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Mathematical Analysis of an Obesity Model with Eating Behaviors

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Abstract. Overweight is a social disease, which is transmitted through social networks. A mathematical model is proposed to simulate the dynamics of social obesity, where the structures of individual heterogeneity and overeating behaviors are incorporated. The basic reproduction number of the disease is calculated and is shown to be a threshold for disease invasion. Sufficient conditions for the global stability of an endemic equilibrium is established by Lyapunov functions. Numerical simulations are provided to reveal how interventions through treatment to eating behaviors and eduction to susceptible individuals suppress the progression of the disease.

AMS subject classifications: 92D30, 34D23

Key words: Overeating behavior, reproduction number, global stability, intervention.

1 Introduction

Obesity is a social disease that influences not only the health of human individuals, but also imposes the social burdens [20, 22, 25, 27, 28]. It is believed that eating behaviors through social networks and genetic factors contribute to overweight. First, relatives and friends facilitate the transmission of overeating patterns through social dinners and so on. Indeed, people tend to eat more when their eating companions eat more. Second, it is estimated that genetic factors account for 40-90% of the population variation in body mass index (BMI) [9]. There have been some mathematical researches in the aim to reveal mechanisms of propagation of obesity [1,6,8,11,17,18,21,22]. Basically, these models divide the population into the compartments of normal population, overweight population and obesity individuals and consider the contagious transmission between normal individuals and overweight individuals. However, genetic contributions to overweight are neglected in these models. Note that both eating habits and inherent factors determine

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the obesity of an individual. In this paper, we improve the modeling of overweight and obesity by incorporating the heterogeneity of population due to genetic factors, where the population is split into two subpopulations: each member in the first one has innately a normal weight, and every individual in the second class has innately an overweight body.

The organization of this paper is as follows. In the next section, we present the model formulations. Section 3 gives the qualitative analysis. In Section 4, we provide numerical simulations to demonstrate the effects of intervention measures. The paper ends with short discussions.

2 Model formulations

We split the population into four groups: the susceptible group (S) in which all individuals admit normal eating behaviors and can be affected to become overeating, the group (I) that every individual exhibits the overeating behaviors, the group (T) in which individuals are treated to enter into the group (R) that all individuals are immune to overeating behaviors. The members in the group S and group I are further divided into two categories according to body mass index (BMI): the category of individuals with the normal BMI (BMI <25 kg/m^2) and the category of overweight members with the higher BMI (BMI≥ 25 kg/m^2) [21]. Let $S_N(t)$ and $S_O(t)$ be the numbers of individuals in group S with normal weights and higher weights at time t respectively, $I_N(t)$ and $I_O(t)$ be the numbers of individuals in group I with normal weights and higher weights at time t respectively, $T(t)$ and $R(t)$ be the numbers of individuals in the group T and group R at time t respectively. Let $S(t)=S_N(t)+S_O(t)$ and $I(t)=I_N(t)+I_O(t)$ denote the total sizes of susceptible group, overeating group at time t respectively.

Previous studies [4] indicate that overeating behaviors are driven by social pressures from friends, siblings, spouses, and neighbors. As a result, it is reasonable to assume that the transition rates of individuals in category S_N and category S_O to overeating group are described respectively by

$$\beta_N(\alpha I_N+I_O)S_N,\ \beta_O(\alpha I_N+I_O)S_O,$$

where β_N and β_O are the transmission coefficients of overeating individuals to susceptible individuals with the normal BMI and the higher BMI respectively, $0\leq\alpha\leq 1$ is a weight coefficient that represents the relative intensity of overeating behaviors with normal BMI to susceptible individuals.

Let μ be the recruitment rate of the population with the fraction p to the category S_N and the fraction $1-p$ to the category S_O. The fraction p is attributed to genetic factors. We assume that only individuals with the higher BMI are treated. Let ξ be the transition rate by which individuals enter into the compartment in treatment, γ be the recovery rate of individuals due to treatment and δ be the relapse rate of treated individuals. For the simplicity of notation, we assume that the removing rate of individuals in all compartments is the same, which is denoted by μ. Motivated by [2,5], we assume that there

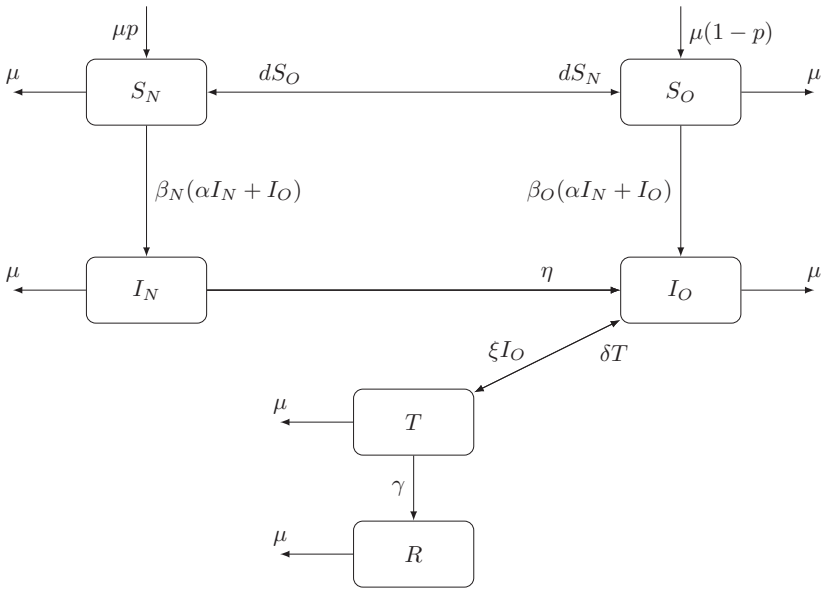

Figure 1: Schematic representation for interactions and transitions of state variables.

is a random transition in the group S due to random perturbations with the transition coefficient d, and the transition in group I is directed from I_N compartment to I_O compartment with the transition coefficient η. The schematic diagram of interactions of these state variables is given in Fig. 1 and the dynamics of the state variables are described by the mathematical model:

$$\frac{dS_N}{dt} = \mu p - \mu S_N - \beta_N(\alpha I_N + I_O)S_N + d(S_O - S_N), \tag{2.1a}$$

$$\frac{dS_O}{dt} = \mu(1-p) - \mu S_O - \beta_O(\alpha I_N + I_O)S_O + d(S_N - S_O), \tag{2.1b}$$

$$\frac{dI_N}{dt} = \beta_N(\alpha I_N + I_O)S_N - (\mu + \eta)I_N, \tag{2.1c}$$

$$\frac{dI_O}{dt} = \beta_O(\alpha I_N + I_O)S_O - (\mu + \xi)I_O + \eta I_N + \delta T, \tag{2.1d}$$

$$\frac{dT}{dt} = \xi I_O - (\mu + \gamma + \delta)T, \tag{2.1e}$$

$$\frac{dR}{dt} = \gamma T - \mu R. \tag{2.1f}$$

Since the first five equations can be decoupled from the system, it suffices to consider

$$\frac{dS_N}{dt} = \mu p - \mu S_N - \beta_N(\alpha I_N + I_O)S_N + d(S_O - S_N), \tag{2.2a}$$

$$\frac{dS_O}{dt} = \mu(1-p) - \mu S_O - \beta_O(\alpha I_N + I_O)S_O + d(S_N - S_O), \tag{2.2b}$$

$$\frac{dI_N}{dt}=\beta_N(\alpha I_N+I_O)S_N-(\mu+\eta)I_N, \tag{2.2c}$$

$$\frac{dI_O}{dt}=\beta_O(\alpha I_N+I_O)S_O-(\mu+\xi)I_O+\eta I_N+\delta T, \tag{2.2d}$$

$$\frac{dT}{dt}=\xi I_O-(\mu+\gamma+\delta)T. \tag{2.2e}$$

It is assumed that all the parameters in (2.2) are positive constants. We will expand this model in the section of numerical simulations to include more biological details.

3 Mathematical analysis

In this section, we start with the preliminary analysis of model (2.2) to present the well-posedness property of the model and the threshold conditions for the disease spread. Then we consider two cases to establish the conditions for the global stability of an endemic equilibrium.

3.1 Preliminary analysis of the model

By similar discussions to those in [3], we get the nonnegativity of solutions of model (2.2), which is stated by the following lemma.

Lemma 3.1. *The solution of model* (2.2) *is nonnegative for* $t>0$ *if* $S_i(0)\geq 0$, $I_i(0)\geq 0$, $T(0)\geq 0$, *where* $i=N,O$.

The next lemma confirms the global existence of the solutions of model (2.2).

Lemma 3.2. *All solutions of model* (2.2) *with nonnegative initial values are ultimately bounded.*

Proof. We consider the change of total size of the population:

$$L=S_N+S_O+I_N+I_O+T.$$

Direct calculations lead to

$$\frac{dL}{dt}\leq \mu-\mu L. \tag{3.1}$$

This, together with Lemma 3.1, implies that the solutions of model (2.2) with nonnegative initial values exit globally, and are ultimately bounded. □

We now compute the basic reproduction number of model (2.2). The model has a unique disease-free equilibrium $E_0=(S_N^0,S_O^0,0,0,0,0)$, where

$$S_N^0=\frac{\mu p+d}{\mu+2d},\quad S_O^0=\frac{d+\mu(1-p)}{\mu+2d}.$$

Following [29], we define the new infection rate matrix F and evolution matrix V of infective individuals under removing and transitions by

$$F=\begin{pmatrix} \alpha\beta_N S_N^0 & \beta_N S_N^0 & 0 \\ \alpha\beta_O S_O^0 & \beta_O S_O^0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad V=\begin{pmatrix} \mu+\eta & 0 & 0 \\ -\eta & \mu+\xi & -\delta \\ 0 & -\xi & \mu+\gamma+\delta \end{pmatrix}.$$

By [7,29], the basic reproduction number R_0 of model (2.2) is the spectral radius of FV^{-1}. Then direct computation yields

$$\begin{aligned} R_0=&\frac{\beta_N S_N^0(\alpha(\mu(\delta+\gamma+\xi+\mu)+\gamma\xi)+\eta(\mu+\gamma+\delta))}{(\mu+\eta)(\mu\delta+\gamma\mu+\gamma\xi+\mu^2+\xi\mu)} \\ &+\frac{\beta_O S_O^0(\mu+\eta)(\mu+\gamma+\delta)}{(\mu+\eta)(\mu\delta+\gamma\mu+\gamma\xi+\mu^2+\xi\mu)}, \end{aligned}$$

where the first term gives the contribution to the basic reproduction number from the channel of normal weight individuals and the second term describes the contribution to the basic reproduction number from the channel of overweight individuals.

We show below that the basic reproduction number acts as the threshold value of disease invasion.

Theorem 3.1. *The disease-free equilibrium E_0 is globally stable if $R_0<1$.*

Proof. The Jacobian matrix J of (2.2) is

$$\begin{pmatrix} -(\mu+d) & d & -\alpha\beta_N S_N^0 & -\beta_N S_N^0 & 0 \\ d & -(\mu+d) & -\alpha\beta_O S_O^0 & -\beta_O S_O^0 & 0 \\ 0 & 0 & \alpha\beta_N S_N^0-(\mu+\eta) & \beta_N S_N^0 & 0 \\ 0 & 0 & \eta+\alpha\beta_O S_O^0 & \beta_O S_O^0-(\mu+\xi) & \delta \\ 0 & 0 & 0 & \xi & -(\mu+\gamma+\delta) \end{pmatrix}.$$

It is clear that the upper left 2-by-2 matrix of J has the eigenvalues of negative real part. The lower right 3-by-3 matrix of J is exactly $F-V$. By [29], $R_0<1$ implies that all the eigenvalues of $F-V$ have negative real parts. As a consequence, J admits eigenvalues with negative real part. Hence, E_0 is asymptotically stable.

Next, we prove that any nonnegative solution of system (2.2) approaches E_0 as $t\to\infty$. By the first two equations of (2.2) and Lemma 3.1 we get

$$\begin{aligned} &\frac{dS_N}{dt}\le \mu p-\mu S_N+d(S_O-S_N), \\ &\frac{dS_O}{dt}\le \mu(1-p)-\mu S_O+d(S_N-S_O). \end{aligned}$$

Notice that the comparison system

$$\frac{dS_N}{dt}=\mu p-\mu S_N+d(S_O-S_N), \tag{3.2a}$$

$$\frac{dS_O}{dt}=\mu(1-p)-\mu S_O+d(S_N-S_O) \tag{3.2b}$$

has the positive equilibrium (S_N^0, S_O^0) that is globally stable. It follows from the comparison principle that for any $\epsilon > 0$,

$$S_N(t) < S_N^0 + \epsilon, \quad S_O(t) < S_O^0 + \epsilon. \tag{3.3}$$

As a result, by the last three equations of (2.2) we obtain

$$\begin{aligned}
\frac{dI_N}{dt} &\le \beta_N(\alpha I_N + I_O)(S_N^0 + \epsilon) - (\mu + \eta) I_N, \\
\frac{dI_O}{dt} &\le \beta_O(\alpha I_N + I_O)(S_O^0 + \epsilon) - (\mu + \xi) I_O + \eta I_N + \delta T, \\
\frac{dT}{dt} &\le \xi I_O - (\mu + \gamma + \delta) T
\end{aligned}$$

for all large t. Let us consider the comparison system

$$\frac{dI_N}{dt} = \beta_N(\alpha I_N + I_O)(S_N^0 + \epsilon) - (\mu + \eta) I_N, \tag{3.4a}$$

$$\frac{dI_O}{dt} = \beta_O(\alpha I_N + I_O)(S_O^0 + \epsilon) - (\mu + \xi) I_O + \eta I_N + \delta T, \tag{3.4b}$$

$$\frac{dT}{dt} = \xi I_O - (\mu + \gamma + \delta) T. \tag{3.4c}$$

Set

$$F_\epsilon = \begin{pmatrix} \alpha\beta_N(S_N^0 + \epsilon) & \beta_N(S_N^0 + \epsilon) & 0 \\ \alpha\beta_O(S_O^0 + \epsilon) & \beta_O(S_O^0 + \epsilon) & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

Then the coefficient matrix of right-side of (3.4) is $F_\epsilon - V$. Note that $F_\epsilon \to F$ as $\epsilon \to 0$ and that all the eigenvalues of $F - V$ have negative real parts. It follows that all the eigenvalues of $F_\epsilon - V$ have negative real parts when $\epsilon > 0$ is small. Therefore, the solutions of the linear system (3.4) approach zero as $t \to \infty$. Consequently, by the comparison principle we see that any nonnegative solution $(S_N(t), S_O(t), I_N(t), I_O(t), T(t))$ of (2.2) satisfies

$$(I_N(t), I_O(t), T(t)) \to (0,0,0) \quad \text{as } t \to \infty.$$

As a consequence, from the first two equations of (2.2) we get

$$(S_N(t), S_O(t)) \to (S_N^0, S_O^0) \quad \text{as } t \to \infty.$$

The proof is complete. □

Theorem 3.2. *If $R_0 > 1$, the disease is uniformly persistent, i.e., there exists a positive constant ϵ such that any positive solution of* (2.2) *satisfies*

$$\liminf_{t\to\infty} I_N(t) > \epsilon, \quad \liminf_{t\to\infty} I_O(t) > \epsilon.$$

Proof. Define

$$X=\{(S_N,S_O,I_N,I_O,T): S_N,S_O,I_N,I_O,T\geq 0\},$$
$$X_0=\{(S_N,S_O,I_N,I_O,T)\in X: I_N,I_O>0\},$$
$$\partial X_0=\{(S_N,S_O,I_N,I_O,T)\in X: I_N=0 \text{ or } I_O=0\}.$$

Then it suffices to show that (2.2) is uniformly persistent with respect to $(X_0,\partial X_0)$. First, Lemma 3.1 implies that X is positively invariant. By similar discussions we see that X_0 is also positively invariant. Furthermore, Lemma 3.2 means that system (2.2) is point dissipative. Then we denote by J_∂ the largest positively invariant set of (2.2) in ∂X_0, and claim

$$J_\partial=\{(S_N,S_O,0,0,0): S_N,S_O\geq 0\}. \tag{3.5}$$

Let us consider a solution $(S_N(t),S_O(t),I_N(t),I_O(t),T(t))$ of (2.2) in the largest positively invariant set J_∂. By contradiction, we assume that there is a $t_0\geq 0$ such that one of $I_N(t_0),I_O(t_0)$, $T(t_0)$ is positive. First, we suppose $I_N(t_0)>0$. Since $I_O(t_0)>0$ means that the solution does not lie in J_∂, we must have $I_O(t_0)=0$. As a result, we get

$$\frac{dI_O}{dt}\Big|_{t=t_0}\geq \eta I_N(t_0)>0.$$

Thus, $I_O(t)>0$ in $(t_0,t_0+\zeta)$ where $\zeta>0$ is small. This, together with $I_N(t_0)>0$, implies that both $I_O(t)$ and $I_N(t)$ are positive when $t>t_0$ and is close to t_0. This leads to a contradiction. Therefore, we must have $I_N(t_0)=0$. For the case where $I_O(t_0)>0$, it follows from the last equation of (2.2) that $T(t)>0$ when $t>t_0$ and is close to t_0. As a result, the fourth equation of (2.2) indicates that $I_O(t)$ is positive when $t>t_0$ and is close to t_0. Since $\mu p>0$ and $I_O(t)>0$ when $t>t_0$ and is close to t_0, it is easy to see from the first equation that $S_N(t)>0$ when $t>t_0$ and is close to t_0. Consequently, the third equation of (2.2) implies that $I_N(t)>0$ when $t>t_0$ and is close to t_0, which contradicts that the solution is in J_∂. The proof is similar for the case where $T_N(t_0)>0$ or $T_L(t_0)>0$. Therefore, (3.5) is verified.

Note that E_0 is globally stable in J_∂. Let us consider a solution of (2.2) in X_0. we claim that there is a positive constant ρ such that the solution satisfies

$$\limsup_{t\to\infty} I_N(t)>\rho, \quad \limsup_{t\to\infty} I_N(t)>\rho. \tag{3.6}$$

Suppose not. Then since $I_N(t)\to 0$ and $I_O(t)\to 0$ as $t\to\infty$, by similar arguments to those in the proof of Theorem 3.1 we obtain

$$S_N(t)>S_N^0-\epsilon, \quad S_O(t)>S_O^0-\epsilon$$

for all large, where $\epsilon>0$ is arbitrarily fixed. It follows from the last three equations of (2.2) that

$$\frac{dI_N}{dt}\geq \beta_N(\alpha I_N+I_O)(S_N^0-\epsilon)-(\mu+\eta)I_N,$$
$$\frac{dI_O}{dt}\geq \beta_O(\alpha I_N+I_O)(S_O^0-\epsilon)-(\mu+\xi)I_O+\eta I_N+\delta T,$$
$$\frac{dT}{dt}\geq \xi I_O-(\mu+\gamma+\delta)T$$

for all large t. Let us consider the comparison system

$$\frac{dI_N}{dt}=\beta_N(\alpha I_N+I_O)(S_N^0-\epsilon)-(\mu+\eta)I_N, \tag{3.7a}$$

$$\frac{dI_O}{dt}=\beta_O(\alpha I_N+I_O)(S_O^0-\epsilon)-(\mu+\xi)I_O+\eta I_N+\delta T, \tag{3.7b}$$

$$\frac{dT}{dt}=\xi I_O-(\mu+\gamma+\delta)T. \tag{3.7c}$$

Set

$$F^\epsilon=\begin{pmatrix}\alpha\beta_N(S_N^0-\epsilon) & \beta(S_N^0-\epsilon) & 0\\ \alpha\beta_O(S_O^0-\epsilon) & \beta_O(S_O^0-\epsilon) & 0\\ 0 & 0 & 0\end{pmatrix}.$$

Then the coefficient matrix of right-side of (3.7) is $F^\epsilon-V$. Note that $F^\epsilon\to F$ as $\epsilon\to 0$ and that $F-V$ is a cooperative matrix. It follow from the Perron-Frobenius theorem that $F-V$ admits a positive eigenvalue with a positive eigenvector [19]. Thus, $F^\epsilon-V$ has a positive eigenvalue with a positive eigenvector for small $\epsilon>0$. Since (3.7) is a linear cooperative system, it is easy to see that

$$(I_N(t),I_O(t),T(t))\to(\infty,\infty,\infty)\quad\text{as } t\to\infty,$$

which contradicts the assumption that $I_N(t)\to 0$ and $I_O(t)\to 0$ as $t\to\infty$. This proves (3.6). Consequently, we conclude from [12,26,30] that the solutions of (2.2) are uniformly persistent with respect to $(X_0,\partial X_0)$. The proof is complete. □

3.2 Global stability when $d=0$

In this subsection, we ignore the random transition between two susceptible groups. This means that we consider the following reduced system:

$$\frac{dS_N}{dt}=\mu p-\mu S_N-\beta_N(\alpha I_N+I_O)S_N, \tag{3.8a}$$

$$\frac{dS_O}{dt}=\mu(1-p)-\mu S_O-\beta_O(\alpha I_N+I_O)S_O, \tag{3.8b}$$

$$\frac{dI_N}{dt}=\beta_N(\alpha I_N+I_O)S_N-(\mu+\eta)I_N, \tag{3.8c}$$

$$\frac{dI_O}{dt}=\beta_O(\alpha I_N+I_O)S_O-(\mu+\xi)I_O+\eta I_N+\delta T, \tag{3.8d}$$

$$\frac{dT}{dt}=\xi I_O-(\mu+\gamma+\delta)T. \tag{3.8e}$$

The mathematical difficulty is that we cannot obtain the explicit expressions for a positive equilibrium and cannot examine the uniqueness of positive equilibrium by algebraic computations. Nevertheless, since model (3.8) is uniformly persistent when $R_0>1$,

it follows from the persistence theory [26, 30] that there exists at least a positive equilibrium $E^*=(S_N^*,S_O^*,I_N^*,I_O^*,T^*)$. Then we use the powerful Lyapunov stability methods [3, 14, 16, 23, 24] to show that this positive equilibrium is globally stable whenever it exists.

Theorem 3.3. *Let $R_0>1$. Then the positive equilibrium E^* of model* (3.8) *is globally stable.*

Proof. We define auxiliary functions by

$$L_1=S_N-S_N^*-S_N^*\ln\frac{S_N}{S_N^*},\quad L_2=S_O-S_O^*-S_O^*\ln\frac{S_O}{S_O^*}, \tag{3.9a}$$

$$L_3=I_N-I_N^*-I_N^*\ln\frac{I_N}{I_N^*},\qquad L_4=I_O-I_O^*-I_O^*\ln\frac{I_O}{I_O^*}, \tag{3.9b}$$

$$L_5=T-T^*-T^*\ln\frac{T}{T^*}. \tag{3.9c}$$

Recall a fundamental inequality (see, for example, [24])

$$1-x-\ln x\le 0,\quad \text{for } x>0. \tag{3.10}$$

Since E^* is a positive equilibrium, we have

$$\mu S_N^*=\mu p-\beta_N(\alpha I_N^*+I_O^*)S_N^*, \tag{3.11a}$$

$$\mu S_O^*=\mu(1-p)-\beta_O(\alpha I_N^*+I_O^*)S_O^*, \tag{3.11b}$$

$$(\mu+\eta)I_N^*=\beta_N(\alpha I_N^*+I_O^*)S_N^*, \tag{3.11c}$$

$$(\mu+\xi)I_O^*=\beta_O(\alpha I_N^*+I_O^*)S_O^*+\eta I_N^*+\delta T^*, \tag{3.11d}$$

$$(\mu+\gamma+\delta)T^*=\xi I_O^*. \tag{3.11e}$$

Calculating the derivatives of L_1 along the solutions of (2.2), with the aid of (3.10) and (3.11) we get

$$\begin{aligned}\frac{dL_1}{dt}&=-\mu\frac{(S_N-S_N^*)^2}{S_N}+[\alpha\beta_N(S_N^*I_N^*-S_NI_N)+\beta_N(S_N^*I_O^*-S_NI_O)]\left(1-\frac{S_N^*}{S_N}\right)\\&\le\alpha\beta_NS_N^*I_N^*\left(\frac{I_N}{I_N^*}-\ln\frac{I_N}{I_N^*}-\frac{S_NI_N}{S_N^*I_N^*}+\ln\frac{S_NI_N}{S_N^*I_N^*}\right)\\&\quad+\beta_NS_N^*I_O^*\left(\frac{I_O}{I_O^*}-\ln\frac{I_O}{I_O^*}-\frac{S_NI_O}{S_N^*I_O^*}+\ln\frac{S_NI_O}{S_N^*I_O^*}\right),\end{aligned} \tag{3.12}$$

$$\begin{aligned}\frac{dL_2}{dt}&=-\mu\frac{(S_O-S_O^*)^2}{S_O}+[\alpha\beta_O(S_O^*I_N^*-S_OI_N)+\beta_O(S_O^*I_O^*-S_OI_O)]\left(1-\frac{S_O^*}{S_O}\right)\\&\le\alpha\beta_OS_O^*I_N^*\left(\frac{I_N}{I_N^*}-\ln\frac{I_N}{I_N^*}-\frac{S_OI_N}{S_O^*I_N^*}+\ln\frac{S_OI_N}{S_O^*I_N^*}\right)\\&\quad+\beta_OS_O^*I_O^*\left(\frac{I_O}{I_O^*}-\ln\frac{I_O}{I_O^*}-\frac{S_OI_O}{S_O^*I_O^*}+\ln\frac{S_OI_O}{S_O^*I_O^*}\right).\end{aligned} \tag{3.13}$$

Similarly, we have

$$\begin{aligned}\frac{dL_3}{dt}=&\alpha\beta_N S_N^* I_N^*\left(\frac{S_N I_N}{S_N^* I_N^*}-\frac{I_N}{I_N^*}+1-\frac{S_N}{S_N^*}\right)\\&+\beta_N S_N^* I_O^*\left(\frac{S_N I_O}{S_N^* I_O^*}-\frac{I_N}{I_N^*}+1-\frac{S_N I_O I_N^*}{S_N^* I_O^* I_N}\right)\\\leq&\alpha\beta_N S_N^* I_N^*\left(\frac{S_N I_N}{S_N^* I_N^*}-\ln\frac{S_N I_N}{S_N^* I_N^*}-\frac{I_N}{I_N^*}+\ln\frac{I_N}{I_N^*}\right)\\&+\beta_N S_N^* I_O^*\left(\frac{S_N I_O}{S_N^* I_O^*}-\ln\frac{S_N I_O}{S_N^* I_O^*}-\frac{I_N}{I_N^*}+\ln\frac{I_N}{I_N^*}\right),\end{aligned}\tag{3.14}$$

$$\begin{aligned}\frac{dL_4}{dt}=&\alpha\beta_O S_O^* I_N^*\left(\frac{S_O I_N}{S_O^* I_N^*}-\frac{I_O}{I_O^*}+1-\frac{S_O I_N I_O^*}{S_O^* I_N^* I_O}\right)\\&+\beta_O S_O^* I_O^*\left(\frac{S_O I_O}{S_O^* I_O^*}-\frac{I_O}{I_O^*}+1-\frac{S_O}{S_O^*}\right)\\&+\eta I_N^*\left(\frac{I_N}{I_N^*}-\frac{I_O}{I_O^*}+1-\frac{I_N I_O^*}{I_N^* I_O}\right)+\delta T^*\left(\frac{T}{T^*}-\frac{I_O}{I_O^*}+1-\frac{T I_O^*}{T^* I_O}\right)\\\leq&\alpha\beta_O S_O^* I_N^*\left(\frac{S_O I_N}{S_O^* I_N^*}-\ln\frac{S_O I_N}{S_O^* I_N^*}-\frac{I_O}{I_O^*}+\ln\frac{I_O}{I_O^*}\right)\\&+\beta_O S_O^* I_O^*\left(\frac{S_O I_O}{S_O^* I_O^*}-\ln\frac{S_O I_O}{S_O^* I_O^*}-\frac{I_O}{I_O^*}+\ln\frac{I_O}{I_O^*}\right)\\&+\eta I_N^*\left(\frac{I_N}{I_N^*}-\ln\frac{I_N}{I_N^*}-\frac{I_O}{I_O^*}+\ln\frac{I_O}{I_O^*}\right)+\delta T^*\left(\frac{T}{T^*}-\ln\frac{T}{T^*}-\frac{I_O}{I_O^*}+\ln\frac{I_O}{I_O^*}\right),\end{aligned}\tag{3.15}$$

and

$$\begin{aligned}\frac{dL_5}{dt}=&\xi I_O^*\left(\frac{I_O}{I_O^*}-\frac{T}{T^*}+1-\frac{I_O T^*}{I_O^* T}\right)\\\leq&\xi I_O^*\left(\frac{I_O}{I_O^*}-\ln\frac{I_O}{I_O^*}-\frac{T}{T^*}+\ln\frac{T}{T^*}\right).\end{aligned}\tag{3.16}$$

Let us define a Lyapunov function by

$$L=(\alpha\beta_O S_O^*+\eta)I_N^*(L_1+L_3)+\beta_N S_N^* I_O^*(L_2+L_4)+\frac{\beta_N S_N^*\delta T^*}{\xi}L_5.\tag{3.17}$$

It follows from (3.12)-(3.16) that $\frac{dL}{dt}\leq 0$. By simple computations, we see that E^* is the unique point of the maximal compact invariant set in $\{(S_N,S_O,I_N,I_O,T)\in \text{int}R_+^5 : \frac{dL}{dt}=0\}$. Consequently, the Lyapunov-LaSalle invariance principle [15] implies the global stability of E^*. □

3.3 Global stability of full model

In this subsection, we study the global stability of an endemic equilibrium of (2.2) when $R_0>1$. Our strategy is to modify the Lyapunov functions in the last subsection to allow the transition coefficient d to be positive. First, since $R_0>1$, it follows from the persistence theory [26,30] that model (2.2) admits a positive equilibrium $E^*=(S_N^*,S_O^*,I_N^*,I_O^*,T^*)$. Next, we derive the estimates to the higher bounds of susceptible groups in model (2.2). By (3.1), we see that a positive solution of (2.2) satisfies

$$\limsup_{t\to\infty}[S_N+S_O+I_N+I_O+T]\leq 1. \tag{3.18}$$

It follows from the first two equations of (2.2) that

$$\frac{dS_N}{dt}\geq \mu p-\mu S_N-\beta_N S_N+d(S_O-S_N), \tag{3.19a}$$

$$\frac{dS_O}{dt}\geq \mu(1-p)-\mu S_O-\beta_O S_O+d(S_N-S_O), \tag{3.19b}$$

where $0\leq\alpha\leq 1$ is used. Let us replace the inequalities in (3.19) by equalities to obtain a linear system, which has a globally stable equilibrium. Then by the comparison method, it is easy to obtain

$$\liminf_{t\to\infty}S_N(t)\geq \frac{\mu(\beta_O p+\mu p+d)}{\beta_N\beta_O+\beta_N d+\beta_N\mu+\beta_O d+\beta_O\mu+2d\mu+\mu^2}:=\underline{S_N}, \tag{3.20a}$$

$$\liminf_{t\to\infty}S_O(t)\geq \frac{\mu(\mu(1-p)+d+\beta_N(1-p))}{\beta_N\beta_O+\beta_N d+\beta_N\mu+\beta_O d+\beta_O\mu+2d\mu+\mu^2}:=\underline{S_O}. \tag{3.20b}$$

Theorem 3.4. *Let $R_0>1$ and*

$$\frac{d^2}{4(\mu+d)^2}<\frac{(\alpha\beta_O S_O^*+\eta)I_N^*\beta_N S_N^* I_O^*\underline{S_O}\,\underline{S_N}}{((\alpha\beta_O S_O^*+\eta)I_N^*+\beta_N S_N^* I_O^*)^2}. \tag{3.21}$$

Then the positive equilibrium E^* of model (2.2) *is globally stable.*

Proof. We adopt the Lyapunov functions defined in (3.9). Calculating the derivative of L_1 along the solutions of (2.2), we have

$$\begin{aligned}\frac{dL_1}{dt}=&-(\mu+d)\frac{(S_N-S_N^*)^2}{S_N}+\frac{d(S_N-S_N^*)(S_O-S_O^*)}{S_N}\\&+[\alpha\beta_N(S_N^*I_N^*-S_NI_N)+\beta_N(S_N^*I_O^*-S_NI_O)]\left(1-\frac{S_N^*}{S_N}\right)\\\leq&-(\mu+d)\frac{(S_N-S_N^*)^2}{S_N}+\frac{d(S_N-S_N^*)(S_O-S_O^*)}{S_N}\\&+\alpha\beta_N S_N^*I_N^*\left(\frac{I_N}{I_N^*}-\ln\frac{I_N}{I_N^*}-\frac{S_NI_N}{S_N^*I_N^*}+\ln\frac{S_NI_N}{S_N^*I_N^*}\right)\\&+\beta_N S_N^*I_O^*\left(\frac{I_O}{I_O^*}-\ln\frac{I_O}{I_O^*}-\frac{S_NI_O}{S_N^*I_O^*}+\ln\frac{S_NI_O}{S_N^*I_O^*}\right).\end{aligned} \tag{3.22}$$

Similarly, we obtain

$$\begin{aligned}\frac{dL_2}{dt}=&-(\mu+d)\frac{(S_O-S_O^*)^2}{S_O}+\frac{d(S_N-S_N^*)(S_O-S_O^*)}{S_O}\\&+[\alpha\beta_O(S_O^*I_N^*-S_OI_N)+\beta_O(S_O^*I_O^*-S_OI_O)]\left(1-\frac{S_O^*}{S_O}\right)\\\leq&-(\mu+d)\frac{(S_O-S_O^*)^2}{S_O}+\frac{d(S_N-S_N^*)(S_O-S_O^*)}{S_O}\\&+\alpha\beta_OS_O^*I_N^*\left(\frac{I_N}{I_N^*}-\ln\frac{I_N}{I_N^*}-\frac{S_OI_N}{S_O^*I_N^*}+\ln\frac{S_OI_N}{S_O^*I_N^*}\right)\\&+\beta_OS_O^*I_O^*\left(\frac{I_O}{I_O^*}-\ln\frac{I_O}{I_O^*}-\frac{S_OI_O}{S_O^*I_O^*}+\ln\frac{S_OI_O}{S_O^*I_O^*}\right).\end{aligned}\tag{3.23}$$

The derivatives of L_3,L_4,L_5 are identical with those in (3.14), (3.15) and (3.16). Using the Lyapunov function L defined in (3.17), it follows that

$$\begin{aligned}\frac{dL}{dt}\leq&-(\alpha\beta_OS_O^*+\eta)I_N^*\left[(\mu+d)\frac{(S_N-S_N^*)^2}{S_N}-\frac{d(S_N-S_N^*)(S_O-S_O^*)}{S_N}\right]\\&-\beta_NS_N^*I_O^*\left[(\mu+d)\frac{(S_O-S_O^*)^2}{S_O}-\frac{d(S_N-S_N^*)(S_O-S_O^*)}{S_O}\right].\end{aligned}\tag{3.24}$$

By direct calculations, we see that the right-hand side of (3.24) is a negative definite quadratic form of $(S_N-S_N^*)$ and $(S_O-S_O^*)$ if

$$\frac{d^2}{4(\mu+d)^2}<\frac{(\alpha\beta_OS_O^*+\eta)I_N^*\beta_NS_N^*I_O^*S_OS_N}{(\alpha\beta_OS_O^*+\eta)I_N^*S_O+\beta_NS_N^*I_O^*S_N)^2}.\tag{3.25}$$

Since (3.21) is satisfied, by (3.18) and (3.20) we see that (3.25) holds for all large t. By simple computations, we see that E^* is the unique point of the maximal compact invariant set in $\{(S_N,S_O,I_N,I_O,T)\in\text{int}R_+^5:\frac{dL}{dt}=0\}$. Consequently, the Lyapunov-LaSalle invariance principle [15] implies the global stability of E^*. □

Notice that the positive equilibrium E^* of (2.2) is confirmed by the persistence theory and is not explicitly expressed. This means that the condition (3.21) cannot be analytically examined. In order to make (3.21) verifiable, we give the estimates to the positive equilibrium. By the equilibrium equations, we get

$$I_N^*=\frac{\mu p-(\mu+d)S_N^*+dS_O^*}{\mu+\eta},\tag{3.26a}$$

$$I_O^*=\frac{[\mu(1-p)+(d-\eta)S_N^*-(d+\eta+\mu)S_O^*]\mu(\mu+\gamma+\delta)}{(\mu+\eta)(\mu\delta+\gamma\mu+\gamma\xi+\mu^2+\xi\mu)}.\tag{3.26b}$$

Note that S_N^* is bounded below by $\underline{S_N}$ and bounded above by 1, and S_O^* is bounded below by $\underline{S_O}$ and bounded above by 1. We define

$$\varpi = \underline{S_O}\,\underline{S_N} \min\left\{ \frac{(\alpha\beta_O S_O^* + \eta) I_N^* \beta_N S_N^* I_O^*}{(\alpha\beta_O S_O^* + \eta) I_N^* + \beta_N S_N^* I_O^*)^2} : \underline{S_N} \le S_N^* \le 1,\ \underline{S_O} \le S_O^* \le 1 \right\},$$

in which I_N^* and I_O^* are given by (3.26). Consequently, we can state the following verifiable conditions for the global stability of the positive equilibrium of (2.2).

Corollary 3.1. *Let* $R_0 > 1$ *and*

$$\frac{d^2}{4(\mu+d)^2} < \varpi. \tag{3.27}$$

Then the positive equilibrium E^* *of model* (2.2) *is globally stable.*

4 Numerical simulations

In this section, we use numerical simulations to reveal how intervention measures affect the dynamics of disease progression. Motivated by [13], we choose the unit of time in weeks and fix $\beta_N = 0.02$, $\mu = 70 \times 365/7$, $\eta = 0.002899976$, $\delta = 0.000423396496$. Then we select $p = 0.95$, $\beta_O = 0.05$, $\alpha = 1$, $d = 0.001$. For the case where $\xi = 0.001$, $\delta = 0$, which means that only natural recovery for overweight individuals plays the role, the percentage of overweight eating members approaches 19.23% as t becomes large (see the left panel of Fig. 2). If the treatment to the members with overweight eating behaviors take effects such that $\xi = 0.005$, then the size of overweight members is reduced to 4.424% (see the right panel of Fig. 2) for large times.

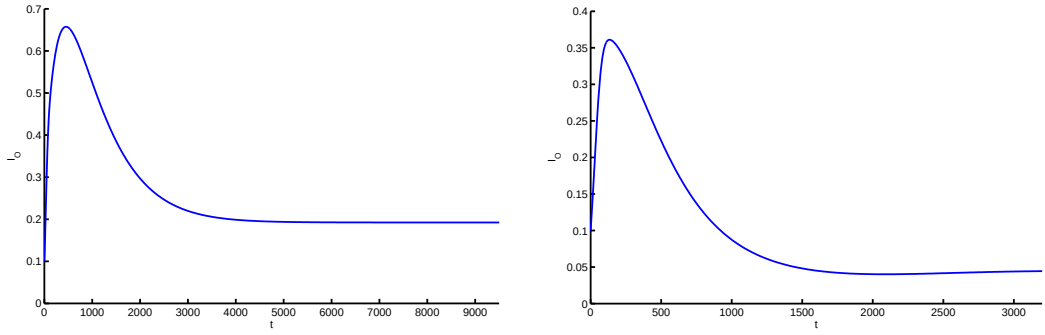

Figure 2: The parameters are fixed as $\mu = 0.0002739726$, $p = 0.95$, $\beta_N = 0.02$, $\beta_O = 0.05$, $\alpha = 1$, $d = 0.001$, $\eta = 0.002899976$, $\delta = 0.000423396496$. The left panel demonstrates that I_O tends to 0.191 as t becomes large where $\xi = 0.001$. The right panel show that the size of obesity is reduced to 0.04464 for large t when $\xi = 0.005$.

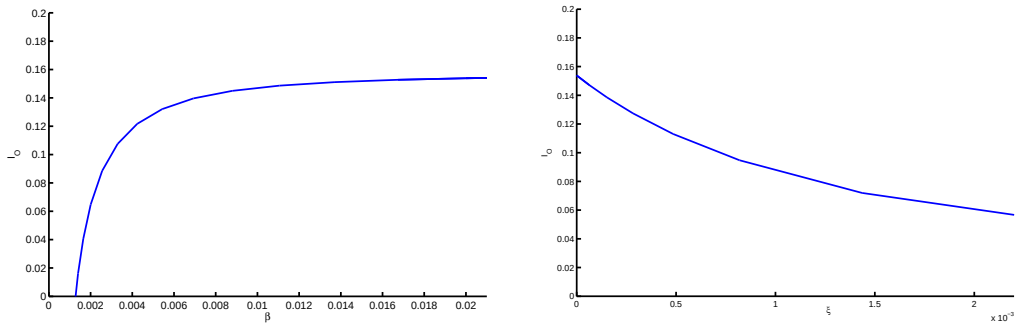

Figure 3: The parameters are fixed as $\mu=0.0002739726$, $p=0.9$, $\alpha=1$, $d=0.001$, $\eta=0.002899976$. The left panel demonstrates that I_O decreases as β decreases where $\xi=0$ and $\delta=0$. The right panel show that I_O decreases quickly as the treatment rate ξ increases where $\delta=0.000423396496$.

In the aim to see the effects of treatment to eating disorder behaviors and education to susceptible members, we modify (2.2) into

$$\frac{dS_N}{dt}=\mu p-\mu S_N-\beta(I_N+I_O)S_N+d(S_O-S_N), \tag{4.1a}$$

$$\frac{dS_O}{dt}=\mu(1-p)-\mu S_O-\beta(I_N+I_O)S_O+d(S_N-S_O), \tag{4.1b}$$

$$\frac{dI_N}{dt}=\beta(I_N+I_O)S_N-(\mu+\eta+r+\xi)I_N, \tag{4.1c}$$

$$\frac{dI_O}{dt}=\beta(I_N+I_O)S_O-(\mu+r+\xi)I_O+\eta I_N+\delta T, \tag{4.1d}$$

$$\frac{dT}{dt}=\xi(I_N+I_O)-(\mu+\gamma+\delta)T, \tag{4.1e}$$

where r is the natural recovery rate of individuals with overeating behaviors, and β is the infection coefficient.

Now, we fix $\mu=70\times365/7$, $\eta=0.002899976$ and select $p=0.9$, $\alpha=1$, $d=0.001$, $r=0.001$. For the case where $\xi=0$ and $\delta=0$, that is, there is no treatment to overeating behaviors, the reduction of infection coefficient β decreases the size of I_O numbers. In fact, the percentage of overweight members with eating disorder behaviors arrives below 5% until β is reduced from 0.02 to 0.002 (see the left panel of Fig. 3). In contrast, the size is below 5% when $\xi=0.0025$, $\delta=0.0004233965$ and $\beta=0.02$, which is shown in the right panel of Fig. 3. Therefore, either the eduction to protect the disease infection or treatment of eating disorder behaviors can control the spread of overweight epidemic disease.

5 Discussions

In this paper, we have incorporated the overeating behaviors and individual heterogeneity into the model of obesity. By mathematical analysis, we have shown that the basic

reproduction number R_0 of the disease is a threshold for disease invasion. That is, the disease dies out if $R_0<1$ and is uniformly persistent if $R_0>1$. Furthermore, by constructing suitable Lyapunov functions, we have obtained the sufficient conditions for the global stability of an endemic equilibrium, even though there is no explicit expressions of the endemic equilibrium or no information for the uniqueness of the endemic equilibrium. In addition, we use numerical simulations to reveal how intervention measures through treatment to eating behaviors or through eduction to susceptible individuals can suppress the progression of the disease. These results give the basis for the design of optimal strategy in controlling the obesity disease.

It will be interesting to fit real data with model (2.2) to give predictive suggestions. Moreover, the sufficient conditions for the global stability of an endemic equilibrium in Theorem 3.4 may be improved because numerous numerical simulations indicate it is always globally stable. Further, the random transition between two susceptible groups can be improved by introducing a larger transition rate from overweight susceptible individuals to normal susceptible members due to interventions from physical exercise or reduction of food uptakes. We leave these as future researches.

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