Nigeria. The positivity and boundedness of solutions were validated for the model. The analytical parameter, effective reproductive ratio, that governed the virus transmissibility in terms of the model parameters was derived and the stability analysed locally and globally around disease-free and disease-endemic equilibria. The disease-free equilibrium of the model is locally and globally asymptotically stable if the effective reproduction number is less than one. Otherwise, it is the endemic equilibrium, that is, asymptotically stable, locally and globally whenever the effective reproduction number is greater than one. Simulation was conducted to justify the theoretical results. Results from the simulation showed that low rates of transmission and mortality from COVID-19 in Nigeria were attributed to the effectiveness of mitigation measures. The results also indicate that implementation of non-pharmaceutical interventions can put Nigeria and other African countries in a good position for combatting subsequent emergence of any form of infectious diseases.

1 Introduction

The ongoing ravaging COVID-19 is a contagious disease instigated by SARS-CoV-2. The first case of the disease was reported in December 2019 in Wuhan, China, and has, within a few weeks, spread across the globe, leading to COVID-19 pandemic [1]. The coronavirus disease 2019 has been regarded as the largest global health crisis in human history as a result of the magnitude of confirmed cases, accompanied with the degree of fatalities across the continents [2]. Reliable data had it that by April 2020, COVID-19 pandemic had led to over 3 million confirmed cases and 230 000 deaths and the disease has spread to over 210 nations globally [3]. As of 1st October, 2020, 10:31 GMT, the figure has skyrocketed to 34, 192, 734 reported cases with 867 347 fatalities [4].

The symptoms and signs of COVID-19 develop within 2 to 14 days [5]. When the disease is fully incubated, the infected individuals may exhibit fever, fatigue, cough and breathing disorder that is similar to those infections instigated by SARS-CoV and MERS-CoV [6, 7]. However, many COVID-19 acute cases and fatalities come from the elderly people (from the age of 65 upward) and individuals with severe health challenges (such as people with kidney disease, hypertension, diabetes, obesity and other health issues that deteriorate the immune system) [3].

The global scourge of COVID-19 pandemic has elicited the attention of scholars in different disciplines, prompting several proposals to examine and envisage the development of the pandemic [8, 9]. Ndairov *et al.* [10] propose a model for the

transmissibility of COVID-19 in the presence of super-spreaders. They performed the stability and sensitivity analyses of the model and discovered that daily reduction in the number of confirmed cases of COVID-19 is a function of the number of hospitalisations. Yang and Wang [11] proposed a model to study the transmission pathways of COVID-19 in terms of human-to-human and environment-to-human spread. Their analysis confirms the tendency of COVID-19 to remain endemic even with prevention and intervention measures. A model for the dynamics of COVID-19 with parameter estimations, sensitivity analysis and data fitting was investigated in [12] while a model for COVID-19 infection that describes the impact of slow diagnosis on the dynamics of COVID-19 is also studied in [13]. In [14], the researchers employ a statistical study of coronavirus disease to calculate time-regulated risk for fatality from the COVID-19 in Wuhan. Their results indicate that movement restrictions and adequate social distancing procedures are capable of reducing the spread of the disease. Furthermore, a data-oriented model that includes behavioural impacts of humans and governmental efforts on the dynamics of COVID-19 in Wuhan was proposed in [15]. A good number of mathematics and non-mathematics studies have also been conducted on COVID-19 [16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 41].

Generally, the experts' prediction that COVID-19 would overrun precarious health systems in many African countries and eliminate millions has not come true as the continent has the second lowest COVID-19 fatality ratio globally as of 31st August 2020 [27]. Particularly, the situation report for the pandemic in Nigeria is better than most African countries especially South Africa with the biggest economy in the continent.

Table 1. Reported Cases and Death of COVID-19 for some African Countries

Countries	Population	Total COVID-19 Cases	Total Recovered	Total Deaths
Mali	20, 250,000	2, 087	-	126
Cameroon	26, 550, 000,	19, 604	-	415
Kenya	53, 770, 000	34, 705	-	585
Ivory Coast	26, 380, 000	18, 208	-	119
Nigeria	206, 984, 347	54, 463	42, 439	1, 027
South Africa	59, 436, 725	630, 595	553, 456	14, 389
Egypt	102, 300, 000	99, 425	-	5, 479
Morocco	36, 910, 000	66, 855	-	1, 253
Algeria	43, 850, 000	45, 469	-	1, 529
Ghana	31, 070, 000	32, 969	-	168

Retrieved 2020-09-03 from https://en.wikipedia.org/wiki/COVID-19_pandemic_in_each_country

After the first case of the pandemic was reported, the government of Nigeria enforced lockdown and social distancing which necessitated closure of schools, worship centres, markets, offices, leisure spots and businesses to check the spread of the virus. COVID-19 phenomenon has caught the attentions of many researchers

both mathematicians and non-mathematicians and several studies have been conducted to monitor and report the situation in Nigeria [28, 29, 30, 31]. Also, the effects of government interventions, policy stringency, policy on demand or application of personal protective equipment (PPEs) have been considered by some researchers [32, 33, 34, 35]. However, the analysis of how mitigation measures succeeded in averting massive infections and mortality from COVID-19 in Nigeria through the use of mathematical modelling is new in the literature. Although COVID-19 is gradually dying off at present, and some countries have started relaxing mitigation measures, while other countries have completely relaxed all measures, this study gives an account of how Nigerian government managed the pandemic in the first six months of emergence and would provide important information about mitigation strategies to adequately equip the country and other African countries towards future outbreak of infectious diseases.

2 Model Formulation and Basic Properties

The model is made up of two components as in [11] - the human population and the environment. The human population is divided into five compartments: the susceptible S , the exposed E , the infected I , the isolated Q and the removed individuals R . Individuals in the infected and the exposed classes are both infectious though infected individuals are more infectious because of the relatively low level of virus in the exposed individuals' systems. Susceptible individuals can contract the virus from the infectious individuals E and I and from the already contaminated environment V at rates α_1 , α_2 and α_3 respectively. However, infectivity is reduced at a constant rate m by various measures that have been put on ground by the government (e.g. lockdown, social distancing, handwashing, use of face masks, environmental sanitation, etc.). Also, some individuals are detected at the exposed stage at rate $(1 - \sigma)$ through measures such as contact tracing and put in isolation Q while the rest who are not detected become infectious at rate σ . Individuals in isolation are receiving treatment and are not allowed to interact with the general public. Therefore, they do not spread COVID-19 to the individuals in the susceptible category. Besides, the exposed and infectious individuals increase the viral loads in the environment at rates β_1 and β_2 respectively. However, the rate of increase in the virus concentration in the environment V by the exposed and infectious individuals is reduced at a constant rate n , the awareness created by the government regarding environmental health. The effects of parameter n on the compartments S , Q and R are not considered because it is assumed that the compartments do not increase the viral load in the environment. It is only the exposed

and infectious individuals that can pass the virus to the environment. Individuals in the infected and isolation classes are treated and recovered at different rates p_1 and p_2 respectively while death due to COVID-19 occurs for infectious and isolated individuals at rates k_1 and k_2 respectively. Death unrelated to COVID-19 occurs for every individual in the population at the same rate μ while virus removal unrelated to human interference occurs at rate τ . The flow of transmission across the compartments is displayed in Figure 1.

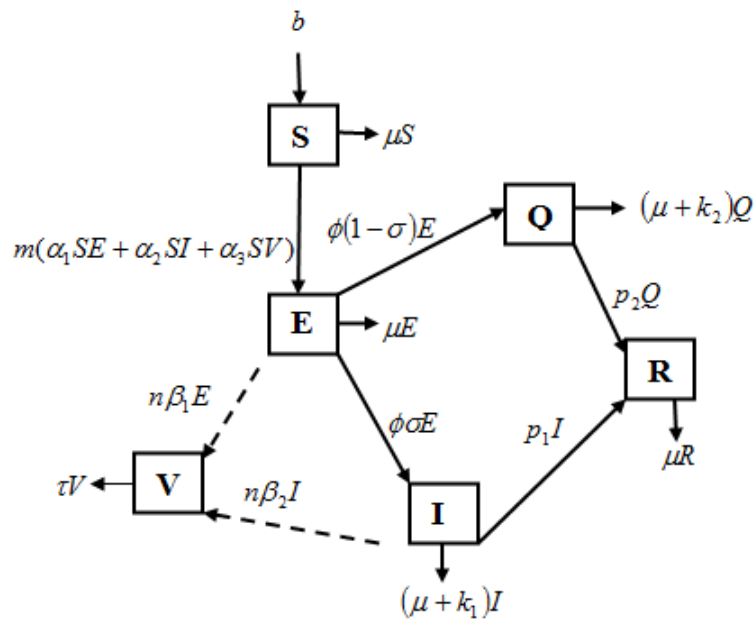


Figure 1: The flow chart of the model

Following the aforementioned assumptions, a model that describes the dynamics and effort to mitigate COVID-19 in Nigeria is introduced thus

$$\frac{dS}{dt} = b - m\alpha_1SE - m\alpha_2SI - m\alpha_3SV - \mu S, \tag{2.1}$$

$$\frac{dE}{dt} = m\alpha_1SE + m\alpha_2SI + m\alpha_3SV - (\phi + \mu)E, \tag{2.2}$$

$$\frac{dI}{dt} = \phi\sigma E - (k_1 + p_1 + \mu)I, \tag{2.3}$$

$$\frac{dQ}{dt} = \phi(1 - \sigma)E - (k_2 + p_2 + \mu)Q, \tag{2.4}$$

$$\frac{dR}{dt} = p_1I + p_2Q - \mu R, \tag{2.5}$$

$$\frac{dV}{dt} = n\beta_1E + n\beta_2I - \tau V, \tag{2.6}$$

with non-negative initial conditions

$$S(0) = S_o, E(0) = E_o, I(0) = I_o, Q(0) = Q_o, R(0) = R_o, V(0) = V_o.$$

Where $k_1 > k_2$, $\beta_2 > \beta_1$, $\alpha_3 < \alpha_1 < \alpha_2$ and $p_1 < p_2$ with parameters nomenclatures stated in Table 2.

Table 2. Description of parameters for the model

Parameters	Nomenclatures	Units
b	human recruitment rate	day^{-1}
m	reduction factor in disease transmission potential	-
α_1	effective contact rate between S and E	day^{-1}
α_2	effective contact rate between S and I	day^{-1}
α_3	effective contact rate between S and V	day^{-1}
μ	natural death rate in all human compartments	day^{-1}
ϕ	transition rate from the exposed stage	day^{-1}
σ	probability of transition from the exposed class into the infected class	-
k_1	death rate due to infection in I class	day^{-1}
k_2	death rate due to infection in Q class	day^{-1}
p_1	recovery rate in I class	day^{-1}
p_2	recovery rate in Q class	day^{-1}
β_1	exposed individuals' rate of contribution to the growth of pathogen	day^{-1}
β_2	infected individuals' rate of contribution to the growth of pathogen	day^{-1}
n	reduction factor in the human contribution to the growth of pathogen	-
τ	removal rate of pathogen	$ml\ day^{-1}$

Basic features of the model

For the system to be valid socially, environmentally, mathematically and epidemiologically, it is necessary to establish that its variables are positive for all $t > 0$ and besides, the region Ω , where the solutions for the model exist, is bounded. The following statements are therefore declared.

Theorem 2.1. *Since $S_o \geq 0, E_o \geq 0, I_o \geq 0, Q_o \geq 0, R_o \geq 0$ and $V_o \geq 0$. Then the coordinate (S, E, I, Q, R, V) , which represents the solutions of the model, is positive.*

Proof. Suppose $t_1 = \sup\{t > 0 : S > 0, E > 0, I > 0, Q > 0, R > 0, V > 0 \in [0, t]\}$. Then $t_1 > 0$ and it is obvious from (2.1), if the positive term is neglected that

$$\frac{dS}{dt} \geq -\Gamma S,$$

where

$$\Gamma = m\alpha_1 E + m\alpha_2 I + m\alpha_3 V + \mu.$$

Hence,

$$\int \frac{dS}{dt} \geq - \int \Gamma S,$$

which implies that

$$S(t_1) = S(0) \exp \left[- \int_0^{t_1} \lambda(r) dr \right] > 0 \quad \forall t_1 > 0$$

from (2.1). Hence, $S(0) > 0$ since $e^p > 0 \quad \forall$ real values of p . Following the same procedure, it can be established that:

$$E > 0, I > 0, Q > 0, R > 0, V > 0.$$

□

Lemma 2.1. *The region*

$\Omega = \left\{ (S, E, I, Q, R, V) \in \mathfrak{R}_+^6; S, E, I, Q, R, V \geq 0; N(t) \leq \frac{b}{\mu}; V(t) \leq \frac{b\lambda}{\tau\mu} \right\}$
is not only positively invariant but attracts all nonnegative solutions of the system.

Proof. Adding system (2.1)-(2.5) for human population,

$$\frac{dN}{dt} = b - (S + E + I + Q + R)\mu - k_1 I - k_2 Q, \Rightarrow$$

$$\frac{dN}{dt} \leq b - \mu N, \Rightarrow$$

$$\frac{dN}{b - \mu N} \leq dt, \Rightarrow$$

$$b - \mu N(t) \geq c_1 e^{-\mu t}.$$

As $t = 0$, $N(t) = N(0)$
then,

$$c_1 = b - \mu N(0).$$

Hence,

$$\begin{aligned} b - \mu N(t) &\geq (b - \mu N(0))e^{-\mu t}, \Rightarrow \\ N(t) &\leq \frac{b}{\mu} - \left(\frac{b - \mu N(0)}{\mu} \right) e^{-\mu t}. \end{aligned}$$

As $t \rightarrow \infty$,

$$0 \leq N(t) \leq \frac{b}{\mu}. \quad (2.7)$$

Hence, the feasible solution set for human population enter the region

$$\Delta = \left\{ (S, E, I, Q, R) \in \mathfrak{R}_+^5; N(t) \leq \frac{b}{\mu} \right\}.$$

Also, the population of the pathogen at time t in the system (2.6) is given by

$$\frac{dV}{dt} = (E + I)\lambda - \tau V, \quad (2.8)$$

with $\lambda = \min(n\beta_1, n\beta_2)$.

λ is the net contribution of infectious individuals (both exposed and infected) to the growth of pathogen in the environment. Notice that both E and I are subset of human population which has been proved to be less than or equal to $\frac{b}{\mu}$ in inequality (2.7).

Therefore, $E + I \leq \frac{b}{\mu}$.

Hence, from (2.8),

$$\begin{aligned} \frac{dV}{dt} &\leq \frac{b\lambda}{\mu} - \tau V, \Rightarrow \\ V(t) &\leq \frac{b\lambda}{\tau\mu} (1 - c_2 e^{-\tau t}). \end{aligned}$$

As $t \rightarrow \infty$ then,

$$V(t) \leq \frac{b\lambda}{\tau\mu}.$$

Therefore, the feasible solutions for the dynamics of coronavirus population in the model exists in the region

$$\Psi = \left\{ V \in \mathfrak{R}^+; V(t) \leq \frac{b\lambda}{\tau\mu} \right\}.$$

Hence, the region Ω that contains $\{\Delta \cup \Psi\}$ attracts every solution in \mathfrak{R}_+^6 . \square

3 Model Analysis

The dynamical behaviour of the model can be studied since the solutions are positive and bounded.

3.1 Equilibria

Two equilibria will be discussed - disease-free and endemic equilibria. Disease-free will be discussed first then followed by the reproduction number before discussing endemic equilibrium. Before February 27 2020, Nigeria was free from COVID-19. The COVID-19-free equilibrium then could be expressed mathematically as

$$\begin{aligned} \mathcal{E}_0 &= (S^\circ, E^\circ, I^\circ, Q^\circ, R^\circ, V^\circ), \\ &= \left(\frac{b}{\mu}, 0, 0, 0, 0, 0 \right). \end{aligned}$$

The agents of infection are in the compartments E , I and V . Therefore, F and V , the new infection and the transition matrices, are derived following Driessche and Watmough [36] as

$$F = \begin{pmatrix} m\alpha_1 S^\circ & m\alpha_2 S^\circ & m\alpha_3 S^\circ \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad (3.1)$$

$$V = \begin{pmatrix} \phi + \mu & 0 & 0 \\ -\phi\sigma & k_1 + p_1 + \mu & 0 \\ -n\beta_1 & -n\beta_2 & \tau \end{pmatrix}. \quad (3.2)$$

Following Driessche and Watmough [36],

$$\begin{aligned} \mathcal{R}_e &= \frac{m\alpha_1 S^\circ}{(\phi + \mu)} + \frac{\phi\sigma m\alpha_2 S^\circ}{(\phi + \mu)(k_1 + p_1 + \mu)} + \frac{[(k_1 + p_1 + \mu)n\beta_1 + \phi\sigma n\beta_2]m\alpha_3 S^\circ}{\tau(\phi + \mu)(k_1 + p_1 + \mu)}, \\ &= \mathcal{R}_a + \mathcal{R}_b + \mathcal{R}_c. \end{aligned} \quad (3.3)$$

\mathcal{R}_e measures disease transmissibility with mitigation measures on ground. It is called effective reproduction number and the first two terms in it quantify disease spread from human-to-human while the third term measures disease transmission into the population from the environment. If all the mitigation measures are not on ground, the effective reproduction number reduces to the basic reproduction number \mathcal{R}_0 given as

$$\begin{aligned}\mathcal{R}_0 &= \frac{\alpha_1 S^\circ}{(\phi + \mu)} + \frac{\phi \alpha_2 S^\circ}{(\phi + \mu)(k_1 + p_1 + \mu)} + \frac{[(k_1 + p_1 + \mu)\beta_1 + \phi\beta_2]\alpha_3 S^\circ}{\tau(\phi + \mu)(k_1 + p_1 + \mu)}, \\ &= \mathcal{R}_x + \mathcal{R}_y + \mathcal{R}_z.\end{aligned}\quad (3.4)$$

After February 27 2020 when coronavirus cases have been confirmed, the endemic equilibrium of the disease could be given as \mathcal{E} with coordinates (S, E, I, Q, R, V) . To obtain the value of each coordinate, the system of equations (2.1)-(2.6) is solved and

$$S = \frac{1}{\mu}[b - (\phi + \mu)E], \quad (3.5)$$

$$I = \left(\frac{\phi\sigma}{k_1 + p_1 + \mu}\right)E, \quad (3.6)$$

$$Q = \left(\frac{\phi(1 - \sigma)}{k_2 + p_2 + \mu}\right)E, \quad (3.7)$$

$$R = \frac{p_1 I + p_2 Q}{\mu}, \quad (3.8)$$

$$V = \frac{[(k_1 + p_1 + \mu)n\beta_1 + \phi\sigma n\beta_2]E}{\tau(k_1 + p_1 + \mu)}. \quad (3.9)$$

Combining (3.6), (3.9) and (2.2) \Rightarrow

$$m\alpha_1 SE + \frac{m\alpha_2 \phi \sigma SE}{(k_1 + p_1 + \mu)} + \frac{m\alpha_3 S[(k_1 + p_1 + \mu)n\beta_1 + \phi\sigma n\beta_2]E}{\tau(k_1 + p_1 + \mu)} - (\phi + \mu)E = 0. \quad (3.10)$$

In (3.10), $E = 0$ corresponds to the disease-free equilibrium before the outbreak of COVID-19.

Also, from (3.10),

$$S = \frac{(\phi + \mu)}{m\alpha_1 + \frac{m\alpha_2 \phi \sigma}{(k_1 + p_1 + \mu)} + \frac{m\alpha_3 [(k_1 + p_1 + \mu)n\beta_1 + \phi\sigma n\beta_2]}{\tau(k_1 + p_1 + \mu)}}. \quad (3.11)$$

Multiply the numerator and denominator of (3.11) by $\frac{S^\circ}{(\phi + \mu)}$ then

$$S = \frac{S^\circ}{\mathcal{R}_0}, \quad (3.12)$$

$$E = \frac{1}{(\phi + \mu)} \left[\pi - \frac{\mu S^\circ}{\mathcal{R}_0} \right], \quad (3.13)$$

$$I = \frac{\phi \sigma}{(\phi + \mu)(k_1 + p_1 + \mu)} \left[\pi - \frac{\mu S^\circ}{\mathcal{R}_0} \right], \quad (3.14)$$

$$Q = \frac{\phi(1 - \sigma)}{(\phi + \mu)(k_2 + p_2 + \mu)} \left[\pi - \frac{\mu S^\circ}{\mathcal{R}_0} \right], \quad (3.15)$$

$$R = \frac{1}{\mu} \left[\frac{p_1 \phi \sigma}{(\phi + \mu)(k_1 + p_1 + \mu)} \left\{ \pi - \frac{\mu S^\circ}{\mathcal{R}_0} \right\} + \frac{p_2 \phi(1 - \sigma)}{(\phi + \mu)(k_2 + p_2 + \mu)} \left\{ \pi - \frac{\mu S^\circ}{\mathcal{R}_0} \right\} \right], \quad (3.16)$$

$$V = \frac{[(k_1 + p_1 + \mu)n\beta_1 + \phi\sigma n\beta_2]}{\tau(\phi + \mu)(k_1 + p_1 + \mu)} \left\{ \pi - \frac{\mu S^\circ}{\mathcal{R}_0} \right\}. \quad (3.17)$$

3.2 Stability of Equilibria

The local and global stability of the model around disease-free equilibrium can be investigated using linearisation approach and Lyapunov functional, respectively.

Theorem 3.1. *The DFE \mathcal{E}_0 of the system (2.1)-(2.6) is locally asymptotically stable if $\mathcal{R}_e < 1$ otherwise it is unstable if $\mathcal{R}_e > 1$.*

Proof. If the system (2.1)-(2.6) is linearised around disease-free equilibrium \mathcal{E}_0 , the variational matrix is obtained as

$$J(\mathcal{E}_0) = \begin{pmatrix} -\mu & -m\alpha_1 S^\circ & -m\alpha_2 S^\circ & 0 & 0 & -m\alpha_3 S^\circ \\ 0 & m\alpha_1 S^\circ - (\phi + \mu) & m\alpha_2 S^\circ & 0 & 0 & m\alpha_3 S^\circ \\ 0 & \phi\sigma & -(k_1 + p_1 + \mu) & 0 & 0 & 0 \\ 0 & \phi(1 - \sigma) & 0 & -(k_2 + p_2 + \mu) & 0 & 0 \\ 0 & 0 & p_1 & p_2 & -\mu & 0 \\ 0 & n\beta_1 & n\beta_2 & 0 & 0 & -\tau \end{pmatrix} \quad (3.18)$$

\mathcal{R}_0 will be less than one and the disease-free equilibrium will be locally asymptotically stable if it can be established that all the eigenvalues of (3.18) are nonpositive.

Evaluating $|J(\mathcal{E}_0) - \lambda I| = 0$, four of the solutions are obtained as

$$\lambda_1 = \lambda_2 = -\mu, \lambda_3 = -\tau \text{ and } \lambda_4 = -(k_2 + p_2 + \mu).$$

The remaining two solutions can be obtained from the equation

$$\lambda^2 + a_1 \lambda + a_2 = 0, \quad (3.19)$$

where

$$a_1 = k_1 + p_1 + \phi + 2\mu - m\alpha_1 S^\circ$$

and,

$$a_2 = (k_1 + p_1 + \mu) \{(\phi + \mu) - m\alpha_1 S^\circ\} - m\alpha_2 \phi \sigma S^\circ.$$

It is obvious that the two solutions in (3.19) are nonpositive if $a_1 > 0$ and $a_2 > 0$. Hence, $\mathcal{R}_0 < 1$ and the disease-free equilibrium, \mathcal{E}_0 of the model is locally asymptotically stable if the condition $a_1 > 0$ and $a_2 > 0$ are satisfied. \square

Having established the necessary and sufficient conditions for the disease-free equilibrium of the model to be locally asymptotically stable, attempt would be made to study the global asymptotic stability behaviour of the model around disease-free equilibrium. Following the popular Lyapunov functional approach as in [28, 29, 37], Lyapunov function W is constructed thus

$$W(t) = A_1 E + A_2 I + A_3 V, \quad (3.20)$$

with the time derivative $\dot{W}(t)$ given as

$$\dot{W}(t) = A_1 \dot{E}(t) + A_2 \dot{I}(t) + A_3 \dot{V}(t). \quad (3.21)$$

Theorem 3.2. *The DFE \mathcal{E}_0 of the model is globally asymptotically stable if the time derivative of the Lyapunov functional $\dot{W}(t) \leq 0 \in \Omega$*

Proof. Putting $\dot{E}(t)$, $\dot{I}(t)$ and $\dot{V}(t)$ in (2.2), (2.3) and (2.6) into (3.21)

$$\begin{aligned} \dot{W}(t) &= A_1 [m\alpha_1 S E + m\alpha_2 S I + m\alpha_3 S V - (\phi + \mu) E] \\ &+ A_2 [\phi \sigma E - (k_1 + p_1 + \mu) I] \\ &+ A_3 [n\beta_1 E + n\beta_2 I - \tau V] \Rightarrow \end{aligned} \quad (3.22)$$

$$\begin{aligned} \dot{W}(t) &= [A_1 m\alpha_1 S - A_1(\phi + \mu) + A_2 \phi \sigma + A_3 n\beta_1] E \\ &+ [A_1 m\alpha_2 S - A_2(k_1 + p_1 + \mu) + A_3 n\beta_2] I \\ &+ [A_1 m\alpha_3 S + \tau] V. \end{aligned} \quad (3.23)$$

At DFE when $\mathcal{R}_e < 1$, $S = S^\circ$, and $E = I = Q = R = V = 0$ but if $\mathcal{R}_e = 1$, the equality $\dot{W}(t) = 0 \Rightarrow$

$$\begin{aligned} &[A_1 m\alpha_1 S - A_1(\phi + \mu) + A_2 \phi \sigma + A_3 n\beta_1] E \\ &+ [A_1 m\alpha_2 S - A_2(k_1 + p_1 + \mu) + A_3 n\beta_2] I \\ &+ [A_1 m\alpha_3 S + \tau] V = 0. \end{aligned} \quad (3.24)$$

It is reasonable in (3.24) for

$$[A_1 m \alpha_1 S - A_1(\phi + \mu) + A_2 \phi \sigma + A_3 n \beta_1] E \leq 0 \quad (3.25)$$

In view of (3.10) and given that the analysis is around the DFE then (3.25) implies that

$$\left[m \alpha_1 S^\circ + \frac{m \alpha_2 \phi \sigma S^\circ}{(k_1 + p_1 + \mu)} + \frac{m \alpha_3 S^\circ [(k_1 + p_1 + \mu) n \beta_1 + \phi \sigma n \beta_2]}{\tau (k_1 + p_1 + \mu)} - (\phi + \mu) \right] E \leq 0, \quad (3.26)$$

$$\begin{aligned} & (\phi + \mu) \left[\frac{m \alpha_1 S^\circ}{(\phi + \mu)} + \frac{m \alpha_2 \phi \sigma S^\circ}{(\phi + \mu)(k_1 + p_1 + \mu)} \right. \\ & \left. + \frac{m \alpha_3 S^\circ [(k_1 + p_1 + \mu) n \beta_1 + \phi \sigma n \beta_2]}{\tau (\phi + \mu)(k_1 + p_1 + \mu)} - 1 \right] E \leq 0, \end{aligned} \quad (3.27)$$

$$\Rightarrow (\phi + \mu) [\mathcal{R}_e - 1] E \leq 0. \quad (3.28)$$

Hence, $\dot{W}(t) = 0$ if $E = 0$. Conversely, $\dot{W}(t) \leq 0$ provided $\mathcal{R}_e \leq 1$. This implies that as

$t \rightarrow \infty$, $(S(t), E(t), I(t), Q(t), R(t), V(t)) \rightarrow \left(\frac{b}{\mu}, 0, 0, 0, 0, 0 \right)$. Therefore, the largest invariant set in $\{S(t), E(t), I(t), Q(t), R(t), V(t) \in \Omega : W(t) = 0\}$ is the singleton $\{\mathcal{E}_0\}$. Hence, by LaSalle's invariance principle [38], the DFE \mathcal{E}_0 is globally asymptotically stable in Ω if $\mathcal{R}_e \leq 1$. \square

To assess the effectiveness of the government mitigation measures in the light of the disease-endemic equilibrium, the stability analysis is extended to the endemic equilibrium so that government mitigation measures are adequately evaluated under a disease- pandemic scenario.

Theorem 3.3. *The disease-endemic equilibrium exists and is locally asymptotically stable if the effective reproductive ratio $\mathcal{R}_e > 1$.*

Proof. Linearising the system (2.1)-(2.6) around disease-pandemic equilibrium

then

$$J(P^*) = \begin{pmatrix} -b_1 & -m\alpha_1 S^* & -m\alpha_2 S^* & 0 & 0 & -m\alpha_3 S^* \\ b_3 & b_2 & m\alpha_2 S^* & 0 & 0 & 0 \\ 0 & \phi\sigma & -(k_1 + p_1 + \mu) & 0 & 0 & 0 \\ 0 & \phi(1 - \sigma) & 0 & -(k_2 + p_1 + \mu) & 0 & 0 \\ 0 & 0 & p_1 & p_2 & -\mu & 0 \\ 0 & n\beta_1 & n\beta_2 & 0 & 0 & -\tau \end{pmatrix}, \quad (3.29)$$

where $b_1 = m(\alpha_1 E^* + \alpha_2 I^* + \alpha_3 V^* + \mu)$, $b_2 = m\alpha_1 S^* - (\mu + \phi)$ and $b_3 = m(\alpha_1 E^* + \alpha_2 I^* + \alpha_3 V^*)$.

Three eigenvalues of (3.29) are $\lambda_1 = -\tau$, $\lambda_2 = -\mu$ and $\lambda_3 = -(k_2 + p_1 + \mu)$. Using row reduced operation, the remaining eigenvalues can be obtained from submatrix U

$$J(U) = \begin{pmatrix} -b_1 & -m\alpha_1 S^* & -m\alpha_2 S^* \\ 0 & \frac{a_1 a_2}{a_3} - m\alpha_1 S^* & \frac{a_1 m\alpha_2 S^*}{a_3} - m\alpha_2 S^* \\ 0 & \phi\sigma & -(k_1 + p_1 + \mu) \end{pmatrix}. \quad (3.30)$$

The matrix in (3.30) has one of the eigenvalues being $\lambda_4 = -b_1$ while the remaining eigenvalues can be obtained from the characteristic equation

$$k_0 \lambda^2 + k_1 \lambda + k_2 = 0, \quad (3.31)$$

where

$$k_0 = 1,$$

$$k_1 = m\alpha_1 S^* - \frac{b_1 b_2}{b_3} - (k_1 + p_1 + \mu),$$

$$k_2 = (k_1 + p_1 + \mu) \left[\frac{b_1 b_2}{b_3} - m\alpha_1 S^* \right] - \phi\sigma \left[\frac{b_1 m\alpha_2 S^*}{b_3} - m\alpha_2 S^* \right].$$

It is straightforward to conclude that the two eigenvalues in (3.31) are negative if $k_1 > 0$ and $k_2 > 0$ so that the effective reproductive ratio \mathcal{R}_e is more than unity. Therefore, the disease-pandemic equilibrium is locally asymptotically stable if $k_1 > 0$ and $k_2 > 0$. \square

Theorem 3.4. *The disease-pandemic equilibrium is globally asymptotically stable if the effective reproductive ratio $\mathcal{R}_e > 1$ such that the derivative of the Lyapunov function \mathcal{J} is negative, i.e., $\frac{d\mathcal{J}}{dt} < 0$.*

Proof. To establish the existence of $\mathcal{R}_e > 1$ under global stability for disease-endemic equilibrium, the derivative of the Lyapunov function \mathcal{J} must be negative.

$$\begin{aligned} J(S^*, E^*, I^*, Q^*, R^*, V^*) = & \left(S - S^* - S^* \ln \frac{S^*}{S} \right) + \left(E - E^* - E^* \ln \frac{E^*}{E} \right) \\ & + \left(I - I^* - S^* \ln \frac{I^*}{I} \right) + \left(Q - Q^* - Q^* \ln \frac{Q^*}{Q} \right) \\ & + \left(R - R^* - R^* \ln \frac{R^*}{R} \right) + \left(V - V^* - V^* \ln \frac{V^*}{V} \right). \end{aligned}$$

So that,

$$\begin{aligned} \frac{d\mathcal{J}}{dt} = & \left(1 - \frac{S^*}{S} \right) \frac{dS}{dt} + \left(1 - \frac{E^*}{E} \right) \frac{dE}{dt} + \left(1 - \frac{I^*}{I} \right) \frac{dI}{dt} \\ & + \left(1 - \frac{Q^*}{Q} \right) \frac{dQ}{dt} + \left(1 - \frac{R^*}{R} \right) \frac{dR}{dt} + \left(1 - \frac{V^*}{V} \right) \frac{dV}{dt}. \end{aligned} \quad (3.32)$$

(3.32) can be written as

$$\frac{d\mathcal{J}}{dt} = M + N, \quad (3.33)$$

where

$$\begin{aligned} M = & b \left(1 - \frac{S^*}{S} \right) + m(\alpha_1 S E + \alpha_2 I S + \alpha_3 S V) \left(1 - \frac{E^*}{E} \right) \\ & + \phi \sigma E \left(1 - \frac{I^*}{I} \right) + \phi(1 - \sigma) E \left(1 - \frac{Q^*}{Q} \right) \\ & + (p_1 I + p_2 Q) \left(1 - \frac{R^*}{R} \right) + (n\beta_1 E + n\beta_2 I) \left(1 - \frac{V^*}{V} \right) \end{aligned}$$

and,

$$\begin{aligned} N = & (m\alpha_1 E + m\alpha_2 I + m\alpha_3 V + \mu) \left(1 - \frac{S}{S^*} \right) S^* + (\mu + \phi) \left(1 - \frac{E}{E^*} \right) E^* \\ & + (k_1 + p_1 + \mu) \left(1 - \frac{I}{I^*} \right) I^* + (k_2 + p_2 + \mu) \left(1 - \frac{Q}{Q^*} \right) Q^* \\ & + \mu \left(1 - \frac{R}{R^*} \right) R^* + \tau \left(1 - \frac{V}{V^*} \right) V^*. \end{aligned}$$

It is observed that $M > 0$ while $N < 0$ because $S^* < S, E^* < E, I^* < I, Q^* < Q, R^* < R$ and $V^* < V$. Therefore, there exists disease-endemic equilibrium and by LaSalle's invariance principle [38], the disease-endemic equilibrium is globally asymptotically stable if $M < N$ so that $\frac{d\mathcal{J}}{dt} < 0$. \square

3.3 Local Sensitivity Analysis

The local sensitivity of the key system parameters to COVID-19 spread is computed in (3.34)-(3.37) by using the sensitivity index formula [39, 40].

$$S_b = \frac{m\alpha_1}{\mu(\phi + \mu)} + \frac{\phi\sigma m\alpha_2}{\mu(\phi + \mu)(k_1 + p_1 + \mu)} + \frac{[(k_1 + p_1 + \mu)n\beta_1 + \phi\sigma n\beta_2]m\alpha_3}{\mu\tau(\phi + \mu)(k_1 + p_1 + \mu)} \times \frac{b}{\mathcal{R}_e}, \quad (3.34)$$

$$S_{\alpha_1} = \frac{mb}{\mu(\phi + \mu)} \times \frac{\alpha_1}{\mathcal{R}_e}, \quad (3.35)$$

$$S_{\alpha_2} = \frac{\phi\sigma mb}{\mu(\phi + \mu)(k_1 + p_1 + \mu)} \times \frac{\alpha_2}{\mathcal{R}_e}, \quad (3.36)$$

$$S_{\alpha_3} = \frac{[(k_1 + p_1 + \mu)n\beta_1 + \phi\sigma n\beta_2]mb}{\mu\tau(\phi + \mu)(k_1 + p_1 + \mu)} \times \frac{\alpha_3}{\mathcal{R}_e}. \quad (3.37)$$

4 Simulation and Discussion

To quantify the analytical results, numerical simulation is carried out using parameters values whose sources are from [28] as well as assumption. Graphical profiles are then displayed to visualise the transmission dynamics and mitigation success of COVID-19 within the first six months of the emergence of the pandemic in Nigeria. To obtain the numerical values for the theoretical results in section 3.0, fix $m = 0.5, p_1 = 0.13978, p_2 = 0.23978, b = 0.349, \alpha_1 = 0.03, \alpha_2 = 0.08, \alpha_3 = 0.01, \phi = 0.2, \sigma = 0.01, \mu = 1/54.5, k_1 = 0.015, k_2 = 0.01, n = 0.3, \tau = 10, \beta_1 = 0.00011, \beta_2 = 0.075, S(0) = 100000, E(0) = 8000, I(0) = 1500, Q(0) = 6000, R(0) = 4262, V(0) = 60$. Then the effective reproduction number \mathcal{R}_e in (3.3) is evaluated as $\mathcal{R}_e = 1.35$ from which

$$\mathcal{R}_a = 1.31, \mathcal{R}_b = 0.04 \text{ and } \mathcal{R}_c = 0.00.$$

$\mathcal{R}_a, \mathcal{R}_b$ and \mathcal{R}_c measure the risk of infection from each transmission pathway. Of all the transmission routes, the largest risk (\mathcal{R}_a) is from the exposed to the susceptible individuals. This is because the exposed individuals are asymptomatic but can spread the virus to the susceptible individuals usually in unintentional modes. However, the transmission from other routes (\mathcal{R}_a) and (\mathcal{R}_b) are insignificant. While the insignificant transmission of the virus from the infected individuals might be attributed to the conspicuousness of disease symptoms in the infected individuals which would make other people to keep away from them, the

insignificant contribution from the environment to the general infection risk might be attributed to the presence of parameter n , the reduction factor in the net contribution of infectious individuals (both E and I) to the growth of pathogen in the environment. The sensitivity indices for the transmission parameters α_1, α_2 and α_3 in (3.35)-(3.37) are 0.97, 0.03 and 0.00 respectively. The sensitivity indices for α_1, α_2 and α_3 correlate with the results for $\mathcal{R}_a, \mathcal{R}_b$ and \mathcal{R}_c and a confirmation that transmission from the exposed to the susceptible individuals poses the greatest threat.

In the same manner, the numerical value of the basic reproduction number (\mathcal{R}_0) in (3.4) is also evaluated and $\mathcal{R}_0 = 10.67$ from which

$$\mathcal{R}_x = 2.61, \mathcal{R}_y = 8.05 \text{ and } \mathcal{R}_z = 0.01.$$

Unlike in effective reproduction number \mathcal{R}_e where the transmission from the infected individuals \mathcal{R}_b is insignificant, the contribution of the infected individuals is enormous in the absence of control with $\mathcal{R}_y = 8.05$. The infection risk from the exposed to the susceptible individuals is also higher with $\mathcal{R}_x = 2.61$ than in the effective reproduction number where $\mathcal{R}_a = 1.31$. Generally, the reproduction number of COVID-19, based on scholars' estimation, is in the region 2.0-3.0 which makes it higher than the reproduction numbers of SARS and MERS coronaviruses [21]. The numerical values of the effective reproduction number \mathcal{R}_e and the basic reproduction number \mathcal{R}_0 in the present analysis for Nigeria, 1.35 and 10.67 respectively, indicate that Nigerian government took the prevention and control of COVID-19 with all seriousness at the start of the pandemic. The timely enforcement of lockdown and social distancing order records an outstanding success in averting massive infection and mortality with $\mathcal{R}_e = 1.35$ below the region estimated by the scholars (2.0-3.0).

The experts' projection that Africa will experience the worst impacts of COVID-19 pandemic given the fragile health system in many African countries would have come to pass in Nigeria [29, 31]. The reproduction number $\mathcal{R}_0 = 10.67$ would have been experienced in Nigeria if the mitigation measures were not taken seriously. COVID-19 first case was confirmed in Nigeria on February 27, 2020. The government instituted lockdown on March 30, 2020 and the lockdown did not relax until July 1, 2020 when the government approved partial ease of lockdown by allowing partial reopening of schools for students in terminal classes and removing of ban on inter-state travels. The schools did not open fully until September 21, 2020 while the international flights did not resume until September 5, 2020. Besides, the government through its agency, Nigeria Centre for Disease Control (NCDC), embarks on massive sensitisation of the public on the signs, symptoms,

dangers and preventions of COVID-19 through various media especially text messages from time to time. Individuals who are infected with the virus are supported and well catered for promptly. They are fed and treated free of charge.

As regards the necessary and sufficient conditions for the disease-free equilibrium of the model to be locally asymptotically stable that are established in subsection 3.2, the initial values of a_1 and a_2 are computed with the usual parameters values to ascertain whether the model is locally asymptotically stable around disease-free equilibrium or not. The value of m , the reduction factor in disease transmission potential is then varied to examine the stability behaviour of the model around the disease-free equilibrium, the results of which are stated in Table 3.

Table 3. Stability behaviour of the model

S/No	k_1	p_1	μ	ϕ	m	α_1	b	α_2	σ	a_1	a_2	DFE
1	0.015	0.13978	1/54.5	0.2	0.5	0.03	0.349	0.08	0.01	+0.11	-0.01	Unstable
2	0.015	0.13978	1/54.5	0.2	0.6	0.03	0.349	0.08	0.01	+0.05	-0.02	Unstable
3	0.015	0.13978	1/54.5	0.2	0.7	0.03	0.349	0.08	0.01	-0.01	-0.03	Stable
4	0.015	0.13978	1/54.5	0.2	0.8	0.03	0.349	0.08	0.01	-0.07	-0.04	Stable
5	0.015	0.13978	1/54.5	0.2	0.9	0.03	0.349	0.08	0.01	-0.12	-0.05	Stable

In Table 3, it is observed that the disease-free equilibrium of the model is stable when the reduction factor m in the disease transmission potential is greater than 0.6. The stability of the disease-free equilibrium implies that COVID-19 would not have spread in Nigeria from the initial infection if adequate measures had been put on ground before February 27 2020 when the first case was reported. Experts argued that Nigeria ought to have closed her borders and stopped international flights before February 27 2020. If the steps had been taken, the Italian businessman who brought the virus to Nigeria would have been prevented and Nigeria would have maintained a stable region in Table 3 for as long as possible. However, since the disease was able to spread from the initial infection, the disease-endemic equilibrium of the model is stable and the stability in endemic equilibrium is observable in the region where the reduction factor m in the disease transmission potential is less than 0.7 In Table 3. The disease-endemic equilibrium becomes stable when the disease-free equilibrium is unstable. It is therefore evident that government mitigation strategies (closure of schools, worship centres, markets, offices, leisure spots, businesses as well as awareness campaigns) play crucial roles in shaping the dynamics of COVID-19 in Nigeria during the first six months of emergence. Although the disease spread, the government of Nigeria was able to avert massive infections and mortality from the pandemic through effective mitigation measures. With the same stated parameters values and initial conditions for the state variables, plots are generated for the model in Figure 2 and Figure 3 to visualise the effect of the parameters on the dynamics of the disease.

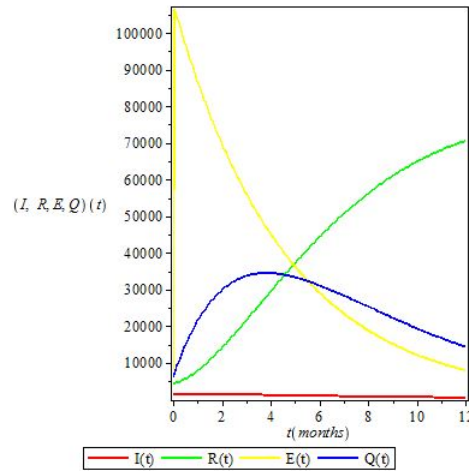


Figure 2: Result of simulation with mitigation measures on ground. $\mathcal{R}_e = 1.35$, $I(t) = 1,038$

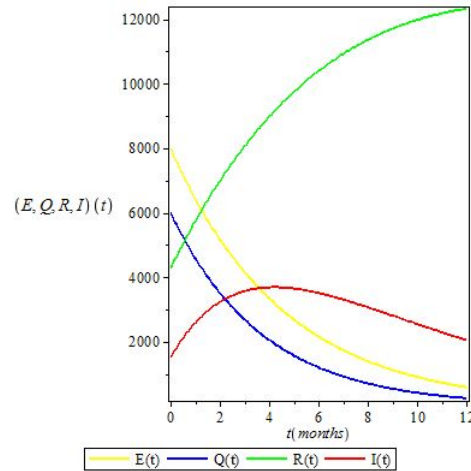


Figure 3: Result of simulation in the absence of mitigation measures. $\mathcal{R}_e = 10.67$, $I(t) = 1,815$

Comparing the initial value of I (i.e., $I(0) = 1500$) with the numerical results of $I(t)$ in Figure 2 and Figure 3, it is evident that things would have gone out of hand if the mitigation measures had not been taken seriously in Nigeria. In Figure 2, the population of infected individuals falls continuously but it rises before it begins to fall in Figure 3. The escalation of the figure of individuals who are

infected with COVID-19 would have spelt doom for Nigeria given the precarious health system but the Nigerian government is able to be on top of the situation with effective mitigation measures that minimise disease transmission and spread to the barest minimum. Key model parameters (transmission parameters α_1 , α_2 and α_3) are varied to perform further numerical simulations to visualise the effect of partial and total mitigation measures on disease spread parameters α_1 , α_2 and α_3 and the population of infectious individuals $I(t)$ in Figure 4 and Figure 5

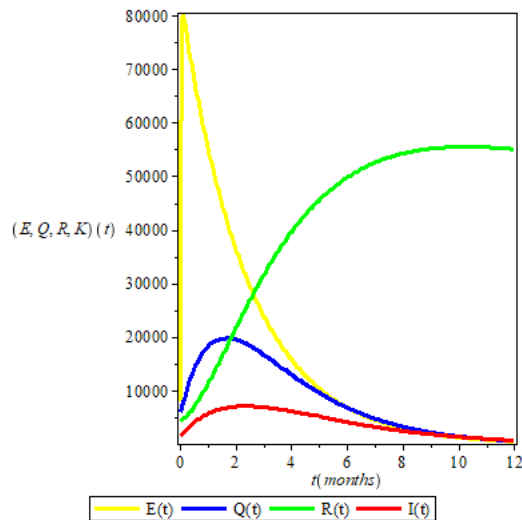


Figure 4: Result of simulation for partial measures.
 $\alpha_1 = 0.003, \alpha_2 = 0.008, \alpha_3 = 0.001$

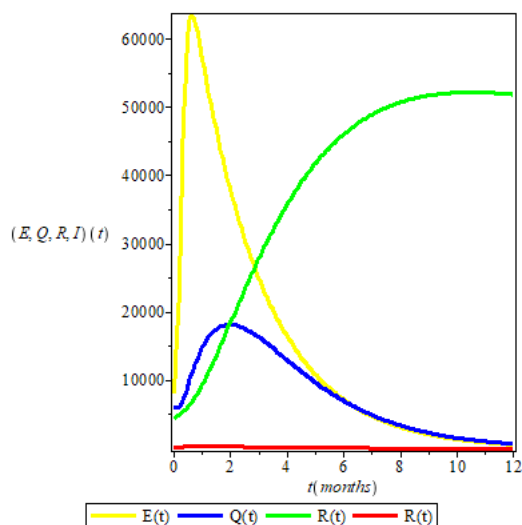


Figure 5: Result of simulation for total measures.

$$\alpha_1 = 0.0003, \alpha_2 = 0.0008, \alpha_3 = 0.0001$$

Whether partial or total, Figure 4 and Figure 5 indicate that government mitigation measures against COVID-19 limit the spread of the disease and the population of individuals who become infectious. The mitigation measures in the form of lockdown and social distancing enforced by the Nigerian government are comprehensive and able to avert massive infections and mortality from COVID-19.

5 Conclusion

The outbreak of COVID-19 brought a fear that the African continent is on the verge of ruin due to widespread poverty and fragile health systems. Nigerians are particularly worried about the fate of the country should the outbreak escalate given the population and the vulnerability index of infectious disease for the county which are in excess of 200 million and 0.27 respectively [31]. However, the pandemic has been effectively managed and the impacts of the outbreak have been largely minimised in Nigeria as of September 3 2020. A mathematical model has been formulated to analyse how massive infections and mortality from COVID-19 have been averted in Nigeria through mitigation measures. The validity of the model is confirmed by establishing positivity and boundedness of its solutions. A thorough analysis is then conducted qualitatively and quantitatively. The qualitative analysis is performed by obtaining the equilibria of the model and deriving the reproduction

numbers both effective reproduction number \mathcal{R}_e and the basic reproduction number \mathcal{R}_0 . The model stability is also examined and it is shown that the disease-free and the disease-endemic equilibria of the model are locally and globally asymptotically stable whenever $\mathcal{R}_e < 1$ and $\mathcal{R}_e > 1$ respectively. The quantitative analysis is finally conducted to justify the theoretical results. Both the theoretical and numerical results indicate that low infections and mortality from COVID-19 during the first six months of COVID-19 emergence in Nigeria are attributed to the effectiveness of government mitigation measures. The effective implementation of non-pharmaceutical interventions can therefore put Nigeria and other African countries in a good position for combatting subsequent emergence of any form of infectious diseases.

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