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INTERVENTION STRATEGY FOR REDUCING ADOLESCENT SMOKING

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ABSTRACT. This study aims to establish and analyze a mathematical model for the transmission dynamics of male adolescent smoking and to determine an optimal control strategy to reduce male adolescent smoking. We consider three groups in the population: smokers, non-smokers, and temporary nonsmokers. In our model to which optimal control theory was applied, the number of smokers decreased sharply and the number of non-smokers increased significantly. Our simulation results under various control scenarios reveal that integrated control measures(such as prevention, education, and treatment) may be necessary to reduce the growth rate of adolescent smoking. Moreover, we concluded that efforts to encourage current smokers and temporary quitters to quit should be sustained longer than efforts to reduce the rate at which nonsmokers become smokers through smoking prevention education.

1. INTRODUCTION

Tobacco smoking remains a major public health menace globally. It inflicts significant mortality and costs billions of dollars in health care. Many adult smokers have problems, but the number of adolescent smokers is also a severe problem.

The survey results on how and why adolescents start smoking are similar worldwide. The biggest reason is peer pressure to smoke in adolescence. Adolescent smoking is more harmful to health than smoking for adults. Further, adolescents fall deeper into nicotine addiction than those who start smoking in adulthood. Accepting smoking in adolescence is likely to lead to the next stage of juvenile crime. Finally, smoking has a severe influence on the emotional and social aspects of adolescents. Factors influencing smoking initiation in adolescents include sociocultural factors, socioenvironmental factors, abilities and skills, and psychological factors [1]. Peer group cohesion is associated with smoking initiation in the teenage years [2]. Also,

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in study [3], they showed social factors such as drinking, and violence were highly related to smoking based on the ecological model. In some studies, peer influence is greater than parental influence.

Encouragement from friends accounted for a high proportion of the opportunities for teenagers to start smoking. In other words, smoking is contagious among friends due to maintaining friendships and pressure from peer groups, and curiosity about tobacco and the characteristics of adolescence further encourages it. The adolescent smoking rate in the group where friends smoked was statistically significantly higher than in the group where friends did not smoke [4]. Smoking among physically and mentally immature adolescents has negative consequences for adolescent development, including stunted growth, memory loss, and reduced learning ability. In addition, the incidence of smoking-related diseases is significantly higher than among adolescent smokers. Furthermore, this behavior is likely to lead to other delinquent behaviors, such as drug use, making it a social and national problem worldwide.

Teenagers are more likely to share smoking and drinking habits with their peers when they attend schools with a relatively substantial number of students who use tobacco or alcohol, according to a study. Teenagers had a high smoking rate when a close friend smoked [4]. Such schools provide increased opportunities for teens susceptible to drinking and smoking to choose friends who share their interests, according to [5].

According to an annual survey of youth health behavior conducted by the Ministry of Education and the Korean Disease Control and Prevention Agency, the smoking rate of male high school students decreased to 23.1% in 2011, 22.4% in 2012, 20.7% in 2013, 20.8% in 2014, 18.3% in 2015, 14.7% in 2016, 13.9% in 2017, 14.1% in 2018, 14.2% in 2019, 10.1% in 2020, and 10% in 2021. However, as of 2021, the overall adolescent smoking rate is 4.5%, and the youth smoking rate is 6.0% for boys and 2.9% for girls. For both boys and girls, the percentage of smokers has increased over the past decade as they move up through the grades from 7th to 12th grade [6]. The age at which they start smoking has been increasingly younger. There are even students who started smoking in elementary school.

This paper analyzed a model mathematically modeled on adolescent smoking based on the fact that adolescent smoking is contagious.

2. Mathematical Model

2.1. **Basic Model.** In recent years, mathematical models of smoking have been developed ([7, 8, 9, 10, 11]). The mathematical models apply the basic SIR model. In a SIR epidemic model, susceptible individuals S become (smokers) infected I but develop immunity (nonsmokers) and enter the immune class R. We assume that there is no population change.

The adolescent population has an influx of new students every year, and a portion of the new students also smoke. The inflow and outflow are the same. If a member of the nonsmoking group (N) is encouraged to smoke by contact with the smoking group (S) and starts smoking, they eventually move to the smoking group (S)(Table 2.1). There is no direct migration from the smoking group (S) to the nonsmoking group (N), but some of them move to the temporarily quitting group $(1-p)$, and some of them move to the steady quitting group p.

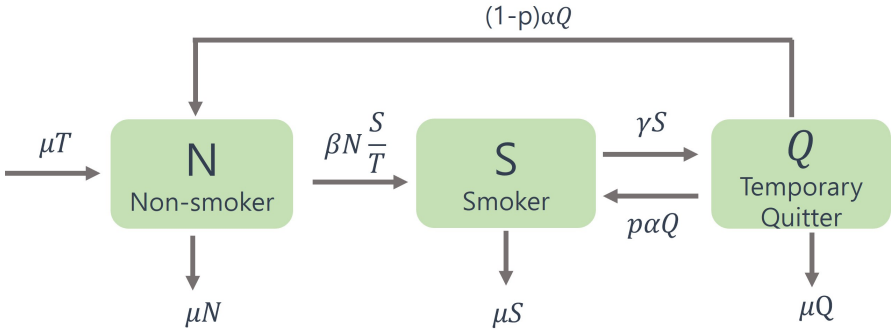

FIGURE 1. Flow chart representing flows between groups, and values representing flow rates.

The model is organized as the dynamics of the three subpopulations of potential nonsmokers $N(t)$, smokers $S(t)$, and smokers who temporarily quit smoking $Q(t)$. Thus, the total population at time t is given by $T(t) = N(t) + S(t) + Q(t)$. We assume that all state variables and parameters are nonnegative for all $0 \leq t$.

As illustrated, in Fig. 1, the dynamical model is as follows:

$$\begin{aligned}
\frac{dN(t)}{dt} &= \mu T(t) - \beta \frac{N(t)S(t)}{T(t)} + (1-p)\alpha Q(t) - \mu N(t) \\
\frac{dS(t)}{dt} &= \beta \frac{N(t)S(t)}{T(t)} + p\alpha Q(t) - \gamma S(t) - \mu S(t) \\
\frac{dQ(t)}{dt} &= \gamma S(t) - \alpha Q(t) - \mu Q(t)
\end{aligned} \tag{2.1}$$

where, $T(t) = N(t)+S(t)+Q(t)$. The parameter μ is the natural inflow and outflow rate in the system, it refers to students entering in the 7th grade and students graduating in the 12th grade in this system. The β represents the transmission rate, however, this means that it includes not only transmission by family and peer group but also personal curiosity and interest. The parameter γ denotes the quitting rate from smoking. The parameters are positive constants.

2.2. **Optimal Control Model.** Measures to reduce smoking among adolescents include prevention and treatment. Prevention is the method of educating students who do not smoke to

TABLE 1. State variables for the smoking model.

State	Description
$N(t)$	No. of adolescent nonsmokers at time t
$S(t)$	No. of adolescent smokers at time t
$Q(t)$	No. of adolescents who temporarily quit smoking at time t

prevent them from smoking in the future. In contrast, treatment is the method of educating students who already smoke to quit smoking. We considered three control strategies to reduce the number of smokers. The first is to reduce the uptake of smoking (i.e., the rate of nonsmoking adolescents moving to the smoking group) and the rate of adolescents who quit smoking and revert to smoking again. The second is to increase the number of those who quit, which is the rate of adolescents who quit smoking for more than 6 months and transition to the nonsmoking group. The mathematical model of smoking adolescents with the control term, considering the above control strategy, is as follows:

$$\begin{aligned}
\frac{dN(t)}{dt} &= \mu T(t) - \beta(1-u_1(t))\frac{N(t)S(t)}{T(t)} + (1-p)\alpha Q(t) + \alpha p u_3(t) Q(t) - \mu N(t), \\
\frac{dS(t)}{dt} &= \beta(1-u_1(t))\frac{N(t)S(t)}{T(t)} + \alpha p(1-u_3(t))Q(t) - \gamma S(t) \\
&\quad -\rho u_2(t) S(t) - \mu S(t) \\
\frac{dQ(t)}{dt} &= \gamma S(t) + \rho u_2(t) S(t) - \alpha Q(t) - \mu Q(t),
\end{aligned} \tag{2.2}$$

where $T(t) = N(t) + S(t) + Q(t)$.

To control, variables u_1, u_2, and u_3 represent control measures for reducing smokers with respect to β, γ, and p, respectively. The factor of $1 - u_1(t)$ reduces the per capita transition rate β from $N(t)$ to $S(t)$. The control variable $u_2(t)$ represents efforts to increase the per capita transition rate γ from $S(t)$ to $Q(t)$. It is assumed that the quitting rate increases at a rate proportional to $u_2(t)$ and where $\rho > 0$ is a rate constant. We should make efforts to permanently quit smoking rather than temporarily quitting smoking. The amount of effort to increase the rate at which students in the temporary smoking cessation group move to the permanent smoking cessation group is $u_3(t)$. To reduce smoking, counseling, family support, and education/campaigns are provided for the students.

TABLE 2. Parameter description and variables for the smoking model.

Parameter	Description	Value	Ref.
α	Leave rate from temporarily quitting smoking per unit of time	$1/6$	6 month
β	Rate of smoking transmission due to social factors contact per unit of time	0.003	Assumed
γ	Rate of transition from smoking to temporarily quitting smoking per unit of time	0.046	Assumed
μ	Leave rate from each subpopulation	$1/72$	6 years
p	Percentage of adolescents returning to smoking among those leaving the temporarily quitting group per unit of time	0.8	[12, 13]

We define the control set as follows:

$$\mathcal{U} = \{(u_1, u_2, u_3) | u_i(t) \text{ which are Lebesgue measurable on } [0, t_f],\ 0 \leq u_{i|i=1,2,3}(t) < 1\},$$

the set of admissible controls, and t_f is the final time.

We focus on the optimal control problem, minimizing the rate of smoking transmission due to social contact and the leave rate from those temporarily quitting smoking. Thus, the smoking problem is reduced to minimizing the cost functional. To specify the cost, we define the cost functional as follows:

$$J(u_1, u_2, u_3) = \int_0^{t_f} \left\{ AS(t) + \frac{B_1}{2} u_1^2(t) + \frac{B_2}{2} u_2^2(t) + \frac{B_3}{2} u_3^2(t) \right\} dt \tag{2.3}$$

subject to the differential equations (2.2). In the objective functional, the quantities A, B_1, B_2, and B_3 represent the weight constants. The costs associated with the controls of the transition rates are described by the terms $\frac{B_1}{2}u_1^2$, $\frac{B_2}{2}u_2^2$, and $\frac{B_3}{2}u_3^2$. We assume the costs are proportional to the square of the corresponding control function.

3. Mathematical Analysis

3.1. **Basic Reproductive Number.** The basic reproductive number can classify the dynamic behavior of the model. This threshold condition determines whether an infectious disease spreads in a susceptible population when the disease is introduced into the population [14]. The basic reproductive number $\mathcal{R}_0$ is a measure of the average number of secondary cases generated by a single primary case in a completely susceptible population [15]. From this viewpoint, van den Driessche and Watmough performed excellent work [14]. We adapted their method to determine the basic reproduction ratio $\mathcal{R}_0$.

We let $x = (x_1, ..., x_n)^t$, with each $x_k \geq 0$, be the number of individuals in each group. We define X_s such that $X_s = \{x \geq 0 \mid x_k = 0,\ k = 1, \ldots, n\}$. We let $\mathcal{F}_k(x)$ be the rate of appearance of new infections in group k and $\mathcal{V}_k^+(x)$ be the rate of transfer of individual into group k by all other means and $\mathcal{V}_k^-(x)$ be the rate of transfer of individuals out of group k. The disease transmission model consists of nonnegative initial conditions with the following system of equations:

$$\frac{x_k}{dt} = f_k(x) = \mathcal{F}_k(x) - \mathcal{V}_k(x), \quad k = 1, ..., n,$$

where $\mathcal{V}_k = \mathcal{V}_k^+ - \mathcal{V}_k^-$.

The threshold is calculated using the next-generation method:

$$\mathcal{R}_0 = \rho(\mathcal{F}\mathcal{V}^{-1}),$$

where ρ is defined as the spectral radius of the next-generation matrix $\mathcal{F}\mathcal{V}^{-1}$, $\mathcal{F}$ is the rate of appearance of new infections in class i, and $\mathcal{V}$ is the transfer of individuals out of class i by all other means [14].

A more general basic reproduction ratio can be defined as the average number of new infections produced by a typical ineffective individual in a population at a disease-free equilibrium

(DFE) $E_0 = (N, 0, 0)$. We obtain

$$\mathcal{R}_0 = \beta \frac{(\alpha + \mu)}{(\alpha + \mu)(\gamma + \mu) - p\alpha\gamma}.$$

The basic reproductive number $\mathcal{R}_0$ measures how quickly smokers spread in their initial phase and predicts whether smoking continues. For the stability of the system, the stability is as follows theorem.

Theorem 3.1. *The disease-free equilibrium E_0 of the basic model 2.1 is locally asymptotically stable if $\mathcal{R}_0 < 1$ and is unstable if $\mathcal{R}_0 > 1$.*

Here, we present the sensitivity analysis of the basic reproductive number $\mathcal{R}_0$:

$$\begin{aligned}
S_\beta &= \frac{\partial \mathcal{R}_0}{\partial \beta} \frac{\beta}{\mathcal{R}_0} = 1, \\
S_\gamma &= \frac{\partial \mathcal{R}_0}{\partial \gamma} \frac{\gamma}{\mathcal{R}_0} = -\gamma\{\mu + (1-p)\alpha\}, \\
S_p &= \frac{\partial \mathcal{R}_0}{\partial p} \frac{p}{\mathcal{R}_0} = \frac{p\alpha\gamma}{\mu(\alpha + \mu + \gamma) + (1-p)\alpha\gamma}.
\end{aligned}$$

Therefore, $S_\beta = 1$ indicates that the relative change in the value of $\mathcal{R}_0$ is the relative change in β, independent of any other parameter values. The positive sign implies that changes are produced in the same direction; that is, an increase or decrease in β produces an increase or decrease in $\mathcal{R}_0$. In contrast to S_β, the value of S_γ has a negative sign, implying that changes are produced in different directions; that is, an increase or decrease in γ produces a decrease or increase in $\mathcal{R}_0$. The sign value of S_p is positive when $p\alpha$ increases; thus, temporary smokers increase the number of students who smoke again.

3.2. **Existence of an Optimal Control.** We aim to determine the optimal solution that minimizes the number of smokers while using as little control as possible. We determine (u_1^*, u_2^*, u_3^*) with

$$J(u_1^*, u_2^*, u_3^*) = min\{J(u_1, u_2, u_3) | (u_1, u_2, u_3)\} \in \mathcal{U},$$

subject to the system of equations given by (2.2), where

$$\mathcal{U} = \{(u_1, u_2, u_3) | u_i(t) \text{ are Lebesgue measurable on } [0, t_f],\ 0 \leq u_{i|i=1,2,3}(t) < 1\}.$$

The necessary and sufficient conditions to be satisfied by the control and corresponding states are derived using Pontryagin's maximum principle [16]. To determine the adjoint equations and transversality conditions, we use the Hamiltonian [17] for $X(t) = (N(t), S(t), Q(t)), \mathcal{U} = (u_1, u_2, u_3)$ and $\mathbf{\Lambda}(t) = (\lambda_1(t), \lambda_2(t), \lambda_3(t))$.

Using the differential equation of the state variable of the model (2.2), we seek the minimal value. To do this, we defined the Hamiltonian $\mathcal{H}$ for the control problem as follows:

$$\begin{aligned}
&\mathcal{H}(\mathbf{X}(t),\mathcal{U}(t),\mathbf{\Lambda}(t)) \\
= \;& AS(t) + \frac{B_1}{2}u_1^2(t) + \frac{B_2}{2}u_2^2(t) + \frac{B_3}{2}u_3^2(t) + \mathbf{\Lambda}(t)\left(\frac{d\mathbf{X}(t)}{dt}\right)^T \\
= \;& AS(t) + \frac{B_1}{2}u_1^2(t) + \frac{B_2}{2}u_2^2(t) + \frac{B_3}{2}u_3^2(t) \\
+ \;& \lambda_1(t)\left\{\mu T(t) - \beta(1-u_1(t))\frac{N(t)S(t)}{T(t)} + (1-p)\alpha Q(t) + p\alpha u_3(t)Q(t) - \mu N(t)\right\} \\
+ \;& \lambda_2(t)\left\{\beta(1-u_1(t))\frac{N(t)S(t)}{T(t)} + p\alpha(1-u_3(t))Q(t) - (\gamma+\mu)S(t) - \rho u_2(t)S(t)\right\} \\
+ \;& \lambda_3(t)\left\{\gamma S(t) + \rho u_2(t)S(t) - (\alpha+\mu)Q(t)\right\}, \qquad (3.1)
\end{aligned}$$

where $\lambda_j (j = 1, 2, 3)$ are the adjoint variables, and the state variables for the population dynamics are denoted by $\mathbf{X}(t) = (N(t), S(t), Q(t))$, the existence of which is guaranteed by Pontryagin's maximum principle [17].

Now, we state and prove the following theorem.

Theorem 3.2. *We let $N(t), S(t)$, and $Q(t)$ be optimal state solutions with associated optimal control variables u_1^*, u_2^*, and u_3^* for the optimal control problem* (2.2) *and* (2.3)*. Then, an adjoint variable exists, such that $\mathbf{\Lambda}(t) = (\lambda_1(t), \lambda_2(t), \lambda_3(t))$ satisfying*

$$\lambda_1'(t) = (\lambda_1 - \lambda_2)\beta(1-u_1(t))\frac{S(t)}{T(t)}$$

$$\lambda_2'(t) = -A - \mu(\lambda_1(t) - \lambda_2(t)) + (\lambda_1(t) - \lambda_2(t))\beta(1-u_1(t))\frac{N(t)}{T(t)} + (\lambda_2(t) - \lambda_3(t))(\gamma + \rho u_2(t))$$

$$\lambda_3'(t) = -\mu\lambda_1(t) - (1-p)\alpha\lambda_1(t) - p\alpha u_3(t)\lambda_1(t)) - p\alpha(1-u_3(t))\lambda_2(t) + (\alpha+\mu)\lambda_3(t)$$

with transversality conditions (or boundary conditions)

$$\lambda_j(T) = 0, j = 1, 2, 3.$$

Furthermore, the optimal controls u_1^*, $u_2^*(t)$, and u_3^* are given by

$$\begin{aligned}
u_1^*(t) &= min\left\{1, max\left\{0, \frac{\beta(\lambda_2(t) - \lambda_1(t))N^*(t)S^*(t)}{B_1 T(t)}\right\}\right\}, \\
u_2^*(t) &= min\left\{1, max\left\{0, \frac{\rho(\lambda_2(t) - \lambda_3(t))S^*(t)}{B_2}\right\}\right\}, \qquad (3.2) \\
u_3^*(t) &= min\left\{1, max\left\{0, \frac{p\alpha(\lambda_2(t) - \lambda_1(t)Q^*(t)}{B_3}\right\}\right\}.
\end{aligned}$$

Proof. To determine the adjoint equations and transversality conditions, From setting $N(t) = N^*(t)$, $S(t) = S^*(t)$, and $Q(t) = Q^*(t)$, and we apply the Hamiltonian (3.1). By Pontryagin's

maximum principle, states $N(t)$, $S(t)$, and $Q(t)$ differentiate the Hamiltonian,

$$\lambda_1' = -\frac{\partial \mathcal{H}}{\partial N}, \quad \lambda_2' = -\frac{\partial \mathcal{H}}{\partial S}, \quad \lambda_3' = -\frac{\partial \mathcal{H}}{\partial Q},$$

then we obtain the costate equations;

$$\lambda_1'(t) = (\lambda_1 - \lambda_2)\beta(1 - u_1(t))\frac{S(t)}{T(t)}$$

$$\lambda_2'(t) = -A - \mu(\lambda_1(t) - \lambda_2(t)) + (\lambda_1(t) - \lambda_2(t))\beta(1 - u_1(t))\frac{N(t)}{T(t)} + (\lambda_2(t) - \lambda_3(t))(\gamma + \rho u_2(t))$$

$$\lambda_3'(t) = -\mu\lambda_1(t) - (1-p)\alpha\lambda_1(t) - p\alpha u_3(t)\lambda_1(t) - p\alpha(1 - u_3(t))\lambda_2(t) + (\alpha + \mu)\lambda_3(t)$$

To obtain the optimality condition (3.2), we also differentiate the Hamiltonian $\mathcal{H}$ with respect to u_1^*, u_2^*, and u_3^* and set each of them equal to zero:

$$\begin{aligned}
0 &= \frac{\partial \mathcal{H}}{\partial u_1} = B_1 u_1(t) + \beta(\lambda_1(t) - \lambda_2(t))\frac{N(t)S(t)}{T(t)}, \\
0 &= \frac{\partial \mathcal{H}}{\partial u_2} = B_2 u_2(t) - \rho(\lambda_2(t) - \lambda_3(t))S, \\
0 &= \frac{\partial \mathcal{H}}{\partial u_3} = B_3 u_3(t) + (\lambda_1(t) - \lambda_2(t))p\alpha Q(t).
\end{aligned}$$

We use a standard optimality technique to determine an explicit expression for the optimal controls for $0 \le u_i^* \le 1, i = 1, 2, 3$, considering the following three cases. On the set $\{t : 0 < u_1^* < 1\}$, we have $\frac{\partial \mathcal{H}}{\partial u_1} = 0$. The optimal control is

$$u_1^* = \frac{\beta(\lambda_2(t) - \lambda_1(t))N^*(t)S^*(t)}{B_1 T(t)}.$$

On the set $\{t : u_1^*(t) = 0\}$, we have $\frac{\partial \mathcal{H}}{\partial u_1} \ge 0$, which implies that

$$u_1^* = \frac{\beta(\lambda_2(t) - \lambda_1(t))N^*(t)S^*(t)}{B_1 T(t)} \ge 0.$$

On the set $\{t : u_1^* = 1\}$, we have $\frac{\partial \mathcal{H}}{\partial u_1} \le 0$, implying that

$$u_1^* = \frac{\beta(\lambda_2(t) - \lambda_1(t))N^*(t)S^*(t)}{B_1 T(t)} \le 0.$$

Combining these three cases determines the characterization of u_1^*:

$$u_1^* = min\left\{1, max\left\{0, \frac{\beta(\lambda_2(t) - \lambda_1(t))N^*(t)S^*(t)}{B_1 T(t)}\right\}\right\}.$$

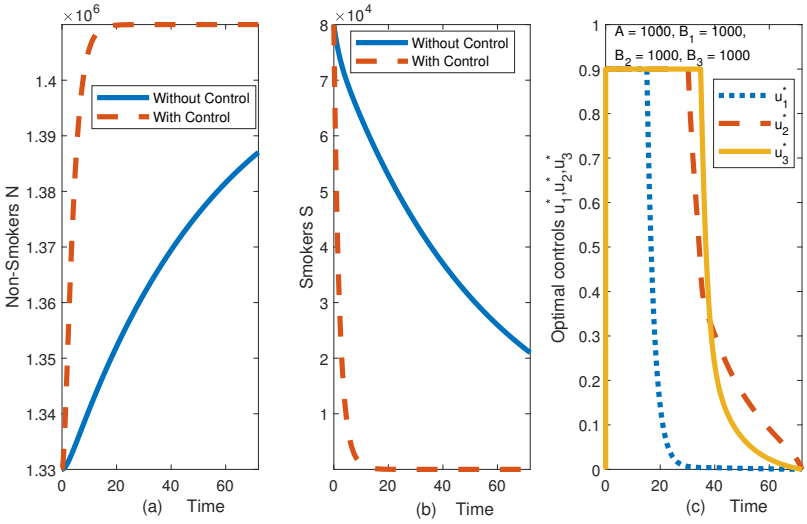

FIGURE 2. Dynamics of states with and without controls.

Using the same arguments, we also obtain the second and third optimal control functions. Therefore, the control functions are

$$u_1^*(t) = min\left\{1, max\left\{0, \frac{\beta(\lambda_2(t) - \lambda_1(t))N^*(t)S^*(t)}{B_1 T(t)}\right\}\right\},$$

$$u_2^*(t) = min\left\{1, max\left\{0, \frac{\rho(\lambda_2(t) - \lambda_3(t))S^*(t)}{B_2}\right\}\right\}, \text{and}$$

$$u_3^*(t) = min\left\{1, max\left\{0, \frac{p\alpha(\lambda_2(t) - \lambda_1(t))Q^*(t)}{B_3}\right\}\right\}.$$

Q.E.D.□

4. NUMERICAL SIMULATIONS AND RESULTS

An optimal control problem has been established based on the SIR model to determine the optimal control strategy for the adolescent smoking epidemic in institutional settings. The optimal control problem consists of six ordinary differential equations describing states and adjoint variables with three control variables. The state variables are nonsmokers N, smokers S, and temporary quitters Q. The control u_1 is associated with prevention, such as reducing the smoking transmission rate from N to S, and the controls u_2 and u_3 are associated with treatment, such as smoking cessation education.

In general, full efficiency of the controls is unfeasible. We assumed the upper bound of each of the controls was 0.9. The problem is solved numerically using the parameter values summarized in Table 2 using the forward-backward sweep method [17] and fourth-order Runge–Kutta

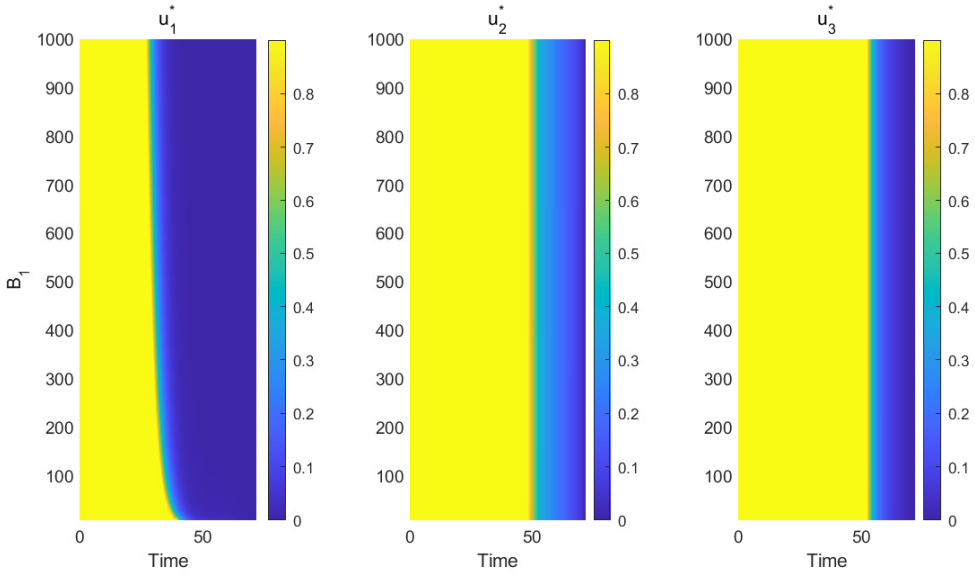

FIGURE 3. Duration of the maximum implementation for the optimal controls u_1, u_2, and u_3 when the cost of B_1 changes.

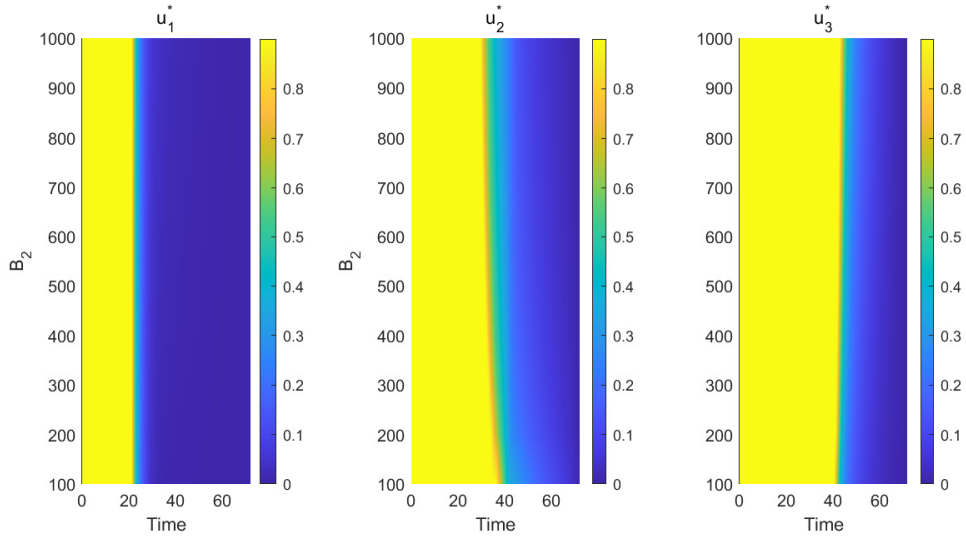

FIGURE 4. Duration of the maximum implementation for the optimal controls u_1, u_2, and u_3 when the cost of B_2 changes.

algorithm, subject to a wide range of plausible values of weight factors A, B_1, B_2, and B_3 because the weights should vary from group to group.

We use data for male students between 13 and 18 (i.e., lower and upper secondary students)[12]. The total population is $T(0) = 1.41 \times 10^6$, where $N(0) = 1.31 \times 10^6$ and $S(0) = 8.0 \times 10^4$ [18]. The rate of male students attempting to quit smoking is approximately 70% [12], and the success rate for quitting smoking among adults is about 30% [13], so we adopt $1 - p = 0.2$. The period for the simulation is $[0, T]$, where $T = 72$ months (i.e., 6 years).

Figure 2 $(a), (b)$ depicts the dynamics of states with and without the controls. The rightmost graph (c) in Fig. 2 presents the time-dependent control strategy in which the controls u_2 and u_3 are implemented longer than the control u_1, maximizing the effort. The controls work well for reducing the number of smokers Fig. $2(a), (b)$. Additionally, control u_1 is faster than u_2 and u_3 in reducing the number of smokers.

As the cost of $Bi_{i=1,2,3}$ and A changes from 0 to 1000, Figs. $3 \sim 6$ show implementation for the optimal controls.

Figures $3 \sim 6$, illustrate the variations in the maximum implementation periods of controls u_1, u_2, and u_3 under different scenarios. We let t_1, t_2, and t_3 be the period for maximum implementation of the optimal controls u_1, u_2, and u_3, respectively.

Figure 3 depicts the change in t_1, t_2, and t_3, where $B_1 = 0 \sim 1000$ while A, B_2, and B_3 are fixed. In this case, as the cost of B1 increases, the period of u_1's maximum effort is decreased, but u_2 and u_3 are not affected by the change in B_1's cost. The controls u_2 and u_3 should maintain maximum effort over a long time.

Figure 4 depicts the change in t_1, t_2, and t_3, where $B_2 = 0 \sim 1000$ while A, B_1, and B_3 are fixed. Although it is not as much of a change as in the case of u_1, we can see that the period of u_2's maximum effort is gradually decreasing. Also, in the case of u_3, it can be seen that the amount of effort of u_3 increases as the cost of B_2 increases. This appears to increase the amount of effort of u_3, which is the control amount of education, as the amount of effort cannot be used due to the price competitiveness of u_2. And it can be seen that u_1 is almost unaffected by the cost of B_2.

Figure 5 depicts the change in t_1, t_2, and t_3 with $B_3 = 0 \sim 1000$ while A, B_1, and B_2 are fixed. It can be seen that as the cost of B_3 increases, the period of maximum effort of u_3 also decreases. And u_1 is almost unaffected by changes in u_3 cost. But, in the case of u_2, it can be seen that the price is not affected from 0 to 400, however, when it exceeds 400, t_2 gradually increases. In this case, as in the Fig. 4, it seems that the amount of effort of u_2 increases instead of that of u_3 due to price competitiveness.

Finally, Fig. 6 shows the change in t_1, t_2, and t_3 when B_1, B_2, and B_3 are fixed and A, the cost related to smokers, is varied from 0 to 1000. In this case, it can be seen that as cost A increases, the maximum effort period increases for all u_1, u_2, and u_3.

5. DISCUSSION

The smoking rate among adolescents is decreasing, as noted in [6] in Korea. To reduce the smoking rate among male adolescents in Korea, we applied the optimal control theory

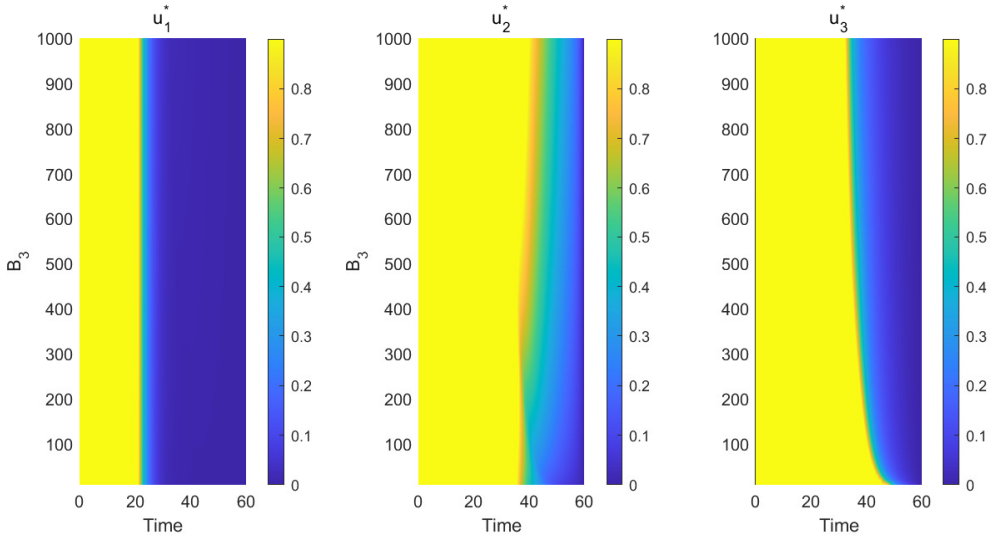

FIGURE 5. Duration of the maximum implementation for the optimal controls u_1, u_2, and u_3 when the cost of B_3 changes.

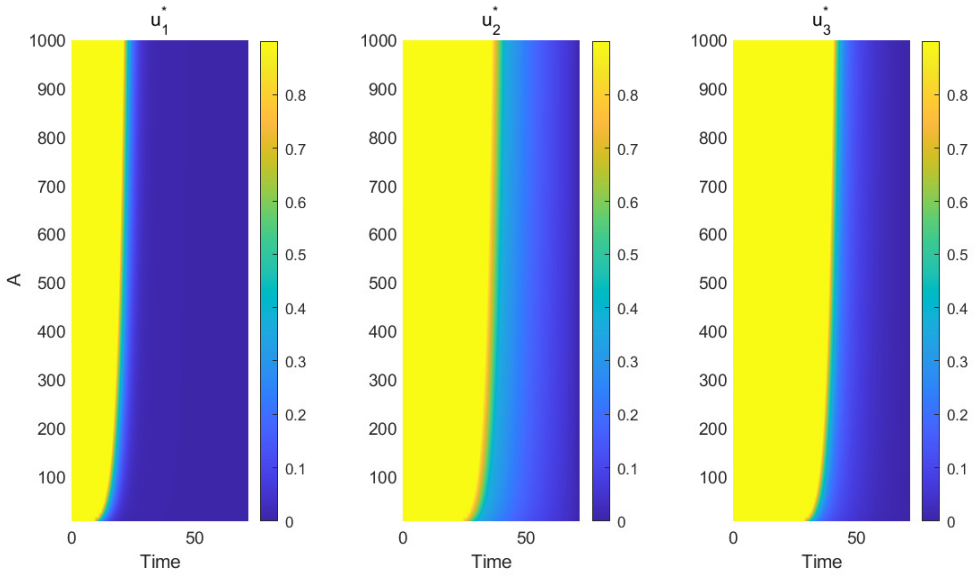

FIGURE 6. Duration of the maximum implementation for the optimal controls u_1, u_2, and u_3 when the cost of A changes.

to the $SIRS$ model, a disease epidemiological mathematical model that examines changes in nonsmokers and smokers among adolescents. As a result, in the model to which optimal control theory was applied, the number of smokers decreased sharply and the number of non-smokers increased significantly.

From the results of the optimal control function, we concluded that efforts to encourage current smokers and temporary quitting smokers should be maintained for a longer period, rather than a period of efforts to reduce the proportion of non-smokers to become smokers through smoking prevention education.

The effects of mass media campaigns on smoking behaviour in youth are very low [19]. This intervention is compared to Siegel's result. Exposure to television antismoking advertisements did not affect the progression to establish smoking among adolescents (ages 14 to 15 years at the baseline), and there were no effects of exposure to radio or outdoor advertisements [20].

Our model depicts that prevention has a shorter duration of maximum effort than education or treatment for smoking cessation, but some methods and efforts must be made to help current smokers and temporary smokers quit permanently. Smoking prevention education in the early stages can be effective to becoming smokers, including counseling, family support, and education/campaign rather than media. Also, we should make efforts to permanently quit smoking rather than temporarily quitting smoking. Although the smoking rate of teenagers in Korea is steadily decreasing, increasing the number of smokers through drug treatment and smoking cessation clinics and continuing management to help smokers continue to quit is an effective strategy to steadily lower the smoking rate. According to [21], the proportion of smokers who experienced physical-mental abuse or neglect was significantly higher than those who did not. Therefore, the family functions of smokers (support, alienation, intimacy, role, sociality, authority) can be controlled to prevent the relapse of smoking.

The results have to increase the number of smokers who quit smoking and encourage quitters to continue to quit smoking through drug treatment smoking cessation clinics and family support for smokers. This finding suggests that continuous management is a more effective strategy. For a successful strategy, additional research is needed on the success factors for smoking cessation.

APPENDIX

A. Basic Reproduction Number $\mathcal{R}_0$. The Jacobian matrix of (2.1) has of the form:

$$J = \begin{pmatrix} \mu - \beta\frac{S}{T} - \mu & -\beta\frac{N}{T} & \mu + (1-p)\alpha \\ \beta\frac{S}{T} & \beta\frac{N}{T} - (\gamma + \mu) & p\alpha \\ 0 & \gamma & -(\alpha + \mu) \end{pmatrix}$$

At a disease-free equilibrium(DFE) $E_0 = (N, 0, 0)$

$$J|_{(N,0,0)} = \begin{pmatrix} 0 & \mu & \mu + (1-p)\alpha \\ 0 & \beta - (\gamma + \mu) & p\alpha \\ 0 & \gamma & -(\alpha + \mu) \end{pmatrix}. \quad F = \begin{pmatrix} \beta & 0 \\ 0 & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} \gamma + \mu & -p\alpha \\ -\gamma & \alpha + \mu \end{pmatrix} \quad \text{and} \quad V^{-1} = \frac{1}{(\alpha+\mu)(\mu+\gamma)-p\alpha\gamma} \begin{pmatrix} \alpha + \mu & p\alpha \\ \gamma & \gamma + \mu \end{pmatrix}.$$

Therefore,

$$\mathcal{R}_0 = \beta \frac{(\alpha + \mu)}{(\alpha + \mu)(\gamma + \mu) - p\alpha\gamma}.$$

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