The Dynamics of Passive Smoking in the Presence of Pro and Anti-Smoking Campaigns

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Abstract

Globally, the effects of passive smoking are considered to be dangerous to non-smoking as that of actual smoking. This paper formulates and analyzes the effect of smoking in the presence of pro and anti-smoking adverts using a deterministic compartmental math- ematical model. In modelling the dynamics of smoking, the population was divided into five subclasses of potential smokers who respond to pro-smoking advert, potential smokers who respond to anti-smoking adverts, and light smokers, chain smokers and quitters. The model was analysed for the existence of smoke free and endemic equilibrium points and their stability. Numerical simulations were carried out to support the analytical results and to determine the parameters influencing the dynamics of the smoking. It was shown that the smoke free equilibrium point is asymptotically stable when the effective reproduction number is less than unit and unstable when an effective reproduction number is greater than unit. The model analysis shows that pro-smoking and anti-smoking adverts have substantial effect on increment or eradication of smoking habits. It was noted that anti-smoking adverts reduces the smoking habit by eighty percent while the pro-smoking advertisement increases the smoking habits. In order to reduce the spread of smoking habits the efficacy rate of the anti-smoking adverts should be sufficiently large while pro-smoking adverts should be minimized.

Keywords: Pro-smoking; Anti-smoking; Adverts; Mathematical modelling; Differential equation.

Introduction

In recent years, the anti-smoking campaigns have gained remarkable considerations around the globe. In growing cities it is common to see many bill boards promoting anti-smoking campaigns. The popularity of the strategy has stemmed from the fact that it reduces disease, disabilities and deaths related to tobacco use. The effect of these campaigns to health and the economy has led to schools of thoughts. Some studies show that anti-smoking campaigns have significant effects to smokers and passive smokers while others do not agree because these campaigns promote smoking habit. Interested readers are referred to visit the work of Verma and Agarwal (2015) for details.

Because of the complexity of this problem, seeking for suitable mathematical models for analyzing the effect of it has been a continuous task of researchers and experts. For example, Alkhudhari *et al.,* (2014) formulated the effect of occasional smokers by using the nonlinear mathematical model to examine the spread of smoking in a population. A square root model for giving up smoking which associate the potential and occasional smokers were presented by Zeb and the colleagues (2011). Lewis *et al.* (2015) proposed the generalized additive models to assess the effectiveness of tobacco control television advertisements in smoke free homes campaign. Findings indicated that mass media campaigns focusing on secondhand smoke can reduce smoking at home. Recently, Verma and Agarwal (2015) proposed a deterministic math-ematical model to analyze the effect of passive

smoking in the presence of anti-smoking campaigns. Results revealed that when the value of the effective education campaigns between the potential smokers and smokers increases, the number of smokers decreases and if the cost of treatment is decreasing, the number of smokers increases.

Castillo-Garsow *et al.,* (2000) determined the effect on the dynamics of tobacco use and the effect of peer pressure, relapse counselling and treatment using a generalized epidemiological model and found that there is a character of tobacco use among adolescents, there is an incre- ment of the population of smokers after 10 years and the recovery efforts were effected by peer cultures. Sharomi and Gumel (2014) refined the model of Castillo-Garsow and the colleagues (2000) by introducing a new class Qt of smokers who temporarily quit smoking. The results showed that the smoking free equilibrium is globally asymptotical stable whenever a certain threshold is reached. They recommended that a number of campaigns for smokers in the community will be effectively controlled or eliminated at equilibrium point if the threshold is made to be less than one unit. Lahrous *et al.,* (2011) employed mathematical model to determine the global stability of the unique smoking. The mathematical model taking into account the occa-

sional smokers and the possibility of quitters to return to smoking has been studied extensively in Zaman (2011a, 2011b). Van Voorn and Kooi (2013) evaluated the three compartment smoking using brute force simulation for the short term dynamics and bifurcation analysis for long term dynamics.

Khalid *et al.* (2016) did a comprehensive review concerning the perturbation iterative algorithm to determine the fractional of giving up smoking. Numerical results from this reference revealed that potential smokers can reach the global equilibrium point while occasional smokers at first increases and slowly decreases and sometimes may attain equilibrium. Acevedo-Estefania *et al.,* (2000) applied the nonlinear system of differential equation to study the effect of secondhand smoking. They suggested that the best way to lower the number of smokers is to educate them about the effect of smoking through campaigns.

However, until today most of the research on the effect of passive smoking in relation to anti-smoking campaigns have overlooked the pro smoking in the analysis. It is not healthy to ignore such a component because anti-smoking campaign emphasize people to quit from smoking while pro smoking stimulate individuals to enjoy smoking. Also, the mathematical aspects for integrating the anti and pro smoking campaigns are limited literature.

In this paper, the pro and anti-smoking campaign is formulated using mathematical model (as nonlinear differential equation) as proposed by Acevedo-Estefania and the colleagues (2000). Simulation results are presented to illustrate the ability of the proposed model in determining the effect of the campaign.

The key motivation of this work includes:

(a) The formulation of the general mathematical model which integrates the effect of pro and anti-smoking campaigns in the presence of passive smoking population.
(b) Since the stability is guaranteed, simulation to demonstrate the efficacy and versatility of the model was provided.

This paper is organized as follows. Section (2) formulates a mathematical model for analyzing the effect of passive smoking in the presence of pro and anti-smoking campaigns. Section (3) proposes the differential equation model for the effects of passive smoking in the presence of pro and anti-smoking campaigns. Section (4) Simulation to show the capability of the proposed model. Finally, concluding remarks plus future directions of research on the effect of passive smoking in the presence of pro and anti smoking campaigns are given in section (5).

Model Formulation

The total population is divided into three sub-populations which consist of Potential smokers,

Smokers and Quitters. Potential smokers are further split into two classes; those who respond to pro-smoking campaigns (S_p) and those who respond to anti smoking campaigns (S_a) . The smokers are also subdivided into two classes of light smokers who smoke less or equal to 15 cigarettes per day (L_s) and chain smokers who smoke more than 15 cigarettes per day (C_s). All smokers are allowed to quit permanently (Q).

Potential smokers are recruited at a rate π, a proportion P responds to anti-smoking campaigns while the remainder $(1 - P)$ respond to the pro-smoking campaigns. Potential smokers are initiated to smoking with a force of infection λ defined by

$$\lambda(t) = \beta\left[\frac{L_s + \eta C_s}{N}\right]$$

where β is the effective contact rate and η is the relative ability of Cs to initiate new smokers when compared to Ls and N is total population. Since chain smokers can show ill-effect of smoking and are likely not to influence non-smokers, we assume that $0 < \eta < 1$. Assuming that all smokers started to smoke as light smokers, we allow light users to quit permanently at a rate ρ1 and can become chain smokers at a rate γ. Chain smokers can either ameliorate to light smokers or quit permanently at rates σ and ρ2 respectively. Individuals in the compartment Cs die naturally as is with all the other classes at a rate μ and an additional death due to the effects of smoking at the rate δ.

Potential smokers who respond to smoking campaigns and join the potential smokers who are anti-smoking class at a rate ε can be measured by the function g(Sp). Usually, when an individual is against smoking, they rarely become pro-smoking. The function g(Sp) can be linear such ε Sp that g(Sp) = ε Sp. A plausible function would be $g(S_p) = \frac{\varepsilon S_p}{1 + S_p}$, a Michaeli's menter type that assumes maturation at a maximum rate ε. For simplicity and illustrations g(Sp) = ε Sp.

Once an individual is in the anti-smoking class, his/her recruitment into smoking can only be due to failure of the anti-smoking campaigns to protect the individuals. The efficacy of the anti-smoking campaigns is denoted by f, with the assumption that $0<f<1$. $f = 1$ means that the campaigns are 100% effective in preventing initiation and $f = 0$ meaning they are not effective at all.

Here is a list of additional notations frequently used in this paper:

- S_p is the population responding to pro smoking campaign(s);
- S_a is the number of individual responding to anti smoking campaign(s);
- L_s is the number of individual smoking less or equal to 15 cigarette per day;
- C_s denotes the number of chain smokers(smoking more than 15 cigarette per day);
- Q denotes all smokers allowed to quit permanently;
- π is the rate of potential smokers;
- p is the proportional rate of anti-smoking campaigns of respondents;
- $1 - p$ is the non-adherence to anti smoking campaigns;
- λ is the rate of potential smokers initiated to smoking with a force of infections;
- β is the rate of effective contact;
- η is the rate of relative ability of chain smoker to influence new smokers;
- $\rho 1$ is the number of light users who can quit permanently;
- γ capability of the individual smoker to progress to chain smokers;
- σ denotes the rate of chain smokers to become light smokers;
- $\rho 2$ is the rate of chain smokers to quit permanently;
- μ is the rate of chain smokers to die naturally;
- δ is the rate of chain smokers who die due to the effect of smoking;
- f is the efficacy of the anti smoking campaigns;
- $g(Sp)$ is the rate of function of potential smokers responding to smoking campaigns;
- $Sp(t)$ number of potential smokers who

respond to pro smoking adverts at time t;
- *Sa(t)* is the number of potential smokers who respond to anti smoking adverts at time t;
- *Ls(t)* is the number of light smokers at time t;
- Cs(t) is the number of chain smokers at time t;
- *Q(t)* is the number of quitters at time t;
- F_i is the spectral radius of the next generation matrix;
- *F* is the matrix at smoke equilibrium point and when $N = \frac{\pi}{\mu}$;
- V_i the dominant eigenvalue of effective reproductive number;
- *V* is the matrix of the positive of the positive eigenvalue;
- *N(t)* is the total population at time t.

The proposed mathematical model which follows the system of nonlinear differential equations:

$$\begin{aligned}
\frac{dS_p}{dt} &= (1-p)\pi - (\varepsilon + \lambda + \mu)S_p \\
\frac{dS_a}{dt} &= p\pi + \varepsilon S_p - (1-f)\lambda S_a - \mu S_a \\
\frac{dL_s}{dt} &= \lambda S_p + (1-f)\lambda S_a + \gamma C_s - (\mu + \rho_1 + \gamma)L_s \\
\frac{dC_s}{dt} &= \gamma L_s - (\gamma + \rho_2 + \delta + \mu)C_s \\
\frac{dQ}{dt} &= \rho_1 L_s + \rho_2 C_s - \mu Q
\end{aligned} \tag{1}$$

The summation of the equations in system equation (1) gives the rate at which the total population is invariant to a particular region *dN*

$$\frac{dN}{dt} = \pi - \mu N - \delta C_s \tag{2}$$

In the absence of smokers, the population size approaches π/μ at t→∞. The feasible solution for the system (1) entering region can be simplified as follows

$$\Omega = \left\{ (S_p, S_a, L_s, C_s, Q) \in \mathbb{R}_+^5 : \mathrm{N} \le \frac{\pi}{\mu} \right\} \tag{3}$$

which is positively invariant and the model now becomes epidemiologically and mathematically sound. All dependent variables and the parameters are taken to be non-negative. This mathematical model permits and sufficiently allows one to study the dynamics of the smoking model in Ω.

Model analysis

The focus of this part analyzes the equilibria and qualitative features of the model by carrying out the stability analysis of the model. This analysis permits one to determine the threshold conditions for the persistence or quitting from smoking. The smoking model is reduced to

$$\frac{dN}{dt} = \pi - \mu N - \delta C_s \tag{4}$$

after adding all the five equations of the system (1). We note that,

$$\frac{dN}{dt} = \pi - \mu N - \delta C_s \le \pi - \mu N \tag{5}$$

Applying Birkoff and Rota's theorem (Michael 2011) on differential inequality (5) by separation of variables, we have

$$\frac{dN}{\pi - \mu N} \le dt \tag{6}$$

Integrating (6) on both sides gives

$$\int \frac{dN}{\pi - \mu N} \le \int dt \Leftrightarrow -\frac{1}{\mu} ln(\pi - \mu N) \le t + k \tag{7}$$

where K is a constant of integration. We thus have, $\pi - \mu N \ge Ae^{-\mu t}$, (8)
where $A = e^k$ is a constant. Applying the initial conditions $N(0) = N_0$ in (6) we get;
$A = \pi - \mu N_0$, (9)
substituting (8) into (9) yields,

$$\pi - \mu N \ge (\pi - \mu N_0)e^{-\mu t}. \tag{10}$$

Making 'N' the subject in (10) we have

$$N \le \frac{\pi}{\mu} - \left[\frac{\pi}{\mu} - N_0 \right] e^{-\mu t} \tag{11}$$

As t→∞ in (11) the population size, $N \to \frac{\pi}{\mu}$, which implies that $0 \le N \le \frac{\pi}{\mu}$. If $N_0 < \frac{\pi}{\mu}$ then as t → ∞ the trajectories approach μ while the N_0 > μ the solution decrease to μ as t → ∞. In $\frac{\pi}{\mu}$ either case the solutions approach $N = \frac{\pi}{\mu}$ μ (??)

enters the region

$$\Omega = \left\{ (S_p, S_a L_s C_s Q) \in \mathbb{R}^5_+ : N \le \frac{\pi}{\mu} \right\}$$

as t → ∞. Thus, the feasible solution set of the system. So every solution with initial conditions to $\mathbb{R}^5_+$ remains or enters the region Ω as t → ∞.

Thus, the region Ω is positively invariant. Therefore, the smoking model is well posed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of the smoking model in Ω.

Model steady states

The steady state of the model becomes

$$\begin{aligned} (1-p)\pi - (\varepsilon + \lambda + \mu)S_p^* &= 0 \\ p\pi + \varepsilon S_p^* - (1-f)\lambda S_a^* - \mu S_a^* &= 0 \\ \lambda S_p^* + (1-f)\lambda S_a^* + \sigma C_s^* - (\mu + \rho_1 + \gamma)L_s &= 0 \\ \gamma L_s^* - (\sigma + \rho_2 + \delta + \mu)C_s^* &= 0 \\ \rho_1 L_s^* + \rho_2 C_s^* - \mu Q^* &= 0 \end{aligned} \quad (12)$$

In the absence of smoking, the smoke free equilibrium of the mathematical model (1) becomes $E_{01} = (S_p^*, S_a^*, L_s^*, C_s^*, Q^*)$.

For $E_{01} = (S_p^*, S_a^*, 0, 0, 0)$

smoke free equilibrium is;

$$E_{01} = \left(\frac{\pi(1-p)}{\varepsilon + \mu}, \frac{\pi(p\mu + \varepsilon)}{\mu(\varepsilon + \mu)}, 0, 0, 0 \right) \quad (13)$$

The reproduction number and local stability of E_0

The reproduction is a vital parameter, which determines the behaviour of the proposed model. The reproduction number can also be determined by various methods including the decompo- sition technique which gives the threshold parameter that governs the spread of disease. The reproduction obtained in this way determines the local stability of the smoking free equilibrium point with local asymptotic stability. Using the matrix theory, the model can be summarized mathematically as the spectral radius of the next generation matrix (Watmough *et al.,* 2002).

The model system (13) gives the following

$$F_i = \begin{bmatrix} \left[\frac{L_s + \eta C_s}{N} \right] \beta S_p + \left[\frac{L_s + \eta C_s}{N} \right] (1-f)\beta S_a \\ 0 \end{bmatrix} \quad (14)$$

The partial derivative of (14) with respect to infected classes (Ls,Cs) are given by;

$$DF_i = \begin{bmatrix} \frac{\beta S_p^* + (1-f)\beta S_a^*}{N} & \frac{\eta\beta S_p^* + (1-f)\eta\beta S_a^*}{N} \\ 0 & 0 \end{bmatrix}' \quad (15)$$

where, at smoke free equilibrium;

$S_p^* = \frac{(1-p)\pi}{\varepsilon + \mu}$ and $S_a^* = \frac{\pi(p\mu + \varepsilon)}{\mu(\varepsilon + \mu)}$

Thus, at smoke equilibrium point E_{01} and when $N \to \frac{\pi}{\mu}$, the matrix becomes

$$F = \begin{bmatrix} \beta\left(\frac{f(\varepsilon + \mu p)}{\varepsilon + \mu}\right) & \eta\beta\left(1 - \frac{f(\varepsilon + \mu p)}{\varepsilon + \mu}\right) \\ 0 & 0 \end{bmatrix}' \quad (16)$$

and When the steady state of the equation 10 equated to zero and substituted in F_i becomes

$$V_i = \begin{bmatrix} (\gamma + \rho_1 + \mu)L_s - \sigma C_s \\ -\gamma L_s + (\sigma + \rho_2 + \mu + \delta)C_s \end{bmatrix} \quad (17)$$

The partial derivative of equation 17 with respect to Ls and Cs yields

$$V = \begin{bmatrix} \gamma + \rho_1 + \mu & -\sigma \\ -\gamma & \sigma + \rho_2 + \mu + \delta \end{bmatrix} \quad (18)$$

The effective reproduction number, is given by: Re f f = ρ (FV −1); where ρ denotes the spectral radius or the dominant eigenvalue.

After some manipulation on (16) and (18) the positive dominant eigenvalues of the matrix FV^{-1} is given by:

$$R_{eff} = \frac{\beta(\varepsilon + \mu - f(\varepsilon + P\mu))(\sigma + \rho_2 + \mu + \eta\gamma + \delta)}{(\varepsilon + \mu)((\gamma + \rho_1 + \mu)(\sigma + \rho_2 + \mu + \delta) - \sigma\gamma)} \quad (19)$$

The threshold quantity R_{eff} is the effective reproduction number of the model system (1) for smoking effects on a population in the presence of pro-smoking and anti-smoking adverts. It measures the average number of new smokers generated by a typical smoker in a potential smokers population. In the absence of pro-

smoking and anti smoking campaigns.
($\varepsilon = f = p = 0$)

the quantity

$$R_{eff} = \frac{\beta(\sigma + \rho_2 + \mu + \delta)}{(\gamma + \rho_1 + \mu)(\gamma + \rho_2 + \mu + \delta) - \sigma\gamma)} = R_0$$

where R_0 is the basic reproduction number (i.e., R_0 represents the average number of new cases generated by a single infected individual in a completely susceptible population).

Local Stability of the Smoke Free-Equilibrium

The smoke free equilibrium of the model system (1) is given by $E_{01} = (S_p^*, S_a^*, L_s^*, C_s^*, Q^*)$ We have

$L_s^* = C_s^* = Q^* = 0$. This gives $E_{01} = (S_p^*, S_a^*, 0, 0, 0)$

Setting the system equal to zero, gives

$$\begin{aligned} 0 &= (1-p)\pi - \varepsilon S_p^* - \mu S_p^*, \\ 0 &= p\pi + \varepsilon S_p^* - \mu S_a^*, \end{aligned} \quad (20)$$

thus, $S_p^* = \frac{(1-p)\pi}{\varepsilon + \mu}$ (21)

Substituting (20) in $0 = p\pi + \varepsilon S_p^* - \mu S_a^*$, (22)

we have, $p\pi + \varepsilon(\frac{\pi(1-p)}{\varepsilon + \mu}) - \mu S_{a^*}$ (23)

Then, $S_a^* = \frac{\pi(p\mu + \varepsilon)}{\mu(\varepsilon + \mu)}$

Therefore, for $E_{01} = (S_p^*, S_a^*, 0, 0, 0)$, (24)

smoke free equilibrium is;

$$E_{01} = \left(\frac{\pi(1-p)}{\varepsilon + \mu}, \frac{\pi(p\mu + \varepsilon)}{\mu(\varepsilon + \mu)}, 0, 0, 0 \right) \quad (25)$$

$$E_{01} = (S_p^*, S_a^*, 0, 0, 0) = \left(\frac{\pi(1-p)}{\varepsilon + \mu}, \frac{\pi(p\mu + \varepsilon)}{\mu(\varepsilon + \mu)}, 0, 0, 0 \right) \quad (26)$$

Theorem 0.1: *The smoke free equilibrium of the smoking model system (1) is locally asymptotically stable if $R_{eff}<1$, and unstable if $R_{eff}>1$* (Acevedo-Estefania *et al.,* 2000).

Global Stability of the Endemic Equilibrium Point

The global stability of the endemic equilibrium is given by constructing the following Lyapunov function. This is for the special case when the population is constant, because if the population is not constant then this will not work (Nyabadza *et al.,* 2012).

Theorem 0.2: *If $R_{eff} > 1$, given that $\delta = 0$ and $\pi = \mu N$, the endemic equilibrium of the model (1) is globally asymptotically stable* (Alkhudhuri *et al.,* 2014).

Numerical results

Numerical simulations of model (1) were carried out using a set of reasonable parameter values given in Table 1. The Parameters were obtained from Acevedo Estefania *et al.,*(2000), Kalula, (2011), Zaman, (2011) and some of the parameters were estimated.

We simulate the model system by using ODE solver coded in MATLAB 8.3.0 (2014a), 64-bit executed in Windows 7 computer. However, it should be known that the parameters used are theoretical so they may (may not) be biologically realistic. Despite the values being unrealistic they are still very useful in illustrating the dynamics of the different populations.

Table 1: Parameters of the smoking model

Symbol	Value	Source
ρ_1	0.5	Acevedo Estefania 2000
ρ_2	0.25	Acevedo Estefania 2000
ε	0.001	Estimate
σ	0.0307	Kalula Asha 2011
γ	0.60	Acevedo Estefania 2000
p	0.8	Estimate
f	1	Estimate
β	2	Acevedo Estefania 2000
η	0.0002	Estimate
$л$	14	Acevedo Estefania 2000
μ	0.031	Zaman 2011
δ	0.01	Estimate

Numerical simulation and graphical illustrations are presented to show the efficacy of the proposed model. Consider a hypothetical population of 10000 individuals with initial conditions of Sp(0) = 8000, Sa(0) = 1970, Ls(0) = 20, Cs(0) = 10 and Q(0) = 0 simulated and presented graphically in Figures (1).

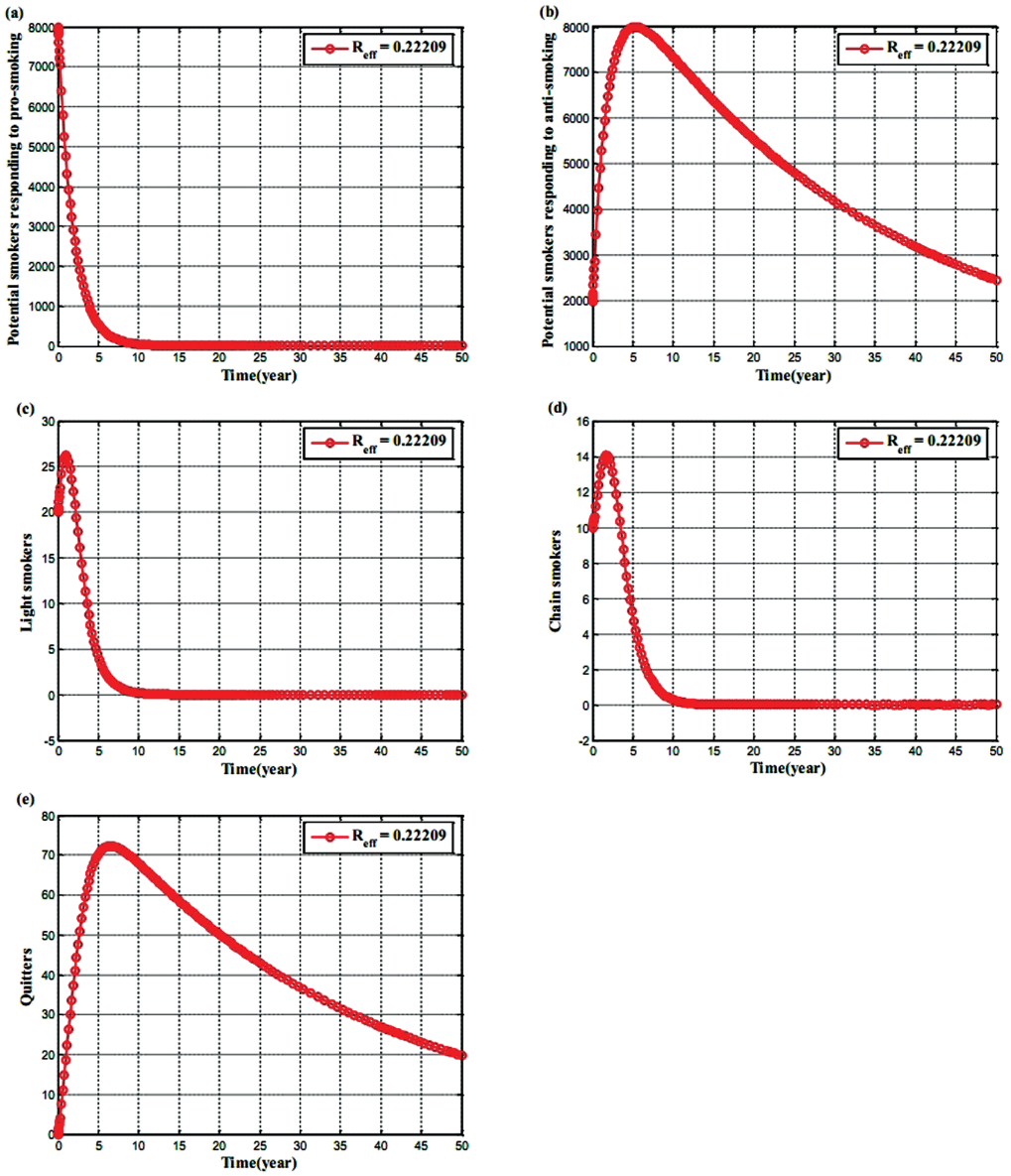


Figure 1: Variation of steady state variables on the smoking dynamics.

Effect of Anti-smoking Adverts on Smoking Dynamics

Figure 2(a)-(b) illustrates the variation of potential smokers at different rates of post expose to individuals (ε) responding to anti-smoking adverts. It shows that as the value of ε becomes large potential smokers who respond to pro-smoking adverts (S_p) decrease with time as presented in Figure 2(a) implies that large population move from this class and joins the anti-smoking adverts class (S_a). Likewise, Figure 2(b) with value denoted by epsilon shows increment of potential smokers who respond to anti-smoking adverts which alternates.

Efficacy of Anti-smoking Adverts on Smoking Dynamics

Figure 3 (a)-(d) gives the variation of steady state

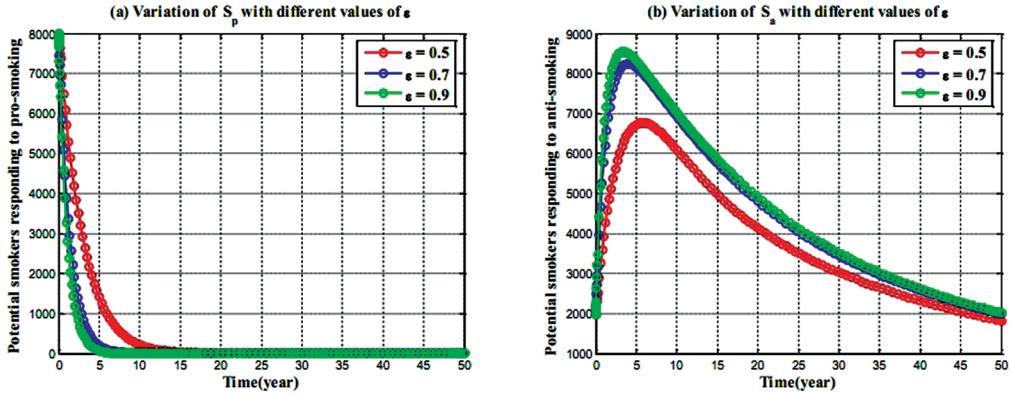


Figure 2: Illustrates the effect of the rate anti-smoking adverts (ε) with different values. The effective reproduction numbers is R_{eff} = 0.22209, 0.16133 and 0.12667

variables with different values of the effect of the efficacy of anti-smoking adverts (*f*). Figure 3(b) reveals that there is an increase of potential smokers who respond to anti-smoking adverts. These results imply that the light smokers (Ls), chain smokers (*C*s) as well as the quitters (*Q*) are expected to decrease see Figure 3(d). As presented in (b)-(d) the anti-smoking campaigns are 100% effective in preventing the initiation. As the R_{eff} becomes less than 1, the smoking habit will die out and when *f* decreases the anti-smoking campaigns are not effective at all.

The Rate of Quitting from Light smoking on the Smoking Dynamics

Figures 4 (a)-(b) illustrates the variation of steady state variables with different values of the rate of quitting from light smoking (ρ_1). If individuals from the light-smoking class quit at a higher rate then chain smokers decrease, that means we will be left with a smaller population of smokers in general. If we make $R_{eff} > 1$, but close to 1 in the simulations, then the smokers (Ls and Cs) will have a small population, but still have a very large portion of the total population,

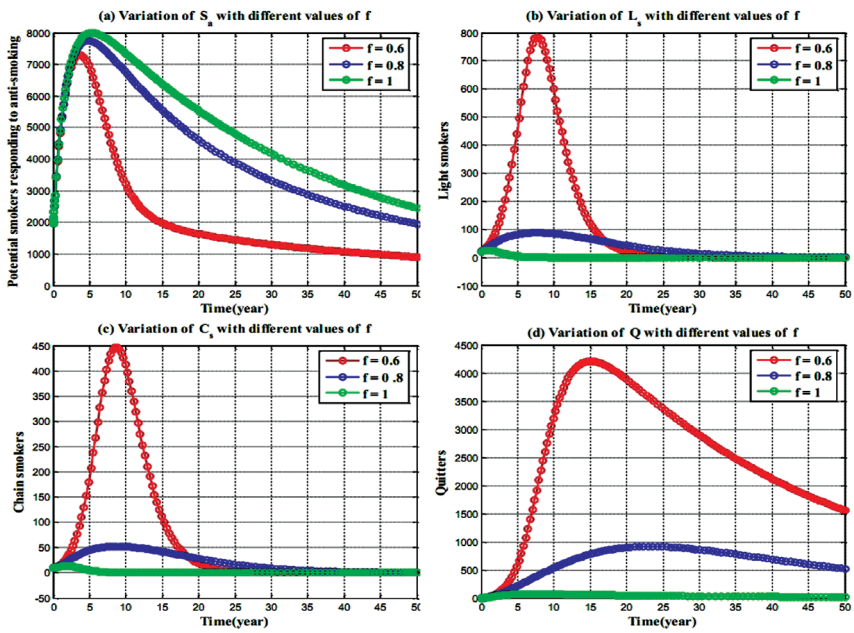
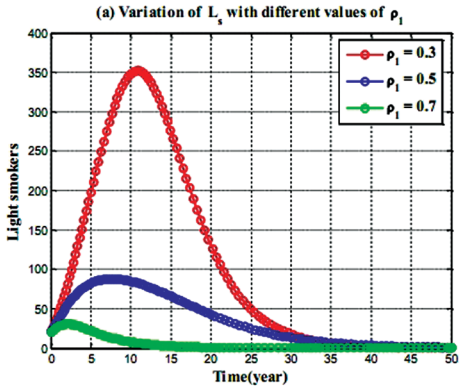


Figure 3: The efficacy of anti-smoking adverts (*f*) with R_{eff} = 0.92139; 0.52403 and 0.12667

Meaning that individuals are still susceptible to smoking in future.



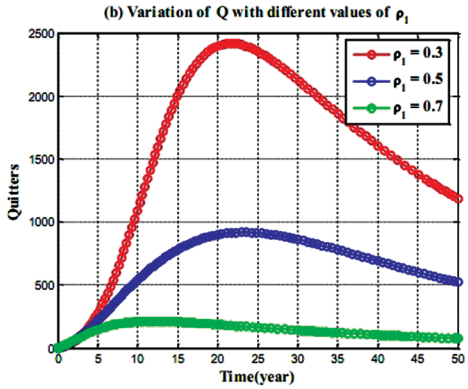


Figure 4: Rate of quitting from light smoking (ρ_1), with R_{eff}= 0.28160; 0.22209 and 0.18335

Effect of Transmission Rate on Reproduction Numbers

Figure 5 shows the relationship between the reproduction numbers as β changes. It is observed that when $R_0 > 1, \beta$ keeps on increasing which implies that when the expected number of secondary cases produced increases, β also increases; for this case β has a direct impact on the dynamics of the smoking habit where without adverts, smoking habit increases. After introducing adverts, it is observed that β keeps on reducing when $R_{eff} < 1$.

Therefore, we can conclude that when adverts are introduced in the community they have a great effect on the smoking habit and the smoking habit can be reduced by 80%.

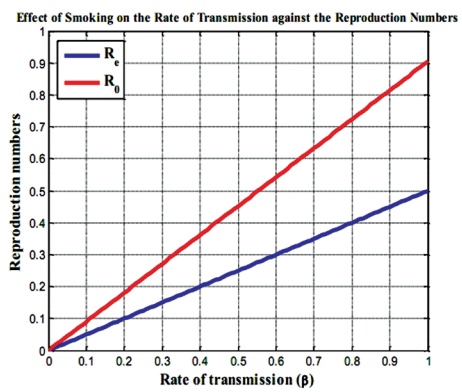


Figure 5: The relationship between reproduction numbers as β changes

Effect of Post-exposure Rate and the Efficacy Rate on the Reproduction Number

Figure 6 shows the relationship between the post exposed individuals (ε) in relation to the reproduction number as well as the relationship of the efficacy of anti-smoking adverts (f) in relation to the reproduction number. We can observe from the figure that as the value of the efficacy of anti-smoking adverts increase the reproduction number decreases which means the anti smoking campaigns are effective.

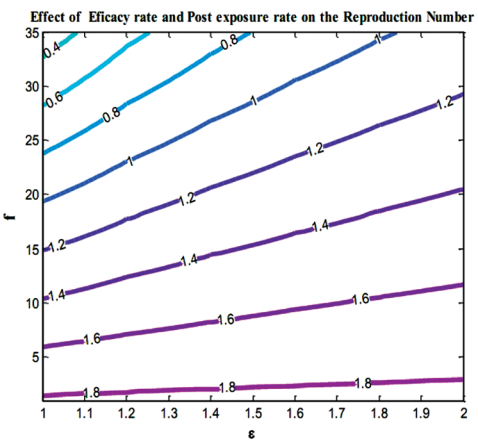


Figure 6: Shows contour plots of the effects of post-exposed individuals and efficacy of anti-smoking adverts on the reproduction number

Effect of Pre-exposure Rate and the Post-exposure Rate on the Reproduction Number

Figure 7, shows the relationship between the pre-exposed individuals (p) in relation to the reproduction number as well as the relationship of the post exposed individuals (ε) in relation to the reproduction number. It is observed from the Figure 7 that as the value of p increases the reproduction number increases and when the value of ε increases the reproduction number decreases.

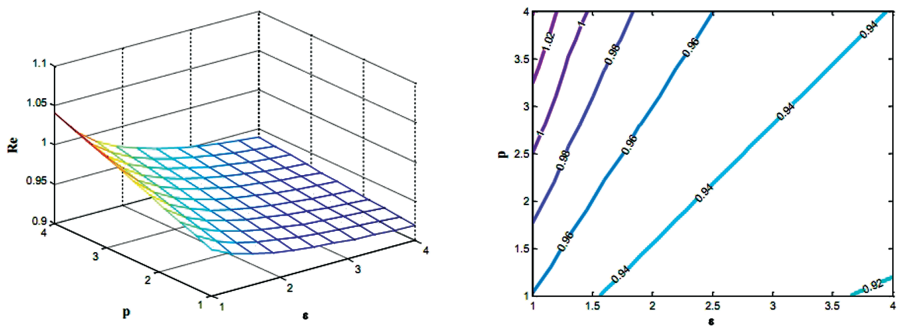


Figure 7: Illustrates the effect of pre-exposed and the post-exposure rates on the reproduction number

Conclusion

In this paper, a mathematical model for the dynamics of smoking is presented and analysed. The model is well posed and exists in a feasible region where smoke free and endemic equilibrium points are obtained and their stability are investigated. The analysis shows the existence of endemic equilibrium which is found to be locally asymptotically stable and globally asymptotically stable under certain conditions due to forward bifurcation at threshold parameter equal to unit. The model incorporates two important aspects, pro-smoking and anti-smoking adverts. The model analysis shows that adverts have significant effect on the spread of smoking habit. The study shows that the higher values of the efficacy of anti-smoking adverts rate (f) the lower the number of smokers and vice-versa. Finally, to keep the smoking habit under control, anti-smoking adverts rate (f) should be sufficiently large and increased.

Recommendations

Smoking eradication is still a challenge world wide, so there is a need to strengthen the control strategies, especially looking for effective strategies which are not yet available. The rate of smoking habit seems to be increasing. Thus from the results of this work it is recommended that, it would be more important to make sure that there are as many adverts as possible to enhance campaign programs as well as the awareness of individuals on the effects of smoking. Also each advert has its own significant impact on the reduction of the smoking habit, if each will be advertised at the targeted scale in many endemic areas in the community, then new generation of smoke free goal can be achieved. Lastly, since there is positive impact of adverts on smoking dynamics, then more education programs should be established to make the community aware on the effects of smoking at large.

As smoking continues to endanger more lives, it is important to have comprehensive researches done in order to explore possible new control strategies of the smoking habit as well as assessing the impact of the existing control strategies. Based on this study it is proposed that future work may consider the cost effectiveness analysis of the control strategies of smoking

models and investigate the behaviour of those who quit from smoking after being recovered.

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