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ANALYSIS OF SMOKING AND COVID-19 MATHEMATICAL MODEL

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ABSTRACT

Smoking and COVID-19 have similar effects in the body system, i.e. damaging the airways and lung function. In this work, a mathematical model to study smoking and COVID-19 transmission was studied. The model was proven to have feasible region and it is well-posed mathematically and epidemiologically, the model was further proven to have positive solutions. The basic reproduction number was computed using the next generation method and sensitivity analysis was carried out. The results show that the disease-free equilibrium is locally and globally stable, the most sensitive parameter is the contact rate. The simulation shows that, curtailing rate of contact between exposed, infected individuals and susceptible human population will reduce the spread of the diseases, also giving attention to recovery strategies and controls will reduce to minimum the disease transmission. Therefore, it is recommended that stakeholders should give attention in controlling smoking habits, and prevention and treatment of COVID-19 infected individuals.

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1. INTRODUCTION

Both COVID-19 and smoking adversely impact the respiratory system, damaging the airways and impairing lung function [1]. Smoking is one of the most challenging public health problems globally, accounting for 9% of all deaths worldwide, and more than half of smokers die from smoking-related diseases. It significantly increases the risk of various diseases, including caners of several organs, respiratory diseases, diabetes and cardiovascular diseases (CVDs) [2], [3]. Smoking can damage nearly all organs of the human body and is one of the main risk factors for respiratory and infectious diseases [4].

Coronavirus disease (COVID-19) is an infectious disease caused by severe acute respiratory syndrome coronavirus (SARS-CoV-2), which was identified in China in December 2019 [5]. Studies shows that those with a history of smoking had a higher risk of getting severe COVID-19 progression compared with those who never smoked [6], [7], [8], [9]. The deadly corona virus primarily affects lungs. Smoking impairs lungs, which makes it hard to fight against corona [10]. A review conducted shows that smokers were 1.4 times more likely to have severe symptoms of COVID-19 and approximately 2.4 times more likely to be admitted to an Intensive Care Unit (ICU) or die compared with non-smokers [11]. In the work of [12], it was concluded that the smoking was positively associated with a higher rate of Infectious Respiratory Diseases (IRD). [7] also carried out a systematic review on smoking and risk of negative outcomes among COVID-19 patients. The result shows that current and former smoking significantly increases the risk of COVID-19 severity and death. Also, [13] in the comprehensive meta-analysis on the role of smoking in COVID-19 progression, it was found that a 30% - 50% excess risk of COVID-19 progression for current and former smokers compared with individuals who have never smoked. Few Mathematical models have been formulated and analyzed to understand disease dynamics among smokers, tuberculosis [14], lung cancer [15], HIV/AIDS [16], but none has considered smoking and Covid-19 dynamics.

COVID-19 continues to be a global threat to public health with million confirmed cases and reported death [17]. One of potential risk factor of COVID-19 infection is smoking [18], [19]. Evidence indicated that COVID-19 patients with any history of smoking were more likely to experience severe or critical COVID-19 health outcomes when compared to individuals without any history of smoking [20]. Understanding the dynamics of how COVID-19 and smoking interact mathematically has not be explored adequately. Therefore, the study is aimed at developing a mathematical model to study smoking and COVID-19 dynamics.

2. RESEARCH METHODS

This research will be conducted using literature method, with the following objectives and steps

1. Formulate a Mathematical Model of Smoking and Covid-19 Infection, here a non-linear differential equation will be used in formulating a system of equations.
2. Compute the Reproduction Number of the Model. In this computation, the next generation method is used to compute the reproduction number of the model equation
3. Carry out Stability analysis of the Model. The disease-free equilibrium of the model is established, the condition for the local and global stability of the disease-free equilibrium is determined.
4. Carry out sensitivity Analysis of the Model. The method established in [21] is applied.

2.1 Model Formulation

A mathematical model is developed to study smoking and COVID-19 dynamics. The model is subdivided into seven classes: Non-Smokers(S_1); Smokers(S_2); Non Smoker Exposed to Covid-19(E_1); Smoker Exposed to Covid-19(E_2); Non Smoker infected with Covid-19(I_1); Smoker infected with Covid-19(I_2); Recovery (R); Population of Non-Smoker (N_1); Population of Smokers (N_2). The Non-Smoker

human population (S_1) is increased by the product recruitment rate and a proportion of the human population that don't smokes $\pi\Lambda$ and leaves via contact rate, natural death rate μ and a the rate at which non-smokers become smokers τ. The smoker population class (S_2) is increased by the rate at which non-smokers become smokers τ, and decreases by the contact rate, natural death rate and smoking induced death rate. The non-smokers COVID-19 exposed class (E_1) is increased by contact rate and reduced by natural death rate μ, rate of progress to non-smoker infected class σ_1 and recovery rate θ_1. The smokers COVID-19 exposed class (E_2) increased by the contact rate for smoker class, reduced by the natural death rate μ, rate of progress to the smoker infected class σ_2 and recovery rate for the exposed class θ_2. The non-smokers infected class (I_1) is increased by the progress rate from non-smoker exposed class σ_1 and rate at which infected smokers become non-smokerρ, it is reduced by natural death rate μ, recovery rate θ_1 and covid-19 induced death rate δ_2. The smoker Covid-19 infected class (I_2) is increased by the progression from the smoker exposed class σ_2, reduced by the natural death rate μ, covid-19 induced death rate δ_3, rate at which smoker becomes non-smokers$\rho$and recovery rate θ_4. The recovery class is increased by the rate of recovery for the exposed classes and infected classes reduced by the natural death rate of the recovery class.

2.1.1 Model Assumptions

1. Both the Exposed and Infected individuals can transmit the virus.
2. The smoker can also die as a result of complication from smoking

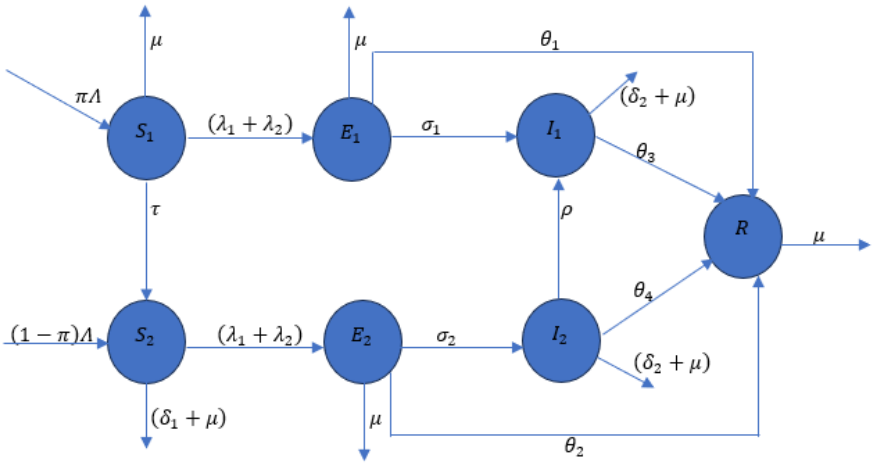

Figure 1. Schematic Diagram

From the schematic diagram above we have the following non-linear differential equations:

$$\frac{dS_1}{dt} = \pi\Lambda - \tau S_1 - (\lambda_1 + \lambda_2 + \mu)S_1, \quad (1)$$

$$\frac{dS_2}{dt} = (1-\pi)\Lambda + \tau S_1 - (\lambda_1 + \lambda_2 + \delta_1 + \mu)S_2, \quad (2)$$

$$\frac{dE_1}{dt} = (\lambda_1 + \lambda_2)S_1 - (\sigma_1 + \mu + \theta_1)E_1, \quad (3)$$

$$\frac{dE_2}{dt} = (\lambda_1 + \lambda_2)S_2 - (\sigma_2 + \mu + \theta_2)\dot{E_2}, \quad (4)$$

$$\frac{dI_1}{dt} = \sigma_1 E_1 + \rho\dot{I_2} - (\delta_2 + \mu + \theta_3)I_1, \quad (5)$$

$$\frac{dI_2}{dt} = \sigma_2 E_2 - (\rho + \delta_2 + \mu + \theta_4)\dot{I_2}, \quad (6)$$

$$\frac{dR}{dt} = \theta_1 E_1 + \theta_2 E_2 + \theta_3 I_1 + \theta_4 I_2 - \mu R. \tag{7}$$

Table 1. Model Parameters

Parameters	Description
Λ	Recruitment Rate
π	Proportion entering non-smoker compartment
$(1-\pi)$	Remaining Proportion in the Smokers compartment
τ	Rate at which non-smokers become smokers
λ_1	Force of infection for non-smokers
λ_2	Force of infection for smokers
$\beta_{1,2}$	Contact Rate
ξ	Account for reduction of infection for non-smokers compared to smokers
ρ	Progression rate from infected smoker to non-smoker
μ	Natural death rate
δ_1	Death as a result of smoking
δ_2	Covid-19 induced death rate
$\theta_{1,2,3,4}$	Recovery rate
$\varphi_{1,2}$	Relative infectiousness of infected individuals compared with exposed individuals
$\sigma_{1,2}$	Progression from Exposed to Infected class
N_1	Population of non-smokers
N_2	Population of smokers
N	Total Population

$$\lambda_1 = \frac{(1-\xi)\beta_1(E_1 + \varphi_1 I_1)}{N_1}; \lambda_2 = \frac{\beta_2(E_2 + \varphi_2 I_2)}{N_2},$$

$$N = N_1 + N_2. \tag{8}$$

3. RESULTS AND DISCUSSION

3.1 Positivity of the Solutions

We show that all variables of the model will remain non-negative for all $t > 0$

Lemma 1. *Let the initial conditions for the model systems in Eqs. (1) - (7) be* $\{S_1(0) > 0, S_2(0) > , E_1(0) > 0, E_2(0) > 0, I_1(0) > 0, I_2(0) > 0, R(0) > 0\} \in \Omega$. *The solution set* $\{S_1(t), S_2(t), E_1(t), E_2(t), I_1(t), I_2(t), R(t)\}$ *of the system Eqs. (1) - (7) are positive for all* $t > 0$.

Proof. From Eq. (1) we have

$$\frac{dS_1}{dt} = \pi\Lambda - \left(\frac{(1-\xi)\beta_1(E_1 + \phi_1 I_1)}{N_1} + \frac{\beta_2(E_2 + \phi_2 I_2)}{N_2} + \tau + \mu\right) S_1$$

$$\frac{dS_1}{dt} \geq -(\lambda_1 + \lambda_2 + \tau + \mu) S_1. \tag{9}$$

Integrating by separating the variables and applying initial conditions, we have

$$S_1(t) \geq S_1(0)\, exp\left(-\int (\lambda_1 + \lambda_2 + \tau + \mu)\right) dt > 0. \tag{10}$$

In the above inequality, $S_1(0)$ denotes the initial population which is positive obviously, and thus $S_1(t)$ is positive. We can follow that the above method to show positivity of the rest model variables, $S_2(t) >, E_1(t) > 0, E_2(t) > 0, I_1(t) > 0, I_2(t) > 0, R(t) > 0$

3.2 Invariant Region

The model solution is bounded in an invariant region,

$$\Omega = \left\{ \left(S_1(t), S_2(t), E_1(t), E_2(t), I_1(t), I_2(t), R(t)\right) \in \Re_+^7 ; N \leq \frac{\pi}{\mu} \right\}. \tag{11}$$

The total population at any time,t, adding all the equations we have

$$\frac{dN}{dt} = \pi - \mu N - \delta_1 S_2 - \delta_2 I_1 - \delta_3 I_2. \tag{12}$$

Neglecting the Smoking and Covid-19 induced death rate, we have

$$\frac{dN}{dt} \leq \pi - \mu N. \tag{13}$$

Solving Eq. (13) we have

$$N(t) \leq \frac{\pi}{\mu} + \left(N_0 - \frac{\pi}{\mu}\right) e^{-\mu t}. \tag{14}$$

It therefore implies that as $t \to \infty, N(t) \leq \frac{\pi}{\mu}$ whenever $N(0) \leq \frac{\pi}{\mu}$. Therefore, the non-negative solution set of the model Eqs. (1) - (7) enters the feasible regionΩ, which is a positively invariant set. Also, the system Eqs. (1) - (7) is epidemiologically and mathematically well-posed in the closed setΩ.

3.3 Disease-Free Equilibrium State (DFE) ($\boldsymbol{\varepsilon_0}$)

Epidemiologically, a disease-free equilibrium can be established when there is no disease in the community. The disease-free equilibrium of the system of model Eqs. (1) - (7) is given by

$$\varepsilon_0 = (S_1^0, S_2^0, E_1^0, E_2^0, I_1^0, I_2^0, R^0) = \left(\frac{\pi\Lambda}{(\tau+\mu)}, \frac{(\tau+\mu-\pi\mu)\Lambda}{(\delta_1+\mu)(\tau+\mu)}, 0,0,0,0,0 \right). \tag{15}$$

3.4 Reproduction Number ($\boldsymbol{R_0}$) and Stability of Disease-Free Equilibrium (DFE)

From an epidemiological viewpoint, the reproduction number is defined as the average number of secondary infections when a typical infection enters an entirely susceptible individual. Applying the next-generation method [20], we have

$$F = \begin{pmatrix} (\lambda_1+\lambda_2)S_1 \\ (\lambda_1+\lambda_2)S_2 \\ 0 \\ 0 \end{pmatrix} V = \begin{pmatrix} (\sigma_1+\mu+\theta_1)E_1 \\ (\sigma_2+\mu+\theta_2)\dot{E}_2 \\ -\sigma_1 E_1 - \rho\dot{I}_2 + (\delta_2+\mu+\theta_3)I_1 \\ -\sigma_2 E_2 + (\rho+\delta_3+\mu+\theta_4)\dot{I}_2 \end{pmatrix}, \tag{16}$$

$$F = \begin{pmatrix} \frac{(1-\xi)\beta_1 S_1}{N_1} & \frac{\beta_2 S_1}{N_2} & \frac{(1-\xi)\varphi_1\beta_1 S_1}{N_1} & \frac{\varphi_2\beta_2 S_1}{N_2} \\ \frac{(1-\xi)\beta_1 S_2}{N_1} & \frac{\beta_2 S_2}{N_2} & \frac{(1-\xi)\varphi_1\beta_1 S_2}{N_1} & \frac{\varphi_2\beta_2 S_2}{N_2} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, V = \begin{pmatrix} \eta_1 & 0 & 0 & 0 \\ 0 & \eta_2 & 0 & 0 \\ -\sigma_1 & 0 & \eta_3 & -\rho \\ 0 & -\sigma_2 & 0 & \eta_4 \end{pmatrix}. \tag{17}$$

Where

$$\begin{aligned} \eta_1 &= (\sigma_1+\theta_1+\mu), \\ \eta_2 &= (\sigma_2+\theta_2+\mu), \\ \eta_3 &= (\delta_2+\theta_3+\mu), \\ \eta_4 &= (\rho+\delta_3+\theta_4+\mu). \end{aligned} \tag{18}$$

At Disease-Free Equilibrium,

$$F=\begin{pmatrix} (1-\xi)\beta_1 & \frac{\beta_2\pi(\delta_1+\mu)}{(\tau+\mu-\pi\mu)} & (1-\xi)\varphi_1\beta_1 & \frac{\varphi_2\beta_2\pi(\delta_1+\mu)}{(\tau+\mu-\pi\mu)} \\ \frac{(1-\xi)\beta_1(\tau+\mu-\pi\mu)}{\pi(\delta_1+\mu)} & \beta_2 & \frac{(1-\xi)\varphi_1\beta_1(\tau+\mu-\pi\mu)}{\pi(\delta_1+\mu)} & \varphi_2\beta_2 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}. \quad (19)$$

Given the following:

$$V^{-1}=\begin{pmatrix} \frac{1}{\eta_1} & 0 & 0 & 0 \\ 0 & \frac{1}{\eta_2} & 0 & 0 \\ \frac{\sigma_1}{\eta_1\eta_3} & \frac{\sigma_2}{\eta_2\eta_3\eta_4} & \frac{1}{\eta_3} & \frac{\rho}{\eta_3\eta_4} \\ 0 & \frac{\sigma_2}{\eta_2\eta_4} & 0 & \frac{1}{\eta_4} \end{pmatrix}, \quad (20)$$

$$FV^{-1}=\begin{pmatrix} \frac{(1-\xi)\beta_1}{\eta_1}+\frac{(1-\xi)\beta_1\kappa_1}{\eta_1\eta_3} & \frac{\beta_2\kappa_4}{\eta_2}+\frac{(1-\xi)\beta_1\kappa_2}{\eta_2\eta_3\eta_4}+\frac{\beta_2\kappa_5}{\eta_2\eta_4} & \frac{(1-\xi)\varphi_1\beta_1}{\eta_3} & \frac{(1-\xi)\beta_1\kappa_3}{\eta_3\eta_4}+\frac{\beta_2\varphi_2\kappa_4}{\eta_4} \\ \frac{(1-\xi)\beta_1\kappa_6}{\eta_1}+\frac{(1-\xi)\beta_1\kappa_1\kappa_6}{\eta_1\eta_3} & \frac{\beta_2}{\eta_2}+\frac{(1-\xi)\beta_1\kappa_2\kappa_6}{\eta_2\eta_3\eta_4}+\frac{\beta_2\varphi_2\sigma_2}{\eta_2\eta_4} & \frac{(1-\xi)\varphi_1\beta_1\kappa_6}{\eta_3} & \frac{(1-\xi)\beta_1\kappa_3\kappa_6}{\eta_3\eta_4}+\frac{\beta_2\varphi_2}{\eta_4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}. \quad (21)$$

The spectral radius of $\rho(FV^{-1})$, gives the reproduction number as

$$R_0=\frac{(1-\xi)\beta_1\left(\rho Q\varphi_1\sigma_2\eta_1+\pi P\eta_2\eta_4(\kappa_1+\eta_3)\right)+(\varphi_2\sigma_2+\eta_4)\pi P\beta_2\eta_1\eta_3}{\pi P\eta_1\eta_2\eta_3\eta_4}, \quad (22)$$

where

$$\left.\begin{aligned} \kappa_1&=\varphi_1\sigma_1 \\ \kappa_2&=\rho\varphi_1\sigma_2 \\ \kappa_3&=\rho\varphi_1 \\ \kappa_4&=\frac{\pi P}{Q} \\ \kappa_5&=\frac{\varphi_2\sigma_2\pi P}{Q} \\ \kappa_6&=\frac{Q}{\pi P} \\ P&=(\delta_1+\mu) \\ Q&=(\tau+\mu-\pi\mu) \end{aligned}\right\}. \quad (23)$$

With the next generation matrix method used, it implies that the disease-free equilibrium point is asymptotically stable, as the method supersedes the stability process of equilibrium point. Hence giving us the theorem below.

Theorem 1. *The disease-free equilibrium ε_0 of the smoking and covid-19 model Eqs. (1) - (7) is locally asymptotically stable (LAS) if and only if $R_0<1$ and unstable if otherwise.*

Proof. We obtain the Jacobian Matrix of the system Eqs. (1) - (7) at the disease-free equilibrium

$$J(S_1^0, S_2^0, 0,0,0,0,0) =$$

$$\begin{pmatrix} -(\tau+\mu) & 0 & -(1-\xi)\beta_1 & -\beta_2\kappa_4 & -(1-\xi)\varphi_1\beta_1 & -\beta_2\varphi_2\kappa_4 & 0 \\ \tau & -(\delta_1+\mu) & -(1-\xi)\beta_1\kappa_6 & -\beta_2 & -(1-\xi)\varphi_1\beta_1\kappa_6 & -\beta_2\varphi_2 & 0 \\ 0 & 0 & -\eta_1+(1-\xi)\beta_1 & \beta_2\kappa_4 & (1-\xi)\varphi_1\beta_1 & \beta_2\varphi_2\kappa_4 & 0 \\ 0 & 0 & (1-\xi)\beta_1\kappa_6 & -\eta_2+\beta_2 & (1-\xi)\varphi_1\beta_1\kappa_6 & \beta_2\varphi_2 & 0 \\ 0 & 0 & \sigma_1 & 0 & -\eta_3 & \rho & 0 \\ 0 & 0 & 0 & \sigma_2 & 0 & -\eta_4 & 0 \\ 0 & 0 & \theta_1 & \theta_2 & \theta_3 & \theta_4 & -\mu \end{pmatrix}. \quad (24)$$

Three of the roots of the Jacobian matrix is $-(\tau+\mu)<0, -(\delta_1+\mu)<0, -\mu<0$, for the remaining roots, we get the following characteristics equation:

$$\lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4 = 0, \quad (25)$$

where

$$b_1 = (\eta_1+\eta_2+\eta_3+\eta_4-\beta_2-(1-\xi)\beta_1),$$
$$b_2 = \begin{pmatrix}\eta_1\eta_2+(\eta_1+\eta_2-\beta_2)\eta_3+(\eta_1+\eta_2+\eta_3-\beta_2)\eta_4\\ -\beta_2(\sigma_2\varphi_2+\eta_1)-(1-\xi)(\sigma_1\varphi_1+\eta_2+\eta_3+\eta_4)\end{pmatrix},$$
$$b_3 = \begin{pmatrix}\eta_1\eta_2(\eta_3+\eta_4)+\eta_3\eta_4(\eta_1+\eta_2)-\beta_2(\eta_1\eta_3+\eta_1\eta_4+\eta_3\eta_4-\sigma_2\varphi_2(\eta_1-\eta_3))-\\ (1-\xi)\beta_1(\sigma_1\varphi_1(\eta_2+\eta_4)+\rho Q\sigma_2\varphi_1+\eta_2\eta_3+\eta_2\eta_4+\eta_3\eta_4)\end{pmatrix},$$
$$b_4 = (1-R_0). \quad (26)$$

The model is locally asymptotically stable when $R_0<1$ and unstable if $R_0>1$.

Theorem 2. *The disease-free equilibrium of equation (1) - (7) is globally asymptotically stable, if $R_0<1$ and unstable if $R_0>1$.*

Proof. We consider the quadratic Lyapunov function for the model equation (1) - (7):

$$V(t) = (S_1 - S_1^* InS_1) + (S_2 - S_2^* InS_2) + E_1 + E_2 + I_1 + I_2 + R. \quad (27)$$

Differentiating with respect to time, we have

$$\dot{V}(t) = \dot{S}_1\left(1-\frac{S_1^*}{S_1}\right) + \dot{S}_2\left(1-\frac{S_2^*}{S_2}\right) + \dot{E}_1 + \dot{E}_2 + \dot{I}_1 + \dot{I}_2 + \dot{R}, \quad (28)$$

$$\begin{aligned}\dot{V}(t) &= \left(1-\frac{S_1^*}{S_1}\right)\left(\pi\Lambda - \tau S_1 - \left(\frac{(1-\xi)\beta_1(E_1+\phi_1 I_1)}{N_1} + \frac{\beta_2(E_2+\phi_2 I_2)}{N_2} + \mu\right)S_1\right)\\ &+\left(1-\frac{S_2^*}{S_2}\right)\left((1-\pi)\Lambda + \tau S_1 - \left(\frac{(1-\xi)\beta_1(E_1+\phi_1 I_1)}{N_1} + \frac{\beta_2(E_2+\phi_2 I_2)}{N_2} + \delta_1 + \mu\right)S_2\right)\\ &+\left(\frac{(1-\xi)\beta_1(E_1+\phi_1 I_1)}{N_1} + \frac{\beta_2(E_2+\phi_2 I_2)}{N_2} + \mu\right)S_1 - (\sigma_1+\mu+\theta_1)E_1\\ &+\left(\frac{(1-\xi)\beta_1(E_1+\phi_1 I_1)}{N_1} + \frac{\beta_2(E_2+\phi_2 I_2)}{N_2} + \mu\right)S_2\\ &-(\sigma_2+\mu+\theta_2)\dot{E}_2 + \sigma_1 E_1 + \rho\dot{I}_2 - (\delta_2+\mu+\theta_3)I_1 + \sigma_2 E_2\\ &-(\rho+\delta_3+\mu+\theta_4)\dot{I}_2 + \theta_1 E_1 + \theta_2 E_2 + \theta_3 I_1 + \theta_4 I_2 - \mu\dot{R}. \end{aligned} \quad (29)$$

Simplifying, we obtain

$$\dot{V}(t) = \pi\Lambda\left(1-\frac{S_1^*}{S_1}\right)+(\tau+\mu)S_1^*\left(1-\frac{S_1}{S_1^*}\right)+\frac{(1-\xi)\beta_1 S_1^*}{N_1}\left(1-\frac{S_1}{S_1^*}\right)E_1$$
$$+\frac{(1-\xi)\beta_1 S_1^*}{N_1}\left(1-\frac{S_1}{S_1^*}\right)\phi_1 I_1+\frac{(1-\xi)\beta_2 S_1^*}{N_2}\left(1-\frac{S_1}{S_1^*}\right)E_2$$
$$+\frac{(1-\xi)\beta_2 S_1^*}{N_2}\left(1-\frac{S_1}{S_1^*}\right)\phi_2 I_2+(1-\pi)\Lambda\left(1-\frac{S_2^*}{S_2}\right)$$
$$+(\delta_1+\mu-\tau)S_2^*\left(1-\frac{S_2}{S_2^*}\right)+\frac{(1-\xi)\beta_1 S_2^*}{N_1}\left(1-\frac{S_2}{S_2^*}\right)E_1$$
$$+\frac{(1-\xi)\beta_1 S_2^*}{N_1}\left(1-\frac{S_2}{S_2^*}\right)\phi_1 I_1+\frac{(1-\xi)\beta_2 S_2^*}{N_2}\left(1-\frac{S_2}{S_2^*}\right)E_2$$
$$+\frac{(1-\xi)\beta_2 S_2^*}{N_2}\left(1-\frac{S_2}{S_2^*}\right)\phi_2 I_2+\left(\frac{(1-\xi)\beta_2(S_1+S_2)}{N_1}-\mu\right)E_1+\left(\frac{\beta_2(S_1+S_2)}{N_2}-\mu\right)E_2$$
$$+\left(\frac{(1-\xi)\beta_1\phi_1(S_1+S_2)}{N_1}-(\delta_2+\mu)\right)I_1+\left(\frac{\beta_2\phi_2(S_1+S_2)}{N_2}-(\delta_3+\mu)\right)I_2-\mu\dot{R}. \quad (30)$$

Using the condition

$$\dot{V}(t) = \pi\Lambda\left(1-\frac{S_1^*}{S_1}\right)+(\tau+\mu)S_1^*\left(1-\frac{S_1}{S_1^*}\right)+(1-\pi)\Lambda\left(1-\frac{S_2^*}{S_2}\right)+(\delta_1+\mu-\tau)S_2^*\left(1-\frac{S_2}{S_2^*}\right)-\mu\dot{R}. \quad (31)$$

However, it is found that

$$S_1^* = \frac{\pi\Lambda}{(\tau+\mu)}, S_2^* = \frac{(\tau+\mu-\pi\mu)\Lambda}{(\delta_1+\mu)(\tau+\mu)}, \quad (32)$$

$$\dot{V}(t) \le \pi\Lambda\left(1-\frac{S_1^*}{S_1}\right)+\pi\Lambda\left(1-\frac{S_1}{S_1^*}\right)+(1-\pi)\Lambda\left(1-\frac{S_2^*}{S_2}\right)+(1-\pi)\Lambda\left(1-\frac{S_2}{S_2^*}\right)-\mu\dot{R}$$
$$\le \pi\Lambda\left(2-\frac{S_1^*}{S_1}-\frac{S_1}{S_1^*}\right)+(1-\pi)\Lambda\left(2-\frac{S_2^*}{S_2}-\frac{S_2}{S_2^*}\right)-\mu\dot{R}. \quad (33)$$

$\dot{V}(t) \le 0$ only when $R = 0$. Therefore, the maximum invariant set in Ω is the singleton set ε_0. Hence, the global stability of ε_0 when $R_0 > 1$follows from LaSalle's invariance principle. This means that the DFE is globally asymptotically stable on Ω.

3.5 Sensitivity Analysis

Sensitivity analysis is carried out to identify the most influential parameters for the spreading and well as curbing the infection in the community. We apply the method established in [22]. The sensitivity index of $R_0$with respect to a parameter, say M, is given by

$$X_M^{R_0} = \frac{\partial R_0}{\partial M}\times\frac{M}{R_0}. \quad (34)$$

Table 2. Model Parameter Values

Parameter	Value	Source
Λ	2500	[23]
π	0.73	Assumed
τ	0.021	[23]
$\beta_{1,2}$	0.82	[22]
ξ	0.87	Assumed
ρ	0.64	Assumed
μ	0.01138	[22]
δ_1	0.0019	[23]
$\delta_{2,3}$	0.031	[22]
$\theta_{1,2,3,4}$	0.63; 0.4; 0.57; 0.43	Assumed
$\varphi_{1,2}$	0.54	Estimated
$\sigma_{1,2}$	0.031	Estimated

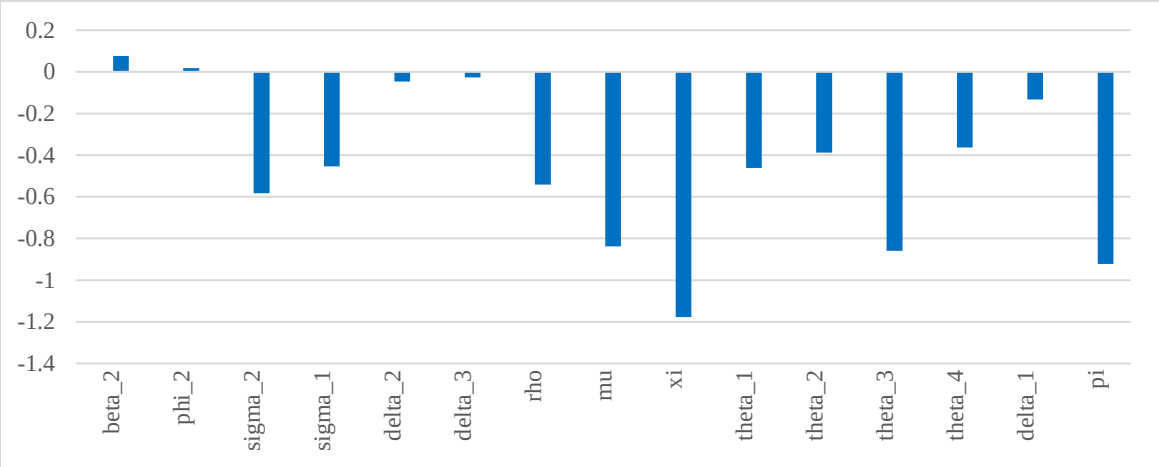

Figure 2. Sensitivity Analysis Plot

As shown in Fig. 2, parameters with positive indices – contact rate (β_2) and relative infection of infected individual compared to exposed individual (φ_2), indicates that an increase in the parameter values would raise the values of R_0. Also, parameters that exhibits negative indices, implies that an increment in their values would lead to a reduction in R_0. From this analysis, policy makers and public health expert should focus on ensuring that the rate of contact between susceptible individual and exposed/infected individual is minimized, as such, more isolation center should be established and individuals exhibiting symptoms of Covid-19 should immediately transferred to the center for proper care. Further-more, as indicated from the parameters with negative indices, attention should also be given to infected and exposed individual so as to adhere to medicals for recovery. In general, efforts should be intensified to ensure that smoking should be discouraged and individuals exposed or infected with Covid-19 should be removed from the population.

3.6 Numerical Simulation

In this section, we use values obtained in Table 2 to present numerical results.

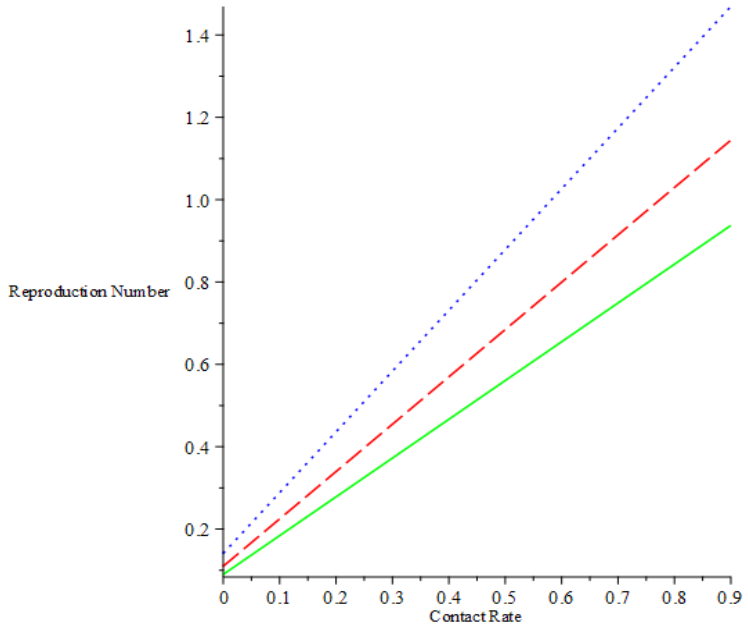

Figure 3. Plot Showing Reproduction Number against Contact Rate

As shown in Fig. 3 above, the reproduction number increases as the contact rate increases. This implies that the disease can be prevented from spreading when precautions are taken to ensure that infected and exposed individuals have no contact with susceptible human population.

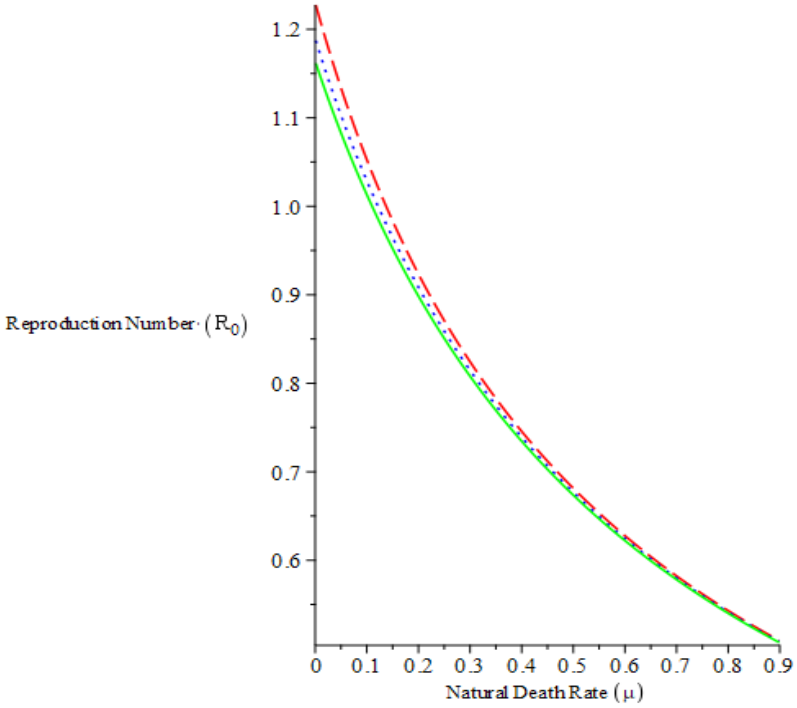

Figure 4. Plot showing Reproduction Number against Natural Death Rate

The graph shown in Fig, 4 above shows the plot of Reproduction Number against Natural death rate. As shown, the secondary infection (reproduction number) decreases as the death rate increases. This can imply, reducing the population of the infected and exposed human population will curb to the minimum the spread of the Covid-19.

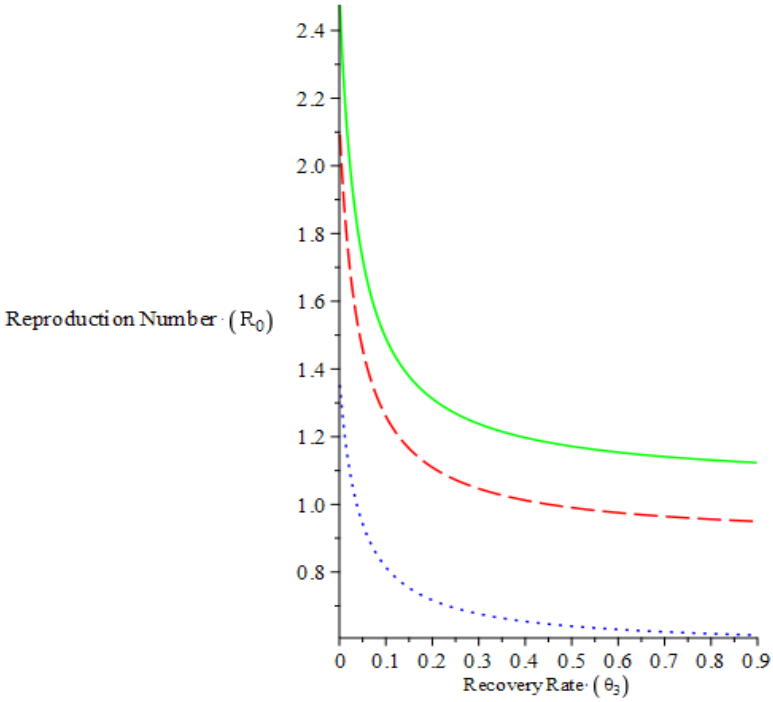

Figure 5. Plot Showing Reproduction Number against Recovery Rate

The graph shown in Fig. 5 shown the relation between the reproduction number and the recovery rate. The plot reveals that giving attention to recovery strategies available is potent in addressing the spread of COVID-19 infection.

4. CONCLUSION

In this paper, we analyzed a mathematical model of smoking and COVID-19. First, we obtained the feasible region where the model is well-posed epidemiologically and mathematically. We further proved the positivity of the solutions. We obtained the disease-free equilibrium point (DFE) there after compute the basic reproduction number using the next-generation matrix method. We analyzed the global stability of the model using the Lyapunov method and we proved that model is globally stable if the reproduction number is less than one. The sensitivity analysis of the Model was carried out, showing that the most sensitive parameter is the contact rate and account of COVID-19 infection. The numerical simulations show that, curtailing rate of contact between exposed, infected individuals and susceptible human population will reduce the spread of the diseases, also giving attention to recovery strategies and controls will reduce to minimum the disease transmission. Therefore, it is recommended that stakeholders should give attention in controlling smoking habits, and prevention and treatment of COVID-19 infected individuals.

Author Contributions

Temidayo Joseph Oluwafemi: Conceptualization, Methodology, Model Analysis, Writing-Original Draft, visualization. Miswanto: Conceptualization, Model Formulation, Methodology, Edit original Draft, visualization. All authors discussed result and contributed to final manuscript.

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Declarations

The authors declares that there are no conflicts of interest to report study.

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