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Analysis of a Mathematical Model of Smoking

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Abstract

This study presents a mathematical model that represents the population growth dynamics of tobacco consumers based on a system of ordinary nonlinear differential equations. The model is used to determine the Basic Reproductive Number (R_0). Two points of equilibrium are found and their local stability is classified. Finally, the Matlab software is used to present numerical simulations using the fourth-order Runge-Kutta method, and it is shown that the solutions approach an asymptotically stable point, under the variation of R_0.

Key words: Smoking, Basic Reproductive Number, Dynamical Systems

1 Introduction

In Colombia and around the world, one of the public health problems that has been recognised in recent years is smoking addict, which has developed into

an epidemic causing many deaths [8]. Environmental pressure, curiosity and stress are factors that influence the development of a smoking habit [2]. The emergence of the habit occurs in stages such as: preparation, trying tobacco for the first time, experimentation and beginning repeated but irregular use, habitual use of tobacco and finally the development of dependence and addiction [6].

Most smokers who know the dangers of tobacco wish to quit and, in theory, after taking up smoking it is possible to stop voluntarily and permanently [6, 1], but counseling and medication can double the likelihood that a smoker who wants to quit will do so. The process of giving up smoking occurs in five phases: pre-contemplation, contemplation, preparation, action and maintenance [6].

Mathematical models are used to interpret the increase in smoking and anticipate the impact of smokers on society [1]. This is how some studies related to the subject have appeared in the literature. The principal characteristics shared by these studies are that a constant population is divided into three groups: potential smokers; smokers; and ex-smokers. The studies consider neither death by illnesses caused by tobacco, nor the possiblilty that a person completely stops being exposed to the comsumption of tobacco. Amongst these, in 1997, C. Castillo-Garsow, G. Jordan-Salivia and A. Rodriguez-Herrera [3] consider a basic model of three ordinary nonlinear differential equations, which consider the use of drugs in general in adolescents and where the factors involved are contagion, recovery and relapse. In [7, 4] a model of four nonlinear differential equations is constructed where groups of people are: potential smokers; smokers; and temporary or permanent ex-smokers. While in [1] a model of five nonlinear differential equations is formulated, distinguishing between to classes of smokers according the the frequency of consumption, high or low. Similar to [1], Sintayehu Matintu [9] studies a non-constant population but holding the other characteristics constant that make a great difference to the model when varied as proposed in this article.

2 Approach of the Model

For this population behavior model of people in the presence of tobacco consumption the following assumptions are taken into account:

- There are deaths as a result of smoking.
- The average number of healthy and completely recovered people are not directly considered in the system.

- People can be in one of two main states: Exposed (people who are not able to pass on the habit, but are at risk of becoming active smokers); and Infected (people who are addicted to tobacco and can pass on the habit). The first state is divided into two sub-states: passive smokers or those at risk of smoking, E_1; and people who have stopped smoking but are at risk of relapsing, E_2.
- There is a constant flow, Δ, of healthy people to the state E_1.
- People leave from the states under study due to factors such as: living in a completely non-smoking population; or the death of the individual.

Table 1 describes the variables and parameters used create the mathematical model that represents the dynamics of transmission of the habit of smoking. The flow diagram in Figure 1 shows the state transitions possible under the model.

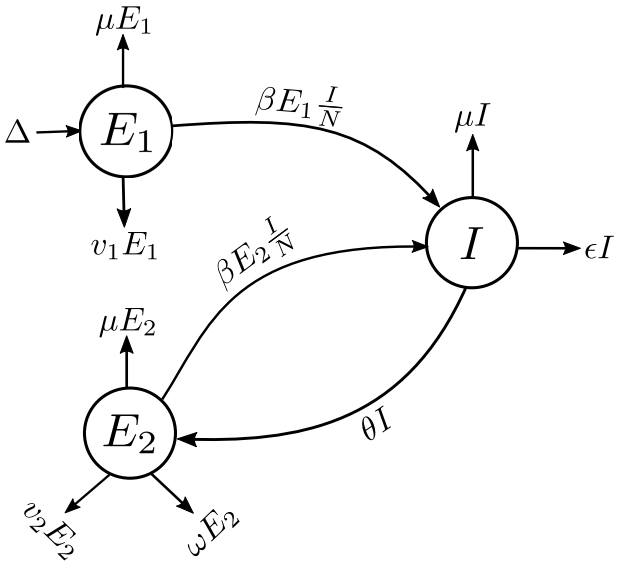

Figure 1: State transmission diagram for the model of smoking habits.

Variable/ parameter	**Description**
E_1	Average number of people over a time t that are at risk of becoming active smokers.
E_2	Average number of people over a time t who have been active smokers and quit, but are at risk of relapsing.
I	Average number of active smokers over a time t.
N	The total size of the population, calculated as $N = E_1+E_2+I$, over each unit of time t.
Δ	Average number of healthy people who become people at risk of becoming active smokers.
μ	The natural death rate over the population.
v_1	The exit rate of people from being at risk of becoming smokers to the healthy population.
v_2	The exit rate of ex-smokers to the healthy population.
ω	Death rate of ex-smokers as a consequence of tobacco consumption.
β	The infection rate of smoking.
ϵ	Death rate of active smokers as a consequence of tobacco consumption.
θ	The exit rate of people from being active smokers to being at risk of relapsing.

Table 1: Variables and parameters of the model

In order to describe the mathematical model by means of a system of differential equations, the following dynamics are taken into account:

- There is a constant inflow of people at risk of becoming an active smoker E_1 given by Δ.
- The number E_1 of people decreases due to: natural death (μE_1 people per unit time); the influence of factors that cause a person to move from the population at risk of being an active smoker to the population of healthy people ($v_1 E_1$ people per unit time); and the impact of smoking ($\beta E_1 \frac{I}{N}$ people per unit time), where $\frac{I}{N}$ is the probablility of meeting an active smoker in the total population.
- The number of active smokers I decreases due to: natural death (μI people per unit time); death as a consequence of smoking (ϵI people per unit time); and the influence of factore that cause a person to leave the population active smokers and join the population of people at risk of relapsing E_2 (θI people per unit time).

- The number of active smokers I increases due to the impact of smoking ($\beta E_2 \frac{I}{N}$ people per unit time), who come from state E_2; and $\beta E_1 \frac{I}{N}$ people per unit time), who come from state E_1.
- The number of people E_2 decreases due to: natural death (μE_2 people per unit time); death as a consequence of having been an active smoker (ωE_2 people per unit time), and the influence of factore that make people move from the population of ex-smokers at risk of relapse to the population of healthy people ($v_2 E_2$ people per unit time).

Thus the mathematical model is described by the following system of ordinary non-linear differential equations:

$$\begin{aligned} E_1'(t) &= \Delta - \beta E_1 \frac{I}{N} - (\mu + v_1) E_1 \\ I'(t) &= \beta \frac{I}{N}(E_1 + E_2) - (\mu + \epsilon + \theta) I \\ E_2'(t) &= \theta I - \beta E_2 \frac{I}{N} - (\mu + v_2 + \omega) E_2. \end{aligned} \tag{1}$$

Where

$\Delta > 0$, $0 < \beta \leq 1$, $0 < \mu < 1$, $0 \leq v_1 \leq 1$, $0 \leq \theta \leq 1$, $0 \leq v_2 \leq 1$, $0 \leq \omega \leq 1$, $0 \leq \epsilon \leq 1$, $v_2 \leq v_1$ and $\omega < \epsilon$.

Defined in the set of biological interest:

$$\Omega = \{(E_1, I, E_2) \in \mathbb{R}^3 : \ E_1, I, E_2 \ \geq \ 0 \ \wedge \ E_1 + I + E_2 \neq 0\}.$$

3 Stability Analysis

In the following section, the points of equilibrium of system (1) are found in terms of the Basic Reproductive Number and an analysis of local stability is carried out at these points.

3.1 Points of Equilibrium

To find the points of equilibrium of system (1) we solve the algebraic system:

$$\Delta - \beta E_1 \frac{I}{N} - (\mu + v_1)E_1 = 0 \tag{2}$$

$$\beta \frac{I}{N}(E_1 + E_2) - (\mu + \epsilon + \theta)I = 0 \tag{3}$$

$$\theta I - \beta E_2 \frac{I}{N} - (\mu + v_2 + \omega)E_2 = 0. \tag{4}$$

- If $I = 0$, then replacing I in (4) we have $E_2 = 0$.

Replacing I in (2) we have $E_1 = \frac{\Delta}{\mu + v_1}$. And so we obtain the point of equilibrium $P_1 = \left(\frac{\Delta}{\mu + v_1}, 0, 0\right)$.

- If $I \neq 0$, from (3) we have

$$\frac{\beta(E_1 + E_2)}{\mu + \epsilon + \theta} = N,$$

that is to say,

$$R_0(E_1 + E_2) = N \tag{5}$$

where

$$R_0 = \frac{\beta}{\mu + \epsilon + \theta}.$$

From (5), replacing N and solving for I, we have

$$(R_0 - 1)(E_1 + E_2) = I. \tag{6}$$

Then, from (5) and (6)

$$\frac{I}{N} = \frac{(R_0 - 1)(E_1 + E_2)}{R_0(E_1 + E_2)} = \frac{R_0 - 1}{R_0}. \tag{7}$$

If we solve (2) for E_1 and substitute in (7), then

$$E_1 = \frac{R_0 \Delta}{(R_0 - 1)\beta + R_0(\mu + v_1)}. \tag{8}$$

Adding (2), (3) and (4) we have

$$\Delta - (\mu + v_1)E_1 - (\mu + \epsilon)I - (\mu + v_2 + \omega)E_2 = 0. \tag{9}$$

From (9) substituting (6) and (8) and solving for E_2 we have:

$$E_2 = \frac{\Delta(R_0 - 1)R_0\theta}{(\beta\,(R_0 - 1) + R_0(\mu + v_1))\,((\mu + \epsilon)\,(R_0 - 1) + \mu + v_2 + \omega)}. \tag{10}$$

Replacing (8) and (10) in (6) we get that

$$I = (R_0 - 1)\Delta\frac{(R_0 - 1)\beta + R_0(\mu + v_2 + \omega)}{(\beta\,(R_0 - 1) + R_0(\mu + v_1))\,((\mu + \epsilon)\,(R_0 - 1) + \mu + v_2 + \omega)}. \tag{11}$$

With this we find the point of equilibrium $P_2 = (E_1^*, I^*, E_2^*)$ where,

$$E_1^* = \frac{R_0\Delta}{(R_0 - 1)\beta + R_0(\mu + v_1)} \tag{12}$$

$$I^* = \frac{(R_0 - 1)\Delta\,[(R_0 - 1)\beta + R_0(\mu + v_2 + \omega)]}{[\beta\,(R_0 - 1) + R_0(\mu + v_1)]\,[(\mu + \epsilon)\,(R_0 - 1) + \mu + v_2 + \omega]} \tag{13}$$

$$E_2^* = \frac{\Delta(R_0 - 1)\theta R_0}{[\beta\,(R_0 - 1) + R_0(\mu + v_1)]\,[(\mu + \epsilon)\,(R_0 - 1) + \mu + v_2 + \omega]}. \tag{14}$$

The point P_1 is called *disease-free equilibrium* and P_2, *endemic equilibrium*. It is important to note that P_2 makes biological sense only if $R_0 > 1$.

3.2 Mathematical definition of the Basic Reproductive Number

To mathematically determine the Basic Reproductive Number for population growth in the presence of tobacco consumption, we use the theory presented by Van den Driessche and Watmough in [5], which consists of finding the spectral radius of the next generation matrix.

Thus, let

$$F = \begin{bmatrix} \Delta \\ \beta\frac{I}{N}\,(E_1 + E_2) \\ \theta I \end{bmatrix} \qquad \text{and} \qquad W = \begin{bmatrix} \beta\frac{I}{N}E_1 + (\mu + v_1)E_1 \\ (\mu + \epsilon + \theta)I \\ \beta\frac{I}{N}E_2 + (\mu + \omega + v_2)E_2 \end{bmatrix}$$

the association matrices of the inflow and outflow of people that consume tobacco in system (1) and let $P_1 = \left(\frac{\Delta}{\mu+v_1}, 0, 0\right)$ the trivial point of equilibrium of (1). Then,

$$G = \left[\begin{array}{ccc} 0 & 0 & 0 \\ \beta\frac{I^2}{(E_1+I+E_2)^2} & \frac{\beta(E_1+E_2)^2}{(E_1+I+E_2)^2} & \beta\frac{I^2}{(E_1+I+E_2)^2} \\ 0 & \theta & 0 \end{array} \right]\Bigg|_{P_1} = \left[\begin{array}{ccc} 0 & 0 & 0 \\ 0 & \beta & 0 \\ 0 & \theta & 0 \end{array} \right]$$

and

$$A = \left[\begin{array}{ccc} \frac{\beta I(I+E_2)}{(E_1+I+E_2)^2} & \frac{\beta E_1(E_1+E_2)}{(E_1+I+E_2)^2} & \frac{\beta I E_1}{(E_1+I+E_2)^2} \\ 0 & \mu+\epsilon+\theta & 0 \\ \frac{\beta I E_2}{(E_1+I+E_2)^2} & \frac{\beta E_2(E_1+E_2)}{(E_1+I+E_2)^2} & \frac{\beta I(I+E_1)}{(E_1+I+E_2)^2}+\mu+v_2+\omega \end{array} \right]\Bigg|_{P_1}$$
$$= \left[\begin{array}{ccc} \mu+v_1 & \beta & 0 \\ 0 & \mu+\epsilon+\theta & 0 \\ 0 & 0 & \mu+v_2+\theta \end{array} \right]$$

Thus the next generation matrix is defined as:

$$GA^{-1} = \left[\begin{array}{ccc} 0 & 0 & 0 \\ 0 & \beta & 0 \\ 0 & \theta & 0 \end{array} \right] \left[\begin{array}{ccc} \frac{1}{\mu+v_1} & -\frac{\beta}{(\mu+v_1)(\mu+\epsilon+\theta)}\beta & 0 \\ 0 & \frac{1}{\mu+\epsilon+\theta} & 0 \\ 0 & 0 & \frac{1}{\mu+v_2+\theta} \end{array} \right] = \left[\begin{array}{ccc} 0 & 0 & 0 \\ 0 & \frac{\beta}{\mu+\epsilon+\theta} & 0 \\ 0 & 0 & 0 \end{array} \right]$$

The eigenvalues of the matrix GA^{-1} are

$$\lambda_1 = \frac{\beta}{\mu+\epsilon+\theta} \text{ and } \lambda_2 = \lambda_3 = 0$$

Thus, the Basic Reproductive Number for population growth in the presence of tobacco consumption is:

$$R_0 = \max\{\lambda_1, \lambda_2\ \lambda_3\} = \lambda_1 = \frac{\beta}{\mu+\epsilon+\theta},$$

which represents the average number of new tobacco addicts that a single addicted person would produce during their period of infection in a population of exposed people.

3.3 Local stability analysis

The local stability of P_1 and P_2 is described in the Theorem 1 with the definitions of stability for dynamic systems [10].

Theorem 1. *Local Stability*

- If $R_0 < 1$ then $P_2 \notin \Omega$ and P_1 is asymptotically stable at the local level.
- If $R_0 > 1$ then $P_1,\ P_2 \in \Omega$ and P_2 is asymptotically stable at the local level, while P_1 is unstable.

Proof. If $R_0 < 1$, and we suppose that $P_2 \in \Omega$, then $R_0 - 1 < 0$ and $E_1^*,\ I^*,\ E_2^* \geq 0$.

Observe that from (12) $(R_0 - 1)\beta + R_0(\mu + v_1) > 0$. Thus, from (14) $(\mu + \epsilon)(R_0 - 1) + \mu + v_2 + \omega < 0$. From this, and from (13) $(R_0 - 1)\beta + R_0(\mu + v_2 + \omega) > 0$. Therefore, for the assumption to be true, the three inequalities must be satisfied, but this is imposible because:

$$\begin{aligned}(R_0 - 1)\beta + R_0(\mu + v_2 + \omega) &= (R_0 - 1)R_0(\mu + \epsilon + \theta) + R_0(\mu + v_2 + \omega) \\ &= R_0[(R_0 - 1)(\mu + \epsilon) + \mu + v_2 + \omega] + R_0(R_0 - 1)\theta \\ &< 0,\end{aligned}$$

and we have shown that $(\mu + \epsilon)(R_0 - 1) + \mu + v_2 + \omega < 0$ and $R_0 - 1 < 0$.

Thus, if $R_0 < 1$, then $P_2 \notin \Omega$ and it is not of interest to analyse its stability.

From system (1) we derive the Jacobean matrix

$$J(P) = \begin{bmatrix} \frac{-\beta I(I+E_2)}{N^2} - (\mu + v_1) & \frac{-\beta E_1(E_1+E_2)}{N^2} & \frac{\beta I E_1}{N^2} \\ \frac{\beta I^2}{N^2} & \frac{\beta(E_1+E_2)^2}{N^2} - (\mu + \epsilon + \theta) & \frac{\beta I^2}{N^2} \\ \frac{\beta I E_2}{N^2} & -\frac{\beta E_2(E_1+E_2)}{N^2} + \theta & -\frac{\beta I(I+E_1)}{N^2} - (\mu + v_2 + \omega) \end{bmatrix}$$

For P_1, we have

$$J(P_1) = \begin{bmatrix} -(\mu + v_1) & -\beta & 0 \\ 0 & -\beta\left(\frac{1-R_0}{R_0}\right) & 0 \\ 0 & \theta & -(\mu + v_2 + \omega) \end{bmatrix}$$

The characteristic polynomial of $J(P_1)$ can be written in the form:

$$p(\lambda) = (\lambda + \mu + v_1)\left(\lambda + \beta\left(\frac{1 - R_0}{R_0}\right)\right)(\lambda + \mu + v_2 + \omega)$$

Thus, the eigenvalues are:

$$\lambda_1 = -(\mu + v_1), \quad \lambda_2 = -\beta\left(\frac{1 - R_0}{R_0}\right) \quad \text{and} \quad \lambda_3 = -(\mu + v_2 + \omega)$$

If $R_0 < 1$ then all the eigenvalues of the matrix $J(P_1)$ are negative and so the equilibrium point P_1 is **asymptotically stable at the local level**. Whereas if $R_0 > 1$, then λ_2 is positive and $\lambda_1,\ \lambda_3$ are negative, making P_1 **locally unstable**.

Evaluating the equilibrium point P_2 in the matrix $J(P)$ we obtain the matrix

$$J(P_2) = \begin{bmatrix} -H\left(1 + \frac{E_2^*}{I^*}\right) - (\mu + v_1) & -K\frac{E_1^*}{I^*} & H\frac{E_1^*}{I^*} \\ H & -K & H \\ H\frac{E_2^*}{I^*} & -K\frac{E_2^*}{I^*} + \theta & -H\left(1 + \frac{E_1^*}{I^*}\right) - (\mu + v_2 + \omega) \end{bmatrix}$$

where $H = \beta\left(\frac{R_0 - 1}{R_0}\right)^2$ and $K = \frac{\beta(R_0 - 1)}{R_0^2}$.

The characteristic polynomial of $J(P_2)$ is written in the form:

$$P(\lambda) = \lambda^3 + b_1\lambda^2 + b_2\lambda + b_3,$$

where

$$\begin{aligned} b_1 =& 2R_0K + \mu + v_1 + \mu + v_2 + \omega. \\ b_2 =& H(\mu + \epsilon) + K(\mu + v_2 + \omega) + (R_0K + \mu + v_2 + \omega)\left(H + H\frac{E_2^*}{I^*} + \mu + v_1\right) \\ &+ H\frac{E_1^*}{I^*}(R_0K + \mu + v_1). \\ b_3 =& [H(\mu + \epsilon) + K(\mu + v_2 + \omega)](R_0K + \mu + v_1). \end{aligned}$$

If $R_0 > 1$, then $R_0 - 1 > 0$, and $K,\ H,\ E_1^*,\ I^*,\ E_2^* > 0$. Thus, $b_1,\ b_2,\ b_3 > 0$.

Furthermore,

$$b_1b_2 - b_3 = [2R_0K + \mu + v_1 + \mu + v_2 + \omega]\left[(R_0K + \mu + v_2 + \omega)\left(H + H\frac{E_2^*}{I^*} + \mu + v_1\right)\right.$$
$$\left. + H\frac{E_1^*}{I^*}(R_0K + \mu + v_1)\right] + [R_0K + \mu + v_2 + \omega][H(\mu + \epsilon) + K(\mu + v_2 + \omega)]$$
$$> 0.$$

Finally, since none of the coefficients of the characteristic polinomial are zero, all the coefficients have the same sign and the Hurwitz determinantes are positive [10], all the roots of the polinomial have a negative real part. Therefore, if $R_0 > 1$, then the point of equilibrium P_2 is locally **stable**.

4 Numerical results

In this section, using the fourth order Runge-Kutta method programmed in the MATLAB software, we find numerical solutions to system (1) for different values of the parameters, showing that these exhibit the points of equilibrium found above and the cualitative behaviour described analytically.

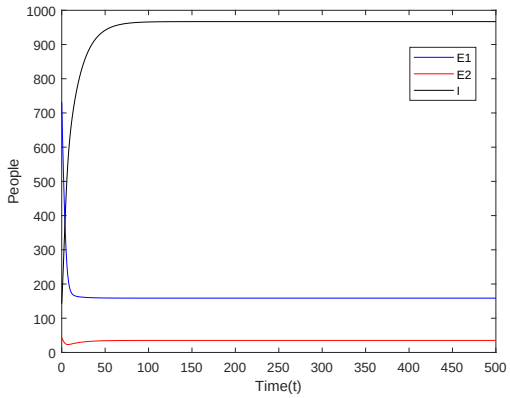

Figure 2: Numerical solutions of system (1) when $R_0 > 1$

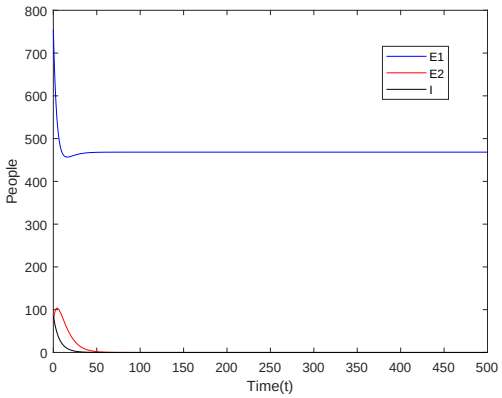

Figure 3: Numerical solutions of system (1) when $R_0 < 1$.

For Figures 2 and 3 a hypothetical initial population of 1000 people was taken, with 850 healthy people that could acquire a smoking habit, 100 people addicted to smoking and 50 ex-smokers at risk of starting again. On average, 100 healthy people became at risk of being active smokers. Furthermore, the approximate probability of a person dying of natural causes was 1.35%, the

probability that an exposed person stops running the risk of becoming an active smoker was 20%, while and ex-smoker might make the same transition with a probability of 10%. It was assumed that an ex-smoker could die as a consequence of having been an active smoker with a probability of 2%.

Due to the authors own interests, each simulation used different values for the remaining parameters; for Figure 2 the probablility that a person acquired a smoking habit was 50%, an active smoker could die from an illness caused by the consumptio of tobacco with a probability of 5% and the probability of quitting smoking was 2% and for Figure 3, the probabilities were 30%, 6% and 35% respectively.

To demonstrate that the models tend to the equilibrium points identified analytically, the models were run for 500 units of time. In effect, these simulations satisfactorily support the analytical results.

5 Conclusions

- The average number of people who catch a smoking habit from a single addicted smoker is given by the product of the probability of infection β and the expected time as a smoker $\frac{1}{\mu+\epsilon+\theta}$.
- When the average number of new tobacco addicts that a single addicted smoker produces is less than one, the population of smokers disappears over time.
- When the average number of new tobacco addicts that a single addicted smoker produces is more than one, the smoking population persists.

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