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# INTERVENTION STRATEGIES FOR THE DYNAMICS OF POPULATION WITH OVEREATING BEHAVIOR

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ABSTRACT. Disordered eating behaviors, such as overeating, are known to be contagious in the general population. The objective of our research is to find an optimal control strategy to reduce the social burden of unhealthy overeating behavior by establishing and analyzing a mathematical model for the social transmission dynamics of unhealthy overeating. We consider four compartments in the population: normal weight with normal eating behavior, normal weight with overeating behavior, overweight with normal eating behavior, and overweight with overeating behavior. Simulation results under various control scenarios show that integrated control measures may be necessary to reduce the growth rate of the overeating population.

## 1. INTRODUCTION

Obesity prevalence has become one of the most prominent issues in global public health [1, 2]. Obesity can be considered a socially transmitted disease, meaning that the social and cultural environment a person inhabits can influence their risk of becoming obese. There are several papers that have used mathematical models to study the dynamics of the obese population. These models can be useful for understanding the factors that contribute to the development of obesity, as well as for evaluating the impact of different interventions to prevent and treat obesity [3, 4, 5, 6, 7, 8].

The complete mechanism behind the social contagion of obesity has not been fully elucidated, but the scientific evidence for social contagion is accumulating [9, 10, 11, 12]. In this paper, we especially focus on the fact that social factors can play a significant role in the development and persistence of overeating patterns and ultimately obesity. The objective of our research is to find optimal intervention strategies to reduce the social burden of unhealthy

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overeating behavior by establishing and analyzing a mathematical model for the social transmission dynamics of unhealthy overeating.

## 2. FORMULATION OF THE MODEL

Comparing the population group with overeating habits with normal eating behaviors. we modify Wang's model [13] and Oh's model [14] for eating behavior transmission as follows:

We divide the population into four groups $S_N, S_O, I_N, I_O$. The susceptible groups $S_N, S_O$, are those in which all individuals report normal eating behaviors but can be socially induced to become overeating. The $S_N$ and $S_O$ groups are individuals with normal weights and those that are overweights/obese, respectively. The infectious group $I_N$ and $I_O$ both exhibit overeating behaviors and have normal weights and are overweights/obese, respectively. Figure 1 shows the schematic diagram of our model.

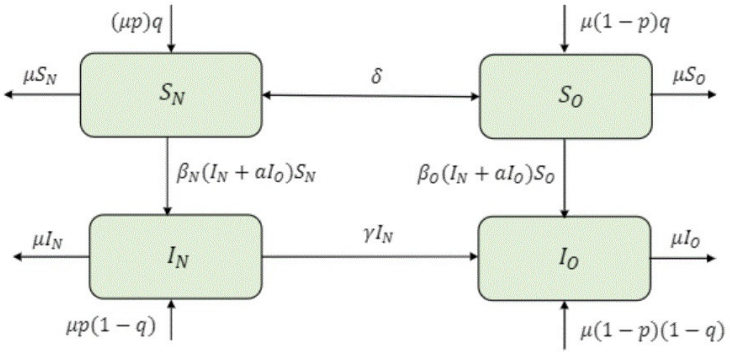


FIGURE 1. Schematic diagram for the model

And its mathematical formulations are

$$\begin{aligned}
\frac{dS_N}{dt} &= \mu pq - \beta_N(I_N + aI_O)S_N - \mu S_N + \delta(S_O - S_N)\\
\frac{dS_O}{dt} &= \mu(1-p)q - \mu S_O - \beta_O(I_N + aI_O)S_O + \delta(S_N - S_O)\\
\frac{dI_N}{dt} &= \mu p(1-q) + \beta_N(I_N + aI_O)S_N - (\mu + \gamma)I_N\\
\frac{dI_O}{dt} &= \mu(1-p)(1-q) + \gamma I_N + \beta_O(I_N + aI_O)S_O - \mu I_O,
\end{aligned} \tag{2.1}$$

where $S_N + S_O + I_N + I_O = 1$.

The variable domain of the model is

$$\Omega = \{(S_N, S_O, I_N, I_O) \in \mathbb{R}^4 \mid 0 \le S_N, S_O, I_N, I_O \le 1\}.$$

In the model, the parameters $\beta_N, \beta_O, a, \delta$, and $\gamma$ are positive constants. The parameter $\mu$ is the recruitment rate of the population, with proportions $p$ recruited to the normal BMI category $S_N \cup I_N$ and $1-p$ to the overweight/obese category $S_O \cup I_O$. We further assume that the normal

BMI category splits with fractions $q$ for category $S_N$ and $1-q$ for category $I_N$, and that the overweight/obese population is also divided into proportions of $q$ and $1-q$ to the category $S_O$ and $I_O$, respectively. Social contact levels of the susceptible population with normal and overweight/obese overeating behaviors are $\beta_N$ and $\beta_O$, respectively. Last, we assume that $I_N$ is more strongly infectious than $I_O$ since the $I_N$ population are not obese despite their overeating behavior.

As in [13], we consider the weight coefficient $0 \leq a \leq 1$ of $I_N$, which represents the relative intensity of overeating behaviors for susceptible individuals with normal BMI, and also considers the random transition in the combined group $S$ due to random perturbations in the transition rate $\delta$. Transition in the group $I$ is directed from $I_N$ to $I_O$ with transition rate $\gamma$.

## 3. Disease-free equilibrium and basic reproductive number

3.1. **Disease-free equilibrium.** We easily find the disease free equilibrium point $(S_N^*, S_O^*, 0, 0)$, where

$$S_N^* = \frac{\mu p + \delta}{\mu + 2\delta} \text{ and } \quad S_O^* = \frac{\{\mu(1-p) + \delta\}}{\mu + 2\delta}.$$

We note that the disease free equilibrium point exists only when $q = 1$.

3.2. **Basic reproductive number.** 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]. The value of $\mathcal{R}_0$ can be calculated using the spectral radius of the next-generation matrix of a model: where $\rho$ is defined as the spectral radius of the next-generation matrix $\mathcal{F}\mathcal{V}^{-1}$, $\mathcal{F}$ is the rate of occurrence of new infections in class $i$, and $\mathcal{V}$ is the transfer of individuals out of class $i$ by all other means [16].

We denote a new infection and the remainder by the matrices $F$ and $V$, respectively.

Then, in the model (2.1), we have

$$F = \begin{bmatrix} \beta_N(I_N + aI_O)S_N \\ \beta_O(I_N + aI_O)S_O \end{bmatrix} \text{ and } V = \begin{bmatrix} -\mu p(1-q) + (\mu+\gamma)I_N \\ -\mu(1-p)(1-q) - \gamma I_N + \mu I_O \end{bmatrix}.$$

Now we compute the matrices $\mathcal{F}$ and $\mathcal{V}$ by evaluating the Jacobian matrices $[\partial F_i/\partial x_j]$ and $[\partial V_i/\partial x_j]$ at the disease-free equilibrium point. Since the disease-free equilibrium point in the model (2.1) is $E^* = (S_N^*, S_O^*, 0, 0)$, we have

$$\mathcal{F} = \begin{bmatrix} \beta_N S_N^* & a\beta_N S_N^* \\ \beta_O S_O^* & a\beta_O S_O^* \end{bmatrix} \text{ and } \mathcal{V} = \begin{bmatrix} \mu+\gamma & 0 \\ -\gamma & \mu \end{bmatrix},$$

Then we obtain the dominant eigenvalue of $\mathcal{F}\mathcal{V}^{-1}$, which gives

$$\mathcal{R}_0 = \rho(\mathcal{F}\mathcal{V}^{-1}) = \frac{(\mu + a\gamma)\beta_N S_N^* + a\beta_O S_O^*(\mu+\gamma)}{\mu(\gamma+\mu)}.$$

But since

$$S_N^* = \frac{\mu p + \delta}{\mu + 2\delta} \text{ and } \quad S_O^* = \frac{\{\mu(1-p) + \delta\}}{\mu + 2\delta},$$

we have

$$\mathcal{R}_0 = \frac{\beta_N(\mu+a\gamma)(\mu p+\delta)+a\beta_O(\mu+\gamma)\{\mu(1-p)+\delta\}}{\mu(\mu+\gamma)(\mu+2\delta)}.$$

Initial transmission is directly related to the basic reproductive number. Sensitivity indices allow us to measure the relative change in a variable when a parameter changes. When the variable is the differentiable function of parameters, the sensitivity index may be alternatively defined using partial derivatives. By the use $\mathcal{R}_0$, the sensitivities are parameters; $\beta_N, \beta_O, \gamma$.

$$\begin{aligned}
S_{\beta_N} &= \frac{\partial \mathcal{R}_0}{\partial \beta_N}\frac{\beta_N}{\mathcal{R}_0} = 1 - \frac{a\beta_O(\mu+\gamma)\{\mu(1-p)+\delta\}}{\beta_N(\mu+a\gamma)(\mu p+\delta)+a\beta_O(\mu+\gamma)\{\mu(1-p)+\delta\}},\\
S_{\beta_O} &= \frac{\partial \mathcal{R}_0}{\partial \beta_O}\frac{\beta_O}{\mathcal{R}_0} = 1 - \frac{\beta_N(\mu+a\gamma)(\mu p+\delta)}{\beta_N(\mu+a\gamma)(\mu p+\delta)+a\beta_O(\mu+\gamma)\{\mu(1-p)+\delta\}},\\
S_{\gamma} &= \frac{\partial \mathcal{R}_0}{\partial \gamma}\frac{\gamma}{\mathcal{R}_0} = \frac{\beta_N\mu^2\gamma(a-1)(\mu p+\delta)(\mu+2\delta)}{\beta_N(\mu+a\gamma)(\mu p+\delta)+a\beta_O(\mu+\gamma)\{\mu(1-p)+\delta\}}.
\end{aligned}$$

Analyzing the individual parameter sensitivities reveals that an elevated value of $\beta_N$ corresponds to an augmentation in $S_{\beta_N}$, while a higher value of $\beta_O$ corresponds to an increase in $S_{\beta_O}$. Furthermore, an increase in $\gamma$ leads to a proportional increase in $S_\gamma$. Consequently, the sensitivity escalates with the augmentation of each parameter.

## 4. Optimal Control

In our model (2.1), we adopt the time-dependent controls $u_1(t)$ and $u_2(t)$ corresponding to $\beta_N$ and $\beta_O$, respectively. The control variables $0 \le u_1(t), u_2(t) \le 1$ represent the amount of intervention at time $t$ to reduce social contact with groups exhibiting the overeating behavior.

We replace the social contact rates $\beta_N$ and $\beta_O$ by $(1-u_1(t))\beta_N$ and by $(1-u_2(t))\beta_O$, respectively. Then the controlled model is as follows:

$$\begin{aligned}
\frac{dS_N}{dt} &= \mu pq - \beta_N(1-u_1(t))(I_N+aI_O)S_N - \mu S_N + \delta(S_O - S_N)\\
\frac{dS_O}{dt} &= \mu(1-p)q - \mu S_O - \beta_O(1-u_2(t))(I_N+aI_O)S_O + \delta(S_N - S_O)\\
\frac{dI_N}{dt} &= \mu p(1-q) + \beta_N(1-u_1(t))(I_N+aI_O)S_N - (\mu+\gamma)I_N\\
\frac{dI_O}{dt} &= \mu(1-p)(1-q) + \gamma I_N + \beta_O(1-u_2(t))(I_N+aI_O)S_O - \mu I_O.
\end{aligned} \tag{4.1}$$

Here we note that, in model system (4.1), the effective reproductive number $\mathcal{R}_t$ is

$$S_N(t)\frac{\beta_N(1-u_1(t))(\mu+a\gamma)(\mu p+\delta)+a\beta_O(1-u_2(t))(\mu+\gamma)\{\mu(1-p)+\delta\}}{\mu(\mu+\gamma)(\mu+2\delta)}.$$

An optimal control problem with the objective cost functional can be given

$$J(u_1,u_2) = \int_0^T \left[A_1 I_N(t) + A_2 I_O(t) + \frac{B_1}{2}u_1^2(t) + \frac{B_2}{2}u_2^2(t)\right]dt \tag{4.2}$$

subject to the state system given by (4.1).

In the objective functional (4.2), the quantities $A_1$ and $A_2$ represent the weight constants of the groups with normal overeating and obese overeating behaviors respectively, while the weight coefficients $B_1$ and $B_2$ are constants that represent cost sizes for the controls $u_1$ and $u_2$ respectively. The terms $\frac{1}{2}B_1u_1^2$ and $\frac{1}{2}B_2u_2^2$ describe the costs associated with transmission by social contact rate for the groups with normal and obese overeating behaviors and with an educational/warning campaign about obesity for minimization of the obese population, respectively.

To solve the optimal control problem $(4.1)$ and $(4.2)$, we need to find a pair $(u_1^*, u_2^*)$ of optimal functions such that

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

subject to the state system given by (4.1), where the control set is defined as

$$\mathcal{U} = \{(u_1, u_2)|\ u_1 \text{ and } u_2 \text{ are Lebesgue measurable functions from } [0, T] \text{ to } [0, 1]\}.$$

We define the Hamiltonian $\mathcal{H}$ for the control problem as follows:

$$\begin{aligned}
&\mathcal{H}(t, \mathbf{X}(t), \mathcal{U}(t), \mathbf{\Lambda}(t)) \\
&= A_1I_N(t) + A_2I_O(t) + \frac{B_1}{2}u_1^2(t) + \frac{B_2}{2}u_2^2(t) + \mathbf{\Lambda}(t)\left(\frac{d\mathbf{X}(t)}{dt}\right)^T \\
&= A_1I_N(t) + A_2I_O(t) + \frac{B_1}{2}u_1^2(t) + \frac{B_2}{2}u_2^2(t) \\
&+ \lambda_1(t)[\mu pq - \beta_N(1 - u_1(t))(I_N + aI_O)S_N - \mu S_N + \delta(S_O - S_N)] \\
&+ \lambda_2(t)[\mu(1 - p)q - \mu S_O - \beta_O(1 - u_2(t))(I_N + aI_O)S_O + \delta(S_N - S_O)] \\
&+ \lambda_3(t)[\mu p(1 - q) + \beta_N(1 - u_1(t))(I_N + aI_O)S_N - (\mu + \gamma)I_N] \\
&+ \lambda_4(t)[\mu(1 - p)(1 - q) + \gamma I_N + \beta_O(1 - u_2(t))(I_N + aI_O)S_O - \mu I_O],
\end{aligned} \tag{4.3}$$

where the adjoint variables are defined by $\mathbf{\Lambda}(t) = (\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t))$ and the state variables are denoted by $\mathbf{X}(t) = (S_N(t), S_O(t), I_N(t), I_O(t))$.

For the necessary condition of our control problem, we state and prove the following theorem:

**Theorem 4.1.** *Let $S_N^*(t), S_O^*(t), I_N^*(t)$ and $I_O^*(t)$ be the optimal state solutions with the associated optimal control variables $u_1^*$ and $u_2^*$ for the optimal control problem $(4.1)$ and $(4.2)$.*

*Then, there exist adjoint variables $\lambda_1(t), \lambda_2(t), \lambda_3(t)$ and $\lambda_4(t)$ that satisfy*

$$\begin{aligned}
\lambda_1'(t) &= (\lambda_1(t) - \lambda_3(t))\beta_N(1 - u_1^*(t))(I_N^*(t) + aI_O^*(t)) + (\lambda_1(t) - \lambda_2(t))\delta + \lambda_1(t)\mu \\
\lambda_2'(t) &= (\lambda_2(t) - \lambda_4(t))\beta_O(1 - u_2^*(t))(I_N^*(t) + aI_O^*(t)) + (\lambda_2(t) - \lambda_1(t))\delta + \lambda_2(t)\mu \\
\lambda_3'(t) &= -A_1 + (\lambda_1(t) - \lambda_3(t))\beta_N(1 - u_1^*(t))S_N^*(t) + (\lambda_2(t) - \lambda_4(t))\beta_O(1 - u_2^*(t))S_O^*(t) \\
&\quad + (\lambda_3(t) - \lambda_4(t))\gamma + \lambda_3(t)\mu \\
\lambda_4'(t) &= -A_2 + (\lambda_1(t) - \lambda_3(t))a\beta_N(1 - u_1^*(t))S_N^*(t) + (\lambda_2(t) - \lambda_4(t))a\beta_O(1 - u_2^*(t))S_O^*(t) \\
&\quad + \lambda_4(t)\mu
\end{aligned}$$

*with transversality conditions (or boundary conditions)*

$$\lambda_j(T) = 0,\ i = 1, 2, 3, 4.$$

*Furthermore, the optimal controls $u_1^*$ and $u_2^*$ are given by*

$$\begin{aligned}
u_1^*(t) &= \min\left\{1, \max\left\{0, \frac{1}{B_1}(\lambda_3(t) - \lambda_1(t))\beta_N(I_N^*(t) + aI_O^*(t))S_N^*(t)\right\}\right\}, \\
u_2^*(t) &= \min\left\{1, \max\left\{0, \frac{1}{B_2}(\lambda_4(t) - \lambda_2(t))\beta_O(I_N^*(t) + aI_O^*(t))S_O^*(t)\right\}\right\}.
\end{aligned} \tag{4.4}$$

*Proof.* To determine the adjoint equations and the transversality conditions, we used the Hamiltonian (4.3). By Pontryagin's Maximum Principle, setting $S_N^*(t), S_O^*(t), I_N^*(t)$ and $I_O^*(t)$ and also differentiating the Hamiltonian (4.3) with respect to $S_N^*(t), S_O^*(t), I_N^*(t)$ and $I_O^*(t)$, we obtain

$$\begin{aligned}
\frac{\partial \mathcal{H}}{\partial S_N} &= (\lambda_3(t) - \lambda_1(t))\beta_N(1 - u_1^*(t))(I_N^*(t) + aI_O^*(t)) + (\lambda_2(t) - \lambda_1(t))\delta - \lambda_1(t)\mu \\
\frac{\partial \mathcal{H}}{\partial S_O} &= (\lambda_4(t) - \lambda_2(t))\beta_O(1 - u_2^*(t))(I_N^*(t) + aI_O^*(t)) + (\lambda_1(t) - \lambda_2(t))\delta - \lambda_2(t)\mu \\
\frac{\partial \mathcal{H}}{\partial I_N} &= A_1 + (\lambda_3(t) - \lambda_1(t))\beta_N(1 - u_1^*(t))S_N^*(t) + (\lambda_4(t) - \lambda_2(t))\beta_O(1 - u_2^*(t))S_O^*(t) \\
&\quad + (\lambda_4(t) - \lambda_3(t))\gamma - \lambda_3(t)\mu \\
\frac{\partial \mathcal{H}}{\partial I_O} &= A_2 + (\lambda_3(t) - \lambda_1(t))a\beta_N(1 - u_1^*(t))S_N^*(t) + (\lambda_4(t) - \lambda_2(t))a\beta_O(1 - u_2^*(t))S_O^*(t) \\
&\quad - \lambda_4(t)\mu
\end{aligned}$$

The costate equations are

$$\lambda_1'(t) = -\frac{\partial \mathcal{H}}{\partial S_N},\ \lambda_2'(t) = -\frac{\partial \mathcal{H}}{\partial S_O},\ \lambda_3'(t) = -\frac{\partial \mathcal{H}}{\partial I_N},\ \lambda_4'(t) = -\frac{\partial \mathcal{H}}{\partial I_O}.$$

To obtain the optimal controls (4.4), we also differentiate the Hamiltonian $\mathcal{H}$ with respect to $u_1, u_2$ and set it equal to zero.

$$\begin{aligned} 0 &= \frac{\partial \mathcal{H}}{\partial u_1^*} = B_1 u_1^*(t) + (\lambda_1(t) - \lambda_3(t))\beta_N(I_N^* + aI_O^*)S_N^*(t) \\ 0 &= \frac{\partial \mathcal{H}}{\partial u_2^*} = B_2 u_2^*(t) + (\lambda_2(t) - \lambda_4(t))\beta_O(I_N^* + aI_O^*)S_O^*(t). \end{aligned} \tag{4.5}$$

Solving for the optimal controls, we obtain

$$\begin{aligned} u_1^*(t) &= \frac{1}{B_1}(\lambda_3(t) - \lambda_1(t))\beta_N(I_N^* + aI_O^*)S_N^*(t) \\ u_2^*(t) &= \frac{1}{B_2}(\lambda_4(t) - \lambda_2(t))\beta_O(I_N^* + aI_O^*)S_O^*(t). \end{aligned} \tag{4.6}$$

To determine an explicit expression for the optimal controls for $0 \leq u_1^* \leq 1$ and $0 \leq u_2^* \leq 1$, we utilize a standard optimality technique, then we obtain characterizations of $u_1^*$ and $u_2^*$:

$$u_1^*(t) = \min\left\{1, \max\left\{0, \frac{1}{B_1}(\lambda_3(t) - \lambda_1(t))\beta_N(I_N^* + aI_O^*)S_N^*(t)\right\}\right\}$$

and

$$u_2^*(t) = \min\left\{1, \max\left\{0, \frac{1}{B_2}(\lambda_4(t) - \lambda_2(t))\beta_O(I_N^* + aI_O^*)S_O^*(t)\right\}\right\}.$$

Q.E.D.□

## 5. Numerical Results

Our goal is to find intervention strategies to prevent people from becoming obese. We simulated the different scenarios using the forward-backward sweep method [17].

All parameter values used in the numerical simulations were estimated based on [13]. The parameters values are $a = 0.7$; $p = 0.85$; $q = 0.05$; $\mu = \frac{1}{70+365/7} = 0.0002739726$; $\beta_N = 0.02$; $\beta_O = 0.05$; $\delta = 0.001$; $\gamma = 0.002899976$. We chose 0.7 as the upper bound for $u_1^*$ and $u_2^*$ due to a person's tolerance. We simulated our model by setting the default values of the weight variables to $A_1 = 0.1, A_2 = 0.1, B_1 = 0.1$ and $B_2 = 0.1$ with initial states $S_N = 0.5, S_O(0) = 0.3, I_N(0) = 0.1$ and $I_O(0) = 0.1$.

We simulated four control scenarios to minimize overweight/obese populations in the model. Figure 2 shows the simulations for the four interventions, which are $A_1 = A_2 = 0.1, A_1 = 0.1; A_2 = 1.0, A_1 = 1.0; A_2 = 0.1$ and $A_1 = A_2 = 1.0$. The green dotted lines in Fig. 2(c) and (d) are the cases where $I_N$ and $I_O$, respectively are not controlled. When not controlled, $I_N$ tends to increase significantly to about 108 and then decreases gradually. The value of $I_O$ tends to increase constantly with no control. From Fig. 2, our simulation results show how the trends of $I_N, I_O$ and the two control variables $u_1^*, u_2^*$ change when controlled with our four scenarios.

First, the solid blue line in Fig. 2 shows the simulation result when $A_1 = A_2 = 0.1$. When we want to minimize the objective cost functional, $u_1^*$ has maximum control only at the

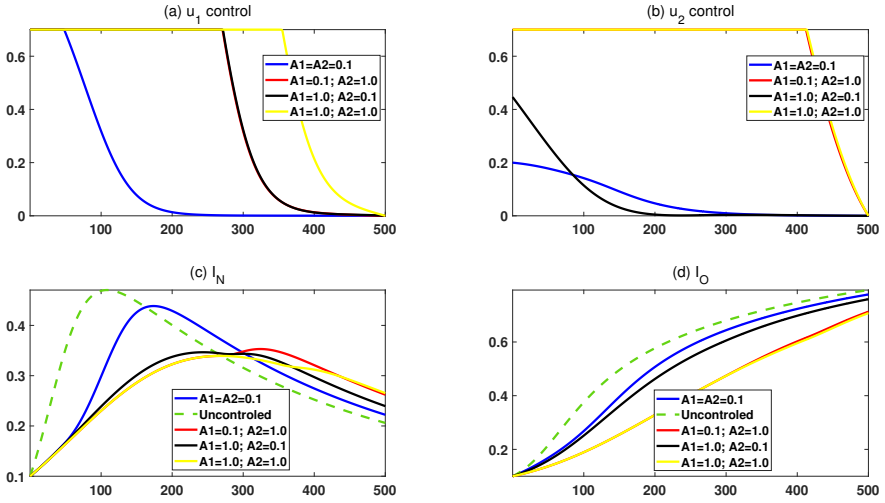


FIGURE 2. Comparison according to changes in values of $A_1$, and $A_2$

beginning, and $u_2^*$ has little control initially. And as a result, the effect of reducing $I_N$ and $I_O$ is relatively low.

Hence the simulation results show that if the weights of $I_N$ and $I_O$ are given equally as $0.1$, a strong reduction effect for the infectious group by controlling $u_1^*, u_2^*$ cannot be expected.

We suggest a variation in the scenario involving the weights $A_1$ and $A_2$. Second, Fig. 2 illustrates the scenario where we select $A_1 = A_2 = 1.0$ on one side and $A_1 = 0.1, A_2 = 1.0$ on the other; the plots are the yellow and red curves of Fig. 2 respectively. Both yellow and red curves of $I_N$ show almost the same trend of about $280$ hours, but $I_N$ does not decrease in the control condition as it does in non-control after about $280$ hours. In this case, $u_1^*$ maintains maximum control until about $280$ hours for the red curve and longer for the yellow line. The maximum implementations of the control $u_2^*$ are both $0.7$. Due to the implementation of control, the overweight/obese population $I_O$ is well controlled as shown in Fig. 2(d). The strong effect of reducing the infectious group by controlling $u_2^*$ can be expected for a relatively long time.

In the case of $I_O$, we can see in Fig. 2(d) that the yellow and red curves show almost the same trend from the beginning. This shows the same effect in controlling $A_1 = A_2 = 1.0$ as in only controlling $A_2 = 1.0$. In this case, $A_1$ and $A_2$ do not need to be heavily weighted.

Last, when $A_1 = 1.0$ and $A_2 = 0.1$ both $I_N$ and $I_O$ experience a greater reduction effect than the baseline value $0.1$. For the black lines in Fig. 2(c) and (d), the state $I_N$ is well controlled before $280$ but loses this effect after that time. The maximum initial implementation of $u_1^*$ is $0.7$, maintained until about $280$. When $A_1 = 1.0$ and $A_2 = 0.1$, the state $I_O$ has considerably less control than when $A_1 = A_2 = 1.0$ or only $A_2 = 1.0$. In this case, $u_2^*$ has

maximum control of about $0.44$ at the beginning and rapidly decreases. The state $I_O$ is steadily controlled, but the reduction effect is not as strong (Fig. 2(d)).

From the above four analyses, $I_N$ is successfully controlled to about $280$ for three control scenarios, while $I_O$ is controlled over the long term for all four. To achieve high effectiveness in reducing infectious, the long-term control is required for $u_2^*$.

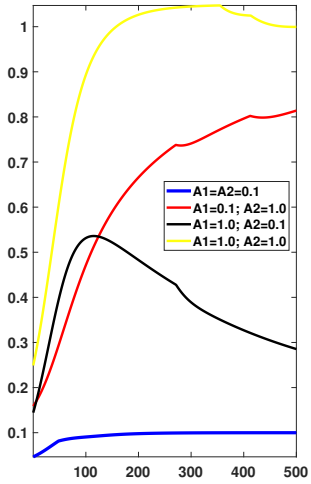


FIGURE 3. Objective cost functional up to $500$

In addition, we conduct simulations to reveal how the cost of intervention is measured through controls of the objective cost functional. Figure 3 shows the simulation results of four scenarios for minimizing the objective functional $J(u_1, u_2)$ for fixed $B_1 = B_2 = 0.1$. We created a scenario for the cost of obesity and investigated intervention strategies over $500$.

In the case of $A_1 = 1.0$ and $A_2 = 0.1$, the yellow curve rises sharply to $0.99$ at about $350$ and decreases slightly after this time. This signifies that a high cost is required until about $350$. In the case $A_1 = 0.1$ and $A_2 = 1.0$, the red curve increases steadily. Therefore, the cost required increases continuously. In the case $A_1 = 1.0$ and $A_2 = 0.1$, the black curve rises sharply to nearly $0.55$ at about $100$ and then gradually decreases. For the last case, $A_1 = A_2 = 0.1$, the blue curve initially shows a slight increases but soon settles to stay at the same value.

## 6. DISCUSSION AND CONCLUSIONS

The simulation exhibits no effect on the proportions $p$ and $q$. Our model shows successful control to reduce the spread of obesity. When one or both weights are ten times the default value, control of $I_N$ increases gently up to about $280$ hours and then decreases. In the case of the baseline value $A_1 = A_2 = 0.1$, $I_N$ is rapidly controlled up to about $180$, after which control decreases. The population $I_N$ of infected people naturally decreases after about $108$.

However, $I_O$ is constantly increasing. In the cases $A_1 = A_2 = 1.0$ and $A_1 = 0.1, A_2 = 1.0$, the population $I_O$ is continuously well controlled by maximum control $u_2^*$. If we hope to delay $I_O$, we should keep control. Therefore, to reduce $I_O$, we can expected a strong effect when both $A_1$ and $A_2$ are high. However, maintaining high $A_1$ and $A_2$ means that the social burden may become high because the intervention must be maintained at maximum for a long time. Similarly, to prevent progression to obesity, [18] suggests that obesity control strategies require intensive, long-term counseling as obesity prevention interventions.

We created a long-term scenario of the cost of obesity. The yellow curve for weights $A_1 = 1.0, A_2 = 1.0$ in Fig. 2 is well-controlled for the states $I_O$ and $I_N$, even when $I_N$ loses control after $280$. Figure 5 illustrates that the cost is more expensive better the control.

Our model shows the necessity for contact intervention with people with normal and overeating behaviors. However, it is necessary to advise an alternative method because it is impossible to manage social contact between people. Also, the social and cultural environment can affect people's risk of becoming obese, so it is necessary to create environments, for instance as social activities, where obesity can be avoided. Even when people are in social contact, limiting unhealthy foods for eating will help them avoid obesity.

The more effectively people are made aware of the risk of obesity, the more they can be expected to avoid it proactively. As concrete examples, Leslie J. Sim et al. propose the implementation of education on a large scale or social marketing approaches as tangible methods to encourage positive reactions to environmental transformations [18]. Mass media campaigns are a commonly used public health strategy. Therefore, the recommended intervention is to encourage human awareness and recognition of the danger of obesity by implementing an intensive media campaign about calorie intake. However, almost half of obesity prevention campaigns have been analyzed as branding advertisements [19], and the evidence is still limited as to whether such campaigns can influence behavior change [20]. Nevertheless, to address the social burdens of obesity, with the rapid advance of digital media and evaluation of other channels such as social media it is necessary to be very careful about various campaign approaches.

We argue that an effective intervention to reduce the transmission of obesity is to help people become smarter about their food consumption. Although it is not possible to prohibit excessive overeating-related entertainment or commercial food advertising through the media, it is suggested that media channels should be given equal weight in presenting information about the risks of obesity.

Governments should consider the health of their citizens by focusing on designing cities that make it easy to use public transportation and that are convenient and enjoyable for people to walk in. There is a need for governments to make it easier for people to shift from a sedentary lifestyle to physical activity.

Future research needs to move beyond overeating behavior in itself and develop a comprehensive model of the factors implicated in the dynamic of diet, such as overeating social patterns.

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