# *Math 585 Term Paper*

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**Abstract**

This is an extended review of *Mathematical Modeling of COVID-19 Epidemic with Effect of Awareness Programs*. Authored by and cited as: Salihu Sabiu Musa, Sania Qureshi, Shi Zhao, Abdullahi Yusuf, Umar Tasiu Mustapha, Daihai He a. Musa et al. *Mathematical Modeling of COVID-19 Epidemic with Effect of Awareness Programs*. Infectious disease modeling 6 (2021): 448-460.

While COVID-19 has presented a seemingly insurmountable public health challenge to the United States of America, and the rest of the developed (modern) world for that matter, it is hard to grasp how such a virus has impacted countries with a lesser health care system and available awareness programs. *Mathematical Modeling of COVID-19 Epidemic with Effect of Awareness Programs* not only provides a gentle introduction to infectious disease modelling, but a solid proposition for a mathematical model to study the transmission dynamics of COVID-19 specifically in the developing country Nigeria.

Providing better health care for a massive population is difficult so the proposed model offers a different solution: assessing the effect of public awareness on the dynamics of COVID-19 in developing countries and how the results of these dynamics can help control infection rates. The dynamical system introduced by this model is constructed from a non-autonomous equation that is used to induce a system of nonlinear ordinary differential equations. While such a system is difficult in practice to satisfy, qualitative analysis is made by the authors to study which data (sets) provide disease free equilibrium with the introduction of parameters that resemble increased public awareness.

The results of the model suggest that even though effective hospitalizations of infected individuals are still the key to controlling infection rates, public awareness is an efficient resource that can be implemented on a global scale to the system. Arriving to such global

conclusions is shown to be directly derived from local analysis of the model in which the authors demonstrate through numerical simulations and sensitivity analysis. Even though the analysis of COVID-19 embarked on in this paper is largely dependent on numerical analysis and the study of dynamical systems, the results provide key insight into how a pandemic level virus can be controlled with simple data solutions. Such data solutions are shown to be directly linked to awareness programs and how they can be used to mitigate control over COVID-19 not only in a third-world country, but also in the rest of the developed world.

**Keywords:** COVID-19, Epidemic, Mathematical modeling, Reproduction number, public awareness

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# 1 Introduction

SARS-COV-2 or Severe acute respiratory coronavirus 2, is a virus that emerged in Wuhan China and induced a pandemic of coronavirus disease in late 2019 which we know today as COVID-19 (Li et al., 2020; Wu, Leung, & Leung, 2020; Lin et al., 2020a; WHO, 2020a, 2020b; CDC, 2020; Nishiura et al., 2020; Zhao et al., 2020a; Musa, Gao, Zhao, et al., 2020). From this point on the outbreak would escalate to inflict more than 200 countries just in 2020 alone and no vaccine for COVID-19 would be available until 2021 (CDC, 2020; WHO, 2020a, 2020b). Come 2021, the world health organization determined that as of 25 January 2021 the COVID-19 pandemic affected 98,794,942 people including 2,124,193 associated deaths worldwide (WHO, 2020a, 2020b). COVID-19 is transmissible through small droplets from an infected individual when sneezing, coughing, or close contact (CDC, 2020; Li et al., 2020; Musa, Zhao, Wang, et al., 2020; Wang et al., 2020; Wu, Leung, & Leung, 2020). It's important to note a few symptoms that we have become aware of throughout the pandemic: fever, coughing, vomiting, new loss of taste or smell, and or difficulty breathing, persistent pain or pressure in chest (CDC, 2020; Li et al., 2020; Tang et al., 2020; Wu, Leung, & Leung, 2020). As you can see, some are closely related to that of a common cold or flu like variant – but nonetheless are persistent across all COVID-19 cases.

While the United States has treatment options for most, if not all, symptoms of COVID-19 – lesser developed countries struggle**.** In particular, and the focus of our study: Nigeria, the most populous country in sub-Saharan Africa. Basing our model data from such a country allows us to focus on a particular health care system fault in underdeveloped countries, noting that their health care does not provide basic and regular health services for its citizens in normal heath emergencies. This is where we insert such a fault Nigeria's health care system: public awareness of the COVID-19 virus and transmission (NCDC, 2020). With this, we move to making our points precise and begin the development of a model fit with the data from our claims regarding Nigeria's health care. Keep in mind, we are considering a population of individuals who are who are at risk of exposure to COVID-19 that do not know they are at risk - it may be useful to derive a dynamical system from a sum of time dependent compartments that represent disjoint portions of the population.

Since there are numerous ways one could approach this we should be entirely transparent as to how in-depth of an analysis and derivation we will perform/explore. With that said, we will not derive the system rigorously here but rather explore its implications and how things are related. This is much more beneficial for the reader as it will shift focus to the methodology and dynamics of the system rather than that of the techniques used in its derivation. However, even though a rigorous derivation will be avoided, we will provide an ODE block diagram and brief explanation as to how one may observe the relation between the equations that induce our system. In addition, we will summarize what data – whether that be quantitative or qualitative – can be gathered from our analysis of such a model and conclude with how said data can be applied to studying not only COVID-19 reduction, but also infectious disease prevention in general.

## 2 Mathematical Model

As mentioned, now we will put together a dynamical system from a sum of time dependent compartments that represent disjoint portions of the population in Nigeria. These disjoint portions of the population, at time $t \in \mathbb{R}_0^+ \coloneqq \{r \in \mathbb{R} : r \geq 0\}$, are given below as values of real valued functions [1] defined on $\mathbb{R}^+$:

**Table 1**
Interpretation of the state variables of the system

| Variable | Description |
|---|---|
| $\boldsymbol{S_a(t)}$ | Individuals who are aware of the disease and follow the preventive health measures |
| $\boldsymbol{S_u(t)}$ | Individuals who are unaware of the disease and do not follow the preventive health measures |
| $\boldsymbol{E(t)}$ | Exposed |
| $\boldsymbol{A(t)}$ | Asymptomatically infectious |
| $\boldsymbol{I(t)}$ | symptomatically infectious |
| $\boldsymbol{H_m(t)}$ | Mild hospitalized |
| $\boldsymbol{H_s(t)}$ | Severe hospitalized |
| $\boldsymbol{R(t)}$ | Recovered individuals |

In the same vein, since each of these compartments represents a disjoint portion of the population, we define a new function $N(t)$ to be the total human population at time $t$ such that

$$N(t) \coloneqq S_a(t) + S_u(t) + E(t) + A(t) + I(t) + H_m(t) + H_s(t) + R(t).$$

It is important to note that even though we consider the quantity $D(t)$, the number of deceased individuals at time $t$, to be a state variable – it is not a summand of $N(t)$. So, $N(t)$ provides the desired model for the human population of Nigeria at time $t$ and is the basis for deriving our dynamical system. However, we first point out that our model considers person-to-person mode of transmission as the potential transmission route and ignored other routes due to their less impact in community transmission (Musa et al, 450). Therefore, our dynamical system that captures the disease dynamics among the population $N(t)$ can be recovered from the following block diagram (or *ODE flow chart*):

[1] The functions we are considering are of the form $f : \mathbb{R}_0^+ \to \mathbb{R}_0^+$. We require the codomain to also be $\mathbb{R}_0^+$ so we avoid obtaining a *negative* population value for any time $t$.

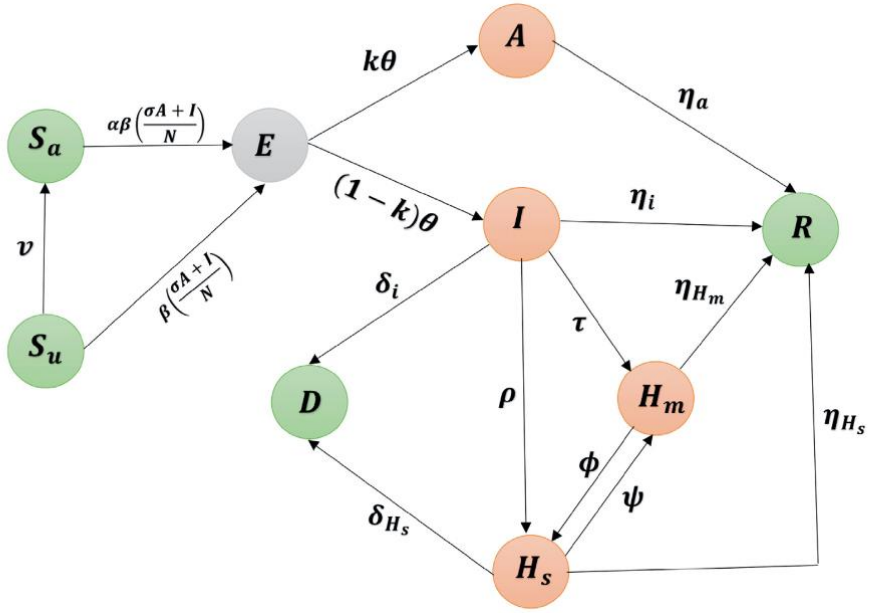


**Fig. 1.** The schematic diagram of the COVID-19 model with awareness programs. The non-infected compartments are represented in green color, the compartment in gray denotes the exposed individuals, while the infected compartments are portrayed in pink color (Musa et al. 450).

In addition, we provide a table of the parameters of the system before the dynamical system so we understand how the rates of change are scaled and influenced:

**Table 2**
Interpretation of the parameters of the system

| Parameter | Description |
|---|---|
| $\boldsymbol{\beta}$ | Community transmission or successful contact rate |
| $\boldsymbol{\alpha}$ | Modification parameter for decrease on infectiousness in $S_a(t)$ |
| $\boldsymbol{\sigma}$ | Infectiousness factor for asymptomatic individuals |
| $\boldsymbol{\nu}$ | Rate at which unaware susceptible will become aware about the disease |
| $\boldsymbol{\theta}$ | Progression rate |
| $\boldsymbol{\kappa}$ | Fraction of infections that become asymptomatic |
| $\boldsymbol{\tau(\rho)}$ | Hospitalization rates from $I(t)$ to $H_m(t)$ |
| $\boldsymbol{\psi}$ | Rate at which the hospitalized individuals more from mild to severe isolation |
| $\boldsymbol{\phi}$ | Rate at which the hospitalized individuals more from severe to mild isolation |
| $\boldsymbol{\delta_j\ (j = i, H_s(t))}$ | COVID-19 induced death rates |
| $\boldsymbol{\eta_l\ (l = a, i, H_m(t), H_s(t))}$ | Recovery Rates |

Thus, keeping in mind **Table 2**, if we follow the derivation arrows in **Fig. 1** we obtain the following system of nonlinear autonomous ordinary differential equations that define our disease dynamics model:

$$\dot{S}_a(t) = -\frac{\alpha\beta S_a(t)(\sigma A(t) + I(t))}{N(t)} + vS_u(t)$$

$$\dot{S}_u(t) = -\frac{\alpha\beta S_a(t)(\sigma A(t) + I(t))}{N(t)} - vS_u(t)$$

$$\dot{E}(t) = \frac{\beta(\sigma A(t) + I(t))\big(\alpha S_a(t) + S_u(t)\big)}{N(t)} - \theta E(t)$$

$$\dot{H}_s(t) = \rho I(t) + \varphi H_m(t) - \big(\eta_{H_s(t)} + \psi + \delta_{H_s(t)}\big)H_s(t)$$

$$\dot{D}(t) = \delta_i I(t) + \delta_{H_s(t)} H_s(t)$$

$$\dot{H}_m(t) = \tau I(t) + \psi H_s(t) - \big(\eta_{H_m(t)} + \varphi\big)H_m(t)$$

$$\dot{I}(t) = (1-k)\theta E(t) - (\eta_i + \tau + \rho + \delta_i)I(t)$$

$$\dot{A}(t) = k\theta E(t) \ - \eta_a A(t)$$

$$\dot{R}(t) = \eta_a A(t) + \eta_i I(t) + \eta_{H_m(t)} H_{m(t)} + \eta_{H_s(t)} H_s(t)$$

Notice how we have adopted the Newtonian time derivative dot notation[2] to denote the derivative of a function with respect to time. This compact notation will serve beneficial as in the next section we will begin a quantitative analysis of the system and explore indications of disease-free equilibrium in the model.

[2] $\dot{f}(t) \equiv \frac{df}{dt}(t)$

## 3 Model Analysis

We now shift our focus to analyzing the dynamics of the system and how we can quantify an indicator of stability for the system. Namely, In the absence of infection the system obtains what is known as a disease-free equilibrium. The local stability of the system can be written rigorously as

$$S_u(t) = E(t) = A(t) = I(t) = H_m(t) = R(t) = D(t) = 0$$

and so, the disease-free equilibrium is given by the 9-tuple

$$Y^0 := \left(S_a^0(t), S_u^0(t), A^0(t), I^0(t), H_m^0(t), H_s^0(t), R^0(t), D^0(t)\right) = (S_a^0(t), 0,0,0,0,0,0,0,0).$$

Furthermore, we can establish the DFE $Y^0$ in terms of a sort of threshold value called *the basic reproduction number* which we will denote as $R_0$. The basic reproduction number intuitively determines whether a disease (in our case COVID-19) can invade a population or not. More precisely, $R_0$ represents the number of secondary COVID-19 cases that would be generated by a typical primary case if placed into a completely susceptible population (Musa et al., 452). While this is a simple concept to grasp, obtaining a closed form expression for $R_0$ is not so straight forward.

To that end, we must employ what is known as a next generation matrix technique to apply to our system. While we will not work through every step to obtain such a closed form expression we will supply the necessary *heavy machinery*, for lack of a better word, to perform such computations. First, let $\mathcal{F}_i(t)$ be the rate of appearance of new infections in compartment $i$, $\mathcal{V}_i^+(t)$ be the rate of transfer of individuals out of compartment $i$, and $\mathcal{V}_i^-(t)$ be the rate of transfer of individuals out of compartment $i$. Note that each of these functions is *at least* twice continuously differentiable (van den Driessche & Watmough, 2002).

**Lemma.** if $t_0$ is a DFE of the system and the system equations are well-defined[3], then the derivatives $D\mathcal{F}(t_0)$ and $D\,\mathcal{V}(t_0)$ – where $\mathcal{V} := \mathcal{V}_i^- - \mathcal{V}_i^+$ – are partitioned as

$$D\mathcal{F}(t_0) = \begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix}, \qquad D\mathcal{V}(t_0) = \begin{pmatrix} V & 0 \\ J_3 & J_4 \end{pmatrix}$$

such that $F$ and $V$ are the $m \times m$ matrices defined by

$$F := \left[\frac{\partial \mathcal{F}_i}{\partial t_j}(t_0)\right], \qquad V := \left[\frac{\partial \mathcal{V}_i}{\partial t_j}(t_0)\right] \quad \text{with } 1 \leq m, \mathrm{j} \leq \mathrm{m}.$$

[3] See the assumptions (A1) - (A5) in van den Driessche & Watmough, 2002 for what makes a transmission model *well defined.*

Note that $F$ is non-negative, $V$ is a non-singular $M$-matrix (block matrix with blocks $J_3$ and $J_4$) and all eigenvalues of $J_4$ have a positive real pat (the proof of this lemma can be found in van den Driessche & Watmough, 2002).

Now, since $F$ is non-negative and $V$ is a non-singular $M$-matrix we have that $V^{-1}$ is non-negative and therefore so is the product $FV^{-1}$. Therefore, we call $FV^{-1}$ *the next generation matrix* for the model and define $R_0 \coloneqq \rho(FV^{-1})$; where $\rho(\cdot)$ is the spectral radius of the matrix of interest. Note that since the spectral radius is the largest absolute value of its eigenvalues, we always obtain a closed form expression for $R_0$; it may not be nice, but we can obtain it. Hence, for our system we write

$$R_0 = \frac{\alpha\beta\sigma\kappa}{\eta_a} + \frac{\alpha\beta(1-\kappa)}{q_1} \quad \text{such that } q_1 = \eta_i + \tau + \rho + \delta_i.$$

Having such a closed form expression allows us to make the following claims regarding the systems *stability*: $Y^0$ is locally-asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$; thus $R_0$ can be thought of as an indicator for our systems stability. While all of this seems very abstract, a nice physical implication of $R_0$ is the following: if $R_0 < 1$, then a small influx of COVID-19 cases will not generate a massive outbreak among the population (Musa et al., 452).

Before we move onto the next section, we should briefly discuss the validation carried out by the authors on the model we have presented. Validating a model of this type is performed by assigning values to the systems parameters and choosing particular initial conditions for our state variables such that the difference between our extrapolation and the number of real COVID-19 cases over a range of time is minimized. Below, we revisit **Table 2** but this time with each parameter assigned the appropriate value alongside a range of values the parameter should run through (with units of days$^{-1}$):

**Table 3**
Baseline values of the parameters used in the model

| Parameter | Value and Range |
|---|---|
| $\boldsymbol{\nu}$ | $1.73768e-02\ \ (0.01-0.5)$ |
| $\boldsymbol{\alpha}$ | $3.06589e-01\ (0.01-0.95)$ |
| $\boldsymbol{\beta}$ | $8.48007e-01\ (0.599-1.68)$ |
| $\boldsymbol{\sigma}$ | $6.74971e-02\ (0.04-0.6)$ |
| $\boldsymbol{\theta}$ | $8.79588e-01\ (0.05-0.95)$ |
| $\boldsymbol{\kappa}$ | $1.63179e-02\ (0-1)$ |
| $\boldsymbol{\eta_a}$ | $3.67068e-02\ (\frac{1}{28}-\frac{1}{3})$ |
| $\boldsymbol{\eta_i}$ | $9.17384e-03\ \ (\frac{1}{1000}-\frac{1}{3})$ |
| $\boldsymbol{\psi}$ | $1.69055e-01\ (0.001-0.5)$ |
| $\boldsymbol{\eta_{H_m(t)}}$ | $0.11624\ (0-1)$ |
| $\boldsymbol{\eta_{H_s(t)}}$ | $0.155\ (0-1)$ |
| $\boldsymbol{\delta_i}$ | $0.015\ (0.01-0.05)$ |

| | |
|---|---|
| $\delta_{H_s(t)}$ | $0.025\ (0.01 - 0.05)$ |
| $\mu$ | $0.00005\ (0.00003 - 0.00006)$ |
| $\tau$ | $0.1259\ (0.09 - 0.51)$ |
| $\rho$ | $0.13266\ (0.001 - 0.5)$ |
| $\phi$ | $0.0341\ (0.001 - 0.5)$ |

Similarly, we apply the following initial conditions to the system variables: $S_a(0) = 130000000$, $S_u(0) = 76000000$, $E(0) = 22$, $A(0) = 10$, $I(0) = 111$, $H_m(0) = 5$, $H_s(0) = 0$, $R(0) = 0$, and $D(0) = 0$. The global error in our approximation with the above choices is roughly $1.2861e - 01$ which, according to the authors (Musa et al., 453), is reasonably small enough. We can visualize this data with the two plots below in **Fig. 2**:

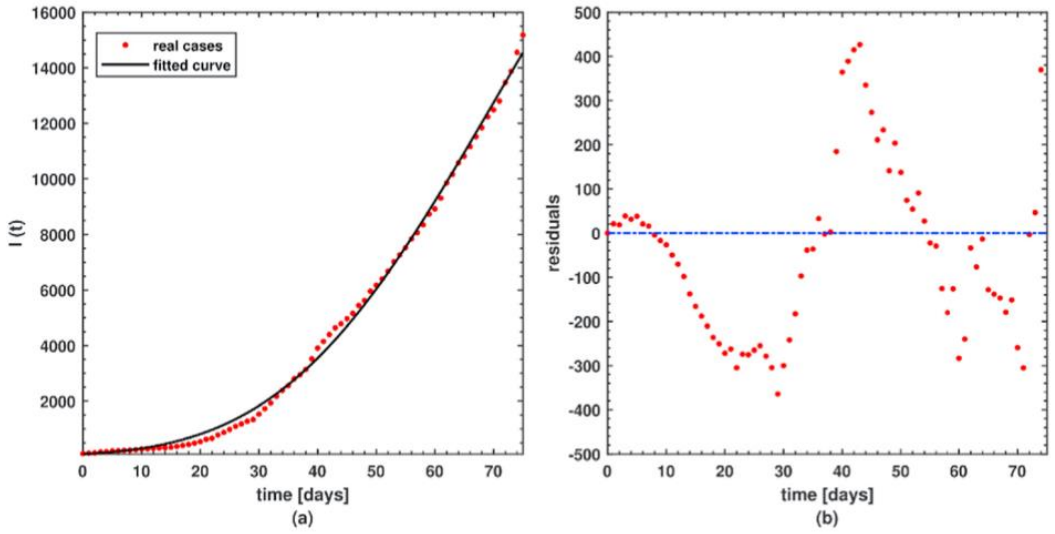


**Fig. 2. (a)** The daily COVID-19 cumulative cases time series in Nigeria from March 29 to June 12, 2020 with the best fitted curve from simulations of the proposed model and (b) the residuals for the best fitted curve (Musa et al., 454)

Note that rather than viewing the whole phase space, we will just observe the curve $I(t)$ to see how effective our approximation has performed vs. the number of real cases in plot **(a)**. The curve on the right in plot **(b)** reports the residual data (which is measured using the $\ell^2 -$ norm[4]) that wasn't "hit" by the fitted curve such that the blue horizontal line represents the approximation (and the legend in plot **(a)** applies to plot **(b)**). An effective way to interpret plot **(b)** from plot **(a)** is the following: whenever the reported data appears under the approximation then the residues will be shown below the horizontal line and vice versa for when the real cases are *above* the fitted curve. Ironically even though **(b)** looks like a scattered mess, when residues

[4] Let $x, y \in \mathbb{R}^2$.The $\ell^2 -$ norm on $\mathbb{R}^2$ of the difference $x - y$ is defined as $\|x - y\|_2 := \sqrt{\sum_{k=1}^{2} |x_k - y_k|^2}$.

are observed to be scattered then the fitting is justified, *i.e.*, the proposed approximation fits the data well (Musa et al., 453).

## 4 Results/Numerical Example

Now that we have a working knowledge of the mathematics used in constructing our model and its DFEs (if multiple exist), we are ready to reap the results of a numerical example that the authors cover. First, it's important to note that what makes this model special is the "disjointness", for lack of a better word, of the individual summands in the original $N(t)$ equation: it allows us to pick apart the dynamics and study how varying parameters influence each component. In particular, we will see the effect of awareness programs on the disease dynamics of COVID-19 in the $I(t)$ compartment once again but this time with a variation of parameters over a time interval of $[0,300]$. Let us consider the values assigned to the parameters in **Table 3** but only this time we will vary $\beta$, $\alpha$, and $\sigma$ in plots **(a), (b),** and **(c)** respectively in **Fig. 3** below.

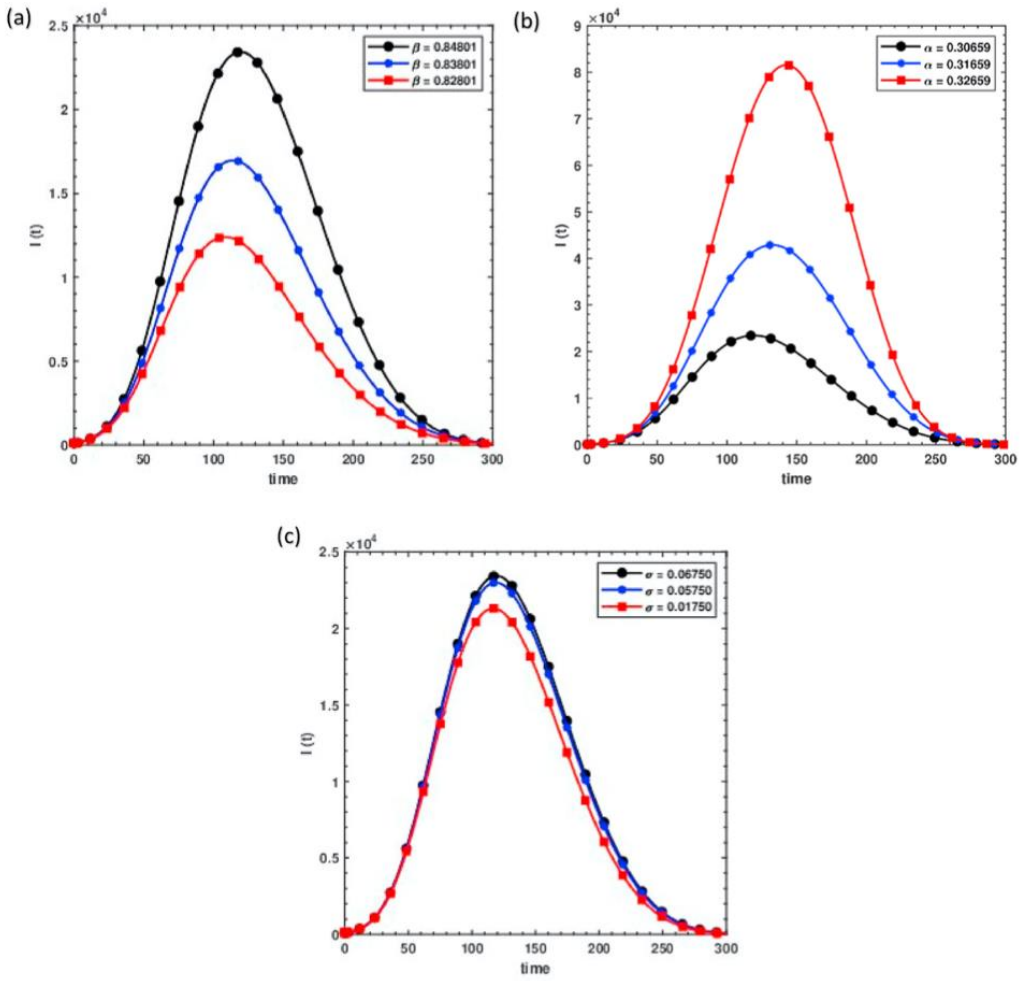


**Fig. 3.** Simulations of the model for various awareness programs under control measures with varying values of **(a)** (community contact rate), **(b)** (modification parameter for deceased on infectiousness in Sa compartment), and **(c)** (infectiousness factor for asymptomatic individuals) while using the parameters' values given in **Table 2** (Musa et al., 455).

Notice first in plot **(c)** if we increase beta, alpha and sigma – all of which influence the rate at which infection spreads – we have an increased number of systematically infected individuals. However, if we consider public awareness programs, in plot **(a)** we see that a slight decrease in community contact rate can seriously decrease the level of infection.

Similarly in plot **(b)**, public awareness programs also means we have a larger value for alpha – which dampens the spread of infection to individuals who are aware of the disease and follow preventative measure – and a smaller value of sigma which is the infectiousness factor for asymptomatic individuals. The results indicate that lack of proper and constant awareness programs for susceptible and exposed individuals could lead to a larger prevalence of COVID-19 especially in a country with already overwhelmed health care system such as Nigeria. With that said, the key biological parameters that are essential to desired results are $\alpha$, $\beta$, $\sigma$ as we saw in **Fig. 3**. Adjusting these through simulation is key to understanding how to best optimize the model for the current population.

## 5 Discussion

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While our model is fairly easy to analyze in different compartments, it is certainly a complicated and dense abstraction of COVID-19 amongst a dense population. In order to even derive the model analytically – whereas we discussed arguably only qualitatively – we would have to preface this review with an entire paper purely of derivation. While there aren't any comparisons in this paper to previous models of the like, the author's do discuss a few techniques they would like to visit in the future that would prove more efficient for numerical simulations. In this paper, the authors apply a nonlinear least-squares curve fitting method with the help of a routine **fminsearch** from the MATLAB Optimization Toolbox for fitting the real cases of the COVID-19 cases in Nigeria to the approximation between 27 February and 12 June 2020. In the future, the authors however wish to revise this model by means of the following quote from the text: "…*Our future studies would include the use of Markov Chain Monte Carlo sampling technique for data fitting process to obtain the unknown biological parameters of the model with associated 95% credible intervals…"* (Musa et al., 457). In addition, they also emphasize that they would like to further the analysis of the dynamics of the system and study ways to improve detail in other compartments.

The model overall is very flexible in the sense that changing the parameters will still allow for the model to perform (as you would hope, otherwise we wouldn't have a very good model at all). However, while there are limitations of this model there is still one that should be considered: could we apply this model globally? The answer to this is a resounding *no* since if we considered the health care of the USA compared to the health care in Nigeria, the simulations of the model, let alone the parameters, would be horribly skewed. However, the purpose of this model is to be applied to an isolated population so global notions need not apply to begin with - although, they are important to recognize. Furthermore, as the authors stated: the precision of the model can only be improved by tuning the analysis and testing different numerical methods for curve fitting to lower the approximation error relative to the true solution points.

# 6 Conclusion

*Mathematical Modeling of COVID-19 Epidemic with Effect of Awareness Programs* provides a gentle introduction into modeling transmission dynamics and mathematical epidemiology in general. Throughout the paper we have seen that model simulations show that a lack of public awareness leads to $R_0 > 1$ and an aggressive spread of COVID-19 throughout the community. Furthermore, Key biological parameters that are essential to implementing public awareness are as follows: $\alpha$, $\beta$, $\sigma$ as we saw in the plots in **Fig.3**. Adjusting these parameters through simulation is key to understanding how our model progresses through time. These simulations show that adequate awareness and enlightenment programs in the most vulnerable communities is an effective way to fight against the disease especially in countries with poor health care systems.

We can generalize these summaries to other infectious diseases other than COVID-19 as we noticed the model doesn't specifically rely on COVID-19 being the virus we are modeling. This notion of the model being well-defined is key for any disease transmission model to be reproducible in further studies. With that said, the model is fit to describe most infectious diseases up to the choice of numerical methods in order to visualize how the model extrapolates the evolution of such a disease. The author's work has shown that not only does public awareness provide an effective disease prevention strategy but the true nature of complexity that is COVD-19 prevention and eradication.

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