The Impact of the Sleeper Effect and Relapse on the Dynamics of Cigarette Smoking Among Adolescents

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Abstract

The Center for Disease Control (CDC) predicts that 6.4 million of today's children will die prematurely from a smoking related illness if environmental conditions remain the same. The percentage of high school students who smoke cigarettes has remained at around 23% for the past three years. Recent research reports a "sleeper effect" in children. That is, children who smoked once before age 11 are twice as likely to become a regular smoker by age 14. We model smoking dynamics among children ages 11 to 18 as a "socially-transmitted" disease, and use it to explore possible mechanisms of the "sleeper effect." Is it due to prior exposure or is it due to higher relapse rates? The model fits the number of smokers for the past 16 years as reported by the CDC. The feasibility of the CDC's goal for 2010 is evaluated. The significance of relapse is

highlighted by a simple bifurcation analysis. The effects of education on this group are explored and recommendations for effective approaches are made.

1 Introduction

The Center for Disease Control (CDC) has identified cigarette smoking as the leading cause of preventable illness and death in the United States, responsible for about 438,000 deaths a year [10, 15]. Approximately 45 million adults are regular smokers, each spending on average $1600-$2000 annually on cigarettes [11, 14]. Altogether, the direct and indirect costs incurred by smokers exceed $167 billion per year [15].

Research has shown that most regular smokers become addicted to cigarettes during adolescence [2]. A recent study has also indicated that cigarette smoking has a "sleeper effect" on children [18, 17]. The term "sleeper effect" refers to the strong correlation between smoking a single cigarette before age 11 and the probability of becoming a regular smoker by age 14. This study was conducted over the course of five years on a group of 2,000 children ages 11 to 16. Of 260 children who had smoked one cigarette by age 11, 18% became regular smokers by the time they were age 14. In contrast, only 7% of the children who had not smoked by age 11 were regular smokers by age 14 [18, 17].

A major goal of cigarette advertising has been to promote and maintain tobacco addiction among children [21]. Old Joe Camel, the cartoon character used in cigarette advertisements by the R.J. Reynolds Tobacco company, who drew much controversy in the 1990's highlights the role of advertisement geared toward teens. A study in 1991 showed that Old Joe Camel and its associated brand name were more readily identified by teens than adults. Teens also found the advertisements more appealing than adults [21]. In 2003, $15.2 billion was spent by cigarette companies to promote their product. This proved very successful as 83% of young smokers chose one of the three most heavily advertised brands: Marlboro, Camel, and Newport [19]. Additionally, a recent study attributes 34% of new experimentation with cigarettes among teens to pro-smoking advertisements [23].

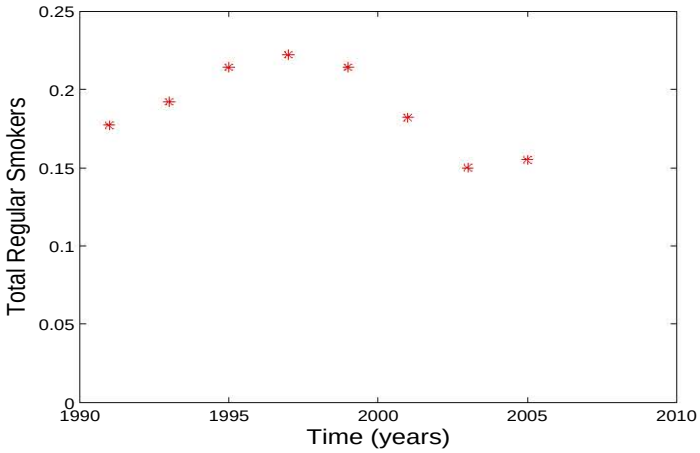

Figure 1: CDC Data: 1991-2005

The CDC defines current smoking for adolescents as having smoked in the past 30 days [7]. The CDC and the Surgeon General have established a goal for the year 2010 as part of their Healthy People 2010 objectives, to reduce the percentage of high school students who smoke to 16%. They also hope to increase attempts to quit smoking by adolescents to 84% and to increase adolescents' disapproval of smoking to 95% [12].

There was a steady decrease in the total number of smokers per year from 1997-2003 [16]. However, recent data from the CDC suggests that the percentage of high school students who smoke in the U.S. has remained constant over the last three years at about 23%, while about 8% of middle school children are reported to be regular smokers [16, 20]. Figure 1 plots the fraction of individuals ages 11 to 18 who smoke per year from 1991 to 2005. If present trends continue, an estimated 6.4 million of today's children are expected to die prematurely from smoking related diseases [19].

There is no scientific explanation for why the rate of becoming a regular smoker is twice as high for children who smoked before age 11 compared to those who did not. The "sleeper effect" suggests that this correlation is a direct result of children experimenting with cigarettes at a young age. Is it possible that the differences in smoking rates are not

caused by children smoking before age 11, but are rather an effect of some other social phenomena?

Using an epidemiological model, we classify cigarette smoking as an "infectious disease" [24]. We use current data to analyze the dynamics of cigarette smoking among adolescents to determine how feasible it is to obtain the CDC's 2010 goal. We explore possible strategies to reduce the prevalence of cigarette smoking and gauge the impact that educational anti-smoking programs must have in order to control cigarette smoking.

2 Formulation of Epidemiological Model for Teen Smoking

Table 1: Parameter Definitions

Parameter	Definition
μ	The rate of leaving a class as a result of aging or death It is also the recruitment rate
r	Fraction of adolescents who smoked before age 11
β_i	Conversion rate of new smokers
ϕ_i	Rate at which individuals quit smoking
ψ_i	Relapse rate
α_i	Rate at which individuals permanently quit smoking
ε_i	Rate at which individuals who have never been regular smokers abstain from smoking

In this section we present a mathematical model to study the dynamics of smoking in the United States among children ages 11 to 18. All children enter the system as susceptible non-smokers with either little or no smoking history. Consequently, a two-string model is used where string 1 is composed of individuals who smoked once before age 11 and string 2 is composed of those who did not, so that the "sleeper effect" can be investigated.

Each string is composed of three classes; N_i, S_i, and Q_i (where $i = 1, 2$ denotes each string). The susceptible non-smoker classes (N_i) are composed of individuals who have

never been regular smokers. The regular smoker classes (S_i) are composed of individuals who have smoked in the last month. The recovered classes (Q_i) are composed of individuals who have quit smoking but are susceptible to relapse. The class of permanent non-smokers (P) is composed of individuals who have decided to permanently quit (from Q_i) or who decided to never become regular smokers (from S_i).

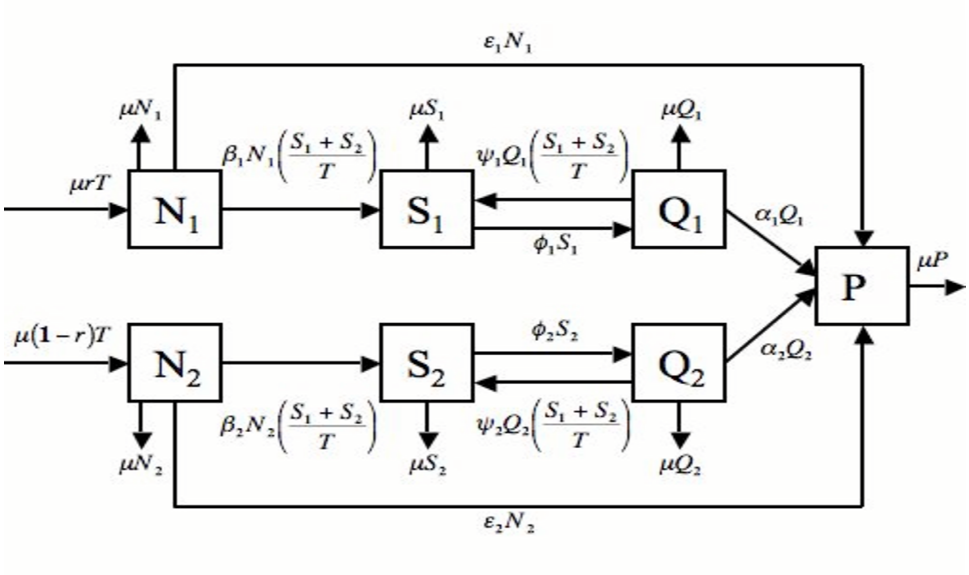

Figure 2: Flow Chart

Individuals who turn 11 years old enter N_1 and N_2 with rates μrT and $\mu(1-r)T$, respectively, where T is the total population of adolescents. Non-smokers (N_i) are susceptible to becoming regular smokers as a result of interactions with regular smokers (S_i). Smokers (S_i) can quit smoking due to personal choice or education. Individuals who have quit (Q_i) can relapse due to social interactions with smokers. All individuals who are not currently regular smokers (N_i, Q_i) can move to class P by choosing to permanently abstain from cigarettes. We assume the total population, T, is constant.

$$\frac{dN_1}{dt} = \mu r T - \beta_1 N_1 \frac{S_1 + S_2}{T} - (\varepsilon_1 + \mu) N_1 \tag{1}$$

$$\frac{dS_1}{dt} = \beta_1 N_1 \frac{S_1 + S_2}{T} + \psi_1 Q_1 \frac{S_1 + S_2}{T} - (\phi_1 + \mu) S_1 \tag{2}$$

$$\frac{dQ_1}{dt} = \phi_1 S_1 - \psi_1 Q_1 \frac{S_1 + S_2}{T} - (\alpha_1 + \mu) Q_1 \tag{3}$$

$$\frac{dN_2}{dt} = \mu (1 - r) T - \beta_2 N_2 \frac{S_1 + S_2}{T} - (\varepsilon_2 + \mu) N_2 \tag{4}$$

$$\frac{dS_2}{dt} = \beta_2 N_2 \frac{S_1 + S_2}{T} + \psi_2 Q_2 \frac{S_1 + S_2}{T} - (\phi_2 + \mu) S_2 \tag{5}$$

$$\frac{dQ_2}{dt} = \phi_2 S_2 - \psi_2 Q_2 \frac{S_1 + S_2}{T} - (\alpha_2 + \mu) Q_2 \tag{6}$$

$$\frac{dP}{dt} = \varepsilon_1 N_1 + \varepsilon_2 N_2 + \alpha_1 Q_1 + \alpha_2 Q_2 - \mu P \tag{7}$$

$$T = N_1 + N_2 + Q_1 + Q_2 + S_1 + S_2 + P \tag{8}$$

Equations (1-3) describe the dynamics of the population that smoked at least once before age 11. Likewise, Equations (4-6) describe the dynamics of the population that did not smoke before age 11.

3 Dynamical Analysis

The basic reproductive number, R_0, represents the average number of secondary smokers generated by a "typical" regular smoker during an average period of regular smoking in a mostly non-smoking population. An epidemic always occurs when $R_0 > 1$.

On average, when $R_0 < 1$, more smokers leave than enter the system and the total number of smokers decreases. It has been observed that in certain socially transmitted processes that incorporate relapse, epidemics are possible even with $R_0 < 1$ [25, 24]. In our model we consider the effects of high relapse rates on the conditions necessary for a smoker free equilibrium. Thus, it is important to analyze the behavior of the system for

Table 2: Explanation of Basic Reproductive Number

Component	Meaning
β_1	Per capita "infection" rate of smokers who smoked at least once before age 11
$\frac{1}{\phi_1+\mu}$	Average smoking period in S_1
$\frac{\mu}{\varepsilon_1+\mu}$	Fraction of "at-risk" non-smokers in N_1
β_2	Per capita "infection" rate of smokers who did not smoke before age 11
$\frac{1}{\phi_2+\mu}$	Average smoking period in S_2
$\frac{\mu}{\varepsilon_2+\mu}$	Fraction of "at-risk" non-smokers in N_2

$R_0 < 1$.

3.1 Basic Reproductive Number

The smoker-free equilibrium (SFE) of the system is:

$$(S_1{}^*, S_2{}^*, N_1{}^*, N_2{}^*, Q_1{}^*, Q_2{}^*, P^*) = \left(0, 0, \frac{\mu r}{\varepsilon_1+\mu}, \frac{\mu\,(1-r)}{\varepsilon_2+\mu}, 0, 0, \frac{\varepsilon_1 r}{\varepsilon_1+\mu} + \frac{\varepsilon_2\,(1-r)}{\varepsilon_2+\mu}\right)$$

and the basic reproductive number is,

$$R_0 = \frac{r\beta_1}{(\phi_1+\mu)} \cdot \frac{\mu}{(\varepsilon_1+\mu)} + \frac{(1-r)\beta_2}{(\phi_2+\mu)} \cdot \frac{\mu}{(\varepsilon_2+\mu)}.$$

See Appendix 10.1 for detailed calculations of R_0. R_0 has distinct contributions from each class of smokers defined in our model. Table 2 explains the components of R_0.

3.2 Backward Bifurcation Analysis

We evaluate the Jacobian of our system of differential equations (1-7) at the SFE and compute appropriate left and right eigenvectors to determine the type of bifurcation our

system exhibits at $R_0 = 1$. From this, a critical value, a, exists when $R_0 = 1$ such that, for $a < 0$ our system has a forward bifurcation[25]. For $a > 0$ our system exhibits a backward bifurcation [25].

$$a = \sum_k \sum_{i,j} \frac{\partial^2 f_k}{\partial x_i \partial x_j} w_i w_j v_k \Bigg|_{(SFE,\ \beta_1 = \beta_1^*)} \tag{9}$$

where Equation (9) is defined in Section 10.2.

Theorem 1. *If*

$$\frac{\psi_1}{m} + \frac{\psi_2}{n} > 1,$$

backward bifurcation occurs at $R_0 = 1$*, where*

$$\begin{aligned}
m &= \frac{C}{A}; \quad n = \frac{C}{B} \\
A &= \frac{\phi_1(\phi_2 + \mu)\left[(\varepsilon_2 + \mu)(\phi_2 + \mu) - \mu\beta_2(1 - r)\right]}{\beta_2(1 - r)\mu(\mu + \alpha_1)} \\
B &= \frac{\phi_2}{\mu + \alpha_2} \\
C &= \frac{(\phi_1 + \mu)\left[(\varepsilon_2 + \mu)(\phi_2 + \mu) - \mu\beta_2(1 - r)\right]^2}{r\mu^2\beta_2(1 - r)(\mu + \varepsilon_2)} + \frac{\beta_2(\mu + \phi_2)}{\mu + \varepsilon_2}.
\end{aligned}$$

See Section 10.2 for proof of Theorem 1.

From Theorem 1 we know that for large relapse rates an "epidemic" of smoking may occur even if $R_0 < 1$. The backward bifurcation curve (Figure 3) illustrates the three types of allowed equilibria for $R_0 < 1$. The backward bifurcation comes from the non-linearity generated by the relapse terms. Figure 4 illustrates the existence of a stable endemic and a stable smoker free equilibrium for $R_0 < 1$.

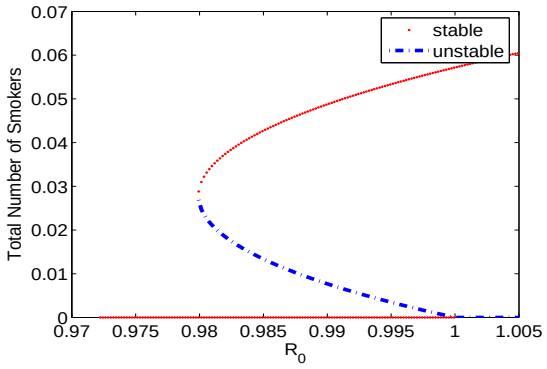

Figure 3: Backward Bifurcation

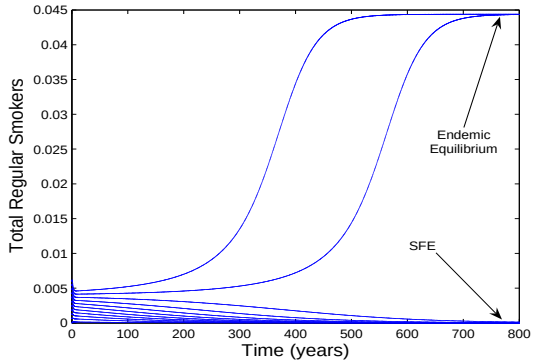

Figure 4: Bi-stable equilibrium for $R_0 < 1$

The cycle of quitting and relapsing due to social interactions is significant because it can create enough secondary cases to compensate for the inability of current smokers to influence other individuals to begin smoking. It is this cycle that allows the existence of endemic equilibria for $R_0 < 1$.

Figure 5 illustrates combinations of ψ_1 and ψ_2 that result in forward bifurcation and combinations that results in backward bifurcation. Parameters used to generate this plot were estimated from US data on adolescent smoking. See Section 4 for details on parameter estimation.

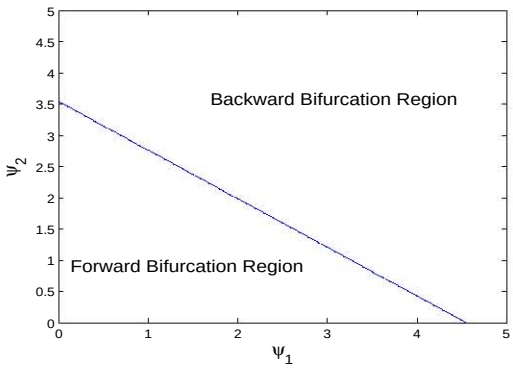

Figure 5: Bifurcation Plane

3.3 Endemic Equilibrium

Theorem 2 guarantees the existence of an endemic equilibrium for $R_0 > 1$ when $\beta_1 = \beta_2$, $\phi_1 = \phi_2$, $\psi_1 = \psi_2$, $\varepsilon_1 = \varepsilon_2$, $\alpha_1 = \alpha_2$, and $r = 1$. This is a special case where experimentation with cigarettes prior to age 11 is not considered.

Theorem 2. *If $R_0 > 1$, there is a unique endemic equilibrium point.*

Proof of Theorem 2 can be found in Appendix 10.3.

We have proven analytically and verified numerically that for $R_0 > 1$ a unique endemic equilibrium exists. The endemic equilibrium is locally asymptotically stable and the SFE is unstable. A value of $R_0 > 1$ always implies that an epidemic will result. These results are significant because they show that we must focus on reducing both R_0 and the relapse rates in order to control teen smoking.

4 Estimation of parameters

There is limited data on the number of individuals per year who begin, quit, and relapse into smoking that specifies whether or not the individuals have smoked before age 11. However, more data is available for the total number of individuals who begin, quit, and

Table 3: Parameter Values

Parameter	r	μ	β	ε	ϕ	ψ	α
Value/Range	0.14	0.125	0.48	0.06	0.546	11-25	0.025-0.1
Source	[17]		[13]	[5]	[2, 8, 6]		

relapse per year. We used this and other data to estimate r, β, ε ϕ, ψ, and α for the entire population [1, 2, 5, 6, 8, 13, 17]. These rates can be used to approximate β_i, ε_i ϕ_i, ψ_i, and α_i for $i = 1, 2$. We used years as the units of time for our model, because most available data is given in years. All rates have units year^{-1}.

$\mu \approx .125$. Individuals spend at most 8 years in our system. Thus the average rate that individuals exit the system due to aging is $\frac{1}{8}$. The natural death rate for this age group is ignored because it is insignificant compared to the the exit rate due to aging [4].

$r \approx .14$. According to a study that was done by Cancer Research UK approximately 14% of 11 year old children have experimented with cigarettes [17].

$\beta \approx .48$. The U.S. Department of Health and Human Services estimates that 11.9% of teens are current smokers. Additionally, 3,900 teens between the ages of 12 and 17 start smoking every day [13]. Using this data, we estimated the percentage of adolescents that start smoking each year. Dividing this percentage by the percentage of adolescents who currently smoke gives the influence of peers on smoking (β).

$\varepsilon \approx .06$. There is no data available for the rate at which non-smokers abstain permanently from cigarettes. We estimated that ε is low relative to the other parameters because most middle school and high school students are not isolated from teens who smoke, so they will remain susceptible due to social interactions with smokers.

$\phi \approx .546$. The CDC estimates that 54.6% of high school students try to quit smoking each year [5]. We use this as the quitting rate.

$11 < \psi < 25$. Estimated relapse rates usually range from 40 to 90% [8, 6]. Since relapse rates are very high, we know ψ should be quite large relative to the other parameters.

Evidence indicates that most people who relapse do so within the first three to six months [1]. We let this be the average amount of time that an individual spends in one of the Q_i classes. This time is denoted by t_q, which can be expressed as one divided by the total rate at which individuals leave the quitting classes.

$$t_q = \frac{1}{\mu + \alpha + \psi \frac{S_1+S_2}{T}} \tag{10}$$

Since data indicates that the number of teen smokers is currently approaching a steady state, we assume that $\frac{S_1+S_2}{T}$ is constant. Currently, 23% of high school students and 8% of middle school students smoke [20]. Assuming half of the individuals between the ages of 11 and 18 are in high school and half are in middle school, 15.5% of the total population, T, are regular smokers, which gives $\frac{S_1+S_2}{T} = .155$. Using this information as well as our estimations for μ and α we found the range for ψ.

$.025 < \alpha < .1$. Reports indicate that every year $2.5 - 10\%$ of individuals who try to quit succeed [2, 6]. We use this to approximate α.

5 Numerical Solutions

Solutions to our system show multiple endemic equilibria for $R_0 < 1$. A unique solution exits for $R_0 > 1$. We use the parameter values and parameter ranges determined in Section 4 along with numerical simulations to obtain insight into the dynamics of current cigarette smoking.

5.1 Curve fitting

Solutions to our model can accurately fit CDC data on cigarette smoking among adolescents for the past 16 years. The parameters values we estimated in Section 4 were similar

to those required for good fits. Figure 6 shows a good fit to CDC data. Initial conditions were: $N_1 = 0.832r$, $N_2 = 0.823(1-r)$, $S_1 = 0.177r$, $S_2 = 0.177(1-r)$, $Q_1 = 0$, $Q_2 = 0$, and $P = 0$. Parameter values were: $r = 0.14$, $\mu = 0.125$, $\beta_1 = 0.95$, $\varepsilon_1 = 0.03$, $\phi_1 = 0.45$, $\psi_1 = 12$, $\alpha_1 = 0.11$, $\beta_2 = 0.73$, $\varepsilon_2 = 0.03$, $\phi_2 = 0.59$, $\psi_2 = 7.21$, and $\alpha_2 = 0.11$.

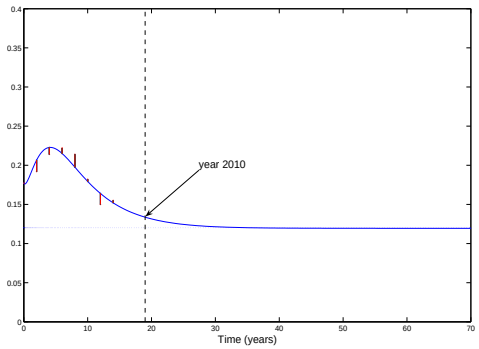

Figure 6: Model fitted to US data

Figure 6 indicates the percentage of adolescents who smoke will slowly decline for 20 years and then reach equilibrium at 12%. This result is in agreement with CDC reports that teen smoking is approaching equilibrium [16].

We assume that children who smoke before age 11 become smokers at a higher rate and relapse at a higher rate due to the "sleeper effect". We assume individuals who did not smoke before age 11 quit smoking at a higher rate. The two rates at which individuals permanently quit smoking are the same.

Parameter values used to obtain a good fit result in $R_0 \approx 0.89$. Parameter values from other good fits also suggest $R_0 < 1$. From Theorem 1 and Section 3.2 we know that high relapse rates are the reason why there is currently an "epidemic" of smoking. This is significant because it means efforts should be focused not only on reducing the rate at which non-smokers begin smoking but also on reducing relapse rates.

Table 4: Distribution of Parameters in R_0

Parameter	Distribution	Mean
r	Beta	.14
μ	Generalized Pareto	.125
β_i	Gamma	0.95, 0.73
ε_i	Gamma	0.06
ϕ_i	Gamma	0.45, 0.59

6 Uncertainty Analysis

We approximate a distribution for each parameter and use this information to perform Monte Carlo simulations of R_0. This uncertainty analysis validates our calculation of R_0.

Values of r have a beta distribution with mean 0.14. Values of r can range from 0 to 1 because it is a proportion. We expect the beta distribution to be left skewed. A value of $r = 0$ means that in a given sample zero children smoked before age 11. Likewise, $r = 1$ means that all sampled children smoked prior to age 11. We expect most samples will include at least some children that experimented, and samples with large numbers of experimenters will be rare.

Values of μ have a generalized Pareto distribution (whose PDF is a shifted exponential function). The minimum value of μ occurs when the natural death rate of individuals ages 11 to 18 is zero. Values of μ increase when natural death is included. There is zero probability μ will be less than 0.125 because individuals cannot spend more than 8 years in our system. The natural death rate for adolescents in the U.S. is very small [4], so the mean should be close to 0.125. For this reason, μ has a generalized Pareto distribution with threshold parameter of 0.125.

Gamma distributions are chosen for β_i because these parameters cannot be less than zero and have a non-zero probability of being any positive number. Data suggest these values are near 0.95 and 0.73 respectively.

Table 5: Monte Carlo Simulations - sample size of 100

Realization	Mean(R_0)	Std(R_0)	P($R_0 > 1$)
1	0.727	0.446	0.200
2	0.681	0.522	0.170
3	0.785	0.455	0.280
4	0.685	0.380	0.160
5	0.723	0.465	0.200
6	0.794	0.613	0.270
7	0.798	0.503	0.290
8	0.671	0.447	0.160
9	0.687	0.454	0.150
10	0.779	0.484	0.240
mean	0.7330	0.4769	0.2120
se	0.0163	0.0194	0.0170
cv	0.0703	0.1283	0.2543

Values of ε_i have gamma distributions with mean 0.06 because values are greater than 0 but are expected to be left skewed. A value of $\varepsilon_i = 0$ means that in a given sample 0% of individuals who have never been regular smokers decide to permanently abstain from smoking per year. Likewise, $\varepsilon_i = 1$ means that 100% of these individuals decide to never smoke. We expect the values of ε_i to be left skewed because most adolescents cannot isolate themselves from peers who smoke because they continue to interact with smokers at school.

Gamma distributions with means 0.45 and 0.59, respectively, are chosen for ϕ_i. Data suggests the rates at which smokers quit are likely to be from 48% to 54%.

Using Monte Carlo simulations we estimate a mean value for R_0 of 0.7330. The probability that $R_0 > 1$ is 0.2120. This validates our estimate in Section 5.1 that R_0 is less than one and high relapse rates are the reason for the smoking "epidemic."

Table 4 is a summary of parameter information used to perform uncertainty analysis on R_0. Table 5 presents results from Monte Carlo simulations of R_0 and Figure 7 plots the distribution of R_0.

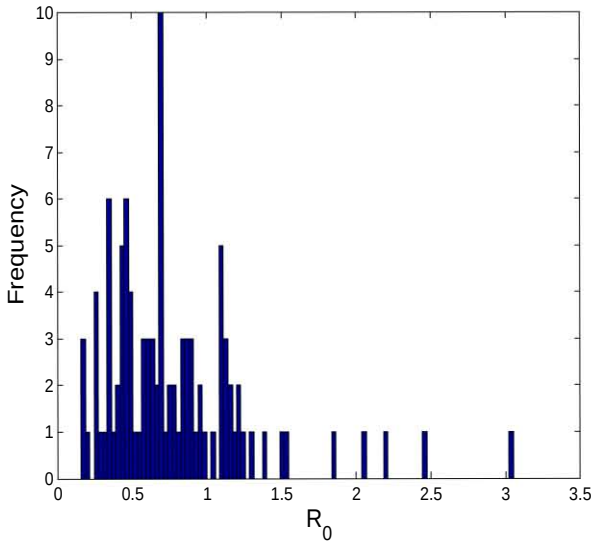

Figure 7: Distribution of R_0 from Monte Carlo Simulations

7 Effects of education

There are many organizations that provide education about the health effects of smoking and support for teens in an effort to stop adolescent smoking [3, 22]. Education on the risks and costs of smoking can decrease the total number of adolescents who smoke. To determine the impact education can have on cigarette smoking we study numerical solutions of our model for various values of r, ε_i, ϕ_i, ψ_i, and α_i. Each of these parameters can be affected through education. We assume education has a minimal effect on the transmission rate (β_i), due to the fact that transmission is a social process.

Children can be educated about the effects of cigarettes through classes in school, after-school programs, and educational advertising. These methods of education can each be used to vary different parameters. Because advertising reaches such a wide range of individuals, it can have an effect on all parameters except μ. Programs about smoking implemented at the elementary school level can lower r. Educational programs in middle

and high schools can influence ε_i because children who are educated about the dangers and expenses associated with smoking are less susceptible to smoking. After-school programs for teens who currently smoke or have quit smoking could affect ϕ_i, ψ_i, and α_i by encouraging teens to quit smoking and giving them the support that they need to remain smoke free.

We used parameter values from Section 5.1 and investigated the effects of varying these parameters starting in the year 2007. These solutions give predictions for the percentage of smokers for the next five decades. We first targeted pairs of parameters and then varied multiple parameters simultaneously. We were able to determine reasonable goals for the reduction of teen smoking and analyze the feasibility of meeting the CDC's goal to reduce smoking among high school students to 16% (or about 12% of 11-18 year olds) by the year 2010. We also compared the effectiveness of each educational approach.

7.1 Varying ψ_1 and ψ_2

From Theorem 1, backward bifurcation is determined by the relapse rates (ψ_1, ψ_2). From Section 5.1 and Section 6 we know these relapse rates are responsible for the current existence of smoking. To control smoking, it is important to decrease these rates to break the cycle of quitting and relapsing. Smoking will eventually decrease to zero with sufficiently small relapse rates when $R_0 < 1$. Because relapse rates are currently very high, significant changes are necessary for this to occur.

From Figure 8 we see that decreasing relapse rates by 25% is enough to meet the CDC's 2010 goal, and the percentage of smokers continues to decrease toward an endemic equilibrium of about 9%. Decreasing relapse rates by 40% reduces the percentage of smokers to about 5% in 50 years. When relapse rates are decreased by 60% the SFE is approached because not enough people are relapsing to make up for the inefficiency of regular smokers to recruit new smokers. This implies that $R_0 < 1$ because ψ_1 and ψ_2 do

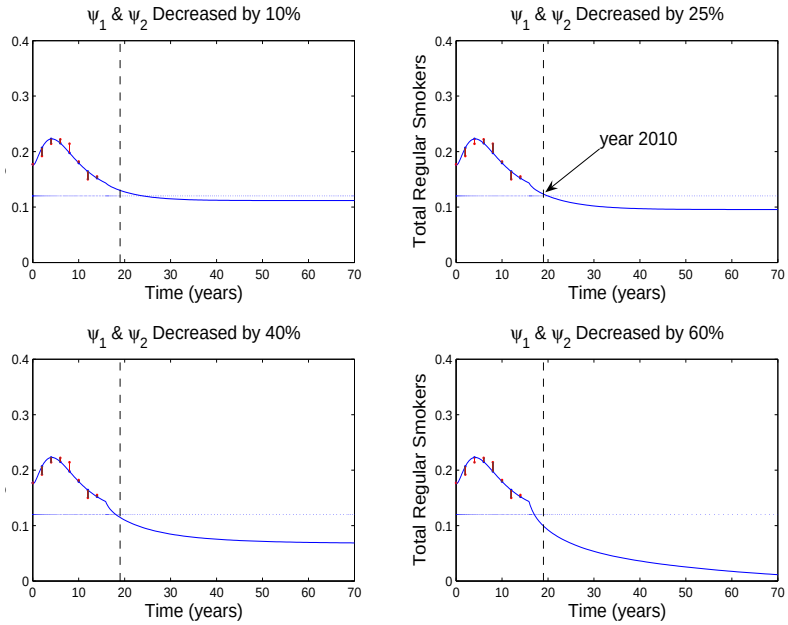

Figure 8: Predictions when ψ_1 and ψ_2 are varied

not contribute to R_0. It is important to note that smoking can be eliminated by targeting only the relapse rates, although such a large reduction in these rates may not be feasible.

The CDC's plans to help them achieve the 2010 goal do not currently include reducing relapse rates. Measures to decrease relapse rates can begin by increasing the number of anti-smoking programs that offer support to individuals who have already quit. Programs designed to help individuals quit should provide follow-up resources so that individuals can have support during the first weeks of being smoke free when they are most likely to relapse [1, 9].

7.2 Varying r

The fraction of children who smoke before age 11 is small ($r = 0.14$) but it affects the dynamics of the smoking population (see Figure 9). When $r = 0$, which means that no

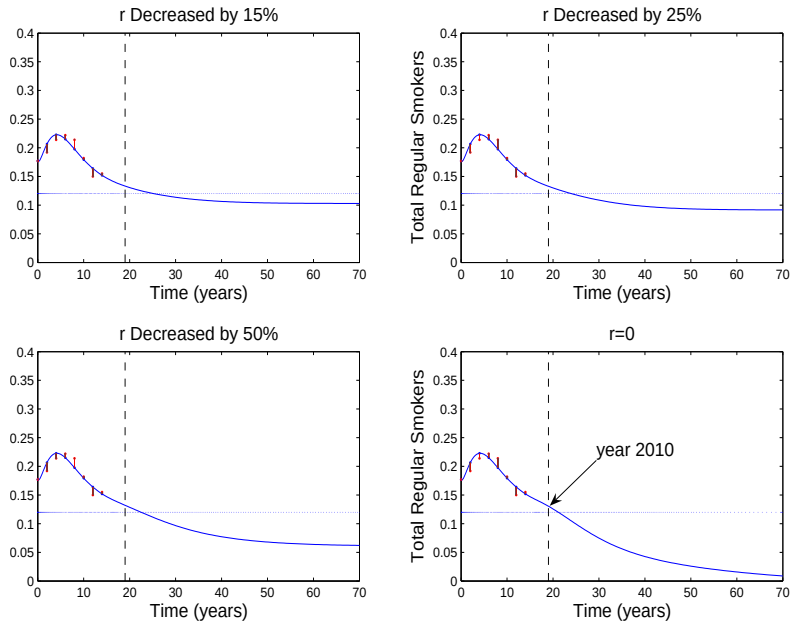

Figure 9: Predictions when r is varied

children smoked before the age of 11, the percentage of adolescent smokers eventually approaches the SFE. However, the CDC's goal for 2010 is still not met. Thus, focusing education only on young children who have never smoked before in order to prevent a "sleeper effect" is not sufficient. Elementary school anti-smoking programs must be used in conjunction with programs for middle and high school students in order to produce significant change.

In addition to educational programs at elementary schools, severe punishments for adults and moderate punishments for minors who facilitate access to cigarettes for anyone under 11 can decrease the number of children who have access to cigarettes and thus decrease r. Decreasing r may be increasingly difficult because some children will always find a way to obtain cigarettes. Although the "sleeper effect" is an interesting phenomenon, it is not the most influential factor affecting adolescent smoking.

7.3 Varying ε_1, ε_2

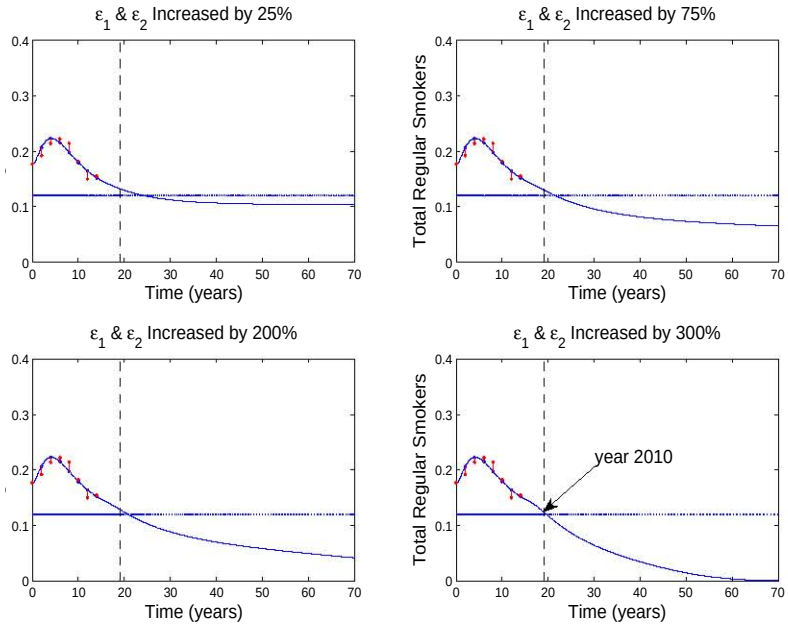

Figure 10: Predictions when ε_1 and ε_2 are varied

Figure 10 shows that increasing the rates at which teens who have never been regular smokers permanently abstain from smoking (ε_1, ε_2) causes a gradual decrease in the percentage of adolescent smokers. A 75% increase will reduce adolescent smoking to about 7% in 50 years. The CDC's 2010 goal would require a 300% increase in these parameters. This change would be enough to also completely eliminate adolescent smoking in about 40 years.

Such a large change in these rates may not seem feasible. However, the rates are currently so small that a 300% increase only makes $\varepsilon_i = 0.09$. This value means that 9% of non-smokers choose to permanently abstain from smoking each year. Although this is a significant change from the present value, it does not seem as unrealistic when considered in these terms. Efforts to educate non-smokers about the dangers of smoking may not be

the most effective solution if used alone, but they are worthwhile if used in combination with other educational efforts.

7.4 Varying ϕ_1 and ϕ_2

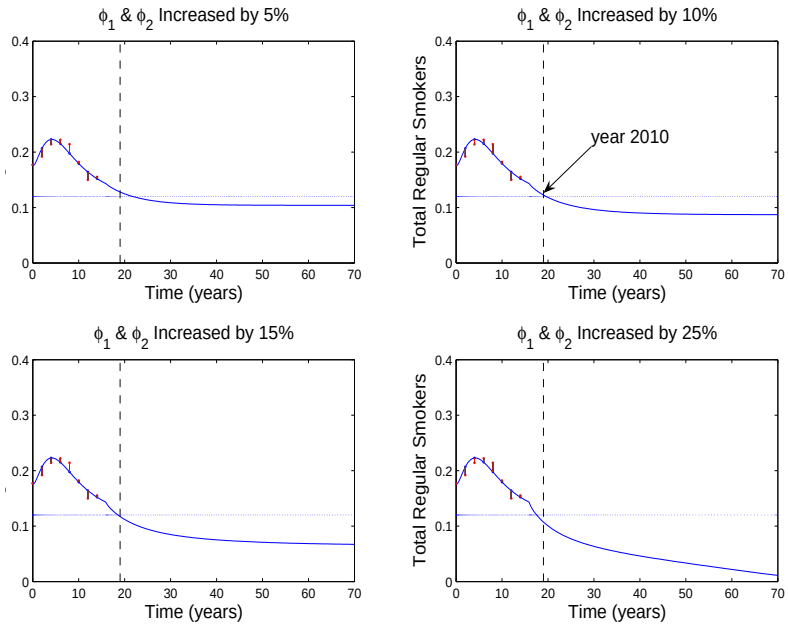

Figure 11: Predictions when ϕ_1 and ϕ_2 are varied

Increasing the rate at which adolescents quit smoking (ϕ_1, ϕ_2) is essential to the reduction of teen smoking. One motivation for a smoker to quit is understanding the effects that smoking has on their body. This understanding can be increased by encouraging smokers to enroll in educational programs and by increasing advertising targeted at adolescents that focuses on the effects and dangers of smoking.

Small changes in the quit rates reduce the number of adolescent smokers very quickly at first, but do not cause significant long-term changes. The CDC's 2010 goal can be met with only a 10% increase in these rates, however, the population of adolescent smokers

declines very slowly after this and approaches an endemic equilibrium of about 9%. This slow decline may be because the high relapse rates are causing these individuals to cycle back into the smoking classes. Increasing the quit rates by 25% is sufficient to eventually eliminate adolescent smoking, although it would take over 50 years to do so.

By targeting quit rates as mentioned in the CDC's plan, their 2010 goal is feasible. However, increasing the quit rates just enough to meet the goal is not enough to eliminate adolescent smoking because many individuals who quit will eventually relapse.

7.5 Varying α_1, α_2

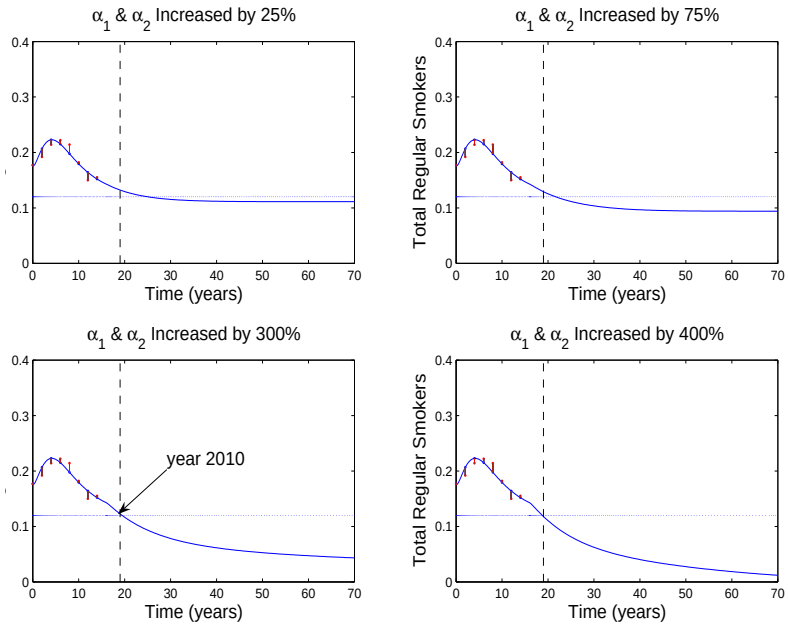

Figure 12: Predictions when α_1 and α_2 are varied

The percentage of smokers who are able to quit successfully each year is under 10% [2, 6]. With such low success rates, most people who quit smoking remain susceptible to relapse. Reducing the number of individuals in the Q_1 and Q_2 classes by increasing

the rates at which they permanently abstain from smoking (α_1, α_2), will decrease the percentage of smokers.

Individuals who have quit are more likely to stick with their decision not to smoke if they interact less with smokers, because relapse is a result of social interactions. One way to encourage this is to create after-school programs that help adolescents who are trying to quit permanently. However, as we can see from Figure 12, focusing our efforts on increasing α_1 and α_2 may not be the most effective solution. A 75% increase in α_1 and α_2 only decreases the current percentage of smokers to 9%. A 300% increase to these rates is necessary to meet the CDC's 2010 goal, and a 400% increase would be needed to eventually eliminate adolescent smoking.

7.6 Varying ϕ_1, ϕ_2, ε_1, and ε_2

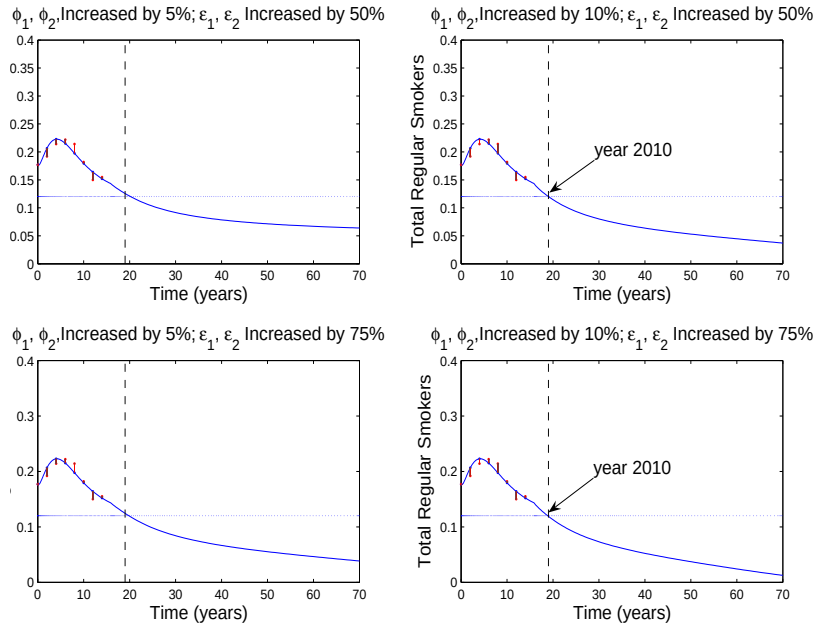

Figure 13: Predictions when ϕ_1, ϕ_2, ε_1, and ε_2 are varied

We analyzed numerical solutions when combinations of parameters were varied. Targeting the rates at which people quit as well as the rates at which non-smokers permanently abstain from smoking causes both short term and long term reductions in the percentage of smokers, as shown in Figure 13. Figure 11 shows that decreasing ϕ_1 and ϕ_2 by 10% will cause an immediate decrease in the number of smokers but the system also soon reaches equilibrium (this is a short term solution). However, decreasing ϵ_1 and ϵ_2 by 75% causes a gradual but prolonged decrease in the number of smokers (this is a long term solution).

To analyze the impact on the total number of smokers that a combination of short and long term solutions have we increase ϕ_1 and ϕ_2 by 10% and ε_1 and ε_2 by 75%. The results not only meets the CDC's goal for 2010, but also completely eliminates adolescent smoking in just over 50 years.

7.7 Varying ϕ_1, ϕ_2, ψ_1, ψ_2, ε_1, and ε_2

Varying ϕ_1, ϕ_2, ψ_1, ψ_2, ε_1, and ε_2 was most effective in meeting the CDC's 2010 goal with minimum change. Quit and relapse rates could be targeted through after-school programs. These programs would encourage adolescents to quit smoking and reduce relapse rates by removing individuals who have quit from an environment of smokers.

From Figure 14 we observe that a small change in all six parameters quickly causes a significant decrease in the percentage of adolescent smokers. By decreasing ψ_1 and ψ_2, increasing ϕ_1 and ϕ_2 by 10% each, and increasing ε_1 and ε_2 by 50%, adolescent smoking will reach 12% by 2010 and will continue to decrease towards a smoking free equilibrium.

8 Results and conclusions

We proposed a mathematical model to describe the dynamics of cigarette smoking among adolescents between the ages of 11 and 18. Through a simple bifurcation analysis we

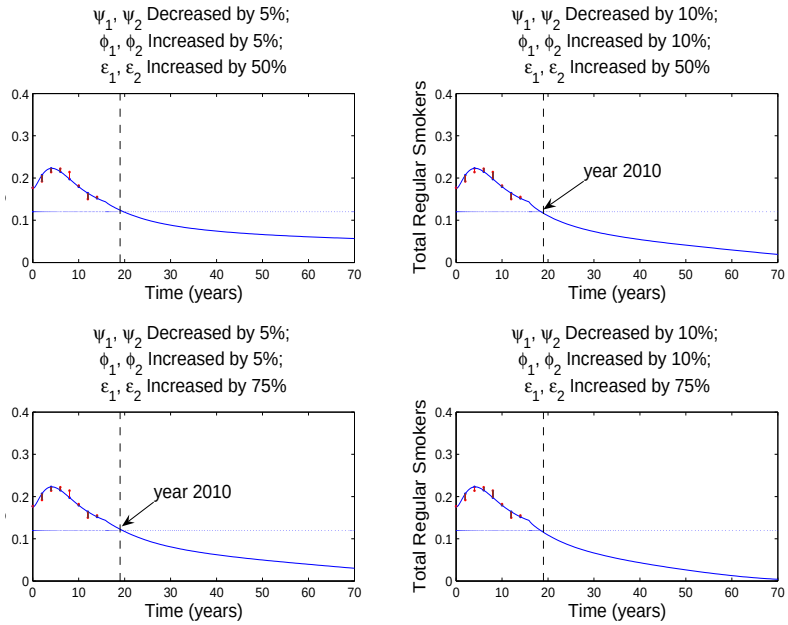

Figure 14: Predictions when ϕ_1, ϕ_2, ψ_1, ψ_2, ε_1, and ε_2 are varied

investigated the significance of relapse rates on current and future trends in cigarette smoking among adolescents. The model produced a good fit to CDC data. The most effective methods of education were determined numerically by varying parameter values and analyzing resulting solutions. We analyzed the CDC's 2010 goals and considered the role of the "sleeper effect" on the dynamics of the system.

We proved analytically that for relapse rates large enough the system supports a backward bifurcation. Numerical solutions that fit CDC data suggest $R_0 < 1$. This R_0 was confirmed by parameter estimation and numerical uncertainty analysis. The same fit to the data indicates that the population of adolescent smokers is approaching an endemic equilibrium. This means that the dynamics of adolescent smoking are currently in or near a region where the population of smokers is maintained by high relapse rates.

This knowledge is essential to choosing the best educational approach. Enough smokers

are quitting each year to eliminate smoking among adolescents. However, adolescents who have quit smoking are not successful at remaining smoke free [1, 9]. Programs designed to help individuals quit may make it harder to eradicate adolescent smoking if the rate at which these individuals relapse is high, because a larger reduction of R_0 will be required in order to approach the SFE. We recommend increasing the number of programs that provide follow-up resources to help ex-smokers permanently quit. The best strategy for accomplishing this is to stress change in social environments because individuals who have quit are more likely to relapse if they continue to associate themselves with smokers.

Consistent with CDC data [16], numerical solutions of our system indicate that current smoking among children ages 11 to 18 has been decreasing since 1997 and will reach 14% by 2010 and settle at an endemic equilibrium of about 12% in another 10 years. Thus, in order to reach 12% by 2010 to meet the CDC's goal, changes in current educational programs must be made. We investigated the effects of implementing various educational programs starting in the year 2007. Solutions highlight which parameters can be realistically changed through education in order to most effectively reduce the percentage of adolescents who smoke.

Numerical solutions indicated that ϕ_1, ϕ_2, ψ_1, and ψ_2 are the parameters that can most quickly reduce the percentage of teen smokers when targeted by education. The other parameters can also significantly influence the numerical solution, but changes occur more slowly at first. Focusing resources on helping individuals to permanently quit smoking would be most beneficial in order to meet the CDC's goal.

We considered focusing educational programs on elementary school children in order to minimize the damage caused by the "sleeper effect." Reducing the proportion of children who smoke before age 11 helps reduce the number of adolescent smokers; however, large decreases in r are necessary in order to cause significant change. Thus we conclude that the "sleeper effect" is not the most influential factor affecting adolescent smoking. Elementary

school anti-smoking programs are not sufficient to eradicate smoking without significant effort at the middle and high school levels.

The CDC's goal for 2010 is feasible, but changes must be made to their current plan. Their public health plan uses surveillance systems and laboratory research to identify causes of adolescent smoking. Using this information they design programs to prevent and reduce smoking. The CDC has created "Guidelines for School Health Programs to Prevent Tobacco Use and Addiction" as well as anti-smoking curricula which has proven effective in reducing or delaying adolescent smoking. However, the help of national, state, and local agencies must be enlisted in order to get these programs implemented, because few schools are currently using them [22].

It is important to note that the CDC's plan focuses primarily on smoking prevention and reduction instead of relapse rates. Our results suggest that it may be more effective to also increase the number of educational programs that specifically target reducing relapse without neglecting existing efforts to increase quit rates and decrease the number of new smokers. Thus, in addition to the CDC's current plan, we recommend that they test and implement programs that encourage adolescents who have quit smoking to remain smoke-free.

The current smoker population will soon reach an endemic equilibrium unless the CDC and other anti-smoking agencies begin targeting the high relapse rates. A feasible change for the CDC to meet their 2010 goal is to decrease relapse and increase the quit rates by 10% while increasing the rate at which at-risk youth chose to abstain from smoking by 50%. With these changes the total percentage of adolescent smokers will steadily decrease and may eventually approach a smoker free equilibrium.

9 Acknowledgments

The Mathematical & Theoretical Biology Institute would like to thank the following grant sources for the Summer 2006 REU program: The National Science Foundation (NSF), award number DMS-0502349. The Alfred P. Sloan Foundation (Sloan). The National Security Agency (NSA), award number H98230-06-1-0097 and Arizona State University.

We would like to take the time to thank the institutions and individuals that made our research possible. A special thanks to Dr. Carlos Castillo-Chavez. Thank you to the Mathematical and Theoretical Biology Institute and Arizona State University.

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10 Appendix

10.1 Calculations of the Basic Reproductive Number

To determine R_0 we must first determine the smoking-free equilibrium (SFE) of the system. The SFE is:

$$({S_1}^*, {S_2}^*, {N_1}^*, {N_2}^*, {Q_1}^*, {Q_2}^*, P^*) = \left(0, 0, \frac{\mu r}{\varepsilon_1 + \mu}, \frac{\mu\,(1-r)}{\varepsilon_2 + \mu}, 0, 0, \frac{\varepsilon_1 r}{\varepsilon_1 + \mu} + \frac{\varepsilon_2\,(1-r)}{\varepsilon_2 + \mu}\right).$$

We only look at the smoking classes to find R_0 [Cite Baojun's reference, which he has not given us]. Let,

$$\begin{aligned} f_1 = \frac{dS_1}{dt} &= \beta_1 N_1 \frac{S_1 + S_2}{T} + \psi_1 Q_1 \frac{S_1 + S_2}{T} - (\phi_1 + \mu)\, S_1, \\ f_2 = \frac{dS_2}{dt} &= \beta_2 N_2 \frac{S_1 + S_2}{T} + \psi_2 Q_2 \frac{S_1 + S_2}{T} - (\phi_2 + \mu)\, S_2, \end{aligned}$$

and,

$$H = \begin{bmatrix} \beta_1 N_1 \frac{S_1+S_2}{T} \\ \beta_2 N_2 \frac{S_1+S_2}{T} \end{bmatrix}; A = \begin{bmatrix} -\psi_1 Q_1 \frac{S_1+S_2}{T} + (\phi_1 + \mu)\, S_1 \\ -\psi_2 Q_2 \frac{S_1+S_2}{T} + (\phi_2 + \mu)\, S_2 \end{bmatrix},$$

here H is composed of all rates of new smokers in S_1 and S_2, and A is composed of all remaining rates.

$$F = \begin{bmatrix} \frac{\partial H_1}{\partial S_1} & \frac{\partial H_1}{\partial S_2} \\ \frac{\partial H_2}{\partial S_1} & \frac{\partial H_2}{\partial S_2} \end{bmatrix} = \begin{bmatrix} \frac{\beta_1 \mu r}{\varepsilon_1 + \mu} & \frac{\beta_1 \mu r}{\varepsilon_1 + \mu} \\ \frac{\beta_2 \mu (1-r)}{\varepsilon_2 + \mu} & \frac{\beta_2 \mu (1-r)}{\varepsilon_2 + \mu} \end{bmatrix}.$$

Likewise,

$$V = \begin{bmatrix} \frac{\partial A_1}{\partial S_1} & \frac{\partial A_1}{\partial S_2} \\ \frac{\partial A_2}{\partial S_1} & \frac{\partial A_2}{\partial S_2} \end{bmatrix} = \begin{bmatrix} \phi_1 + \mu & 0 \\ 0 & \phi_2 + \mu \end{bmatrix} \Rightarrow V^{-1} = \begin{bmatrix} \frac{1}{\phi_1+\mu} & 0 \\ 0 & \frac{1}{\phi_2+\mu} \end{bmatrix},$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta_1\mu r}{(\varepsilon_1+\mu)(\phi_1+\mu)} & \frac{\beta_1\mu r}{(\varepsilon_1+\mu)(\phi_2+\mu)} \\ \frac{\beta_2\mu(1-r)}{(\varepsilon_2+\mu)(\phi_1+\mu)} & \frac{\beta_2\mu(1-r)}{(\varepsilon_2+\mu)(\phi_2+\mu)} \end{bmatrix}.$$

R_0 is the dominant eigenvalue of FV^{-1}.

$$R_0 = \frac{\beta_1\mu r}{(\varepsilon_1 + \mu)(\phi_1 + \mu)} + \frac{\beta_2\mu(1-r)}{(\varepsilon_2 + \mu)(\phi_2 + \mu)}.$$

10.2 Proof of Theorem 1

Proof. For analytical purposes we re-scale the system of equations with the following substitutions:

$$\frac{N_1}{T} = x_1, \frac{N_2}{T} = x_2, \frac{S_1}{T} = x_3, \frac{S_2}{T} = x_4, \frac{Q_1}{T} = x_5, \frac{Q_2}{T} = x_6.$$

The rescaled system of differential equations is as follows:

$$\frac{dx_1}{dt} = \mu r - \beta_1 x_1 (x_3 + x_4) - (\varepsilon_1 + \mu) x_1 := f_1 \tag{11}$$
$$\frac{dx_2}{dt} = \mu (1 - r) - \beta_2 x_2 (x_3 + x_4) - (\varepsilon_2 + \mu) x_2 := f_2 \tag{12}$$
$$\frac{dx_3}{dt} = \beta_1 x_1 (x_3 + x_4) + \psi_1 x_5 (x_3 + x_4) - (\phi_1 + \mu) x_3 := f_3 \tag{13}$$
$$\frac{dx_4}{dt} = \beta_2 x_2 (x_3 + x_4) + \psi_2 x_6 (x_3 + x_4) - (\phi_2 + \mu) x_4 := f_4 \tag{14}$$
$$\frac{dx_5}{dt} = \phi_1 x_3 - \psi_1 x_5 (x_3 + x_4) - (\alpha_1 + \mu) x_5 := f_5 \tag{15}$$
$$\frac{dx_6}{dt} = \phi_2 x_4 - \psi_2 x_6 (x_3 + x_4) - (\alpha_2 + \mu) x_6 := f_6 \tag{16}$$

We analyzed the bifurcation of our model in order to better understand the dynamics of our system. Using equations (11-16) we determine the conditions for backward bifurcation [25].

We chose β_1 as the bifurcation parameter. We set $R_0 = 1$,

$$1 = \frac{\beta_1 \mu r}{(\varepsilon_1 + \mu)(\phi + \mu)} + \frac{\beta_2 \mu (1-r)}{(\varepsilon_2 + \mu)(\phi_2 + \mu)} \tag{17}$$

and solve for β_1 to obtain

$$\beta_1 = \beta_1^* = \frac{(\varepsilon_1 + \mu)(\phi_1 + \mu)\left[(\varepsilon_2 + \mu)(\phi_2 + \mu) - \beta_2 \mu (1-r)\right]}{\mu r (\varepsilon_2 + \mu)(\phi_2 + \mu)}. \tag{18}$$

We then look at the Jacobian of the system evaluated at the SFE and $\beta_1 = \beta_1^*$

$$J = \begin{bmatrix} -(\varepsilon_1 + \mu) & 0 & -\frac{\beta_1^* \mu r}{\varepsilon_1 + \mu} & -\frac{\beta_1^* \mu r}{\varepsilon_1 + \mu} & 0 & 0 \\ 0 & -(\varepsilon_2 + \mu) & -\frac{\beta_2 \mu (1-r)}{\varepsilon_2 + \mu} & -\frac{\beta_2 \mu (1-r)}{\varepsilon_2 + \mu} & 0 & 0 \\ 0 & 0 & \frac{\beta_1^* \mu r}{\varepsilon_1 + \mu} - (\phi_1 + \mu) & \frac{\beta_1^* \mu r}{\varepsilon_1 + \mu} & 0 & 0 \\ 0 & 0 & \frac{\beta_2 \mu (1-r)}{\varepsilon_2 + \mu} & \frac{\beta_2 \mu (1-r)}{\varepsilon_2 + \mu} - (\phi_2 + \mu) & 0 & 0 \\ 0 & 0 & \phi_1 & 0 & -(\alpha_1 + \mu) & 0 \\ 0 & 0 & 0 & \phi_2 & 0 & -(\alpha_2 + \mu) \end{bmatrix}$$

A right eigenvector of J for the eigenvalue $\lambda = 0$ is

$$w = \begin{bmatrix} w_1 & w_2 & w_3 & w_4 & w_5 & w_6 \end{bmatrix}^T.$$

where,

$$
\begin{aligned}
w_1 &= -\frac{\beta_1^* r(\phi_2+\mu)(\varepsilon_2+\mu)}{\beta_2(1-r)(\varepsilon_1+\mu)^2} \\
w_2 &= -\frac{\phi_2+\mu}{\varepsilon_2+\mu} \\
w_3 &= \frac{(\phi_2+\mu)(\varepsilon_2+\mu)}{\beta_2(1-r)\mu} - 1 \\
w_4 &= 1 \\
w_5 &= \frac{\phi_1\left((\phi_2+\mu)(\varepsilon_2+\mu) - \beta_2\mu(1-r)\right)}{\beta_2\mu(1-r)(\alpha_1+\mu)} \\
w_6 &= \frac{\phi_2}{\alpha_2+\mu}
\end{aligned}
$$

and a left eigenvector is

$$
v = \begin{bmatrix} v_1 & v_2 & v_3 & v_4 & v_5 & v_6 \end{bmatrix} = \begin{bmatrix} 0 & 0 & \frac{\phi_2+\mu}{\phi_1+\mu} & 1 & 0 & 0 \end{bmatrix}.
$$

Recall that

$$
a = \sum_k \sum_{i,j} \frac{\partial^2 f_k}{\partial x_i \partial x_j} w_i w_j v_k \Bigg|_{(SFE,\ \beta_1=\beta_1^*)}. \tag{19}
$$

We compute the non-zero partial derivatives appearing in equation (19) to be the following,

$$
\begin{aligned}
&\frac{\partial^2 f_3}{\partial x_1 \partial x_3} = \beta_1^*; \quad \frac{\partial^2 f_3}{\partial x_1 \partial x_4} = \beta_1^*; \quad \frac{\partial^2 f_3}{\partial x_5 \partial x_3} = \psi_1; \quad \frac{\partial^2 f_3}{\partial x_5 \partial x_4} = \psi_1; \\
&\frac{\partial^2 f_4}{\partial x_2 \partial x_3} = \beta_2; \quad \frac{\partial^2 f_4}{\partial x_2 \partial x_4} = \beta_2; \quad \frac{\partial^2 f_4}{\partial x_6 \partial x_3} = \psi_2; \quad \frac{\partial^2 f_4}{\partial x_6 \partial x_4} = \psi_2.
\end{aligned}
$$

Expanding equation (19) we get the following,

$$a = 2(w_3 + w_4)(\beta_1^* w_1 v_3 + \psi_1 w_5 v_3 + \beta_2 w_2 v_4 + \psi_2 w_6 v_4).$$

Since $2(w_3 + w_4)$ is always positive we now consider the sign of the rest of the expression. Thus our system exhibits backward bifurcation when

$$(\beta_1^* w_1 v_3 + \psi_1 w_5 v_3 + \beta_2 w_2 v_4 + \psi_2 w_6 v_4) > 0. \tag{20}$$

Let

$$A = w_5 v_3; \quad B = w_6 v_4; \quad C = -(\beta_1^* w_1 v_3 + \beta_2 w_2 v_4)$$
$$m = \frac{C}{A}; \quad n = \frac{C}{B}.$$

Equation (20) is true when ψ_1, ψ_2 large enough such that

$$\frac{\psi_1}{m} + \frac{\psi_2}{n} > 1.$$

Thus we have a backward bifurcation for large enough values of ψ_1 and ψ_2. □

10.3 Proof of Theorem 2

Proof. To prove existence of endemic equilibrium when $R_0 > 1$, in the case of $\beta_1 = \beta_2$, $\phi_1 = \phi_2$, $\psi_1 = \psi_2$, $\varepsilon_1 = \varepsilon_2$, $\alpha_1 = \alpha_2$, and we let $r = 1$.

$$0 = \mu T - \beta N \frac{S}{T} - (\varepsilon + \mu) N \tag{21}$$

$$0 = \beta N \frac{S}{T} + \psi Q \frac{S}{T} - (\phi + \mu) S \tag{22}$$

$$0 = \phi S - \psi Q \frac{S}{T} - (\alpha + \mu) Q \tag{23}$$

$$0 = \varepsilon N + \alpha Q - \mu P \tag{24}$$

From Equation (21),

$$\begin{aligned} 0 &= \mu - \beta N S - (\varepsilon + \mu) N \\ \mu &= N \left[\beta S + (\varepsilon + \mu)\right] \\ N &= \frac{\mu}{\beta S + (\varepsilon + \mu)}. \end{aligned} \tag{25}$$

From Equation (22),

$$\begin{aligned} 0 &= \beta N S + \psi Q S - (\phi + \mu) S \\ 0 &= S \left[\beta N + \psi Q - (\phi + \mu)\right]. \end{aligned}$$

Let $S \neq 0$ and $0 = [\beta N + \psi Q - (\phi + \mu)]$. Then,

$$N = \frac{(\phi + \mu) - \psi Q}{\beta}. \tag{26}$$

Equating Equations (25) and (26) we obtain,

$$\frac{(\phi+\mu)-\psi Q}{\beta} = \frac{\mu}{\beta S+(\varepsilon+\mu)}. \tag{27}$$

From Equation (23),

$$\begin{aligned} 0 &= \phi S - \psi Q S - (\alpha+\mu)Q \\ \phi S &= Q[\psi S + (\alpha+\mu)] \\ Q &= \frac{\phi S}{\psi S+(\alpha+\mu)}. \end{aligned}$$

From Equation (27),

$$\begin{aligned} \beta S+(\varepsilon+\mu) &= \frac{\beta\mu}{(\phi+\mu)-\psi Q} \\ &= \frac{\beta\mu}{(\phi+\mu)-\frac{\psi\phi S}{\psi S+(\alpha+\mu)}} \\ &= \frac{\beta\mu[\psi S+(\alpha+\mu)]}{(\phi+\mu)[\psi S+(\alpha+\mu)]-\psi\phi S} \\ &= \frac{\beta\mu[\psi S+(\alpha+\mu)]}{(\phi+\mu)\psi S+(\phi+\mu)(\alpha+\mu)-\psi\phi S} \end{aligned}$$

$$\beta S+(\varepsilon+\mu) = \frac{\beta\mu[\psi S+(\alpha+\mu)]}{\mu\psi S+(\phi+\mu)(\alpha+\mu)} \tag{28}$$

We make the following substitutions,

$$a=(\varepsilon+\mu),\ b=(\phi+\mu),\ c=(\alpha+\mu).$$

Rewriting Equation (28),

$$(\beta S + a)(\mu\psi S + bc) = \beta\mu(\psi S + c)$$
$$0 = \beta\mu\psi S^2 + [\beta bc + a\mu\psi - \beta\mu\psi]S + abc - \beta\mu c. \tag{29}$$

Let Equation 29 equal $F(S)$. Therefore,

$$F(S) = \beta\mu\psi S^2 + [\beta bc + a\mu\psi - \beta\mu\psi]S + abc - \beta\mu c$$
$$= \beta\mu\psi S^2 + [\beta bc + a\mu\psi - \beta\mu\psi]S + abc(1 - R_0) \tag{30}$$
$$= AS^2 + BS + C. \tag{31}$$

For $R_0 > 1$

$$C = abc(1 - R_0) < 0.$$

From the Quadratic Theorem,

$$z_1,\ z_2 = \frac{C}{A},$$

where

$$A = \beta\mu\psi,\ B = \beta bc + a\mu\psi - \beta\mu\psi,\ C = abc(1 - R_0).$$

Since $C < 0$ and $A > 0$, $\frac{C}{A} < 0$.

Thus there exists exactly one positive endemic equilibrium when $R_0 > 1$.

□