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Global Stability Indices of the Transmission and Bilinear Control Functions for Tobacco Smoking Epidemic

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ABSTRACT

Background: The seeming insurmountable effect of tobacco-smoking, coupled with intense laxity by society towards the avalanche consequential effects of tobacco-smoking consumption and the yet-to-be-available mathematical model for comprehensive treatment of multiple effects of tobacco-smoking, necessitated this present investigation. The present research arguably presented an insight into the global stability indices of not just the impact of smoking transmission but explicitly demonstrated the methodological application of designated bilinear control functions in the presence of screening techniques for the eradication of consequential effects of smoking and tobacco consumption.

Methods: The model explored deterministic 6-subpopulations, formulated using first-order differential equations. System interactions was investigated using bilinear control functions with system well-posedness established. Furthermore, system smoking generation number for both off- and onset-treatment scenarios was determined. Analytic predictions for both local and global indices explored second additive compound matrix in conjunction with Lozinski measure . Numerical simulations evolved Runge-Kutta of order of precision 4 in a Mathcad surface.

Results: Accompanying simulations indicated protracted initial asymptomatic saddle period of smoking infection for $t_f \leq 140$ days under the off-treatment scenario. Inducement of bilinear control functions under screening method yielded drastic reduction to near zero within the first $t_f \leq 80$ days as vindicated by the exponential rejuvenation of potential smokers.

Conclusion: Investigation concludes that eradication of consequential effects of tobacco-smoking is achievable provided both smokers, tobacco companies and the governments implement concurrently, the application of designated bilinear control functions the world over. Immensely, the study exhibits mutual benefits to the society and scientists in the field of Bio-mathematics and epidemiologists.

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Introduction

Tobacco consumption versa-vice cigarette smoking is known to constitute a preventable yet major health risk the world-over. About 1.3 billion of the world population is known to be consuming tobacco substance, yet t the adverse effect of chronic tobacco consumption often lead to premature mortality with an estimated annual death rate of 5 million (an average of 9%) people [1,2]. Transmission of the effect of smoking and tobacco consumption is socio-environmental with human-to-human transmissibility and having the environment as it reservoir. Zoonotic science has considered smoking as the causative agent of a number of non-communicable diseases of the form: chronic lung diseases, asthma, obstructive pulmonary disease, stroke, diabetes, premature heart attack, cancer, cardiovascular [1,3-5]. Moreso, death rate emanating from cancers of the mouth, ling, stomach cancers, kidney, pancreas, cervix and liver cancer are as a result of chronic smoking [5].

As a socio-environmental component of the society, behavioral smoking pattern spread through both close and distance social links with trend of cluster among socially isolated groups, [6,7]. Overlooking the adverse effect of smoking, most persons smoke for the sake of either pleasure, to regulate body weight or for suppression of hunger along with sense of taste and smell [8].

Apart from health organizations that could run direct adverts and campaigns on the effect of smoking, one pertinent approach in the investigation of the dynamics for the spread of smoking effects and control interventions have been through the use of mathematical modeling. For instance, the first simplest mathematical model in 1997, considered a 3-Dimensional state-space (potential smokers P, smokers S and quitters Q) [9]. The behavioral attitude and consequential effect of smoking by mild and chain smokers was later studied [10]. Using the Brownian motion for the perturbation of derived model equations, the stochastic study for smoking model was investigated and its sufficient conditions for their mean square established [11]. Accounting for saturated incidence rate for a smoking model, the study on mathematical analysis and optimal control of giving up smoking was conducted [2]. Recently, the application of fractional theory have been very useful in studying smoking dynamics models. Example, the mathematical assessment of the dynamics of tobacco smoking was investigated using fractional theory [12]. The study explore Caputo operator accounting for tobacco in the form of snuffing.

Remarkably, the vast adverse effect of tobacco consumptions often manifest in the deteriorating health of many contagious diseases. For instance, patience with COVID-19, who are smokers do have high risk factor with rapid health deterioration than non-smokers. More specifically, the consequential effect of tobacco-smking can adversely accelerate the deteriorating health of patience with HIV/AIDS and tritrophic rate of COVID-19 infections [13-15]. Therefore, the main goal of this present investigation is that of seeking for the methodological application of a bilinear control functions (nicotine replacement therapy and non-nicotine medication) under coherent screening method, geared towards optimal recovery and reunion with the susceptible population.

The organizational structure of this paper are as follows: section 2, is devoted to the material and methods. This section account for the problem statement of the study and the derivation of system basic model. The mathematical analysis for derived model is contained in section 3. In section 4, we discuss the stability analysis in relation to its local and global conditions. In section5, we present numerical illustrations of our derived analytical predictions. Comparative analysis of established results are explicitly presented in section 6. Finally, section 7, account for study incisive conclusion and study recommendations. The entire work is anticipated to unveil the insight to the methodological treatment of smoking effect as an epidemic.

Materials and Methods

The materials and methods for this present proposed study is constituted by a set of varying subpopulations having access to tobacco reservoir (host-vector, and environment). The consequential effect of smoking is investigated using a bilinear control functions (nicotine replacement therapy - NRT and non-nicotine medications – NNM). The fact behind this investigation is unveil by the problem statement of the study, followed by derived set of mathematical equations for the proposed model. Method of mathematical analysis explores the fundamental theory of differential equations, while the derivation of system basic reproduction number explores the next generation matrix with the incorporation of linearization method. We shall investigate the local asymptotic stability using LaSalle's invariant principle with Routh Hurwitz criterion and the global stability conditions via the Lozinskii measure *lB* with the incorporation of second additive compound matrix. The aspect of the numerical simulations shall explore Runge-Kutta of order of precision 4 in a Mathcad surface.

Problem Statement for a Tobacco Model

It is obvious that following the adverse effect of smoking of tobacco, government and non-governmental agencies the world over, are increasingly running all forms of campaigns and adverts aimed at reducing the rate of smoking. Often embedded in those adverts, is the consequential impact upon smoking of tobacco. The study on the dynamical effect of smoking and its social impact as well as control measures have severally been conducted using mathematical modeling among other known health methodologies. In this present study, we bring to bear a mathematical analysis of a smoking model with social factor, [8]. In that model, a set of 5-dimensioanl mathematical subpopulations in the form of: non-smoking population P, light smokers S_i, chain smokers S_c, temporal quit smokers Q_t and permanent quitters Q_p were explored for model formulation. For more details on the model, we capture here, the schematic diagram as deduced by Figure 1 below:

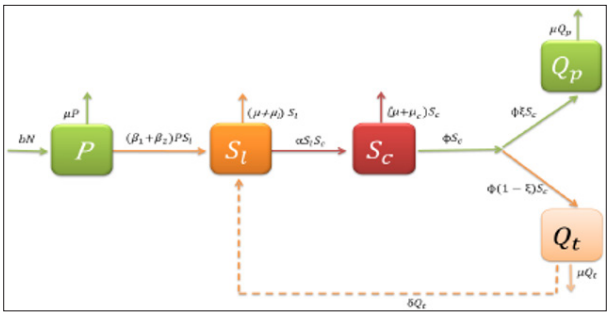


Figure 1: Schematic Diagram for Model (1), [8].

The mathematical equations is as depicted by Equation (1) below:

$$\begin{aligned}
\frac{dP}{dt} &= bN-(\beta_1+\beta_2)PS_l-\mu P\\
\frac{dS_l}{dt} &= (\beta_1+\beta_2)PS_l+\delta Q_t-\alpha S_lS_c-(\mu+\mu_l)S_l\\
\frac{dS_c}{dt} &= \alpha S_lS_c-\phi S_c-(\mu+\mu_l)S_c \qquad (1)\\
\frac{dQ_t}{dt} &= \phi(1-\varepsilon)S_c-\delta Q_t-\mu Q_t\\
\frac{dQ_p}{dt} &= \phi\varepsilon S_c-\delta Q_t-\mu Q_p
\end{aligned}$$

with initial conditions $P(0)\geq 0, S_l(0)\geq 0, S_c(0)\geq 0, Q_t(0)\geq 0, Q_p\geq 0$

A critical review of the above model indicated that the incidence rates $(\beta_1+\beta_2)$ was not derived. Rather, initial values were assigned to them. Furthermore, no screening method applied to the infection dynamics through the i^{th} compartments and moreso, the model was devoid of any control functions. Therefore, in attempt to incorporate the aforementioned lapses of model (1), the present study sought to formulate a socio-epidemiological 6-Dimensional mathematical model to be investigated using a bilinear control functions in the presence of screening technique.

Derivation of System Mathematical Equations

Here, we attempt to overcome the aforementioned lapses of model (1) with the incorporation of enhance state components, screening method and the introduction of bilinear control functions. Suppose the population understudy is segregated into 6-Dimensional subpopulation measured in $cellsmm^{-3}$, then the system compartment is constituted by potential smokers P_s, mild smokers S_m, chronic smokers S_c, permanent quitters Q_p, temporal quitters Q_t and recovered population R_p. The interactions between these subpopulations and tobacco reservoir is investigated using a bilinear control functions (nicotine replacement therapy –

nasal spray/lozenges and non-nicotine medication – bupropion/ varenicline) in the presence of a screening method denoted by ϕ. Furthermore, our proposed model is built on the following assumptions:

Assumption 1

i. Only temporal and permanent quitters undergo screening, i.e. $Q_t, Q_p > 0$.
ii. Control functions (u_1, v_1) are only introduced to Q_t, Q_p upon screening for all $(u_1, v_1) > 0$.
iii. Only temporal and permanent quitters die due to smoking, i.e. $u_i > 0$.
iv. Temporal quitters may revert to mild smoking, i.e. $Q_t \rightarrow S_m$ for all $Q_t \leq S_m$.
v. Only screened permanent quitters apply nicotine therapy, $\eta > 0$.
vi. Recovered reunion with the potential smoking population, $R_p < P_s$.

Thus, using assumption 1, the socio-epidemiological model equations for the present study is derive as:

$$\begin{aligned}
\frac{dP_s}{dt} &= b_p + \eta R_p - \beta_i(\hat{N})P_s - \mu P_s \\
\frac{dS_m}{dt} &= \beta_i(\hat{N})P_s + \delta Q_t - \alpha S_m - (\mu + \mu_i)S_m \\
\frac{dS_c}{dt} &= \alpha S_m - (1-u_1)\phi\varepsilon S_c - (1-v_1)(1-\varepsilon)\phi S_c - (\mu + \mu_i)S_c \\
\frac{dQ_p}{dt} &= (1-u_1)\phi\varepsilon S_c - (\tau_1 + \mu)Q_p \\
\frac{dQ_t}{dt} &= (1-v_1)(1-\varepsilon)\phi S_c - (\tau_2 + \delta + \mu)Q_t \\
\frac{dR_p}{dt} &= \tau_1 Q_p + \tau_2 Q_t - \eta R_p - \mu R_p
\end{aligned} \tag{2}$$

with initial values $(P_s, S_m, S_c, Q_p, Q_t, R_p) \geq 0$ for all $t = t_0 = 0$, $(u_1, v_1) = 0$ where

$$\beta_i(\hat{N}) = \sum_{i=1}^{4} \beta_i c_i(\hat{N}_i) \qquad \beta_i \hat{N}) > 0 \quad i = 1,...,4 \tag{3}$$

and having $N = P_s + S_m + S_c + Q_p + Q_t + R_p; \hat{N} = S_m + S_c + Q_p + Q_t \;\forall\; \hat{N} < N$

If $(u_1, v_1) > 0$, then Equation (3) becomes

$$\beta_i(\hat{N}) = (1-u_1)(1-v_1)\left[\sum_{i=1}^{4} \beta_i c_i(\hat{N}_i)\right] \quad \beta_i \hat{N}) > 0 \;\; i = 1,...,4 \tag{4}$$

The socio-epidemiological implication of system (2) is explain thus: from the first equation, we observe that the differential outcome for potential smokers is constituted by natural birth rate and recovery rate from smoking $(b_p + \eta R_p)$with depleting proportion from incidence rate and natural death rate given by $-(\beta_i(\hat{N})P_s + \mu P_s)$ The second equation depicts mild smokers, which is sustained by the incidence rate of smoking and the proportion that reverted back to smoking after quitting temporally denote by $-(\beta_i(\hat{N})P_s + \delta Q_t)$ The clearance rate here include the proportion that transit to chronic smoking and the death rate due to either natural and/or due to smoking presented by $-[\alpha S_m + (\mu + \mu_i)S_m]$. The third equation has its source rate from mild smokers that transit to chronic smokers αS_m. Accounting for varying treatment, if chronic smokers are subjected to screening method at the rate ϕ, then the proportion of chronic smokers that desired to quit smoking permanently is presented by $-(1-u_1)\phi\varepsilon S_c$ where ε is the rate at which permanent quitters receive treatment. That is, the proportion that desired to temporally quit smoking is denoted by $-(1-v_1)(1-\varepsilon)\phi S_c$. The clearance rate for this compartment is given by $-(\mu+\mu_i)S_c$, which denote natural death and death due to infection.

From the fourth equation, we observe that quitting smoking permanently is a function of nicotine replacement therapy $(1-u_1)$ $(1-\mu_1)$ i.e. $(1-\mu_1)\,\phi\varepsilon S_c$. The inducement of control function could lead to recovery rate and having spatial natural death rate of $-\mu Q_p$. Similarly, from the fifth equation, the proportion quitting smoking temporally determine by non-nicotine medication is given by $-(1-v_1)(1-\varepsilon)\phi S_c$ and having depletive ratio of $-(\tau_2 + \delta + \mu)Q_t$ Finally, sixth equation describe the proportion that recovered from smoking. Here, the source rate is given by $\tau_1 Q_p + \tau_2 Q_t$. Those that recover actually reunion with potential smoker at $-\eta R_p$ and having natural clearance rate of $-\mu R_p$. Thus, with Equations (3) and (4) derived, system (2) completely represent the mathematical dynamics of the effect of global tobacco epidemic model with bilinear control functions under enhanced screening technique. Figure 2, below, gives the graphic image of system (2), while Table 2, depicts the detail description for both the state-space and parameter variables of Figure 2.

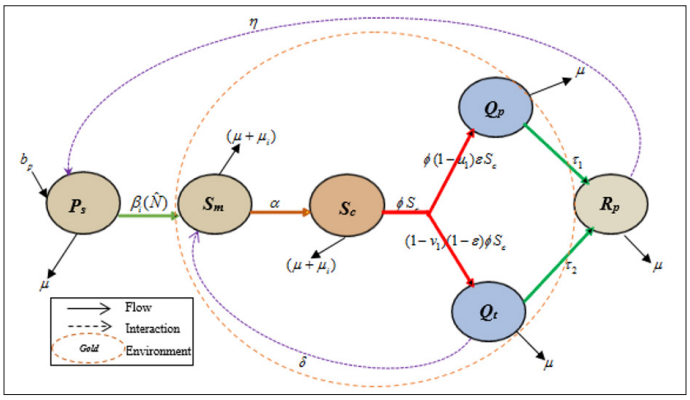


Figure 2: Graphic Image of P-2S-2Q-R Smoking Model with Control Functions

Table 1: Description of State-space and Parameter Variables for Model (2)

Description of State-space and Parameter Variables			
State-space		**Parameter Variables**	
Symbols	**Description**	**Symbols**	**Description**
P_s	Potential smokers	b_p	Natural birth rate
S_m	Mild smokers	μ	Natural death rate
S_c	Chronic smokers	$\beta_{i(i=1,...,4)}$	Incidence rates
Q_p	Permanent quitters	μ_i	Clearance rate due to smoking
Q_t	Temporal quitters	α	Intensity rate of smoking
R_p	Recovered from smoking	ϕ	Screening rate
		ε	Rate at which screen Q_p receive μ_1
		$c_{i(i=1,..4)}$	Varying rate of interactions within state-space

		$(1-\varepsilon)$	Rate at which screen Q_t receive v_1
		τ_1, τ_2	Recovery rates from Q_p and Q_t respectively
		u_1, v_1	Nicotine replacement therapy; non-nicotine medication
		η	Recovered becoming potential smokers
		δ	Temporal quitters reverting to smoking again

Mathematical Analysis for Derived Model

Since system (2) represent a set of living organisms, it is paramount that we verify and prove for the following:
the existence and uniqueness of system solution, the positivity of solution and the boundedness of solution within certain invariant region denoted by .

Existence of Solution

Let $\Omega : R \to R_+^6$ such that

$t \mapsto \left(P_s(t), S_m(t), S_c(t), Q_p(t), Q_t(t), R_p(t)\right)$ and G: $R^n \to R^6$ such that

$\Omega(t) \to G(\Omega(t)) = \left(P_s^\circ(t), S_m^\circ(t), S_c^\circ(t), Q_p^\circ(t), Q_t^\circ(t), R_p^\circ(t)\right)$

Then, $\Omega(t) = G(\Omega(t), \Omega(0)) = \Omega_o$.

Definition 1: *(Cauchy-Lipschitz condition)*

In particular, Lipschitz condition is defined as: a function $f : [a,b] \to \Re$ is said to satisfy the Lipschitz condition if there is a constant such that $|f(x) - f(x')| \le M|x - x'| \ \forall \ x, x' \in [a,b]$ where M is the Lipschitz constant.

Theorem 1: (Existence and uniqueness)

The system (2) is continuous and satisfies Cauchy-Lipschitz condition.

Proof: We explore existence and uniqueness results, [13]. Then, we show from system (2), taking the first equation, while the rest follow similar procedures. Now, let

$$P(t,S) = \frac{dP_s}{dt} = b_p + \eta R_p - \beta_i(\hat{N})P_s - \mu P_s \qquad (5)$$

Then, using Equation (4), the partial derivative of Equation (5) becomes

$$\frac{\partial P(t,S)}{\partial S} = -(1-u_1)(1-v_1)\left[\sum_{i=1}^{4}\beta_i c_i(\hat{N}_i)\right] - \mu \qquad (6)$$

This shows that the function $P(t,S)$ and its partial derivative $\frac{\partial P(t,S)}{\partial S}$ are defined and continuous at all point (t,S). Similarly, the right hand functions of other equations and their respective partial derivatives satisfy these conditions. This imply that by existence and uniqueness theorem, there exists a unique solution for $P_s(t)$, $S_m(t)$, $S_c(t)$, $Q_p(t)$, $Q_t(t)$ and $R_p(t)$ in some open intervals centred t_0. We then have to show that the solution satisfies the Lipschitz condition. Now, using Equation (5), we see at once that

$$\left|P(t,S_{P_s(1)}) - P(t,S_{P_s(2)})\right| = \begin{vmatrix} b_p + \eta R_p - (1-u_1)(1-v_1)\sum_{i=1}^{4}\beta_i c_i(\hat{N}_i)P_s - \mu P_s \\ -b_p + \eta R_p - (1-u_1)(1-v_1)\sum_{i=1}^{4}\beta_i c_i(\hat{N}_i)P_s - \mu P_s \end{vmatrix}$$

$$= \left|(-)\left[(1-u_1)(1-v_1)\left(\sum_{i=1}^{4}\beta_i c_i(\hat{N}_i)\right) + \mu\right]\left(P_{s,(1)} - P_{s,(2)}\right)\right|$$

$$\le \left[(1-u_1)(1-v_1)\left(\sum_{i=1}^{4}\beta_i c_i(\hat{N}_i)\right) + \mu\right]\left|P_{s,(1)} - P_{s,(2)}\right|$$

This implies that $\left|P(t,S_{P_s(1)}) - P(t,S_{P_s(2)})\right| \le M\left|P_{s,(1)} - P_{s,(2)}\right|$

where $M = \left((1-u_1)(1-v_1)\left(\sum_{i=1}^{4}\beta_i c_i(\hat{N}_i)\right) + \mu\right)$ is a Lipschitz constant. In a similar procedure, we show that the remaining variables satisfy Lipschitz condition. Therefore, there exists a unique solution $P_s(t)$, $S_m(t)$, $S_c(t)$, $Q_p(t)$, $Q_t(t)$, $R_p(t)$ for all $t \ge 0$.

Positivity and Boundedness of Solutions

The following theorems justifies the positivity and boundedness of the solutions of the system (2).

Theorem 2: (Positivity of solutions)

Suppose system (2) is bounded by the initial conditions $\left\{P_s(0), S_m(0), S_c(0), Q_p(0), Q_t(0), R_p(0)\right\} \ge 0 \in \mathbb{R}_+^6$ Then, the unique solution of Theorem 1, forms a set of solutions $\left\{P_s(t), S_m(t), S_c(t), Q_p(t), Q_t(t), R_p(t)\right\}$ and is non-negative for all t ≥ 0.

Proof: Invoking existing result for positivity of solutions, [14]. Then, we prove for the first equation of system (2) and then deduce for the rest of the equations for all t ≥ 0. That is,

$$\frac{dP_s}{dt} \ge -\left((1-u_1)(1-v_1)\left(\sum_{i=1}^{4}\beta_i c_i(\hat{N}_i)\right) + \mu\right)P_s \cdot \qquad (7)$$

Suppose $\gamma = (1-u_1)(1-v_1)\left(\sum_{i=1}^{4}\beta_i c_i(\hat{N}_i)\right) + \mu$, then Equation (7) becomes

$$\frac{dP_s}{dt} \ge -\gamma P_s \qquad (8)$$

Taking the integrating factor, we have

$$\ln|P_s| \ge \int -\gamma dt + C$$

This imply that

$$|P_s| \ge e^{\int -\gamma dt e^c}$$

$$\ge P_s(0)e^{\int -\gamma dt}$$

Hence,

$$P_s \ge P_s(0)e^{-\int (1-u_1)(1-v_1)\left(\sum_{i=1}^{4}\beta_i c_i(\hat{N}_i) + \mu\right)dt} \ge 0$$

where $P_s(0)$ represent the potential smoking population at $t = 0$. Thus, by recursive argument, we verify for the rest of the equation of system (2). Therefore, we see that any solution of system (2) is non-negative i.e. $\left\{P_s(t), S_m(t), S_c(t), Q_p(t), Q_t(t), R_p(t)\right\} \in \mathbb{R}_+^6$ and

prove is completed.

Theorem 3: (Boundedness of solution)
All solutions of system (2) is bounded and positively invariant in the region $\prod_D$, where

$$\prod_D = \left\{ \left(P_s, S_m, S_c, Q_p, Q_t, R_p\right) \in \mathbb{R}_+^6 : 0 \le \left(P_s(t), S_m(t), S_c(t), Q_p(t), Q_t(t), R_p(t)\right) \le \frac{b_p}{\mu} \right\}. \quad (9)$$

Proof: Here, we investigate the prove using existing result, [15]. Now, taking on system (2), the sum of the differentiation gives

$$\frac{dN}{dt} = b_p - (\mu + \mu_i)N .$$

If the population is free from tobacco smoking and its effect, then $\mu_i = 0$ and we have

$$\frac{dN}{dt} = b_p - \mu N$$

or

$$\frac{dN}{dt} + \mu N \le b_p .$$

This is a first order homogeneous differential inequality. Applying the integrating factor $IF = e^{\int \mu dt} = e^{\mu t}$, we have

$$e^{\mu t}.\frac{dN(t)}{dt} + \mu N(t) e^{\mu t} \le b_p e^{\mu t} ,$$

or

$$\frac{d}{dt}\left[\mu N(t) e^{\mu t}\right] \le b_p e^{\mu t} .$$

Integrating, we have

$$N(t) e^{\mu t} \le \frac{b_p}{\mu} e^{\mu t} + C ,$$

where C, is the constant of integration. Now, simplifying, we have

$$N(t) \le \frac{b_p}{\mu} + C e^{-\mu t} .$$

Solving for C and taking initial condition for t=0, and then by substituting the resulting value, we have

$$N(t) \le \frac{b_p}{\mu} + \left(N(0) - \frac{b_p}{\mu} \right) e^{-\mu t} .$$

Therefore, taking the limit as $t \to \infty$ we have

$$\lim_{t \to \infty} N(t) \le \frac{b_p}{\mu} \quad (10).$$

Equation (10) shows that system (2) is biologically feasible in the region $\prod_D$. Therefore, the solution of system (2) with initial conditions is bounded in the invariant region of Equation (9) for all $t \in [0, \infty)$. Hence, system (2) is well posed.

Equilibrium Points Analysis
The analytic nature of system (2) is concerned with the study steady-space and their local stability, which of course is challenging due to the complex non-linearities of derived model (2). Yet, it is pertinent that we investigate the system multiple local asymptotic stability as well as their global stability conditions.

Existence of System Steady States
Since we denoted the vectorial capacity of system (2) by $\prod_D$, then from Equation (9), we have

$$\prod_D = \left(P_s, S_m, S_c, Q_p, Q_t, R_p\right)$$

That is, system (2) can be written in the form:

$$\frac{d\prod(t)}{dt} = f(t, \prod_D, z) \quad (11)$$

where $f(t, \prod_D, z)$ is the right side of the ODE of system (2) and z, is the vector parameters as depicted by Table 2, then, we compute as following:

i) Tobacco Smoking Free-Equilibrium (T-SFE)
At T-SFE, there is no infection, which implies no spreading of smoking effect i.e. $S_m = S_c = Q_p = R_p = 0$. Therefore, the T-SFE for system (2) denoted by E^0 is derive as: $E^0 = \left(P_s^o, 0, 0, 0, 0, 0\right)$

where from Equation (10), $P_s = \frac{b_p}{\mu}$, i.e.

$$E^0 = \left(\frac{b_p}{\mu}, 0, 0, 0, 0, 0 \right) . \quad (12)$$

That is, T-SFE at no smoking effect is only a function of recruitment rate with respect to natural death rate.

ii) Tobacco Smoking Endemic Equilibrium (T-SEE)
With the spreading of smoking effect, endemic state is bound to occur. Then from system (2), for any arbitrary endemic equilibrium denoted by E^*, we have

$$E^* = \left(P_s^*, S_m^*, S_c^*, Q_p^*, Q_t^*, R_p^*\right), \quad (13)$$

Such that

$$N^* = P_s^* + S_m^* + S_c^* + Q_p^* + Q_t^* + R_p^* .$$

Then, solving equations of system (2) step-wisely by using Equation (11), we have from the first equation,

$$0 = b_p - \beta_i(\hat{N}) P_s - \mu P_s ,$$

or

$$P_s^* = \frac{b_p}{\beta_i^* + \mu} .$$

Taking on the second equation, we have

$$0 = \beta_i^*(\hat{N}) P_s^* + \delta Q_t - (\alpha + \mu + \mu_i) S_m^*$$

or

$$S_m^* = \frac{\beta_i^*(\hat{N}) P_s^*}{(\alpha + \mu + \mu_i)} = \frac{b_p}{(\alpha + \mu + \mu_i)} \frac{\beta_i^*}{\beta_i^* + \mu}$$

This implies that

$$S_m^* = \frac{b_p}{q_1} \left(\frac{\beta_i^*}{\beta_i^* + \mu} \right),$$

where, $q_1 = \alpha + \mu + \mu_i$. Thus, by recursive argument, each of the remaining equations are computed to give the required results for E^* as:

$$E^* = \begin{cases} P_s^* &= \dfrac{b_p}{\beta_i^* + \mu} \\ S_m^* &= \dfrac{b_p}{q_1}\left(\dfrac{\beta_i^*}{\beta_i^* + \mu}\right) \\ S_c^* &= \dfrac{b_p \alpha}{q_2}\left(\dfrac{\beta_i^*}{\beta_i^* + \mu}\right) \\ Q_p^* &= \dfrac{b_p \alpha q_3}{q_2 q_4}\left(\dfrac{\beta_i^*}{\beta_i^* + \mu}\right) \\ Q_t^* &= \dfrac{b_p \alpha q_5}{q_2 q_6}\left(\dfrac{\beta_i^*}{\beta_i^* + \mu}\right) \\ R_p^* &= \dfrac{b_p \alpha q_{10}}{q_2 q_8}\left(\dfrac{\beta_i^*}{\beta_i^* + \mu}\right) \end{cases}, \quad (14)$$

where

$$\begin{cases} q_1 = \alpha + \mu + \mu_i, & q_2 = (1-u_1)\phi\varepsilon + (1-v_1)(1-\varepsilon)\phi + (\mu + \mu_i) \\ q_3 = (1-u_1)\phi\varepsilon & q_4 = (\tau_1 + \mu) \\ q_5 = (1-v_1)(1-\varepsilon)\phi & q_6 = (\tau_2 + \mu) \\ q_7 = (\tau_1 + \tau_2) & q_8 = (\eta + \mu) \\ q_9 = (\tau_2 + \delta + \mu) & q_{10} = q_7(q_3 + q_4) \end{cases} . \quad (15)$$

Clearly, if Equation (14) is substituted into Equation (11) at $\beta_i^*=0$, we return to Equation (12), which define the existence of T-SFE. Therefore, the endemic state for system (2) at which $\beta_i^* \neq 0$, as depicted by Equation (14) satisfies Equation (13). Obviously, these two indices E^0 and E^* are important components for the computation of system basic reproduction number denoted by R_0.

System Basic Reproduction Number, R_0

The spread of tobacco effect is in itself, a social adverse factor and thus, a virus by all ramifications. That is, permanent smokers among other forms, constitute reservoir of tobacco smoking effect. Therefore, the intensity to have a clear flow of smoking transmission pattern and its biological effect is defined by the basic reproduction number R_0. For simplicity, the reproduction number for smoking effect is also known as smoking generating number $S_{g(0)}$ i.e. $R_0 \approx S_{g(0)}$, [8]. Then, we shall investigate $S_{g(0)}$ using existing approach known as next generation matrix method, [16].

From system (2), to account for the infectious state variables, we let

$$X = (S_m, S_c, Q_p, Q_t).$$

Mathematically, $S_{g(0)}$ is define as: [17].

$$R_0 \approx S_{g(0)} = \rho(FV^{-1}) = \left[\frac{\partial F_i}{\partial x_j}(E^0)\right].\left[\frac{\partial V_i}{\partial x_j}(E^0)\right]^{-1}, \quad (16)$$

where the notations F_i and V_i represent the matrices of new spread in compartment i and the transfer terms at T-SFE into the i^{th} compartment, while E^0 is the T-SFE. Then, accounting for the actual transmutable state-space from system (2), we have

$$F_i = \begin{pmatrix} F_1 \\ F_2 \\ F_3 \\ F_4 \end{pmatrix} = \begin{pmatrix} [\beta_1 c_1 S_m + \beta_2 c_2 S_c + \beta_3 c_3 Q_p + \beta_4 c_4 Q_t]P_s + \delta Q_t \\ 0 \\ 0 \\ \delta \end{pmatrix}$$

At T-SFE, using Equation (12), the linearization of F_i gives

$$F_0 = \begin{pmatrix} \beta_1 c_1 \dfrac{b_p}{\mu} & \beta_2 c_2 \dfrac{b_p}{\mu} & \beta_3 c_3 \dfrac{b_p}{\mu} & \beta_4 c_{41} \dfrac{b_p}{\mu} + \delta \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}. \quad (17)$$

Now, computing for V_i, we have

$$V_{i=1,..4} = \begin{pmatrix} (\alpha + \mu + \mu_i)S_m \\ (1-u_1)\phi\varepsilon + (1-v_1)(1-\varepsilon)\phi + (\mu + \mu_i)S_c \\ (\tau_1 + \mu)Q_p \\ (\tau_2 + \delta + \mu)Q_t \end{pmatrix}$$

or

$$V_{i=1,..4} = \begin{pmatrix} q_1 S_m \\ q_2 S_c \\ q_4 Q_p \\ q_9 Q_t \end{pmatrix},$$

where q_1 q_2 q_4 and q_9 is defined from Equation (15). The linearization of V_i, gives

$$V_0 = \begin{pmatrix} q_1 & 0 & 0 & 0 \\ 0 & q_2 & 0 & 0 \\ 0 & 0 & q_4 & 0 \\ 0 & 0 & 0 & q_9 \end{pmatrix}. \quad (18)$$

Thus, by Equation (16), the T-smoking generation number, which correspond to the spectral radius $F_0V_0^-$ is computed as: [18,19].

$$S_{g(1)} = \rho(F_0 V_0^-) = \frac{b_p}{\mu}\left(\frac{\beta_1 c_1}{q_1} + \frac{\beta_2 c_2}{q_2} + \frac{\beta_3 c_3}{q_4} + \frac{\beta_4 c_4}{q_9}\right)$$

or

$$S_{g(1)} = \frac{b_p}{\mu}\left(\sum_i^4 G_j\right), \quad (19)$$

where $G_{j=1,..4}$ represent the reproduction numbers for the infectious state-space. Clearly, Equation (19), depicts the system reproduction number at off-treatment scenario (i.e. $(u_1, v_1) = 0$) with computed value of $S_{g(1)} = 7.293 > 1$

If $(u_1, v_1) > 0$ represent treatment functions, then at $(1–u_1)$ $(1–v_1)$ control functions, Equation (19) becomes

$$S_{g(2)} = \rho(F_0 V_0^-) = \varphi\frac{b_p}{\mu}\left(\frac{\beta_1 c_1}{q_1} + \frac{\beta_2 c_2}{q_2} + \frac{\beta_3 c_3}{q_4} + \frac{\beta_4 c_4}{q_9}\right),$$

or

$$S_{g(2)} = \varphi\frac{b_p}{\mu}\left(\sum_i^4 G_j\right), \quad (20)$$

where $\varphi = (1–u_1)$ $(1–v_1)$. Equation (20) is known as the system effective smoking generation number with value computed as $S_{g(2)} = 0.178 < 1$.

Analysis of Stability Indices for T-SFE and T-SEE

For simplicity, we shall consider the system stability analysis in terms of its local asymptotes for both T-SFE and T-SEE, using the critical role of $S_{g(0)}$ with the incorporation of the LaSalle's invariant principle. The global asymptotes aspect shall be investigated

using second additive compound matrix with incorporation of Lozinskii measure *lB*.

Asymptotic Local Stability in Terms of $S_{g(0)}$

Reproduction number $S_{g(0)}$, is an important tool that determine the rate of transmission of any infectious disease both at off and onset-treatment scenarios. The following theorems investigates the cases for local asymptotic T-SFE and local asymptotic T-SEE.

Tobacco – Smoking Free Equilibrium (T-SFE)

At disease free-state, then $S_{g(0)} < 1$, which implies that no transmission of smoking effect i.e. $\beta_i c_i(\hat{N}) = 0$

The following theorem holds.

Theorem 4

System (2) is locally asymptotically stable at T-SFE (E^0) if $S_{g(0)}<1$, otherwise, unstable if $S_{g(0)} > 1$.

Proof: We linearize system (2) by using existing result for the local stability analysis, [17].

Let *J* denote the Jacobian matrix for system (2). Then, *J* at T-SFE is derive as:

$$J_{(E^0)} = \begin{pmatrix} -\mu & -\beta_1 c_1 & -\beta_2 c_2 & -\beta_3 c_3 & -\beta_4 c_4 & \eta \\ 0 & -(q_1+\beta_1 c_1) & 0 & \beta_3 c_3 & \beta_4 c_4+\delta & 0 \\ 0 & \alpha & -q_2 & 0 & 0 & 0 \\ 0 & 0 & q_3 & -q_4 & 0 & 0 \\ 0 & 0 & q_5 & 0 & -q_9 & 0 \\ 0 & 0 & 0 & \tau_1 & \tau_2 & -q_8 \end{pmatrix}. \quad (21)$$

Here, the characteristic equation for Equation (21) is given by

$$\det[J_{(E^0)} - \lambda I] = 0 \quad (22)$$

Taking the eigenvalues of Equation (21) with respect to Equation (22), gives: $-\mu$, $-(q_1+\beta_1 c_1)$, $-q_2$, $-q_4$, $-q_9$ and $-q_8$. This shows that all the eigenvalues are all negative and having real parts. Therefore, the localization of infection for system (2) at T-SFE is locally asymptotically stable for all $S_{g(0)} < 1$ and unstable otherwise.

Tobacco – Smoking Endemic Equilibrium (T-SEE)

Clearly, at $\beta_i c_i(\hat{N}) \neq 0$ implies that $S_{g(0)} > 1$, which will ignite introduction of control functions i.e. $(u_1, v_1) > 0$, such that $(1-u_1) > 0$ denotes that rate at which $Q_p(t)$ administer nicotine replacement therapy and $(1-v_1) > 0$ represent the intake of non-nicotine medications by $Q_p(t)$. Then, at $S_{g(0)} > 1$, system (2) is bound to exhibit disease endemicity. Thus, the following theorem justify the existence of T-SEE.

Theorem 5

If $S_{g(0)} > 1$, then for system (2), there exists locally asymptotic stability at T-SEE (E^*), otherwise unstable.

Proof We shall investigate the proof of this theorem in two folds:

Method 1 Invoking existing result for local asymptotic stability of endemic equilibrium [20].

From system (2), the differential sum is derive as:

$$\frac{dN}{dt} = b_p - \mu N - \mu_i(S_m + S_c)$$

or

$$\frac{dN}{dt} = b_p - \mu N - \mu_i(\bar{N}),$$

where $\bar{N} = S_m + S_c$ and μ_i is the death rate due to tobacco smoking.

But $\frac{dN}{dt} = 0$, then we have

$$0 = b_p - \mu N - \mu_i(\bar{N}) \quad (23)$$

or

$$b_p - \mu N^* - \mu_i(\bar{N}) = 0,$$

where N^* is the endemic population. Solving for N^*, we have

$$N^* = \frac{b_p - \mu_i(\bar{N})}{\mu} \quad (24)$$

which corresponds to the fact that at equilibrium, $N^* = \frac{b_p}{\mu}$ if $\beta_i^* = 0$. If $\beta_i^* \neq 0$, then there exists endemic infection and Equation (24) can be rewritten in terms of $S_{g(0)}$ i.e.

$$\beta_i^*(1 + \beta_i^* \hat{\Omega} - S_{g(0)}) = 0$$

or

$$\beta_i^* = \frac{S_{g(0)} - 1}{\hat{\Omega}}, \quad (25)$$

where $\hat{\Omega}$ is disease constant derive from Equation (15). Equation (25) holds provided $S_{g(0)} > 1$. Hence prove completed.

Method 2 By adopting the linearization method, we invoking existing result by Routh Hurwitz, [21].

From system (2), the linearization at T-SEE (E^*), yield the following Jacobian matrix $J_{(E^*)}$ as:

$$J_{(E^*)} = \begin{pmatrix} -\mu-\beta_i(\hat{N}) & -(\beta_i(\hat{N})+\beta_1 c_1)\frac{b_p}{\mu} & -(\beta_i(\hat{N})+\beta_2 c_2)\frac{b_p}{\mu} & -(\beta_i(\hat{N})+\beta_3 c_3)\frac{b_p}{\mu} & -(\beta_i(\hat{N})+\beta_4 c_4)\frac{b_p}{\mu} & \eta \\ \beta_i(\hat{N}) & -(q_1+(\beta_i(\hat{N})+\beta_1 c_1)\frac{b_p}{\mu}) & (\beta_i(\hat{N})-\beta_2 c_2)\frac{b_p}{\mu} & (\beta_i(\hat{N})-\beta_3 c_3)\frac{b_p}{\mu} & -[\delta+(\beta_i(\hat{N})+\beta_4 c_4)\frac{b_p}{\mu}] & 0 \\ 0 & \alpha & -q_2 & 0 & 0 & 0 \\ 0 & 0 & -q_3 & -q_4 & 0 & 0 \\ 0 & 0 & -q_5 & 0 & -(q_9+q_5) & 0 \\ 0 & 0 & 0 & \tau_1 & \tau_2 & -(\mu+\eta) \end{pmatrix}. \quad (26)$$

The auxiliary equation for Equation (26) takes the form:

$$(\lambda+\mu)(\lambda+\delta+\mu)(\lambda+\phi+\mu+\mu_i)(\lambda+\tau_1+\tau_2+\mu)a_1\lambda + a_2 = 0$$

where $a_1 = -[x_{11} - x_{22}] = -(\mu - \beta_i(\hat{N}) - (-q_1 + (\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu})$ and

$$a_2 = \{(x_{11}x_{22}) - (x_{12}x_{21})\} = \{(-\mu + \beta_i(\hat{N}))(-(q_1 + (\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu} - (-\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu}(-\beta_i(\hat{N}))\}$$

Then, using the Routh Hurwitz criterion, the equilibrium state E^* is asymptotically stable provided a_1, a_2 >0. Hence, for $S_{g(0)} > 1$ system (2) is locally asymptotically stable.

Remark 1

i. Theorem 4, satisfies the existence of T-FEE (E^0) at $S_{g(0)} < 1$.

ii. Theorem 5, validates the existence of T-SEE (E^*) at $S_{g(0)} > 1$.

Asymptotic Global Stability Analysis

Here, we adopt the second additive compound matrix approach with the incorporation of Lozinskii measure *lB* to investigate the existence of system asymptotic global stability of our model. The following lemma and theorem holds:

Lemma 1 If $\dot{x} = \{F(x) \setminus F : D \to \Re^n\}$ be an endemic equilibrium of the form $\dot{x}$ and there is a compact absorbing set, then this system is globally asymptotically stable around that equilibrium provided there exists a function $D(x)$ and a Lozinskii measure l, such that

$$\underset{\to\infty}{Lim} \sup\sup - \int l(B)dt \quad 0,$$

where l is the Lozinskii measure, B , is the field (or integrand) and D, the compact set.

Theorem 6

If $S_{g(0)} > 1$ then, the smoking model (2) is globally asymptotically stable at endemic equilibrium E^*.

Proof

Let J be the Jacobian matrix and $J^{|2|}$ be the second additive compound matrix contained in the first three equations of system (2), since these equations are actual targets of endemic infection. Then,

$$J = \begin{bmatrix} -\mu - \beta_i(\hat{N}) & -(\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu} & -(\beta_i(\hat{N}) + \beta_2 c_2)\frac{b_p}{\mu} \\ \beta_i(\hat{N}) & -(q_1 + (\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu}) & (\beta_i(\hat{N}) - \beta_2 c_2)\frac{b_p}{\mu} \\ 0 & \alpha & -q_2 \end{bmatrix}. \quad (27)$$

Now, if

$$J^{|2|} = \begin{vmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{12} + a_{32} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{vmatrix}. \quad (28)$$

Then, from Equation (28), Equation (27) becomes

$$J^{|2|} = \begin{vmatrix} h_1 & (\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu} & (\beta_i(\hat{N}) + \beta_2 c_2)\frac{b_p}{\mu} \\ \alpha & h_2 & -(\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu} \\ 0 & -\beta_i(\hat{N}) & h_3 \end{vmatrix}, \quad (29)$$

where $h_1 = -\mu - \beta_i(\hat{N}) - q_1 - (\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu}$

$h_2 = -\mu - \beta_i(\hat{N}) - q_2$ and $h_3 = -(q_1 + (\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu}) - q_2$.

Using Lemma 1, we let the function $F(x)$, be defined by

$$F(x) = (P_s, S_m S_c) = diag\left\{\frac{P_s}{S_m}, \frac{P_s}{S_m}, \frac{P_s}{S_m}\right\},$$

which, implies that $F^{-1}(x) = diag\left\{\frac{S_m}{P_s}, \frac{S_m}{P_s}, \frac{S_m}{P_s}\right\}$.

Taking the derivative with respect to time, we have

$$F'(x) = diag\left\{\frac{\dot{P}_m}{P_s} - \frac{P_S\dot{S}_m}{S_m^2}, \frac{\dot{P}_m}{P_s} - \frac{P_s\dot{S}_m}{S_m^2}, \frac{\dot{P}_m}{P_s} - \frac{P_S\dot{S}_m}{S_m^2}\right\}. \quad (30)$$

Now, the product of inverse and derivative of the function $F(x)$, gives

$$F'F^{-1} = diag\left\{\frac{P_m}{P_s} - \frac{S_m}{S_m}, \frac{P_m}{P_s} - \frac{S_m}{S_m}, \frac{P_m}{P_s} - \frac{S_m}{S_m}\right\}$$

and

$F'J^{|2|}F^{-1} = J^{|2|}$.

Thus, we can take

$$B = F'F^{-1} + FJ^{|2|}F^{-1}. \quad (31)$$

In matrix form, LHS of Equation (31), is given by

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix},$$

where $B_{11} = \frac{P_m}{P_s} - \frac{S_m}{S_m} - h_1, B_{12} = (\beta_i(\hat{N}) + \beta_2 c_2)\frac{b_p}{\mu} - (\beta_i(\hat{N}) + \beta_2 c_2)\frac{b_p}{\mu}$

$B_{21} = (\alpha, 0)^t$ and $B_{22} = \begin{pmatrix} h_2 & -(\beta_i(\hat{N}) + \beta_1 c_1)\frac{b_p}{\mu} \\ -\beta_i(\hat{N}) & h_3 \end{pmatrix}$

Let (x_1, x_2, x_3) be a vector in $\Re^3$ and its norm $\|\|$ defined by $\|x_1, x_2, x_3\| = \max\{\|x_1\|, \|x_2\|, \|x_3\|\}$. Let be the Lozinskii measure with respect to the norms. Then, from existing results, we have, [22].

$l(B) \le \sup\{f_1, f_2\} = \sup\{l(B_{11}) + \|(B_{12})\|, l(B_{22}) + \|(B_{21})\|\}$,

where $f_i = l(B_{ii}) + \|l(B_{ij})\|, \forall \ i = 1,2, i \neq j$. This implies that

$f_1 = \{l(B_{11}) + \|l(B_{12})\|\}$, $f_2 = \{l(B_{22}) + \|l(B_{21})\|\}$,

where $l(B_{11}) = \frac{P_s}{P_s} - \frac{S_m}{S_m} - h_1$, $l(B_{12}) = 0$, $l(B_{21}) = \alpha$,

$l(B_{22}) = \max\left(\frac{P_s}{P_s} - \frac{S_m}{S_m} - h_2, \frac{P_s}{P_s} - \frac{S_m}{S_m} - h_3\right)$.

This implies that

$l(B_{22}) = \frac{P_s}{P_s} - \frac{S_m}{S_m} - h_2 - h_3$ and $\|l(B_{21})\| = \alpha$

Therefore, f_1 and f_2 becomes

$f_1 = \frac{P_s}{P_s} - \frac{S_m}{S_m} - h_3$, $f_2 = \frac{P_s}{P_s} - \frac{S_m}{S_m} - h_2$.

If $h_i := [h_2, h_3]$, then

$$\begin{cases} f_1 \le \frac{P_s}{P_s} - h_i - 2h_i \\ f_2 \le \frac{P_s}{P_s} - \phi - 2h - h \end{cases} \quad (32)$$

which implies that $l(B) \le \left\{ \frac{P_s}{P_s} - 2h_i - \min hl - \phi - \mu_i \right\}$.

Hence, $l(B) \le P_s P_s - 2h$

Now, integrating the Lozinskii measure lB with respect to $t \in [0,t]$ and taking limit as $t \to \infty$, we obtain

$$\underset{t\to\infty}{Lim} \sup\sup \frac{1}{t}\int_0^t l(B)dt \le -2h < 0 \cdot \quad (33)$$

Equation (33) can be written in the form

$$\hat{s} = \underset{t\to\infty}{Lim} \sup\sup \frac{1}{t}\int_0^t l(B)ds < 0 \cdot \quad (34)$$

Thus, the system containing the first three equations of system (2) is globally asymptotically stable around the interior equilibrium (P_s^*, S_m^*, S_c^*).

Now, from the last three equations of system (2), we have

$$\begin{aligned} \frac{dQ_p}{dt} &= (1-u_1)\phi\varepsilon S_c - (\tau_1+\mu)Q_p \\ \frac{dQ_t}{dt} &= (1-v_1)(1-\varepsilon)\phi S_c - (\tau_2+\delta+\mu)Q_t \cdot \\ \frac{dR_p}{dt} &= \tau_1 Q_p + \tau_2 Q_t - \eta R_p - \mu R_p \end{aligned} \quad (35)$$

Taking limits of Equation (35), we get

$$\begin{aligned} \frac{dQ_p}{dt} &= (1-u_1)\phi\varepsilon S_c^* - (\tau_1+\mu)Q_p \\ \frac{dQ_t}{dt} &= (1-v_1)(1-\varepsilon)\phi S_c^* - (\tau_2+\delta+\mu)Q_t \cdot \\ \frac{dR_p}{dt} &= \tau_1 Q_p + \tau_2 Q_t - \eta R_p - \mu R_p \end{aligned} \quad (36)$$

Solving Equation (36), and using the initial conditions $Q_p(0)$, $Q_t(0)$, $R_p(0)$ for large time t i.e. $t \to \infty$, $Q_p \to Q_p^*$, $Q_t \to Q_t^*$ and $R_p \to R_p^*$, which is sufficient to prove that the endemic equilibrium point E^* is globally asymptotically stable.

Numerical Computations

Here, we explore classical numerical simulation to verify system derived analytical predictions of sections 2-4, noting that analytic computations are invariably imperative due to complexity of system non-linear equations. In the course of our investigation, two indices have been paramount, which include: the system force of infection $\beta_i(\hat{N})$ and the system smoking reproduction numbers $S_{g(0)} \cong (S_{g(1)}, S_{g(2)})$. Notably, smoking generation numbers $(S_{g(1)}, S_{g(2)})$ are functions of system mass action $\beta_i(\hat{N})$, intensity rate of smoking α and screening rate ϕ. Therefore, we this important component to numerically illustrate its inclusion in present study.

Next, we shall simulate the system basic model (2) at off-treatment scenario, which is anticipated to induce the contributive role of $S_{g(1)}$. Finally, with the introduction of bilinear control functions to endemic smoking epidemic, we shall simulate derived model for treatment on-set scenario at $S_{g(1)}$. The entire simulations explore in-built rkfixed Runge-Kutta of order of precision 4 in a Mathcad surface in relation to established data as in Table 2.

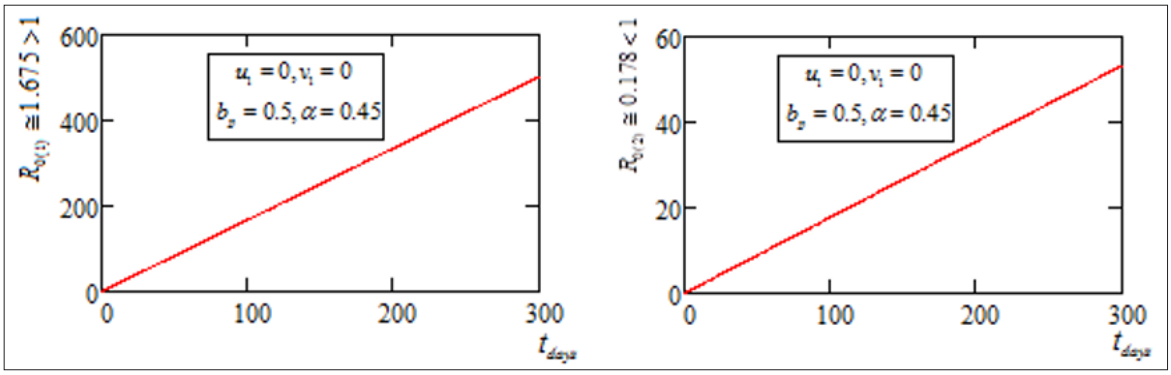
Table 2: State-space and Parameter Variables with Initial Values for Model (2)

State-space				Parameter variables			
Symbols	**Initial values**	**Units**	**References**	**Symbols**	**Initial values**	**Units**	**References**
P_s	0.5	$cellsmm^{-3}$	Estimated	b_p	0.25		[22,23]
S_m	0.19			μ	0.014		[2]
S_c	0.18			$\beta_{i(i=1,,,,4)}$	0.004,		[21]
					0.0012,		
					0.0029,		
					0.005		
Q_p	0.17			μ_i	0.00021		[8,16]
Q_t	0.16			α	0.45		[8,16]
R_p	0.1			ϕ	0.5	$Time^{-1}$	UCTH
				ε	0.628		Estimated
				$c_{i(i=1,..4)}$	0.5, 0.4, 0.3, 0.2		Estimated
				$(1-\varepsilon)$	0.312		Estimated
				τ_1, τ_2	[0,1]		[16, 20,24]
				η	[0,1]		UCTH
				δ	0.025		[5]
				u_1, v_1	$(u_1, v_1) \in [0,1]$		UCTH

Note: UCTH – University of Calabar Teaching Hospital

Simulations of System Generation Numbers, $S_{g(1)}, S_{g(2)}$

In the analytical predictions of this study, we observed that transmission rate and effect of control functions is further ascertained by the numerical computation of the model smoking generation numbers $S_{g(1)}, S_{g(2)}$. Figure 3(a-b) below, depict the observed simulations.



a) Smoking generation number for tobacco smoking smoking under off-treatment with $\beta_i > 0, \mu_i = 0.00021$

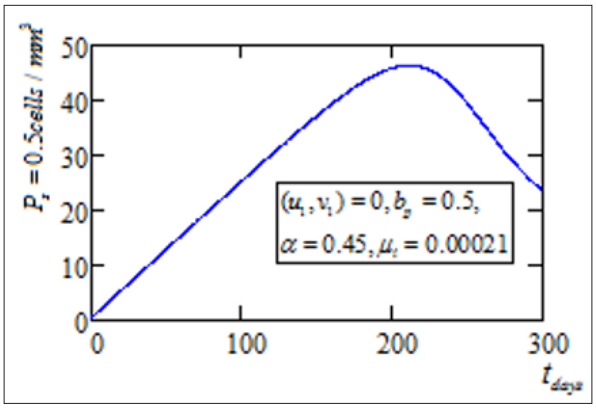
a) Smoking generation number for tobacco under onset-treatment with $\beta_i > 0, \mu_i = 0.00021$

Figure 3 (a-b): Graphical images of smoking generation numbers for off/onset – treatments of tobacco smoking, $\Re_{0(1)}, \Re_{0(2)}$

From Figure 3(a), it is observed that spread of tobacco-smoking incidence rate is portray by smooth rapid incline linear curve indicating geometric overture of adverse spread of smoking infection under off-treatment scenario. That is, smoking generation number is valid in the range $1.675 \leq S_{g(1)} \leq 502.527$ for all $t_f < 300$ *days*. With introduction of bilinear control functions, Figure 3(a), exhibit some smooth non-rapid linear curve portraying drastic reduction in the spread of smoking effect with value range of $0.178 \leq S_{g(2)} \leq 53.254$ for all $t_f \leq 300$ *days*.

Numerical Simulations of System Model Under Off-Treatment, $(u_1 = 0, v_1 = 0)$

Here, we explicitly implement the vital input of system basic smoking generation number $S_{g(1)}$ as demonstrated by the simulation of system basic model at off-treatment scenario i.e. $(u_1, v_1) = 0$, $\beta_i > 0$. Using computed algorithm and corresponding results of appendix *A*1 and *A*2, we present the smoking dynamics as depicted by Figure 4(a-f), below:



From Figure 4(a), we observe that potential smoking population exhibit initial steady smooth incline population, which depicts asymptotic infection stage under off-treatment scenario with value of $0.5 \leq P_s(t) \leq 46.334$ *cellsmm*$^{-3}$ for all $t_f \leq 210$ days. Furthermore, the curve exhibits convex-like declination, indicating the consequential effect of smoking with decline value of $46.334 \geq P_s(t) \geq 23$ *cellsmm*$^{-3}$ for all $210 \leq t_f \leq 300$ days.

a) Potential smokers under off-treatment, $S_{g(1)} \geq 1.675$

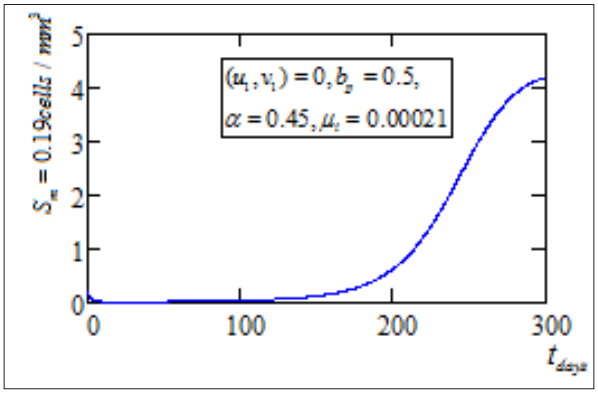
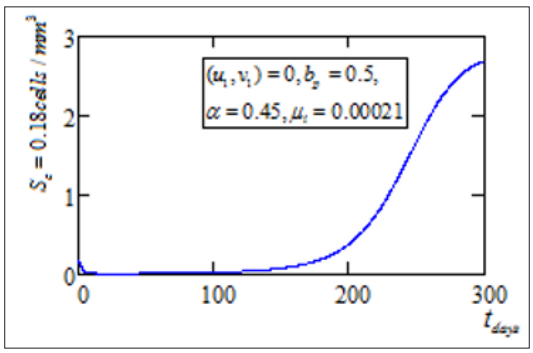


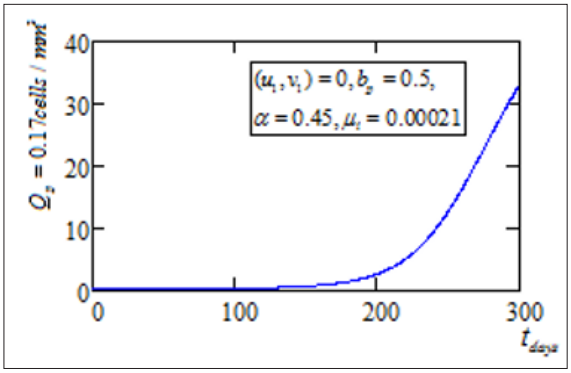
Figure 4(b), which depicts the subpopulation of mild smokers exhibit initial asymptotic saddle point, indicating insignificant mild spread of smoking infection at the early stage of smoking with value range of $0.018 \leq S_m(t) \leq 0.5$ *cellsmm*$^{-3}$ for all $t_f \leq 200$days. The curve thereafter exhibits incline symptomatic infection rate with value range of $0.5 \leq S_m(t) \leq 4.193$ *cellsmm*$^{-3}$ for all $200 \leq t_f \leq 300$ days.

b) Mild smokers under off-treatment, $S_{g(1)} \geq 1.675$



Under off-treatment control functions, Figure 4(c) exhibit similar undulating linear asymptotic saddle-point like curve as in Figure 4(b). Here, asymptotic initial infection rate lies in the interval $0.012 S_c(t) \leq 0.018$ *cellsmm*$^{-3}$ for all $t_f \leq 200$ days. A variation of the infection flow shows symptomatic incline infection rate with value at $0.018 \leq S_c(t) \leq 2.687$ *cellsmm*$^{-3}$ for all $200 \leq t_f \leq 300$ days.

c) Chronic smokers under off-treatment, $S_{g(1)} > 1.675$,



In the absence of control functions, Figure 4(d) depicts the proportion of permanent quitters of tobacco smoking. The population exhibit similar asymptotic saddle point at $0.012 \leq Q_p(t) \leq 0.17$ *cellsmm*$^{-3}$ for all $t_f \leq 120$ days. Thereafter, the effect of lack of control functions is vindicated by the concave-like inclination of infection rate with value range of $0.17 \leq Q_p(t) \leq 33.137$ *cellsmm*$^{-3}$ for all $120 \leq t_f \leq 300$ days.

d) Permanent quitters under off-treatment ,

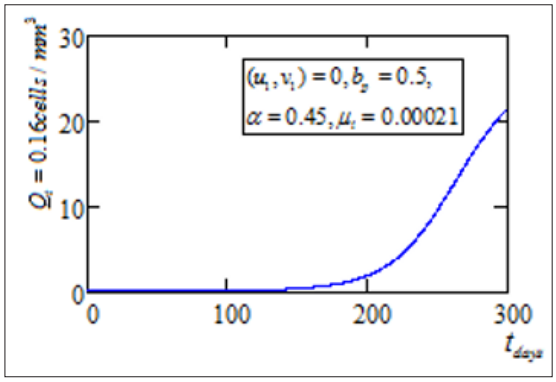
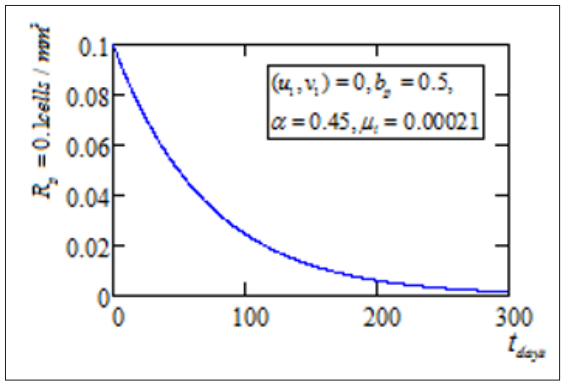


Figure 4(e) exhibit similar diminish charactisitcs of Figure 4(d), which portrait early asymptotic saddle infection rate $0.012 \leq Q_t(t) \leq 0.142$ *cellsmm*$^{-3}$ for all $t_f \leq 140$ days. With zero control functions, the subpopulation exhibits gradual convex-like spread of smoking infection with value range of $0.142 \leq Q_t(t) \leq 21.512$ *cellsmm*$^{-3}$ for all $140 \leq t_f \leq 300$ days.

e) Temporal quitters under off-treatment, $S_{g(1)} \geq 1.675$,



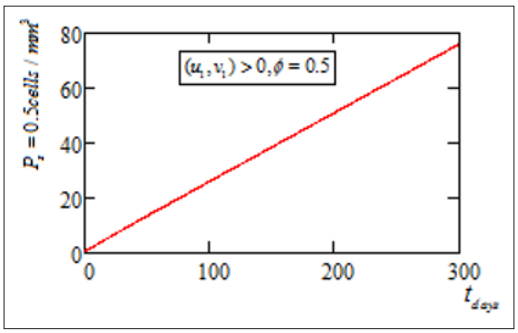
From Figure 4(f), which represent the recovery population, we observe rapid decline, following the non-availbility of any control functions. Here, the initial population of $R_p(t) \leq 0.1 cellsmm^{-3}$, decay with concave-like linear curve of $0.1 \leq R_p(t) \leq 1.5 \times 10^{-3}$ *cellsmm*$^{-3}$ for all $t_f \leq 300$ days.

f) Recovered smokers under onset-treatment, $S_{g(1)} \geq 1.675$,

Figure 4(a-f): Graphical images for computed off-treatment of tobacco-smoking dynamics $S_{g(1)} \geq 1.675 > 1$

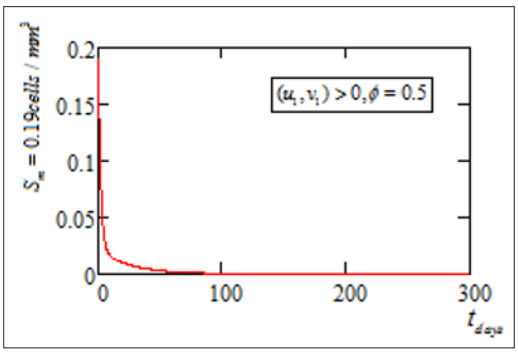
Numerical Simulations of System Endemic Effect Under Bilinear Controls ($u_1 > 0$, $v_1 > 0$)

In this subsection, we compute numerically, derive theoretical endemic indices to further uncover derived globall stability conditions following the application of designated bilinear control functions ($u_1 > 0$, $v_1 > 0$) , noting that at this poin, smoking generation index as computed is $S_{g(2)} \geq 0.176 < 1$ and $\varepsilon' = (1-\varepsilon)$, $\tau_i = (\tau_1 + \tau_2)$ respectvely. Thus, using program algorithm and corresponding results of appendix *B*1 and *B*2, we present the smoking effect and treatment dynamics as depicted by Fig. 5(a-f), below:



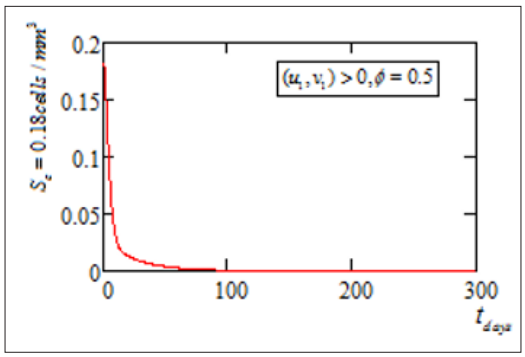
From Figure 5(a), we observe that with the introduction of bilinear control functions in the presence of screening method, potential smokers exhibit smooth accelerated incline linear curve with rejuvenated value range of $0.5 \leq P_s(t) \leq 76.045$ *cellsmm*$^{-3}$ for all $t_f \leq 300$ days.

a) Potential smokers under onset-treatment, $S_{g(2)} \geq 0.178$, $\eta = 0.125$



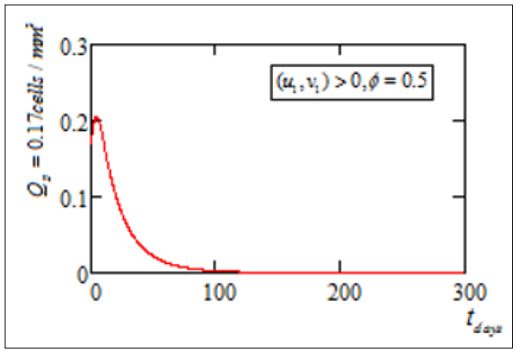
With the introduction of both screening and bilinear control functions, mild smokers depicted by Figure 5(b), exhibits sharp spontaneous decline of smoking infection. Moreso, diligent elimination of smoking effect by the mild subpopulation is seen after $t_f \leq 90$ days with diminish value range of $0.19 \leq S_m(t) \geq 1.774 \times 10^{-6}$ *cellsmm*$^{-3}$.

b) Mild smokers under onset-treatment, $S_{g(2)} \geq 0.178$, $\delta = 0.025$,



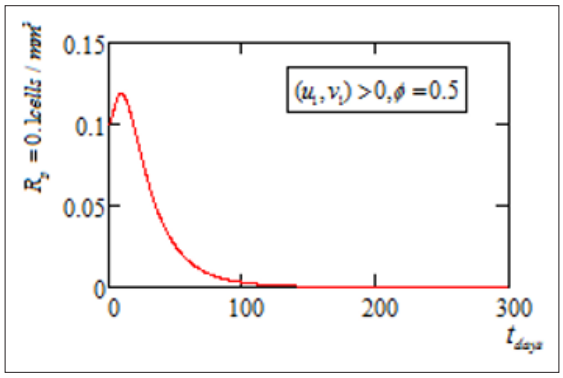
Under induce bilinear control mechanism with screening method, chronic smokers compartment represented by Figure 5(c), exhibit enhance sharper decline of smoking infection. Near zero reduction of smoking effect is seem with value range of $0.182 \leq S_c(t) \geq 2.128 \times 10^{-6}$ *cellsmm*$^{-3}$ for all $t_f \leq 96$ days. Thereafter, the curve exhbits infection saddle point at $96 \leq t_f \leq 90$ days.

c) Chronic smokers under onset-treatment, $S_{g(2)} \geq 0.178$, $\mu_i = 0.00021$



From Figure 5(e), we observe that subjecting temporal quitters of tobacco smokers to both designated bilinear controls and screening method lead to initialslight incline infection but exhibit rapid decline thereafter due to onset-treatment. Here, tobacco infection is reduce to near zero status with value range of $0.253 \geq Q_t(t) \geq 1.182 \times 10^{-5}$ *cellsmm*$^{-3}$ for all $10 \leq t_f \leq 140$ days. Compartment curve attain saddle stability through $140 \leq t_f \leq 300$ days.

e) Temporal quitters under onset-treatment, $S_{g(2)} \geq 0.178, \varepsilon' = 0.312$



Recovered compartment depicted by Figure 5(f), clearly exhibit initial inclination indicating massive recovery, which can be attributed to observatory medication in the first $t_f \leq 10$ days. The decline thereafter indicates gradual discharge fo recovered population to interface with potential smokers after $10 \leq t_f \leq 140$ days for all value of $0.119 \geq R_p(t) \geq 5.735 \times 10^{-6}$ *cellsmm*$^{-3}$ and thereafter the curve exhibit saddle point for all $140 \leq t_f \leq 300$ days.

f) Recovered smokers under onset-treatment, $S_{g(2)} \geq 0.178$, $\tau_i = 0.085$

Figure 5(a-f): Graphical Images for Computed Onset-Treatment of Tobacco-Smoking Dynamics, $S_{g(2)} \geq 0.178 < 1$

Discussion of Results

Ignited by the limitations of system motivating model, the present investigation have been formulated as an extended 6-Dimisional deterministic mathematical dynamic model [8]. Investigating the interactions between designated subpopulations and the vector (tobacco-smoking effect), the system explored bilinear control functions (nicotine replacement therapy with non-nicotine medications for permanent quitters and non-nicotine medications with over-counter nicotine for temporal quitters) in the presence of screening mechanism. The main objective of research was the investigation of the methodological application of bilinear control functions in the presence of screening method, geared towards the eradication of adverse effect of tobacco smoking in an epidemic scenario.

In the construction of the present model, a number of core predominant assumptions were considered. Among which include: only the screened are subjeceted to application of control functions and that rate of death due to infection are only appicablr to both temporal and permanent smokers and may vary. Model formulation and investigation of system well-posedness explored the concept of fundamental theory of ordinary differential equations. Furthermore, analytic predictions of model constituted by system local and global behavior, was extensively conducted using classical theory of stability analysis inconjunction with second additive compound matrix and Lozinski measure . Remarkably, system analytic predictions were simplified to off-treatment and onset treatment scenarios and not only were the system basic and effective tobacco-smoking generation numbers determined but correspondin numerical illustrations explicitly computed.it is aloso nited tha due to the complexity of system non-linear equations, model simulations explored highly in-built Runge-Kutta of order of precision 4 in a Mathcad surface.

Following numerical computations, results showed that for untreated smoking effect with mass action $\beta_i(\hat{N}) \cong 7.743 \times 10^{-3}$, the amount of concentrated smoking generation number required to contaminate a proportion of potential smokers of range $0.5 \leq P_s(t) \leq 46.334$ *cellsmm*$^{-3}$ is in domain of $S_{g(1)} \geq 1.675 > 1$ for all days. Under off-treatment scenario, $Q_p(t) \leq 33.137$ *cellmm*$^{-3}$ of the infected proportion are liable to die after $t_f \leq 300$ days due to non-quitting of smoking with no access to medications. This result is in confirmation with that of motivating model under off-treatemnt, [8]. Recovery at this circumstance is near zero with value at $R_p \leq 1.5 \times 10^{-3}$ for all $t_f \leq 300$ days.

Impressively, with the introduction of designated bilinear controls at specificated state-space, under screening techniques, it was observed that not only did rate of infection $\beta_i(\hat{N}) \cong 2.304 \times 10^4$, drastically reduced but smoking generation number equally declined to a low value of $S_{g(2)} \cong 0.178 < 1$. This is an insignificant infection rate, which give way to rejuvenation of potential smokers to some geometric inclined range of $0.5 \leq P_s(t) \leq 76.045$ *cellsmm*$^{-3}$ for all $t_f \leq 300$ days. This overwhelming result not only out-class those of [8,12]., but most interestingly depicted a more indept outcome when compared with that from, where optimal control techniques was deeply applied [2].

Furthermore, following the induced bilinear controls, other compartments inclusive of $S_m(t)$,$S_c(t)$, $Q_t(t)$, and $Q_t(t)$ exhibited bear zero elimination of smoking infection with varying time range of $90 \leq t_f \leq 140$ days. More explicitly, is the fact that recovery compartment $R_p(t)$, which had been reduced to near zero, clearly indicated rapid proliferation of recovered to the potential subpopulation. Objectively, the inducement of bilinear control functions under enhance screening method, have presented a first kind analysis of smoking effect transmission and sequential aaplication of bilinear treatment dynamics.

Conclusion

Triggered by the avalanche of consequential effect of tobacco-smoking and the non-avaliability of mathematical predictions that explicitly accounted for application of treatment functions, this present investigation accidiously attempted to profer an insight to the methodological application of bilinear control functions geared towards the eradication of the effect of tobacco-smoking the world-over. Essentially, the present investigation have been systematically designed to account for global stability indices of infection transmission and application of bilinear control fucntions for the consequebntial effect of tobacco-smoking epidemic.

In using fundamental theory of ordinary differential equations for model formulation, this research explored extensively, the application of classical theory of stability analysis in conjunction with second additive compound matrix induced by Lozinski measure *lB* for the determination of both system local and global indices of smoking transmission and treatment dynamics. Furthermore, system analytic predictiions were illustrated numerically at both off-treatment and onset-treatment scenarios. Results of numerical computstions clearly indicated largely asymptomatic spread of adverse effect of tobacco consumption at off-treatment scenario with smoking geberation number $S_{g(1)} \geq 1.675$. This outcome clearly definied why consequential effect of smoking are not dictated early enough for all $t_f \leq 140$ days. Moreso, the outcome in reality represent real-ife expectant of asymptotmatic stage of smoking effect. A result that can be attributed to human adaptive immune system. Collapse of immune system was thereafter observed following consistent smoking habit for all $140 \leq t_f \leq 300$ days of clinical investigation. A result that conformed with that of for infectious population without control programs [2].

Further simulations under bilinear controls with smoking generation number $S_{g(2)} \geq 0.178$, saw a linear exponential rejuvenation of potential smokers with rising value range of $0.5 \leq P_s(t) \leq 76.045$ $cellsmm^{-3}$ for all $t_f \leq 300$ days. Active infectious population under bilinear control investigations indicated near complete eradication of smoking effect within time frame of $80 \leq t_f \leq 140$ days wth mild smokers getting rid of infection much more earlier $t_f \leq 80$ days. At $t_f \leq 100$ days, recovered population exhibited proliferation to potential compartment. This outcome was an improvement to those of with only screening approach and with induced social controls explored optimal control techniques [2,8]. Thus, to stem down the increasing adverse effect of tobacco-smoking, this present control techniques is highly recommended for immense implementation by both tobacco (cigratte) companies and governments of various countries the world-over [23-27].

Data Availability

The data generated for this mathematical modeling include those obtained from city of Calabar and UCTH–Calabar and Cross River State Ministry of Health, in affiliation with published parameter values as cited accordingly.

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Declaration of Competing Interest

The corresponding author attest to the fact that there exists no conflict of interest in the submission of this manuscript and that the entire content remains the original recipes of the author, which had not been submitted for consideration to any journal outfit.

Credit Authorship Contribution Statement

The authors' contributions to this manuscript were thus - **Bassey Echeng Bassey:** conceptualization, methodology, data collections, writing – original draft, writing – reviews and editing, algorithm and software programing, analysis and writing of final version. **Iwge O. Ewona:** Methodology, supervision, formal analysis, editing and valuations. **Adagba Odey Henry:** Supervision, methodology, review, funding, editing and validation.

Delphine Rexson Bassey: Methodology, data collection, formal analysis, editing and simulations

Appendix A

Appendix A1: **Program algorithm for Fig. 4(a-f)**

$$u1 := 0.(\qquad v1 := 0.($$

$$H := (0.5 \;\; 0.19 \;\; 0.18 \;\; 0.17 \;\; 0.16 \;\; 0.1)^T$$

$$F(t,H) := \left| \begin{array}{l} \beta i \leftarrow (1-u1)\cdot(1-v1)\cdot\left(\beta 1\cdot c1\cdot H_2 + \beta 2\cdot c2\cdot H_3 + \beta 3\cdot c3\cdot H_4 + \beta 4\cdot c4\cdot H_5\right) \\ F_1 \leftarrow bp + \eta\cdot H_6 - \left(\beta i\cdot H_1\right)\cdot\mu\cdot H_1 \\ F_2 \leftarrow \left(\beta i\cdot H_1\right) + \left(\delta\cdot H_5\right) - \left(\alpha\cdot H_2\right) - (\mu + \mu i)\cdot H_2 \\ F_3 \leftarrow \left(\alpha\cdot H_2\right) - \left\lceil (1-u1)\cdot\phi\cdot\varepsilon\cdot H_3 \right\rceil - \left\lceil (1-v1)\cdot(1-\varepsilon)\cdot H_3 \right\rceil - (\mu + \mu i)\cdot H_3 \\ F_4 \leftarrow (1-u1)\cdot\phi\cdot\varepsilon\cdot H_3 - (\tau 1 + \mu)\cdot H_4 \\ F_5 \leftarrow \left\lceil (1-v1)\cdot(1-\varepsilon)\cdot H_3 \right\rceil - (\tau 2 + \delta + \mu)\cdot H_5 \\ F_6 \leftarrow \tau 1\cdot H_4 + \tau 2\cdot H_5 - \eta\cdot H_6 - \mu\cdot H_6 \\ F \end{array} \right.$$

Appendix A2: **Results for program algorithm A1**

$J := \text{rkfixed}(H, 0, T, n, F) =$

	1	2	3	4	5	6	7
1	0	0.5	0.19	0.18	0.17	0.16	0.1
2	0.3	0.575	0.167	0.168	0.186	0.177	0.1
3	0.6	0.65	0.146	0.155	0.2	0.193	0.099
4	0.9	0.725	0.129	0.142	0.213	0.207	0.099
5	1.2	0.8	0.114	0.13	0.225	0.22	0.098
6	1.5	0.875	0.101	0.118	0.236	0.231	0.098
7	1.8	0.95	0.089	0.107	0.245	0.241	0.098
8	2.1	1.025	0.08	0.097	0.254	0.25	0.097
9	2.4	1.1	0.071	0.088	0.262	0.257	0.097
10	2.7	1.175	0.064	0.08	0.268	0.263	0.096
11	3	1.25	0.058	0.072	0.274	0.269	0.096
12	3.3	1.325	0.052	0.065	0.28	0.273	0.095
13	3.6	1.4	0.048	0.059	0.284	0.277	0.095
14	3.9	1.475	0.044	0.053	0.288	0.28	0.095
15	4.2	1.55	0.04	0.048	0.292	0.282	0.094
16	4.5	1.625	0.037	0.044	0.295	0.284	...

Appendix B

Appendix B1: **Program algorithm for Fig. 5(a-f)**

$u1 := 0.5$ $\quad v1 := 0.3$

$H := (0.5 \;\; 0.19 \;\; 0.18 \;\; 0.17 \;\; 0.16 \;\; 0.1)^T$

$$
F(t,H) := \left|
\begin{array}{l}
\beta i \leftarrow (1-u1)\cdot(1-v1)\cdot\left(\beta 1\cdot c1\cdot H_2+\beta 2\cdot c2\cdot H_3+\beta 3\cdot c3\cdot H_4+\beta 4\cdot c4\cdot H_5\right)\\
F_1 \leftarrow bp+\eta\cdot H_6-\left(\beta i\cdot H_1\right)\cdot\mu\cdot H_1\\
F_2 \leftarrow \left(\beta i\cdot H_1\right)+\left(\delta\cdot H_5\right)-\left(\alpha\cdot H_2\right)-(\mu+\mu i)\cdot H_2\\
F_3 \leftarrow \left(\alpha\cdot H_2\right)-\left[(1-u1)\cdot\phi\cdot\varepsilon\cdot H_3\right]-\left[(1-v1)\cdot(1-\varepsilon)\cdot H_3\right]-(\mu+\mu i)\cdot H_3\\
F_4 \leftarrow (1-u1)\cdot\phi\cdot\varepsilon\cdot H_3-(\tau 1+\mu)\cdot H_4\\
F_5 \leftarrow \left[(1-v1)\cdot(1-\varepsilon)\cdot H_3\right]-(\tau 2+\delta+\mu)\cdot H_5\\
F_6 \leftarrow \tau 1\cdot H_4+\tau 2\cdot H_5-\eta\cdot H_6-\mu\cdot H_6\\
F
\end{array}
\right.
$$

Appendix B2: **Results for program algorithm B2**

$J := rkfixed(H, 0, T, n, F) =$

	1	2	3	4	5
1	0	0.5	0.19	0.18	0.17
2	0.3	0.579	0.166	0.182	0.174
3	0.6	0.658	0.146	0.182	0.179
4	0.9	0.736	0.128	0.179	0.183
5	1.2	0.815	0.113	0.174	0.187
6	1.5	0.894	0.1	0.168	0.19
7	1.8	0.973	0.088	0.161	0.194
8	2.1	1.052	0.078	0.154	0.196
9	2.4	1.131	0.07	0.146	0.199
10	2.7	1.209	0.062	0.138	0.201
11	3	1.288	0.056	0.13	0.202
12	3.3	1.367	0.05	0.122	0.204
13	3.6	1.446	0.046	0.115	0.204
14	3.9	1.526	0.041	0.108	0.205
15	4.2	1.605	0.038	0.101	0.205
16	4.5	1.684	0.035	0.094	...

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