Transactions of the Nigerian Association of Mathematical Physics
Volume 4, (July, 2017), pp135 – 142

Effect of Environment On The Dynamics of Tobacco Smoking: A Mathematical Modelling Approach

Yayock J.F.[1], Mayaki Z.I.[1], Anka G.H.[2] and Yusuf I.[3]

[1]Department of Mathematics, Usmanu Danfodiyo University Sokoto, Nigeria.
[2]Department of Mathematics, Gov't. Girls College of Arts, Sci., & Islamic Studies Sec. Sch. Sokoto.
[3]Department of Computer Science, Niger State College of Education Minna, Nigeria.

Abstract

A deterministic model for assessing the effect of the nature of environment and urbanization on the dynamics and control of tobacco smoking was developed and analyzed. The effective reproduction number (R_S) ***which is a threshold for the spread of tobacco smoking was obtained. Numerical simulations revealed that once the deadly behavior is re-introduced into the population, the morbidity and mortality of the disease continue to rise unless rural-urban migration is checked and people are discouraged from promoting the sale and consumption of tobacco.***

Keywords: Disease-free equilibrium, effective reproduction number, urbanization, tobacco smoking

1.0 INTRODUCTION

"The true face of smoking is disease, death and horror – not the glamour and sophistication the pushers in the tobacco industry try to portray" – David Byrne

Tobacco smoking involves burning of tobacco leaves which results to smoke breathed in to be tasted and absorbed into the blood stream [1]. It is the practice of burning tobacco and inhaling the smoke (consisting of particle and gaseous phases). Most commonly the substance is the dried leaves of the tobacco plant which have been rolled to create a small, round cylinder called a "cigarette". According to World No Tobacco Day [2], Tobacco smoking is the most popular form, being practiced by over one billion people globally, of whom the majority are in the developing world.

Tobacco is one of the most used additive substances that contain several chemicals which are injurious to health. It is often used in different forms such as chewing, snuffing and smoking. The fact remains however that, tobacco use is harmful because it is known to contain over 4,000 hazardous chemical substances, 50 of which are carcinogenic [3]. The most dangerous chemicals in tobacco are nicotine and carbon monoxide. Nicotine, in its concentrated form, is one of the most powerful poisons known to mankind. One drop of nicotine solution, if directly injected into the human body, may lead to death within minutes. Nicotine does not kill a person as he smokes because it is absorbed gradually by the body following slight biochemical modifications. It has no medicinal property whatsoever and is supposed to be marketed as an insecticide. It has high addictive tendency as cocaine, heroin and morphine [4]. Carbon monoxide is the same as the gas that emanates from car's exhaust. It easily interacts with the hemoglobin in red blood cells and impairs its oxygen carrying capacity thereby causing difficulties in breathing. Carbon monoxide, combined with nicotine, predisposes the user to coronary thrombosis and stroke, commonly known as cardiovascular accidents.

The tobacco epidemic is one of the biggest public health threats the world has ever faced, killing more than 7 million people a year. More than 6 million of those deaths are the result of direct tobacco use while around 890 000 are the result of non-smokers being exposed to second-hand smoke. Nearly 80% of the more than 1 billion smokers worldwide live in low- and middle-income countries, where the burden of tobacco-related illness and death is heaviest. Tobacco users who die prematurely deprive their families of income, raise the cost of health care and hinder economic development. In some countries, children from poor households are frequently employed in tobacco farming to provide family income. These children are especially vulnerable to "green tobacco sickness", which is caused by the nicotine that is absorbed through the skin from the handling of wet tobacco leaves [5].

Correspondence Author: Anka G.H., Email: gazalianka@gmail.com, Tel: +2348060266665

Tobacco use is the single greatest cause of preventable death globally [6]. As many as half of people who use tobacco die from the results of this use. Tobacco is the only legal drug that kills many of its users when used exactly as intended by the manufacturers. WHO has estimated that tobacco use (smokers and smokeless) is currently responsible for the death of about six million people across the world each year with many of these occurring prematurely. This total includes 600,000 people who are also estimated to die from the effects of second-hand smoke.
The past years have witnessed several studies on the dynamics of smoking. Several authors [7…13], had worked on various mathematical models to describe the dynamics of tobacco use, effects, spread and control among others. The works of these authors is complemented by incorporating smokers that are not aware of the health impediments associated with tobacco smoking and smokers that are aware of the health impediments associated with tobacco smoking compartments and the effect of the nature of environment on the smoking dynamics which are very essential in the prevalence dynamics and control of smoking.

2.0 Materials and Methods

2.1 Model Formulation

The model contains seven (7) compartments, namely: The at-risk rural potential smokers P_R representing people who are likely to become infected with smoking; the at-risk urban potential smokers (P_U), representing people who also are likely to become infected with smoking, part-time smokers (S_P), the unaware chain smokers (C), representing people that are not aware of the health impediments associated with smoking; the aware smokers (C_A), representing people that are aware of the health impediments associated with smoking; temporary quitters (Q_T), and permanent quitters (Q_P). The following assumptions are considered when constructing the model:

(1) Recruited individuals are assumed to be through birth and rural-urban transfer of potential smokers
(2) The population is homogenous and depends on time, t
(3) Urban-rural transfer is assumed to be very rare, hence, is not considered
(4) Part-time smokers neither quit temporarily nor permanently
(5) Temporary quitters are considered non-infectious

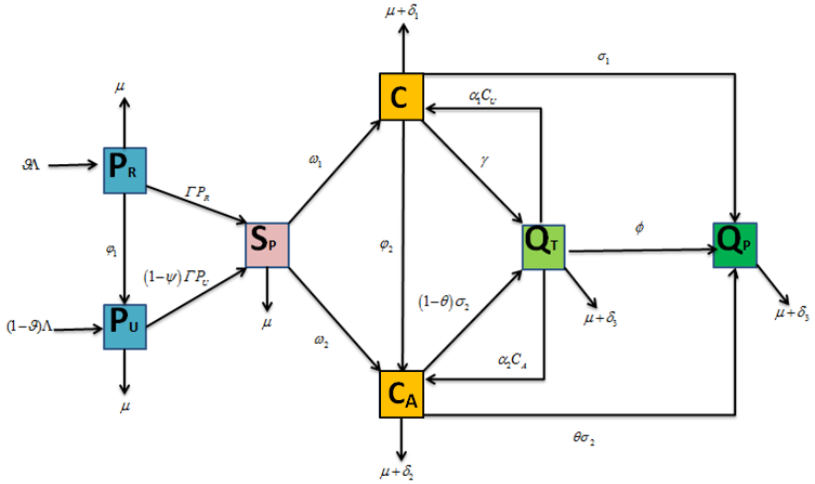

Figure 1: Schematic Diagram of Tobacco Smoking Dynamics Model

$$\frac{dP_R}{dt} = \vartheta\Lambda - \beta\left(S_P + \eta_1 C + \eta_2 C_A\right)P_R - (\varphi_1 + \mu)P_R \qquad (1)$$

The P_R population is generated from daily recruitment of uninfected individuals through births given by Λ while ϑ is the proportion of the recruited individuals from rural area and is reduced by the effective social contact rate Γ between P_R, S_P, C and C_A. It is also reduced by the number of people who moves from P_R to P_U due to migration at the rate φ_1 and those that die naturally at the rate μ.

$$\frac{dP_U}{dt} = (1-\vartheta)\Lambda + \varphi_1 P_R - \beta\left(S_P + \eta_1 C + \eta_2 C_A\right)(1-\Psi)P_U - \mu P_U \qquad (2)$$

The P_U population is generated from daily recruitment of uninfected individuals through births given by Λ while $(1-\vartheta)$ is the proportion of the recruited individuals from urban area and is reduced by the effective social contact rate Γ while $(1-\Psi)$ is the effectiveness of urbanization of P_U. It is reduced by the number of those that die naturally at the rate μ.

$$\frac{dS_P}{dt} = \beta\left(S_P + \eta_1 C + \eta_2 C_A\right)\left(P_R + (1-\Psi)P_U\right) - (\omega_1 + \omega_2 + \mu)\mathrm{S}_P \quad (3)$$

Individuals in class S_P may progress to class C or C_A at rate ω_1 and ω_2 respectively, and some will die naturally.

$$\frac{dC}{dt} = \omega_1 \mathrm{S}_P + \alpha_1 \mathrm{C}\mathrm{Q}_T - (\gamma + \sigma_1 + \varphi_2 + \delta_1 + \mu)\mathrm{C} \quad (4)$$

Individuals in class C may then be aware of the health impediments of smoking and progress to class C_A at the rate φ_2 or decide to quit temporarily or permanently at the rates γ and σ_1 respectively.

$$\frac{dC_A}{dt} = \omega_2 \mathrm{S}_P + \varphi_2 \mathrm{C} + \alpha_2 \mathrm{C}_A\mathrm{Q}_T - (\sigma_2 + \delta_2 + \mu)\mathrm{C}_A \quad (5)$$

Individuals in class C_A may decide to quit smoking temporarily and progress to class Q_T or permanently and progress class Q_P at the rates $(1-\theta)\sigma_2$ and $\theta\sigma_2$ respectively, where θ is the proportion of individuals that are quitting permanently.

$$\frac{dQ_T}{dt} = \gamma C + (1-\theta)\sigma_2 C_A - (\alpha_1 C + \alpha_2 C_A)\mathrm{Q}_T - \left(\phi + \delta_3 + \mu\right)Q_T \quad (6)$$

Individuals in class Q_T may decide to quit smoking permanently at the rate ϕ and progress to class Q_P or relapse to C or C_A via peer pressure rates α_1 and α_2 respectively.

$$\frac{dQ_P}{dt} = \sigma_1 \mathrm{C} + \theta\sigma_2 \mathrm{C}_A + \phi Q_T - \left(\delta_3 + \mu\right)Q_P \quad (7)$$

Individuals in C, C_A *or* Q_T quit smoking permanently at the rates σ_1, σ_2 *and* ϕ respectively and move to class Q_P. Natural death occurs in all classes at rate μ. Smoking induced death occurs in class C and C_A at the rates δ_1 and δ_2 respectively and δ_3 for both Q_T and Q_P.

The model equations (1) – (7) are in the biological-feasible region:

$$\Omega = \left\{P_R, P_U, S_P, C, C_A, Q_T, Q_P \in \Re_+^7\right\} \quad (8)$$

Let

$$\left.\begin{aligned} \varepsilon_1 &= 1-\theta \\ \varepsilon_2 &= 1-\Psi \\ \varepsilon_3 &= 1-\vartheta \\ k_1 &= \varphi_1 + \mu \\ k_2 &= \omega_1 + \omega_2 + \mu \\ k_3 &= \gamma + \delta_1 + \varphi_2 + \delta_3 + \mu \\ k_4 &= \sigma_2 + \delta_2 + \mu \\ k_5 &= \phi + \delta_3 + \mu \end{aligned}\right\} \quad (9)$$

So that (1) – (7) becomes

$$\frac{dP_R}{dt} = \vartheta\Lambda - \beta\left(S_P + \eta_1 C + \eta_2 C_A\right)P_R - k_1 P_R \quad (10)$$

$$\frac{dP_U}{dt} = \varepsilon_3\Lambda + \varphi_1 P_R - \beta\left(S_P + \eta_1 C + \eta_2 C_A\right)\varepsilon_2 P_U - \mu P_U \quad (11)$$

$$\frac{dS_P}{dt} = \beta\left(S_P + \eta_1 C + \eta_2 C_A\right)\left(P_R + \varepsilon_2 P_U\right) - k_2 \mathrm{S}_P \tag{12}$$

$$\frac{dC}{dt} = \omega_1 \mathrm{S}_P + \alpha_1 \mathrm{C}\mathrm{Q}_T - k_3 \mathrm{C} \tag{13}$$

$$\frac{dC_A}{dt} = \omega_2 \mathrm{S}_P + \varphi_2 \mathrm{C} + \alpha_2 \mathrm{C}_A \mathrm{Q}_T - k_4 \mathrm{C}_A \tag{14}$$

$$\frac{dQ_T}{dt} = \gamma C + \varepsilon_1 \sigma_2 C_A - (\alpha_1 C + \alpha_2 C_A)\mathrm{Q}_T - k_5 Q_T \tag{15}$$

$$\frac{dQ_P}{dt} = \sigma_1 \mathrm{C} + \theta\sigma_2 \mathrm{C}_A + \phi Q_T - k_5 Q_P \tag{16}$$

2.2 Effective Reproduction Number, $\left(R_S\right)$

It is utmost important in mathematical epidemiology to find the threshold for the spread of an infection in a population as it tells whether or not if the disease has ability to invade a population [14]. Effective reproduction number is the number of new tobacco smoking cases recorded when a single tobacco smoker is introduced into a population where everyone is a potential smoker in the presence of antismoking campaigns. The next generation operator technique described by Diekmann and Heesterbek [15] was used to obtain the effective reproduction number $\left(R_S\right)$ of the model. It is expressed as the spectral radius of the next generation matrix $K = FV^{-1}$. The matrices F and V are obtained from the infected classes $\left(S_p, C_U, C_A\right)$ at smoking-free equilibrium $\left(E^0\right)$.

$$\begin{pmatrix} P_R^{\,0} \\ P_U^{\,0} \\ S_P^{\,0} \\ C^0 \\ C_A^{\,0} \\ Q_P^{\,0} \\ Q_T^{\,0} \end{pmatrix} = \begin{pmatrix} \dfrac{\vartheta\Lambda}{k_1} \\ \dfrac{\Lambda\left(\varepsilon_3 k_1 + \vartheta\varphi_1\right)}{\mu k_1} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{17}$$

and thus we have

$$F = \begin{pmatrix} \beta\left(P_R^{\,0} + \varepsilon_2 P_U^{\,0}\right) & \beta\eta_1\left(P_R^{\,0} + \varepsilon_2 P_U^{\,0}\right) & \beta\eta_2\left(P_R^{\,0} + \varepsilon_2 P_U^{\,0}\right) \\ 0 & \alpha_1 Q_T^{\,0} & 0 \\ 0 & 0 & \alpha_2 Q_T^{\,0} \end{pmatrix} \tag{18}$$

And

$$V^{-1} = \begin{pmatrix} \dfrac{1}{k_2} & 0 & 0 \\ \dfrac{\omega_1}{k_2 k_3} & \dfrac{1}{k_3} & 0 \\ \dfrac{\omega_1\varphi_2 + k_3\omega_2}{k_2 k_3 k_4} & \dfrac{\varphi_2}{k_2 k_4} & \dfrac{1}{k_4} \end{pmatrix} \tag{19}$$

The effective reproduction number is given as:

$$R_S = \frac{\beta\left(P_R{}^0 + \varepsilon_2 P_U{}^0\right)\left[k_3k_4 + k_4\eta_1\omega_1 + \eta_2\left(\omega_1\varphi_2 + k_3\omega_2\right)\right]}{k_2k_3k_4} \tag{20}$$

3.0 Results and Discussion

Table 1: Value of Parameters for the Model

Parameter	Value	Reference
Λ	14	Acevedo Estefania [16]
μ	0.0031	Estimated
β	0.04	Alkhudhari *et al*. [12]
η_1	0.0002	Muhaya [13]
α_1	0.03	Estimated
α_2	0.01	Estimated
θ	0.75	Assumed
η_2	0.0001	Muhaya [13]
φ_1	0.33	Assumed
φ_2	0.33	Assumed
σ_1	0.25	Estimated
σ_2	0.45	Estimated
γ	0.25	Estimated
ω_1	0.0307	Kalula [17]
ω_2	0.0107	Kalula [17]
Ψ, ϕ, ϑ	$(0,1)$	Varied
δ_1	0.01	Muhaya [13]
δ_2	0.001	Estimated
δ_3	0.0001	Estimated

3.1 Effects of Rural-urban migration, Effectiveness of urbanization and Quitting Smoking permanently on the dynamics and Control of Smoking

The following figures revealed different effects of rural-urban migration, effectiveness of urbanization and quitting smoking permanently on the dynamics and control of smoking. Since the rate of control parameters can vary between 0 and 1, we have three cases of control i.e low (25%), moderate (50%) and high (75%) except otherwise where specified.

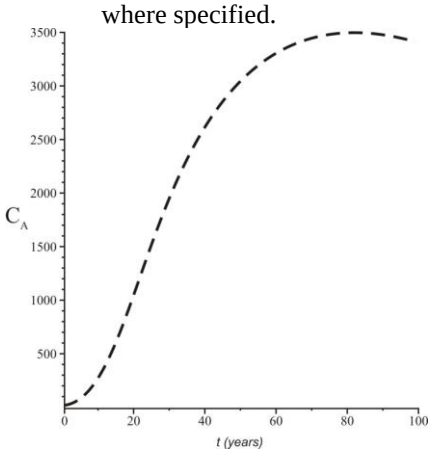

Figure 2: Effect of zero control measures on the number of mortality due to smoking. Parameters used are as in Table 1 with $\vartheta = \psi = \phi = 0$ which gives $R_S = 5346.1$. Figure 2 reveals that if no control measures are put in place by the government of a country, the mortality due to smoking will continue to rise above 3500 in the first 100 years with zero initial population of chain smokers. Smoking remains endemic even though mortality reduces due to lack of more potential smokers to infect.

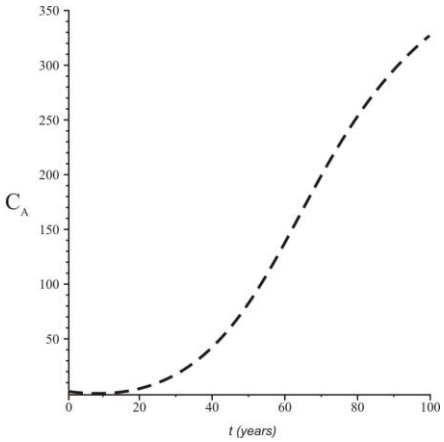

Figure 3: Effect of low (25%) control measures on the number of mortality due to smoking. Parameters used are as in Table 1 with $\vartheta = \psi = \phi = 0.25$ which gives $R_S = 11.47$

In similar way, we observe from Figure 3 that a low control measures does not bring about the control of smoking. Although low control measures are better than no control at all. Even though the mortality decreased within the first 10 years, the death toll exceeds 300 in the first 100 years without any sign of decreasing. This is not a good measure to control smoking in a population.

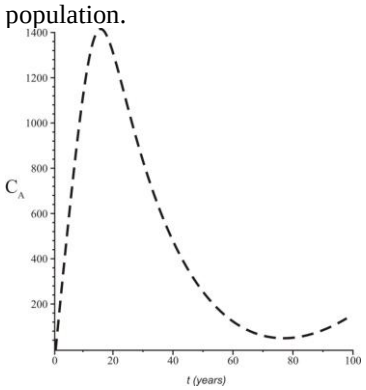

Figure 4: Effect of moderate (50%) control measures on the number of mortality due to smoking. Parameters used are as in Table 1 with $\vartheta = \psi = \phi = 0.5$ which gives $R_S = 2.15$

Figure 4 shows that a moderate control measures does not also bring about the control of smoking. Although moderate control is better than low control measures because even though the mortality increased within the first 20 years, the death toll continues to fall even below 100 in the first 80 years after which it continues to show sign of increase. This is also not a good measure to control smoking in a population.

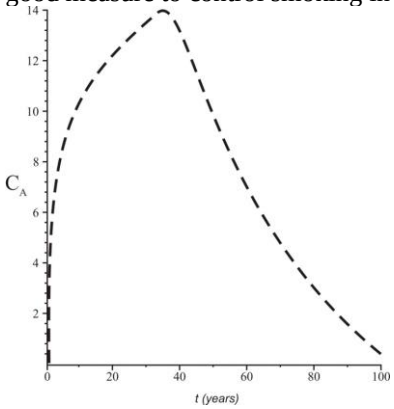

Figure 5: Effect of high (75%) control measures on the number of mortality due to smoking. Parameters used are as in Table 1 with $\vartheta = \psi = \phi = 0.75$ which gives $R_S = 0.21$

Figure 5 shows how deadly smoking is. This is because total eradication of smokers is not obtainable even with a 75% control measures put in place. Although the mortality is reducing, individuals still suffers death even after 100 years. This calls for a 100% universal control measures for tobacco smoking to be totally and immediately eradicated from a population.

4. CONCLUSION

We developed and analysed a new mathematical model for the dynamics and control of tobacco smoking in a homogenous population. The effective reproduction number (R_S) was obtained. Our results showed that rural-urban migration, urbanisation and the rate at which smokers quit tobacco smoking permanently have great impact on the dynamics and control of tobacco smoking. It is therefore recommended that

(1) the law makers through law enforcement agencies should come up with rules and regulations guiding smoking and possible sanctions in the case of people who will go against the laws,
(2) the government should impose high tax rate on the sellers and promoters of tobacco, and
(3) curriculum planners should incorporate the hazards of tobacco smoking so as to discourage student from tobacco smoking at tender ages

References

[1] Cerami C, Founds H, Nicholl I, Mitsuhashi T, Giordano D, Vanpatten S, Lee A, Al-Abed Y, Vlassara H, Bucala R, Cerami A (1997). "Tobacco smoke is a source of toxic reactive glycation products". *Proceedings of the National Academy of Sciences of the United States of America*. 94 (25): 13915–20.

[2] World No Tobacco Day: Matters arising. News Agency of Nigeria (NAN), May 28,2015.

[3] Abubakar, U. (2007a). Tobacco: Why the smoke Billows Higher. Daily Trust, Abuja, Nigeria 22-29.

[4] Hu, T.W; Mao, Z. *et al*. (2006). "China at the Crossroads: The Economics of Tobacco and Health". *Tobacco Control*. 15: i37–i41

[5] World Health Organisation (2017). Tobacco Fact sheet. Retrieved on July 1, 2017 from http://www.who.int/mediacentre/factsheets/fs339/en/

[6] World Health Organization (2008). *WHO Report on the Global Tobacco Epidemics 2008: The MPOWER Package* (PDF). Geneva: World Health Organization. P.8.

[7] Darby SC, Pike MC (1988). Lung cancer and passive smoking, predicted effects from a mathematical model for cigarette smoking and lung cancer. The Macmillan press Ltd.

[8] Castillo-Garsow C., Jordan- Salivia G, and Rodriguez Herrera A (1997). Mathematical models for the dynamics of tobacco use, recovery and relapse. Technical Report Series BU-1505-M.

[9] Sharomi, O., Gumel A.B. (2008). Curtailing smoking dynamics: A mathematical modeling approach. Applied mathematics and computations 195: 475-499.

[10] Lahrouz *et al.* (2011). Deterministic and stochastic stability of a mathematical model of smoking, statistics and probability letters 81. 1276-1284.

[11] Erturk, V.S., Zaman, G., Momani, S. (2012). A numeric- analytic method for approximating a giving up smoking model containing fractional derivatives, Computers and Mathematics with Applications. 64, 3065-3074

[12] Alkhudhari, Z., Al-Sheikh, S., Al-Tuwairqi, S. (2014). Global Dynamics of a Mathematical Model on Smoking. *ISRN Applied Mathematics*. Vol., Article ID 847075

[16] Muhaya K. (2013). Mathematical modelling on the effects of Passive smoking in the presence of pro and Anti-smoking campaigns. MSc. Dissertation submitted to the department of Mathematics, University of Dar es Salaam.

[14] Yusuf, I., Abdulrahman, S., Musa, B. And Adamu, G. (2016). Controlling the Spread of Corruption through Social Media: A Mathematical Modelling Approach. *IOSR Journal of Mathematics*. 12(6): 75 – 81.

[15] Diekmann, O., & Heesterbeek, J. A. P. (2000). *Mathematical epidemiology of infectious diseases: Model building, analysis and integration*. New York: Wiley.

[16] Acevedo-Estefania, C., Gonzalez, C., Soto, K.R., Eric, D., Summerville, E.D., Song, B., Castillo-Chavez, C. (2000) A mathematical model for lung cancer, the effects of secondhandsmoke and education. BU-1525-M: 521-548.

[17] Kalula, S.A. (2011). Modelling the dynamics of methamphetamine abuse in the Western Cape. Lee.