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MODELING AND ANALYSIS OF SOCIAL OBESITY EPIDEMIC

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Abstract Overweight and obesity have become a global epidemic due to increasing unhealthy eating habits and sedentary lifestyles. An individual can gain weight excessively through social influence, and understanding its underlying interpersonal dynamics is crucial for effective intervention and prevention programs. By considering the social effects on weight gain, this paper presents a compartment model to describe the social spread of overweight and obesity. Bifurcation analysis suggests that a backward bifurcation exists when the relative hazard of weight regain is a larger value. Strategies for eliminating the overweight and obesity epidemic are provided by analyzing the obesity-free equilibrium globally by incorporating Lyapunov functions and the method of fluctuations. Since the pervasiveness of overweight and obesity in the United States seems to be stabilized, we analyze the local stability of the obesity-endemic equilibrium to establish a condition for the plateau, by applying a matrix theoretic method that utilizes compound matrices. The results suggest that weight loss programs can help maintain the plateau; however, weight loss maintenance programs should be promoted to eliminate the disease.

Keywords Obesity, saddle-node bifurcation, bi-stability, global stability.

MSC(2010) 37N25, 34C20, 34C23, 34D23.

1. Introduction

Being overweight or obese is a major health concern in many countries, often leading to various non-infectious diseases including cardiovascular diseases, diabetes, and certain types of cancers. The root cause of overweight and obesity is the lack of balance between calorie intake and expenditure. Globally, inactivity and high-calorie food consumption have increased due to the sedentary nature of life. Furthermore, changes in eating habits and physical activity are mainly the consequences of social and environmental changes. Social contacts and peer pressure [6] attributed to excess weight gain. As a result, the global overweight and obesity pandemic has recently been identified as a social contagion disease. Another feature of the overweight and obesity epidemic is that its prevalence appears to stabilize at a plateau, according to the National Center for Health Statistics (NCHS) [14]. To design a

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better control strategy, it is crucial to consider the social influence of relationships and identify the causal mechanisms of excess weight gain.

An individual's weight tends to gradually become closer to those of his social circle [19]. More precisely, social contagion in excess weight gain conveys the idea that the weight outcomes of individuals in a social network increase the probability of weight changes in other individuals in the current social network due to changing norms or mirroring [6]. According to Dater and Nicosia [6], studying the social contagion nature of excess weight gain would greatly favor interventions targeting social networks. Therefore, a comprehensive understanding of the overweight and obesity epidemic and its control is likely to include social influence [17]. In addition, due to the rapid growth of the obesity epidemic, public awareness is significantly increased. Thanks to the efforts of public health officials and government organizations, the overall prevalence of excess weight gain in the United States has stabilized in recent years [26]. However, further interventions in this plateau condition should be needed to ensure that obesity prevalence does not reflect a plateau, but also declines. Therefore, it is essential to understand the reasons behind the plateau to take adequate measures to decrease the current trend of overweight and obesity prevalence or maintain the currently observed leveling curve.

Mathematical models provide a powerful tool for describing disease spread and can provide general insight into the biological and sociological mechanisms of a disease. Numerous studies specifically examine the interconnection between peer pressure and obesity [5, 9, 15, 22]. The approach that considers interpersonal excess weight transmission has resulted in wide-ranging studies on mathematical modeling of the obesity epidemic. In this manner, the study [16] proposes a compartmental model to analyze the social obesity epidemic in Valencia, Spain. The authors adopt a bi-linear incidence rate and consider healthy-weight, overweight and obese subpopulations. Using sensitivity analysis through numerical simulations, they suggest strategies to control the increasing trend of obesity and conclude that preventive health campaigns are more effective than treatment programs. The same effect is considered in the study [11] to discuss the dynamics of infant obesity. The presented model has six sub-populations: healthy weight, latent, overweight, obese, overweight on a diet, and obese on a diet. The authors use numerical simulations to predict the growing trend of childhood obesity in the future. Considering the same subpopulations, the study [1] analyses a non-autonomous obesity model and reveals the periodic behavior of the solutions. They obtain sufficient conditions to ensure the existence of a periodic positive solution. Another mathematical model [21] provides mechanistic insights into the leveling obesity prevalence and predicts overweight, obesity, and extreme obesity prevalence trends. The authors establish numerical conditions to make the obesity prevalence a plateau. Due to the complexity of the models, most of the previous work explores the obesity prevalence trends numerically. The current literature still lacks analytical formulas for plateau and complete overweight and obesity epidemic elimination, which can better perceive the relation between critical parameters and the model dynamics [3]. In this project, we incorporate social influence to model the obesity dynamics and derive both the threshold for overweight and obesity epidemic eradication under the appearance of backward bifurcation and the sufficient parameter conditions for the stable obesity-endemic equilibrium representing the overweight and obesity epidemic plateau. Moreover, by performing symbolic computations, we overcome computational challenges in analysis.

We have organized the paper as follows. Section 2 presents the model formulation, and later we reduce the system to a three-dimensional one. Sections 3 and 4 bring the basic properties of the solution and discuss the existence of equilibrium points in the feasible region. Next, in Section 5, we calculate the basic reproduction number $\mathcal{R}_0$ and conduct a sensitivity analysis for $\mathcal{R}_0$. Furthermore, by applying the center manifold theorem, we establish a closed-form formula for the occurrence of backward bifurcation in Section 6. Section 7 discusses two strategies to eliminate the overweight and obesity epidemic by analyzing the global stability of the obesity-free equilibrium. In Section 8, we carry out the local stability analysis of the obesity-endemic equilibrium to provide parameter conditions behind the plateau. We further compute the normal form of the saddle-node bifurcation for obesity-endemic equilibrium. Section 9 provides a discussion of the results, and the final section presents our conclusions for the study.

2. Model formation

Excess weight gain (overweight/ obesity) can be treated as a disease transmitted by social influence through habits of an unhealthy lifestyle. By considering the social contagion of obesity, our model is written as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \mu N - \lambda(W,B)\,S - \mu S,\\
\frac{dW}{dt} &= \lambda(W,B)\,S + \sigma\lambda(W,B)\,R + \gamma_1 B - (\alpha+\gamma_2+\mu)W,\\
\frac{dB}{dt} &= \alpha W - (\gamma_1+\mu)B,\\
\frac{dR}{dt} &= \gamma_2 W - \sigma\lambda(W,B)\,R - \mu R, \quad \text{where}\\
\lambda(W,B) &= m_1 W + m_2 B.
\end{aligned} \tag{2.1}$$

Model features are listed as follows.

1. Based on an established model [7], which considers healthy weight, obese and ex-obese sub-populations, we further include the overweight sub-population because overweight people have an easier experience of recovering the healthy weight and have a different level of social influence than that of obese people.
2. According to the classification of body mass index (BMI), our model (2.1) has four compartments, which are never overweight ($S(t)$ with BMI below $25kg/\ m^2$), overweight ($W(t)$ with $25 < BMI < 30kg/m^2$), obese ($B(t)$ with $30 < BMI < 40kg/m^2$), and recovered to healthy weight($R(t)$ with BMI below $25kg/m^2$).
3. The birth and death rate is μ.
4. The transmission rates from healthy weight to overweight under the influence of overweight and obese are m_1 and m_2, respectively.
5. According to a report by the World Health Organization (WHO), about 39% of the global adult population was overweight and 13% were obese in 2016 [27], we assume $m_1 > m_2$.

6. The transmission rate from overweight to obesity is α.
7. The natural recovery rates from obesity to the overweight stage and from overweight to the healthy weight stage are γ_1 and γ_2, respectively.
8. In the recovered stage, the risk of weight regain in recovered individuals is much higher than the weight gain of a never-overweight individual to an overweight. The term of relapse is given by $\sigma\lambda = \sigma(m_1W + m_2B)$. The parameter σ is the relative risk of weight regain among recovered individuals. Its value exceeds one due to the high risk of returning to the overweight state.
9. The parameter N denotes the population size and, for simplicity and clarity, we normalize it to unity. All other parameters are positive and are given in Table 1.

The flow diagram of the model dynamics is shown in Fig. 1. Here, it is assumed that the contagious risks from the overweight and obesity compartments are independent of each other. Healthy weight individuals, who are socially influenced by overweight and obese people and addicted to unhealthy lifestyles, begin to progress toward being overweight. Overweight can progress to becoming obese if they choose to continue their sedentary lifestyle or can be recovered with a healthy weight if they choose to take actions to prevent gaining excess weight. Obese people can also reduce their weight to the overweight stage if they choose a healthy lifestyle.

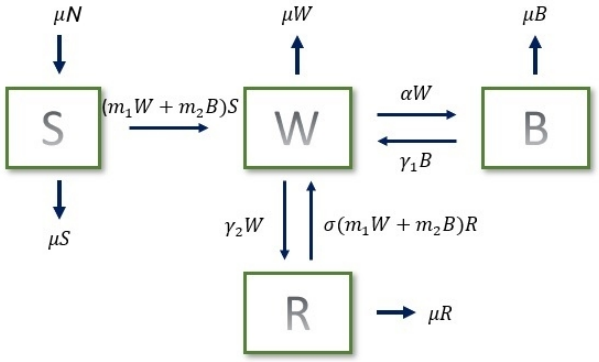


Figure 1. Flow diagram of model (2.1) for overweight and obesity dynamics.

3. Forward invariance

Proposition 3.1. *For model* (2.1)*, the set*

$$\Gamma = \left\{(S, W, B, R) \in \mathbb{R}^4_+ : S + W + B + R = 1\right\}, \tag{3.1}$$

is forward invariant. Moreover, the solution of model (2.1) *exists globally.*

Proof. For a given set of initial conditions, $(S(0), W(0), B(0), R(0)) \in \overline{\mathbb{R}^4_+}$, the smooth functions in the right-hand side of model (2.1) guarantee the local existence and uniqueness of the solutions.

Table 1. Descriptions and values [7,21] of parameters in model (2.1).

Param.	Definition	Value
μ	Natural birth/death rate	0.0144
m_1	Transmission rate from never overweight to overweight by social influence of overweight	0.04
m_2	Transmission rate from never overweight to overweight by social influence of obese	0.02
α	Spontaneous rate of weight gain from overweight to obesity	0.08
γ_1	Rate of weight loss from obesity to overweight	0.03
γ_2	Rate of weight loss from overweight to healthy weight	0.033
σ	Rate of relative hazard of weight regain from normal to overweight	5

Defining the total population as $N_T(t) = S(t) + W(t) + B(t) + R(t)$, we have

$$\frac{dN_T(t)}{dt} = \frac{d}{dt}[S(t) + W(t) + B(t) + R(t)] = \mu[N - N_T(t)], \tag{3.2}$$

or

$$\frac{d}{dt}[N - N_T(t)] = -\mu[N - N_T(t)]. \tag{3.3}$$

Furthermore, considering the initial condition $N_T(0) = S(0) + W(0) + B(0) + R(0)$, it follows

$$N - N_T(t) = (N - N_T(0))e^{-\mu t}, \quad t \geq 0. \tag{3.4}$$

Equation (3.4) provides two cases for $N_T(t)$. Case 1: If $N_T(0) = N$, then $N_T(t) = N$ for all $t \geq 0$. Case 2: If $N_T(0) \neq N$, then $\lim_{t\to+\infty} N_T(t) = N$ for all $t \geq 0$. Without loss of generality, normalize the total population to unity for all $t \geq 0$, where $N_T = S(t) + W(t) + B(t) + R(t) = 1$. In summary, the total population either remains a constant value of one or approaches one in the forward time.

Moreover, the solution of model (2.1) with $(S(0), W(0), B(0), R(0)) \in \overline{\mathbb{R}^4_+}$ will remain in the non-negative cone $\overline{\mathbb{R}^4_+}$ for all $t \geq 0$, because

$$\begin{aligned}
&\frac{dS}{dt}|_{S=0} = \mu N > 0,\\
&\frac{dW}{dt}|_{W=0} = m_2 B(S + \sigma R) + \gamma_1 B > 0,\\
&\frac{dB}{dt}|_{B=0} = \alpha W > 0, \quad \text{and}\\
&\frac{dR}{dt}|_{R=0} = \gamma_2 W > 0.
\end{aligned} \tag{3.5}$$

That is, the vector field of the model (2.1) on $\partial\mathbb{R}^4_+$ (the boundary of $\mathbb{R}^4_+$) is either tangential to $\partial\mathbb{R}^4_+$ or pointing towards $\mathbb{R}^4_+$.

Therefore, all solutions of model (2.1) starting from $\overline{\mathbb{R}^4_+}$ will eventually enter the closed region (3.1) forward in time. □

4. Existence of equilibrium solutions

Noticing that the total population is conserved, that is, $\lim_{t\to+\infty} N_T(t) = 1$ for all $t \geq 0$, we have the identity $S(t) = N_T(t) - W(t) - B(t) - R(t) = 1 - W(t) - B(t) - R(t)$, thus we can ignore the first equation in model (2.1) and investigate its equivalent system

$$\begin{aligned} \frac{dW}{dt} &= (m_1 W + m_2 B)(1 - W - B - R + \sigma R) + \gamma_1 B - (\mu + \alpha + \gamma_2)W, \\ \frac{dB}{dt} &= \alpha W - (\gamma_1 + \mu)B, \\ \frac{dR}{dt} &= \gamma_2 W - \sigma(m_1 W + m_2 B)R - \mu R, \end{aligned} \tag{4.1}$$

in the following restricted region:

$$\Omega = \{(W, B, R) : 0 \leq W, B, R \leq 1, 0 \leq W + B + R \leq 1\}. \tag{4.2}$$

Setting $\frac{dW}{dt} = \frac{dB}{dt} = \frac{dR}{dt} = 0$, we derive the equilibrium solutions $E^* = (W^*, B^*, R^*)$, where

$$\begin{aligned} B^* &= \frac{\alpha W^*}{(\gamma_1 + \mu)}, \\ R^* &= \frac{(\gamma_1 + \mu)\gamma_2 W^*}{\sigma(m_1\,\gamma_1 + m_1\mu + m_2\,\alpha)W^* + \gamma_1\mu + \mu^2}. \end{aligned} \tag{4.3}$$

Here W^* is the root of the cubic equation

$$W(a_2 W^2 + a_1 W + a_0) = 0, \tag{4.4}$$

where

$$\begin{aligned} a_2 &= \sigma(\alpha + \gamma_1 + \mu)(m_1\gamma_1 + m_1\mu + m_2\alpha)^2 > 0, \\ a_1 &= (\sigma + 1)\mu^2 - [(m_1 - \alpha - \gamma_1)\sigma - \alpha - \gamma_1 - \gamma_2]\mu \\ &\quad -(m_1\gamma_1 + m_2\alpha)\sigma + \gamma_1\gamma_2, \\ a_0 &= \alpha\mu(\gamma_1 + m_2)(\gamma_1 + \mu)^2\frac{1 - \mathcal{R}_0^2}{\mathcal{R}_0^2}, \\ \mathcal{R}_0^2 &= \frac{\alpha\ (m_2 + \gamma_1)}{(\gamma_1 + \mu)\,(\alpha + \gamma_2 + \mu - m_1)}. \end{aligned} \tag{4.5}$$

We have the following result for the existence of the solutions.

Theorem 4.1. *For equation* (4.4)*, the trivial root* $W^* = 0$ *always exits and corresponds to the boundary obesity-free equilibrium* $E_0 = (W^*, B^*, R^*) = (0, 0, 0)$*. The non-trivial root of equation* (4.4) *exists if and only if* $\Delta = a_1^2 - 4\,a_2\,a_0 > 0$*. Furthermore, only one positive non-trivial root exists if* $a_0 < 0$*, and two positive non-trivial roots exist if* $a_1 < 0$ *and* $a_0 > 0$*. The non-trivial root derives obesity-endemic equilibrium* $E^* = (W^*, B^*, R^*)$ *for model* (2.1)*.*

5. Basic reproduction number $\mathcal{R}_0$

The basic reproduction number ($\mathcal{R}_0$) is the number of individuals with unhealthy weight generated by one overweight or obese individual in a population where all individuals are susceptible to weight gain. The overweight and obesity epidemic will spread out if $\mathcal{R}_0 > 1$, or die out if $\mathcal{R}_0 < 1$. Hence, the value $\mathcal{R}_0$ is a crucial threshold when determining the elimination of the overweight and obesity epidemic. We calculate the basic reproduction number by using the next-generation matrix method [24]. Consider the state variables $x = (x_1, x_2, x_3)^T = (W, B, R)^T$ in the model (4.1). For the unhealthy weight class $(x_1, x_2)^T$, we have $\frac{dx_i}{dt} = \mathcal{F}_i(x) - \mathcal{V}_i(x)$ for $i = 1, 2$, where $\mathcal{F}(x)$ denote the rates of new overweight and obesity appearances and $\mathcal{V}(x)$ denote rates of transitions between compartments. By defining F and V as

$$\begin{aligned} F &= \left[\frac{\partial \mathcal{F}_i(x)}{\partial x_j}|_{x=E_0}\right]_{i=1,2} = \begin{bmatrix} 0 & m_2+\gamma_1 \\ \alpha & 0 \end{bmatrix} \quad \text{and,} \\ V &= \left[\frac{\partial \mathcal{V}_i(x)}{\partial x_j}|_{x=E_0}\right]]_{i=1,2} = \begin{bmatrix} \alpha+\gamma_2+\mu-m_1 & 0 \\ 0 & \gamma_1+\mu \end{bmatrix}, \end{aligned} \tag{5.1}$$

we have

$$\mathcal{R}_0 = \rho\left(FV^{-1}\right) = \sqrt{\frac{\alpha\,(m_2+\gamma_1)}{(\gamma_1+\mu)\,(\alpha+\gamma_2+\mu-m_1)}}, \tag{5.2}$$

where $\rho(\cdot)$ denotes the spectral radius and we assume

$$\alpha+\gamma_2+\mu-m_1 > 0. \tag{5.3}$$

Moreover, the threshold value $\mathcal{R}_0(m_1) = 1$ is equivalent to

$$m_1 \triangleq \hat{m}_1 = \frac{\mu^2+(\alpha+\gamma_1+\gamma_2)\,\mu-\alpha\,m_2+\gamma_1\,\gamma_2}{(\gamma_1+\mu)}. \tag{5.4}$$

5.1. Sensitivity analysis

To obtain the impact of the parameters on $\mathcal{R}_0$ and the unhealthy weight class ($W(t)$ and $B(t)$), we conduct a sensitivity analysis. First, we calculate the normalized forward sensitivity index of $\mathcal{R}_0$ [4] to measure the relative variance of $\mathcal{R}_0$ to the relative variation of the parameters, as follows:

$$\gamma_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0}. \tag{5.5}$$

The higher $\gamma_p^{\mathcal{R}_0}$ value means the more sensitivity the parameter to $\mathcal{R}_0$. Furthermore, the positive (negative) sign denotes the positive (negative) relation between $\mathcal{R}_0$ and the parameter p. Taking into account all the model parameters, we have the

following.

$$\begin{aligned}
\gamma_{\mu}^{\mathcal{R}_0} &= \frac{-1}{2}\frac{(m_1-\alpha-\gamma_1-\gamma_2-2\mu)\,\mu}{(m_1-\alpha-\gamma_2-\mu)\,(\gamma_1+\mu)}, & \gamma_{m_1}^{\mathcal{R}_0} &= \frac{-m_1}{2m_1-2\alpha-2\gamma_2-2\mu},\\
\gamma_{m_2}^{\mathcal{R}_0} &= \frac{m_2}{2m_2+2\gamma_1}, & \gamma_{\alpha}^{\mathcal{R}_0} &= \frac{m_1-\gamma_2-\mu}{2m_1-2\alpha-2\gamma_2-2\mu},\\
\gamma_{\gamma_1}^{\mathcal{R}_0} &= -\frac{1}{2}\frac{(m_2-\mu)\,\gamma_1}{(m_2+\gamma_1)\,(\gamma_1+\mu)}, & \gamma_{\gamma_2}^{\mathcal{R}_0} &= \frac{\gamma_2}{2m_1-2\alpha-2\gamma_2-2\mu},\\
\gamma_{\sigma}^{\mathcal{R}_0} &= 0.
\end{aligned} \tag{5.6}$$

Taking the parameter values in Table 1, the calculated sensitivity indices are $\gamma_{\mu}^{\mathcal{R}_0} = -0.24454$, $\gamma_{m_1}^{\mathcal{R}_0} = 0.22883$, $\gamma_{m_2}^{\mathcal{R}_0} = 0.2$, $\gamma_{\alpha}^{\mathcal{R}_0} = 0.04233$, $\gamma_{\gamma_1}^{\mathcal{R}_0} = -0.0378$, and $\gamma_{\gamma_2}^{\mathcal{R}_0} = -0.1888$. The most sensitive parameter to $\mathcal{R}_0$ is the natural birth/death rate μ. A higher birth / death rate produces a larger category of healthy weight. It implies an inverse effect of increasing the basic reproduction number. i.e., an increase in the birth / death rate by 10% will decrease $\mathcal{R}_0$ by 2.4454%. Similarly, an increase of 10% in the values of γ_1, α, m_2, and m_1 will increase the basic reproduction number by 0.3784%, 1.8602%, 2 %, and 2.2883 %, respectively. While an increase in γ_2 of 10% will reduce the basic reproduction number by 1.8879%.

6. Backward bifurcation near E_0 and $\mathcal{R}_0 = 1$

The local stability of the obesity-free equilibrium $E_0 = (0, 0, 0)$ is determined by the eigenvalues of the Jacobian matrix

$$J|_{E_0} = \begin{bmatrix} -(\alpha+\gamma_2+\mu-m_1) & m_2+\gamma_1 & 0\\ \alpha & -(\gamma_1+\mu) & 0\\ \gamma_2 & 0 & -\mu \end{bmatrix}, \tag{6.1}$$

which are $\lambda_1 = -\mu$ and

$$\begin{aligned}
\lambda_{2,3} &= \frac{-(M_1+M_2) \pm \sqrt{(M_1+M_2)^2 - 4\,M_1\,M_2(1-\mathcal{R}_0^2)}}{2},\\
M_1 &= \alpha+\gamma_2+\mu-m_1 > 0,\\
M_2 &= \gamma_1+\mu.
\end{aligned} \tag{6.2}$$

Here, $\lambda_{2,3}$ are both negative if $0 < \mathcal{R}_0 < 1$, have opposite signs if $\mathcal{R}_0 > 1$, and have one zero value if $\mathcal{R}_0 = 1$.

Theorem 6.1. *E_0 is locally asymptotically stable if $0 < \mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

To determine the dynamics of the model at $\mathcal{R}_0 = 1$, where $J|_{E_0}$ in (6.1) possesses one zero and two negative eigenvalues, we derive the corresponding center

manifold [25, 29]. Defining $x = (x_1, x_2, x_3)^T = (W, B, R)^T$ and $f = (f_1, f_2, f_3)^T = \left(\frac{dW}{dt}, \frac{dB}{dt}, \frac{dR}{dt}\right)^T$, model (4.1) is transformed to

$$\frac{dx}{dt} = f(x, \phi), \tag{6.3}$$

where $\phi = (\phi_f, \phi_b) \in \mathbb{R}^8_+$ represents all the parameters for model (4.1). ϕ_f and ϕ_b denote the fixed and bifurcation parameters, respectively. Being one of the most sensitive parameters to $\mathcal{R}_0$ from the results in Subsection 5.1 and can be changed by public health efforts, m_1, the transmission rate from a healthy weight to overweight is selected as the bifurcation parameter. Then, the Jacobian matrix for the obesity model (4.1) at E_0 and $m_1 = \hat{m}_1$ at (5.4) (or $\mathcal{R}_0 = 1$)

$$J|_{E_0,\hat{m}_1} = \begin{bmatrix} -\dfrac{\alpha\,(m_2+\gamma_1)}{\gamma_1+\mu} & m_2+\gamma_1 & 0 \\ \alpha & -(\gamma_1+\mu) & 0 \\ \gamma_2 & 0 & -\mu \end{bmatrix}, \tag{6.4}$$

has one zero eigenvalue and two negative eigenvalues $-\mu$ and $-\frac{\alpha m_2 N + \gamma_1\,\alpha + (\gamma_1+\mu)^2}{\gamma_1+\mu}$. Next, we choose the eigenvectors (right and left) for the zero eigenvalue as

$$w = \left(\frac{\gamma_1+\mu}{\alpha}, 1, \frac{\gamma_2\,(\gamma_1+\mu)}{\alpha\,\mu}\right)^T \quad \text{and} \quad v = \left(\frac{1}{n}, \frac{m_2+\gamma_1}{n(\gamma_1+\mu)}, 0\right), \tag{6.5}$$

where $n = \frac{\gamma_1+\mu}{\alpha} + \frac{m_2+\gamma_1}{\gamma_1+\mu}$ and $< w, v >= 1$.

Defining the perturbation as $\nu = m_1 - \hat{m}_1$, the center manifold up to the second order is

$$\frac{dc}{dt} = \frac{1}{2}ac^2 + b\nu c, \tag{6.6}$$

where the coefficients a and b are

$$\begin{aligned} a &= \textstyle\sum_{i,j,k=1}^{3} v_i w_j w_k \dfrac{\partial^2 f_i}{\partial x_j \partial x_k}|_{(E_0,\hat{m}_1)} \\ &= -\frac{2(\gamma_1+\mu)\left(\mu^2 + (\alpha+\gamma_1+\gamma_2)\,\mu + \gamma_1\,\gamma_2\right) a_m}{\alpha\,\left({\gamma_1}^2 + (\alpha+2\,\mu)\,\gamma_1 + m_2\alpha + \mu^2\right)\mu} \quad \text{and,} \\ b &= \textstyle\sum_{i,k=1}^{3} v_k w_i \dfrac{\partial^2 f_k}{\partial x_i \partial \nu}|_{(E_0,\hat{m}_1)} = \dfrac{(\gamma_1+\mu)^2}{{\gamma_1}^2 + (\alpha+2\,\mu)\,\gamma_1 + \alpha\,m_2 + \mu^2} > 0. \end{aligned} \tag{6.7}$$

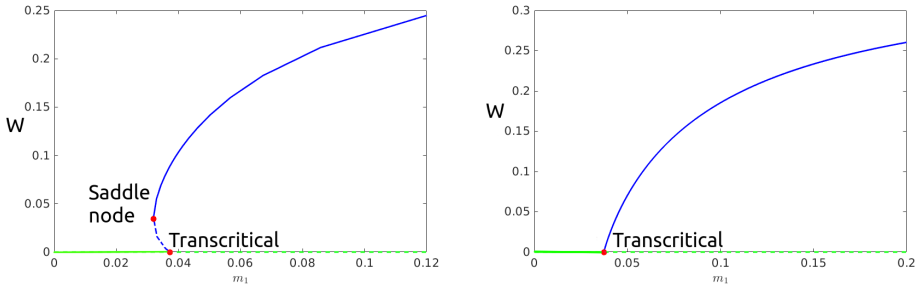
Here, $a_m = \mu^2 + [(1-\sigma)\,\gamma_2 + \gamma_1 + \alpha]\,\mu - \gamma_1\,\gamma_2\,(\sigma - 1)$. Since the value of b is always positive, the sign of a, which is decided by the sign of a_m, determines the direction of this transcritical bifurcation.

Also, note that all other non-zero factors in (6.7) are $\frac{\partial^2 f_1}{\partial x_1^2}|_{(E_0,\hat{m}_1)} = -2\hat{m}_1$, $\frac{\partial^2 f_1}{\partial x_1 \partial x_2}|_{(E_0,\hat{m}_1)} = \frac{\partial^2 f_1}{\partial x_2 \partial x_1}|_{(E_0,\hat{m}_1)} = -\hat{m}_1 - m_2$, $\frac{\partial^2 f_1}{\partial x_2^2}|_{(E_0,\hat{m}_1)} = -2m_2$, $\frac{\partial^2 f_1}{\partial x_2 \partial x_3}|_{(E_0,\hat{m}_1)} = \frac{\partial^2 f_1}{\partial x_3 \partial x_2}|_{(E_0,\hat{m}_1)} = m_2(\sigma - 1)$ $\frac{\partial^2 f_1}{\partial x_1 \partial x_3}|_{(E_0,\hat{m}_1)} = \frac{\partial^2 f_1}{\partial x_3 \partial x_1}|_{(E_0,\hat{m}_1)} = \hat{m}_1(\sigma - 1)$, and $\frac{\partial^2 f_1}{\partial x_1 \partial m_1}|_{(E_0,\hat{m}_1)} = 1$.

Theorem 6.2. *The obesity epidemic model* (4.1) *admits a forward (backward) bifurcation if* $a_m > 0\,(a_m < 0)$ *and* $\mathcal{R}_0 = 1$. *Furthermore,* $a_m > 0$ *is equivalent to*

$$\sigma < \sigma^* = \frac{\mu^2 + (\alpha + \gamma_1)\,\mu}{\gamma_2\,(\gamma_1 + \mu)} + 1. \tag{6.8}$$

The parameter values in Table 1 derive a threshold value for σ as $\sigma^* = \frac{11113}{5000} \approx 2.22$. If we take $\sigma = 1.5 < 2.22$, Theorem 6.2 predicts a forward bifurcation. If we take $\sigma = 5 > 2.22$, Theorem 6.2 predicts a backward bifurcation. The bifurcation diagrams in Fig. 2 verify the backward and forward bifurcation cases.



(a) $\sigma = 5$ (b) $\sigma = 1.5$

Figure 2. Backward and forward bifurcation cases are plotted in (a) and (b). The obesity-free equilibrium E_0 (in green) and the obesity-endemic equilibrium E^* (in blue) intersect at the transcritical bifurcation point and change their stability. Solid and dashed lines represent stable and unstable equilibria.

7. Two strategies for complete obesity elimination

7.1. Strategy I: Reduce $\mathcal{R}_0$ less than the obesity elimination threshold

Due to the appearance of the backward bifurcation, being $\mathcal{R}_0 < 1$ is not sufficient for the complete elimination of the obesity and overweight epidemic. In this subsection, we will derive a new threshold to eliminate the obesity epidemic, guaranteeing the global stability of the obesity-free equilibrium, E_0.

Proposition 3.1 states that, starting from non-negative, the solutions of system (4.1) remain non-negative and bounded. By the Bolzano-Weierstrass theorem, infinite bounded sequences $W(\tau_n)$, $B(\tau_n)$, and $R(\tau_n)$ have convergent subsequences, respectively. By the method of fluctuations [20, 30, 31], there exists a sequence $\{\tau_n\} \in \mathbb{R}$ satisfying $\lim_{n\to+\infty} \tau_n = +\infty$, such that

$$\lim_{n\to+\infty} W(\tau_n) = W^\infty, \lim_{n\to+\infty} B(\tau_n) = B^\infty, \lim_{n\to+\infty} R(\tau_n) = R^\infty,$$
$$\lim_{n\to+\infty} \dot{W}(\tau_n) = 0, \lim_{n\to+\infty} \dot{B}(\tau_n) = 0, \quad \text{and} \quad \lim_{n\to+\infty} \dot{R}(\tau_n) = 0,$$

where the superscript $^\infty$ and the subscript $_\infty$ denote the upper and lower limits. That is, $x^\infty = \limsup_{t\to+\infty} x(t)$ and $x_\infty = \liminf_{t\to+\infty} x(t)$ for a continuous and bounded

function $x : [0, +\infty) \to \mathbb{R}$. Moreover, Proposition 3.1 also guarantees that $0 \leq W_\infty < W^\infty$, $0 \leq B_\infty < B^\infty$, and $0 \leq R_\infty < R^\infty$.

The second equation of (4.1) yields

$$\dot{B}(\tau_n) + (\gamma_1 + \mu)\, B(\tau_n) = \alpha\, W(\tau_n). \tag{7.1}$$

It follows

$$B^\infty \leq \frac{\alpha}{\gamma_1 + \mu} W^\infty, \quad \text{as} \quad n \to +\infty. \tag{7.2}$$

The third equations of (4.1) derives

$$\dot{R}(\tau_n) + \sigma\,(m_1\, W(\tau_n) + m_2\, B(\tau_n))\, R(\tau_n) + \mu\, R(\tau_n) = \gamma_2\, W(\tau_n). \tag{7.3}$$

It follows

$$\sigma\,(m_1\, W^\infty + m_2\, B^\infty)\, R^\infty \leq \sigma\,(m_1\, W^\infty + m_2\, B^\infty)\, R^\infty + \mu\, R^\infty \leq \gamma_2\, W^\infty, \tag{7.4}$$

as $n \to +\infty$. Moreover, the first equation of (4.1) yields

$$\begin{aligned} &(\mu + \alpha + \gamma_2)\, W^\infty \\ &\leq (\mu + \alpha + \gamma_2)\, W^\infty + (m_2\, B^\infty + m_1\, W^\infty)(W^\infty + B^\infty + R^\infty) \\ &\leq (m_1\, W^\infty + m_2\, B^\infty) + \sigma\,(m_1\, W^\infty + m_2\, B^\infty)\, R^\infty + \gamma_1 B^\infty. \end{aligned} \tag{7.5}$$

Recalling $B^\infty \leq \frac{\alpha}{\gamma_1+\mu} W^\infty$ in (7.2) and $\sigma\,(m_1\, W^\infty + m_2\, B^\infty)\, R^\infty \leq \gamma_2\, W^\infty$ in (7.4), inequality (7.5) yields

$$(\mu + \alpha + \gamma_2)\, W^\infty \leq m_1 W^\infty + m_2 \frac{\alpha}{\gamma_1 + \mu} W^\infty + \gamma_2\, W^\infty + \frac{\alpha\gamma_1}{\gamma_1 + \mu} W^\infty. \tag{7.6}$$

The preceding inequality is equivalent to

$$W^\infty\,(\mu + \alpha + \gamma_2 - m_1)(1 - \mathcal{R}_1^2) \leq 0, \tag{7.7}$$

where

$$\begin{aligned} \mathcal{R}_1^2 &= \frac{\alpha(m_2 + \gamma_1)}{(\gamma_1 + \mu)(\mu + \alpha + \gamma_2 - m_1)} + \frac{\gamma_2}{\mu + \alpha + \gamma_2 - m_1} \\ &= \mathcal{R}_0^2 + \frac{\gamma_2}{(\mu + \alpha + \gamma_2 - m_1)}. \end{aligned} \tag{7.8}$$

Here $\mathcal{R}_0$ is defined in (5.2) under the assumption $\mu+\alpha+\gamma_2-m_1 > 0$ in (5.3). In the case $\mathcal{R}_1^2 < 1$, recalling $0 \leq W_\infty \leq W^\infty$, inequality (7.7) yields $W_\infty = W^\infty = 0$; since $R^\infty \leq \frac{\gamma_2}{\mu} W^\infty$ from (7.4) together with $0 \leq R_\infty \leq R^\infty$, we have $R_\infty = R^\infty = 0$; the inequality $B^\infty \leq \frac{\alpha}{\gamma_1+\mu} W^\infty$ from (7.2) together with $0 \leq B_\infty \leq B^\infty$ yields $B_\infty = B^\infty = 0$. We conclude that $\lim_{t\to+\infty} W(t) = 0$, $\lim_{t\to+\infty} B(t) = 0$, and $\lim_{t\to+\infty} R(t) = 0$ when $\mathcal{R}_1^2 < 1$. This obesity elimination result provides the following theorem.

Theorem 7.1. *The obesity-free equilibrium E_0 of the obesity model* (4.1) *is globally asymptotically stable if $\mathcal{R}_0 < \mathcal{R}_1 < 1$, where $\mathcal{R}_1 > 0$ in* (7.8) *is defined as the obesity elimination threshold.*

7.2. Strategy II: Reduce the relative weight regain hazard σ less than unity

According to the literature [8, 10], the ex-overweight population has a high risk of weight regain. Fortunately, appropriate weight loss maintenance programs using several approaches: use of sequential medications, sequential dieting, and individual and group follow-up programs [8] can increase the efficacy of effective long-term weight regain prevention, then reduce the relative hazard of weight regain. In model (4.1), the relative rate of weight regain is denoted as σ. We derive the following theorem as a complete obesity elimination strategy.

Theorem 7.2. *The obesity epidemic can be completely eliminated if both the hazard of weight regain and the basic reproduction number is less than one. That is the obesity-free equilibrium E_0 is globally asymptotically stable if $\sigma \leq 1$ and $\mathcal{R}_0 < 1$.*

Proof. Following Theorem 2.1 in the paper [18], we take the Lyapunov function $Q(W, B) = z\, V^{-1} X$ for model (4.1) on Ω in (4.2). Here, $X = (W, B)^T$ denotes the disease compartments, and $z = \left(\frac{\alpha+\gamma_2+\mu-m_1}{m_2+\gamma_1}\mathcal{R}_0, 1\right)$ is a left Perron eigenvector associated with the largest eigenvalue $\mathcal{R}_0$ of the matrix

$$V^{-1}F = \begin{bmatrix} 0 & \dfrac{m_2+\gamma_1}{\mu+\alpha+\gamma_2-m_1} \\ \dfrac{\alpha}{\gamma_1+\mu} & 0 \end{bmatrix}.$$

Here, matrices V and F are given in equation (5.1).

The Lyapunov function $Q(W, B) = \frac{\mathcal{R}_0}{m_2+\gamma_1}W + \frac{1}{\gamma_1+\mu}B$ satisfies $Q(0, 0) = 0$ and $Q(W, B) > 0$ for all positive values W and B, and

$$\begin{aligned}
\frac{dQ}{dt} &= \frac{\mathcal{R}_0}{m_2+\gamma_1}\frac{dW}{dt} + \frac{1}{\gamma_1+\mu}\frac{dB}{dt} \\
&= \frac{\mathcal{R}_0}{m_2+\gamma_1}\{-(\alpha+\gamma_2+\mu-m_1)W + (m_2+\gamma_1)B \\
&\quad -(m_1W+m_2B)(B+W+(1-\sigma)R)\} + \frac{1}{\gamma_1+\mu}[\alpha W - (\gamma_1+\mu)B] \\
&\leq \frac{\mathcal{R}_0}{m_2+\gamma_1}[-(\alpha+\gamma_2+\mu-m_1)W + (m_2+\gamma_1)B] + \frac{1}{\gamma_1+\mu}[\alpha W - (\gamma_1+\mu)B] \\
&= -\frac{\alpha+\gamma_2+\mu-m_1}{m_2+\gamma_1}\mathcal{R}_0\, W + \frac{\alpha}{\gamma_1+\mu}W \\
&= -\frac{\alpha+\gamma_2+\mu-m_1}{m_2+\gamma_1}\mathcal{R}_0\left[1 - \frac{\alpha(m_2+\gamma_1)}{\mathcal{R}_0(\alpha+\gamma_2+\mu-m_1)(\gamma_1+\mu)}\right] W \\
&= -\frac{\alpha+\gamma_2+\mu-m_1}{m_2+\gamma_1}\mathcal{R}_0\,(1-\mathcal{R}_0)\, W \\
&\leq 0, \quad \text{when} \quad \mathcal{R}_0 < 1 \quad \text{and} \quad \sigma < 1.
\end{aligned}$$

The preceding inequality suggests that $E_0 = (W, B, R) = (0, 0, 0)$ is the largest invariant subset on Ω and is globally asymptotically stable, when $\mathcal{R}_0 < 1$ and $\sigma < 1$. □

Moreover, the globally stable obesity-free equilibrium E_0 indicates the complete elimination of the obesity epidemic. This scenario occurs under conditions $\sigma < 1$ and $\mathcal{R}_0 < 1$, which satisfy Theorem 6.2 and demonstrate a forward bifurcation.

8. Conditions for the obesity plateau via local stability analysis for the obesity-endemic equilibrium

In this section, we will derive the conditions to reach the obesity plateau when the overweight $W(t)$ and obese $B(t)$ populations are non-zero. First, we will derive the property of uniform persistence in Proposition 8.1, which indicates that overweight and obesity populations remain positive if starting from positive initial data under the condition $\mathcal{R}_0 > 1$.

Proposition 8.1. *If $\mathcal{R}_0 > 1$, the system (4.1) is uniformly persistent in $\dot{\Omega}$.*

Proof. When $R_0 > 1$, E_0 has two negative and one positive eigenvalue, thus repels nearby solutions. Since the system (4.1) is forward invariant, solutions starting with positive initial values stay positive in the interior of Ω.

Let $\dot{\Omega}$ be the interior of Ω. A solution (W, B, R) of the system (4.1) with initial values $(W^0, B^0, R^0) \in \mathring{\Omega}$ satisfies

$$\liminf_{t\to\infty} W(t) > \epsilon_0, \quad \liminf_{t\to\infty} B(t) > \epsilon_0, \quad \liminf_{t\to\infty} R(t) > \epsilon_0,$$

$$\liminf_{t\to\infty} 1 - W(t) - B(t) - R(t) > \epsilon_0, \quad \text{for} \quad 0 < \epsilon_0 < 1.$$

□

8.1. Strategy: Increase the weight loss rate from overweight to healthy weight and decrease the relative hazard of weight regain so that the plateau condition is satisfied

Understanding the underlying mechanism of the currently observed plateau is essential to prevent further increases in the prevalence of overweight and obesity. A strategy to maintain the plateau can be obtained by analyzing the local stability of the obesity-endemic equilibrium E^* and is given in Theorem 8.1.

The usual procedure for proof of local stability is to verify the Ruth-Hurwitz conditions. Since the Jacobian matrix for the system (4.1) is slightly more complicated, to determine the signs of the eigenvalues of the 3×3 matrix (6.1), we apply a lemma from the study by McCluskey and van den Driessche [13] as follows:

Lemma 8.1. (Lemma 3 [13]) *Let M be a 3×3 real matrix. If $tr(M)$, $det(M)$, and $det(M^{[2]})$ are all negative, then all the eigenvalues of M have the negative real part.*

Then the following theorem is established for the local stability of the obesity-endemic equilibrium by employing Lemma 8.1.

Theorem 8.1. *If $\mathcal{R}_0 > 1$, then the obesity model (4.1) has a unique obesity-endemic equilibrium E^* in $\dot{\Omega}$ and E^* is asymptotically stable when $(\sigma-1)\mu < \gamma_2$.*

Proof. To prove the asymptotic stability of the obesity-endemic equilibrium E^* we first calculate the Jacobian at E^*

$$J|_{E^*} = \begin{bmatrix} b_{11} & b_{12} & b_{13} \\ \alpha & -\gamma_1 - \mu & 0 \\ b_{31} & -\sigma m_2 R & -\sigma(m_1 W + m_2 B) - \mu \end{bmatrix}, \tag{8.1}$$

where

$$\begin{aligned} b_{11} &= ((\sigma-1)R - B - W)\, m_1 - (m_1 W + m_2 B) - (\mu + \alpha + \gamma_2) + m_1, \\ b_{12} &= ((\sigma-1)R - B - W)\, m_2 - (m_1 W + m_2 B) + (m_2 + \gamma_1), \\ b_{13} &= (\sigma-1)(m_1 W + m_2 B) > 0, \\ b_{31} &= -\sigma m_1 R + \gamma_2. \end{aligned} \tag{8.2}$$

Now, we identify the signs of the elements of the matrix $J|_{E^*}$. By the equations in (4.1), at the equilibrium E^*, we have

$$\begin{aligned} &(1 - W - B - R + \sigma R)m_1 - \mu - \alpha - \gamma_2 \\ &= -\{(1 - W - B - R)m_2 + \sigma m_2 R + \gamma_1\}\frac{B}{W}, \end{aligned} \tag{8.3}$$

$$\gamma_1 + \mu = \alpha\frac{W}{B}, \tag{8.4}$$

$$-\sigma m_1 R + \gamma_2 = (\sigma m_2 B + \mu)\frac{R}{W}. \tag{8.5}$$

By substituting equations (8.3)-(8.5) and by rearranging b_{11}

$$\begin{aligned} b_{11} &= (1 - W - B - R + \sigma R)m_1 - (\mu + \alpha + \gamma_2) - (m_1 W + m_2 B) \\ &= -\left[(1 - W - B - R)m_2 + \sigma m_2 R + \gamma_1\right]\tfrac{B}{W} - (m_1 W + m_2 B) < 0, \end{aligned} \tag{8.6}$$

and $b_{31} > 0$.

Next, we establish that the system (4.1) is a competitive system when $b_{12} > 0$, an important property that is useful for studying global stability when the overweight and obesity epidemic persists. Consider a smooth vector field defined for x such that $x \to f(x)$ in an open set $D \in \mathbb{R}^n$. The system $x' = f(x)$, $x \in D$ is considered competitive if a diagonal matrix $H = diag\,(\varsigma_1, \varsigma_2, ..., \varsigma_n,)$, where ς_i is 1 or -1 and $H(\partial f/\partial x)$H contains non-positive off-diagonal elements for all $x \in D$ [23]. According to the Jacobian matrix for the model (4.1), we choose the

matrix $H = diag(1, -1, -1)$. Then the matrices H and J give;

$$H(J)H = \begin{bmatrix} b_{11} & -b_{12} & -b_{13} \\ -\alpha & -\gamma_1 - \mu & 0 \\ -b_{31} & -\sigma m_2 R & -\sigma(m_1 W + m_2 B) - \mu \end{bmatrix}. \tag{8.7}$$

The matrix in (8.7) shows that the proposed obesity system (4.1) is competitive in $\dot{\Omega}$ for $b_{12} > 0$ with respect to the partial ordering defined by the orthant $\{(W, B, R) \in \mathbb{R}^3 : W \geq 0, B \leq 0, R \leq 0\}$. Hence, the three-dimensional competitive system has the Poincaré-Bendixon property.

Now, we apply the Lemma 8.1 to identify the signs of the eigenvalues of the matrix $J|_{E^*}$.

Step 1: Proof of $tr(J) < 0$. By the equation (8.6), we have

$$tr(J|_{E^*}) = b_{11} - (\gamma_1 + \mu) - \sigma(m_1 W + m_2 B) - \mu < 0.$$

Step 2: Proof of $det(J^{[2]}) < 0$. Now, consider the second additive compound matrix $J^{[2]}$ of the Jacobian, $J = \frac{\partial f}{\partial x}$. Thus, if $J = (b_{ij})_{3\times 3}$, then

$$J^{[2]} = \begin{bmatrix} b_{11} + b_{22} & b_{23} & -b_{13} \\ b_{32} & b_{11} + b_{33} & b_{12} \\ -b_{31} & b_{21} & b_{22} + b_{33} \end{bmatrix}. \tag{8.8}$$

Thus

$$J^{[2]} = \begin{bmatrix} b_{11} - (\gamma_1 + \mu) & 0 & -b_{13} \\ -\sigma m_2 R & b_{11} - (\sigma(m_1 W + m_2 B) + \mu) & b_{12} \\ -b_{31} & \alpha & -(\sigma(m_1 W + m_2 B) + \mu) \\ & & -(\gamma_1 + \mu) \end{bmatrix}. \tag{8.9}$$

Take $P = diag(R, B, W)$. Then the matrix $J^{[2]}$ is similar to the matrix $T = PJ^{[2]}P^{-1}$. If T is stable (all eigenvalues are negative), then $J^{[2]}$ is stable. To demonstrate that $det(J^{[2]}) < 0$, we prove the stability of T. The calculations give

$$T = \begin{bmatrix} t_{11} & 0 & (\sigma - 1)(m_1 W + m_2 B)\frac{R}{W} \\ -m_2 \sigma B & t_{22} & t_{23} \\ (m_1 \sigma R - \gamma_2)\frac{W}{R} & \alpha \frac{W}{B} & -\sigma(m_1 W + m_2 B) - (\gamma_1 + \mu) - \mu \end{bmatrix}, \tag{8.10}$$

where

$$\begin{aligned} t_{11} &= ((\sigma-1)R - W - B)m_1 + m_1 - (m_1W + m_2B) - (\alpha + \gamma_2 + \mu) \\ &\quad -(\gamma_1 + \mu) < 0, \\ t_{22} &= ((\sigma-1)R - W - B)m_1 + m_1 - (\sigma+1)(m_1W + m_2B) - (\alpha + \gamma_2 + \mu) \\ &\quad -(\gamma_1 + \mu) - \mu < 0, \\ t_{23} &= [((\sigma-1)R - W - B)m_2 + m_2 - (m_1W + m_2B) + \gamma_1]\tfrac{B}{W} > 0. \end{aligned} \tag{8.11}$$

Since the diagonal elements of the matrix T are negative, if we can prove that the matrix is diagonally dominant, followed by the Gershgorin circle theorem [2], T is stable. Take the following.

$$h_1 = t_{11} + 0 + \left|-(\sigma-1)(m_1W + m_2B)\frac{R}{W}\right| = t_{11} + (\sigma-1)(m_1W + m_2B)\frac{R}{W}. \tag{8.12}$$

From the third equation of (4.1), we have

$$\sigma(m_1W + m_2B)\frac{R}{W} < \gamma_2. \tag{8.13}$$

It is reasonable to assume that when the overweight and obesity epidemic persists $\gamma_2 < \gamma_1 + \mu$ and by the equation (8.13), we have $h_1 < 0$. Consider

$$h_2 = t_{22} + |-m_2\sigma B| + |t_{23}| = t_{22} + m_2\sigma B + t_{23} < 0. \tag{8.14}$$

Now, we take

$$\begin{aligned} h_3 &= -\sigma(m_1W + m_2B) - (\gamma_1 + \mu) - \mu + \left|(m_1\sigma R - \gamma_2)\tfrac{W}{R}\right| + \left|\alpha\tfrac{W}{B}\right| \\ &= -\sigma(m_1W + m_2B) - (\gamma_1 + \mu) - \mu + (-m_1\sigma R + \gamma_2)\tfrac{W}{R} + \alpha\tfrac{W}{B}. \end{aligned} \tag{8.15}$$

By equations (8.4) and (8.5), we obtain that $h_3 < 0$. Therefore, T is diagonally dominant and, hence T is stable. Thus $J^{[2]}$ is stable, and then $det(J^{[2]}|_{E^*}) < 0$.

Step 3: Proof of $det(J) < 0$. Now we consider the determinant of J,

$$det(J) = b_{13}[b_{31}(\gamma_1 + \mu) - \sigma m_2\alpha R] - [\sigma(m_1W + m_2B) + \mu][-b_{11}(\gamma_1 + \mu) - \alpha b_{12}]. \tag{8.16}$$

By the equations (8.3)-(8.5), we have

$$\begin{aligned} b_{31}(\gamma_1 + \mu) - \sigma m_2\alpha R &= (\gamma_1 + \mu)\left(\gamma_2 W - \sigma(m_1W + m_2B)R\right)\frac{1}{W} \\ &= \mu(\gamma_1 + \mu)\frac{R}{W}, \end{aligned} \tag{8.17}$$

and

$$\begin{aligned} -b_{11}(\gamma_1 + \mu) - \alpha b_{12} &= (\gamma_1 + \mu)\left(-b_{11} - \frac{\alpha}{\gamma_1 + \mu}b_{12}\right) \\ &= (\gamma_1 + \mu)(m_1W + m_2B)(1 + \tfrac{B}{W}). \end{aligned} \tag{8.18}$$

Then

$$det(J|_{E^*}) \leq (\gamma_1 + \mu)(m_1 W + m_2 B)\,[(\sigma - 1)\mu - \gamma_2]\frac{R}{W} < 0, \tag{8.19}$$

provided $(\sigma - 1)\mu < \gamma_2$. □

As local stability of E^* is ensured for the parameter condition $(\sigma - 1)\mu < \gamma_2$, the weight loss rate from overweight to healthy weight (γ_2) should be large enough to achieve the plateau. Moreover, it takes a longer period of time to change natural birth/death rates μ compared to other parameters. Then the realistic option is to decrease the relative hazard of weight regain to a value small enough to satisfy the parameter condition in Theorem 8.1.

8.2. Saddle-node bifurcation for E^*

As excess weight gain is highly affected by the transmission rate from a healthy weight to overweight by the social influence of overweight individuals, we choose m_1 as the bifurcation parameter. The characteristic polynomial P_3 of the Jacobian matrix (8.1) is

$$P_3(L; W^*, m_1) = L^3 + c_1 L^2 + c_2 L + c_3,$$

where

$$\begin{aligned}
c_1 &= \sigma(m_1 W^* + m_2 B^* - \mu) + \mu - b_{11} + \gamma_1,\\
c_2 &= (\gamma_1 + \mu - b_{11})(m_1 W^* + m_2 B^* - \mu)\sigma - \alpha b_{12} - b_{11}(\gamma_1 + \mu) - b_{13} b_{31},\\
c_3 &= -\sigma(\gamma_1 + \mu)(m_1 W^* + m_2 B^* - \mu) b_{11} - b_{13} b_{31}(\gamma_1 + \mu)\\
&\quad -\alpha\sigma(-\mu b_{12} + (b_{12} B^* - b_{13} R^*) m_2 + m_1 b_{12} W^*).
\end{aligned} \tag{8.20}$$

Here, b_{11}, b_{12}, b_{13}, and b_{31} are given by the equation (8.6).

A static bifurcation occurs if $c_3 = 0$ and the corresponding Hurwitz criteria $\Delta_1 = c_1$ and $\Delta_2 = c_1 c_2 - c_3$ are positive [28].

A zero-eigenvalue bifurcation occurs when $c_3 = 0$ or $m_1 = m_{1s}$, where

$$m_{1s} = \frac{2}{\sigma}\sqrt{\frac{\mu(\sigma - 1)(\alpha + \gamma_1 + \mu)\gamma_2}{(\gamma_1 + \mu)}} + \frac{[(\sigma - 1)\mu - \sigma m_2]\alpha}{(\gamma_1 + \mu)\sigma} + \frac{(\gamma_2 - \mu)}{\sigma} + \mu. \tag{8.21}$$

Assuming the equilibrium at $m_1 = m_{1s}$ is $E_s^* = (W_s^*, B_s^*, R_s^*)$. Taking the transformation $W = y_1 + W_s^*, B = y_2 + B_s^*, R = y_3 + R_s^*$ and $m_1 = \tilde{m}_1 + m_{1s}$, we transform system (4.1) to

$$\dot{y} = Ay + F(y), \tag{8.22}$$

where $A = J|_{(E_s^*, m_{1s})}$ and $F(y) = \frac{1}{2}\tilde{B}(y, y) + O(||y||^3)$ and $y = (y_1, y_2, y_3)^T$ [12].

The system (8.22) contains an equilibrium at $y = 0$ and $\tilde{m}_1 = 0$ with simple zero eigenvalue $\lambda_1 = 0$. Let v_0 be an eigenvector to the zero eigenvalues such that

$Av_0 = 0$ and w_0 be the adjoint eigenvector such that $A^T w_0 = 0$. We have

$$v_0 = \begin{bmatrix} 1 \\ \dfrac{\alpha}{\gamma_1 + \mu} \\ v_{03} \end{bmatrix}, \; w_0 = \begin{bmatrix} \dfrac{1}{n} \\ \dfrac{w_{02}}{n} \\ \dfrac{(\sigma-1)(m_{1s}W_s^* + m_2 B_s^*)}{n\,[\sigma(m_{1s}W_s^* + m_2 B_s^*) + \mu]} \end{bmatrix}, \tag{8.23}$$

where

$$\begin{aligned} v_{03} &= \frac{-R_s^*[\alpha m_2 + m_{1s}(\gamma_1+\mu)]\sigma + \gamma_2(\gamma_1+\mu)}{(\gamma_1+\mu)(\sigma(m_{1s}W_s^* + m_2 B_s^*) + \mu)}, \\ w_{02} &= \frac{1}{(\gamma_1+\mu)(\sigma(m_{1s}W_s^* + m_2 B_s^*) + \mu)} \times \{-(2B_s^* + W_s^* - 1)B_s^* \sigma {m_2}^2 \\ &\quad + [((1 - 3B_s^* - W_s^*)m_{1s}W_s^* + B_s^*\gamma_1 + \mu R_s^*)\sigma - (2B_s^* + R_s^* + W_s^* - 1)\mu]m_2 \\ &\quad - (m_{1s}W^* - \gamma_1)(m_{1s}\sigma W^* + \mu)\}, \end{aligned}$$

and

$$\begin{aligned} n = \frac{1}{(\gamma_1+\mu)(\sigma(m_{1s}W_s^* + m_2 B_s^*) + \mu)} &\times \{\mu^2 - [(m_{1s}W_s^* + m_2 B_s^*)v_{03} - w_{03}\alpha]\mu \\ &- \gamma_1\mu + [(v_{03}+1)\mu + (v_{03}+1)\gamma_1 + w_{03}\alpha](m_{1s}W_s^* + m_2 B_s^*)\sigma \\ &- \gamma_1 v_{03}(m_{1s}W_s^* + m_2 B_s^*)\}. \end{aligned}$$

The one-dimensional center manifold near $y = 0$ and $\tilde{m}_1 = 0$ is topologically equivalent to the normal form

$$\dot{\eta} = \tilde{b}\,\eta^2 + \epsilon, \quad \eta \in \mathbb{R}^1, \tag{8.24}$$

where ϵ is the unfolding parameter and

$$\begin{aligned} \tilde{b} &= \frac{1}{2}\langle w_0, \tilde{B}(v_0, v_0)\rangle \\ &= \frac{-(\alpha m_2 + m_{1s}(\gamma_1+\mu))}{n(\gamma_1+\mu)^2(\sigma(m_{1s}W_s^* + m_2 B_s^*) + \mu)} \times \{((1-\sigma)v_{03} + 1)\mu^2 \\ &\quad + ((m_{1s}W_s^* + m_2 B_s^* - \gamma_1 v_{03})\sigma + (v_{03}+1)\gamma_1 + \alpha)\mu \\ &\quad + \sigma(\gamma_1 + \alpha)(m_{1s}W_s^* + m_2 B_s^*)\}. \end{aligned}$$

Setting parameters as in Table 1, but leaving m_1 free, we calculate a zero-eigenvalue bifurcation critical point at $m_{1s} = \frac{32031}{1000000}$. The other two eigenvalues are $-\frac{19}{718}$ and $-\frac{38}{273}$. Choosing the normalized eigenvector and adjoint eigenvector associated with zero eigenvalues as $v_0 = (1, \frac{200}{111}, \frac{152}{217})^T$ and $w_0 = (\frac{27}{86}, \frac{30}{89}, \frac{21}{187})^T$. we derive the quadratic coefficient in (8.24) as $\tilde{b} = -\frac{17}{635}$. Moreover, the transversality condition is satisfied as $\sum_{i,j=1}^{n} w_{0j} \frac{\partial F_i(y,\tilde{m}_1)}{\partial m_1}\Big|_{y=0,\tilde{m}_1=0} = \frac{16}{1841} \neq 0$. Thus, the equilibrium $E_s^* = (\frac{9}{263}, \frac{23}{373}, \frac{15}{346})$ undergoes a saddle-node bifurcation at $m_1 = m_{1s} = \frac{32031}{1000000}$ as shown in Fig. 2(a).

9. Discussion and biological implications

This paper presents a compartment model to predict overweight and obesity prevalence by considering interactions and transitions between healthy weight, overweight, and obese populations described by BMI measures. Here, we have considered the effect of social contacts and peer pressure on excess weight gain. We showed that for a higher value of relative hazard of weight regain σ, the suggested model can show a backward bifurcation. In the backward bifurcation scenario, the upper and lower endemic equilibrium branches showed asymptotic stability and instability, respectively. Moreover, the horizontal branch for the obesity-free equilibrium becomes stable to unstable when the parameter m_1 varies. As shown by Fig. 2(a), a bi-stable region is observed when the system exhibits different branches of equilibrium curves with the varying parameter m_1. In this case, the solutions converge to the stable obesity-free equilibrium for the lower initial overweight population located below the unstable branch of obesity-endemic equilibrium. Otherwise, the solutions will be directed to a stable positive equilibrium. However, regardless of the initial size of the overweight or obese population, for the values $\mathcal{R}_0$ on the left and right of the saddle-node bifurcation, the solutions converged to stable obesity-free equilibrium and stable obesity-endemic equilibrium, respectively (Fig. 3).

By mathematical analysis, we have shown that for the overweight and obesity epidemic control, the parameter σ (relative hazard of weight regain) plays an important role. An ex-overweight person who is now in healthy weight may be aware of the health risks of being overweight or obese; he or she is more resistant to gaining weight. In this scenario, we can take $\sigma < 1$. Then, the model shows only the forward bifurcation, i.e., bringing down the basic reproduction number below unity results in complete overweight and obesity epidemic eradication. Although many overweight and obese individuals have been involved in weight loss programs, maintaining good shape is challenging in the long run once they lose weight to a healthy weight. This leads to the chronic nature of overweight and obesity, making it challenging to lower the overweight and obese population size. In this sense, public health policymakers must consider enhancing awareness and promoting weight loss maintenance programs targeting individuals who lose their weight to a healthy weight to effectively control the overweight and obesity epidemic. To be more precise, this suggests that the individuals who lose weight can participate in weight loss maintenance programs that contain several approaches such as sequential medication, sequential dieting, and group and individual-based follow-up programs for a while until they adapt to a healthy and active lifestyle that prevents them from regaining weight.

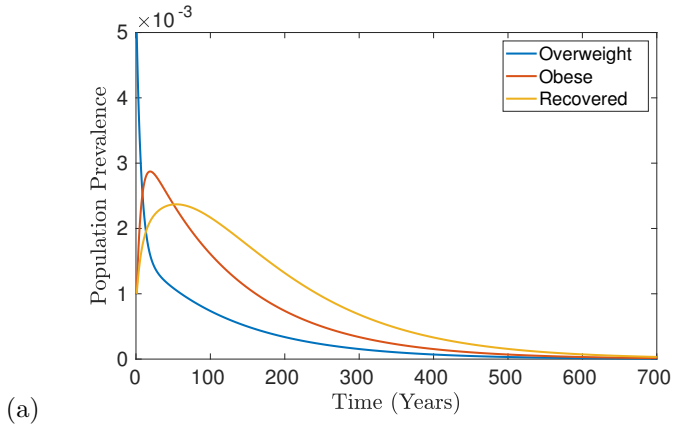
Furthermore, it should be noted that overweight and obesity epidemic elimination is not an easy task and takes a very long time (Fig. 3(a)). According to studies, the prevalence of overweight and obesity is increasing in a high number. Therefore, public health policymakers aim to prevent further growth of the overweight and obese population. By the local stability analysis of the obesity-endemic equilibrium, we suggest that the plateau in obesity is guaranteed if we have a higher recovery rate from overweight to a healthy weight or a lower relative weight regain hazard. Furthermore, as we observed in the bifurcation analysis, as shown in Fig. 2, the overweight population can be reduced by reducing $\mathcal{R}_0$. According to the sensitivity analysis, by increasing the parameters natural birth/death rates μ and the recovery rate from overweight γ_2 and obese γ_1, $\mathcal{R}_0$ can be decreased. However,

changing the natural birth and death rates is a slower process and not a realistic option for depleting the basic reproduction number. Increasing γ_2 and γ_1 is a faster solution as overweight individuals lose weight and achieve a healthy weight. This can be done by having a healthy diet and an active lifestyle. Public health policymakers should increase the opportunities for people to have a healthy and active lifestyle by conducting intervention programs, such as healthy lifestyle campaigns that can be applied to both overweight and obese groups. In addition, treatment programs such as medications and surgeries must be introduced into the obese class, as reducing weight is much more difficult in this phase. On the other hand, the basic reproduction number increases when the social influence rates m_1 and m_2 increase. These parameter value increments and decrements can be achieved by enhancing awareness of having a healthy and balanced diet and an active lifestyle. To promote a supportive environment and healthy behavior to prevent overweight and obesity, local and state programs can make the resources available to the public on public health recommendations and evidence-based practices. Also, the effort to reverse obesity patterns can be established in several settings, such as early childhood care, hospitals, schools, and food venues.

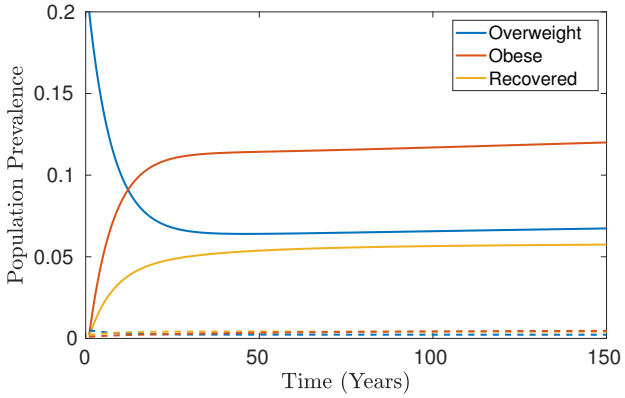
10. Conclusions

Our study aimed to discuss the reasons behind the plateau in the overweight and obesity epidemic and, if possible, eliminate it. The practical feasibility of eradicating the obesity epidemic is subject to discussion; however, at least public health policymakers can establish adequate prevention and intervention strategies to decrease excess weight gain prevalence. Throughout the study, we intended to provide control strategies through stability analysis and bifurcation theory. The stability analysis for obesity-free and obesity-endemic equilibrium provided parameters for eradication and plateau of excess weight gain, respectively. We looked for sufficient parameter conditions for the plateau in the overweight and obesity epidemic by local stability analysis. The results suggested that promoting weight loss and maintenance programs can help keep the plateau. The analysis indicated that much attention should be paid to reducing the risk of weight regain as the backward bifurcation exists for higher values of relative risk of weight regain. Therefore, weight loss maintenance programs should be promoted to eliminate the epidemic, while weight loss programs help maintain the plateau.

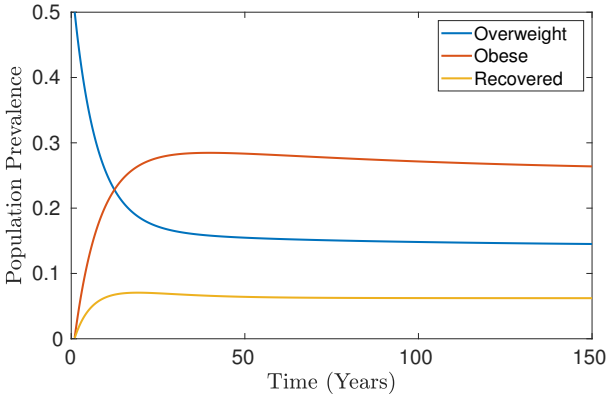
We considered that social influence causes an excess weight gain in the proposed model. However, non-social conditions such as genetics and lifestyle habits also affect excess weight gain. Therefore, the proposed model can be further extended considering the risk of non-contagious factors. Other than that, there are several factors that we can consider to expand the model, such as disease-related deaths and social influence on weight loss. In addition, an optimal control process can be introduced to the proposed model to better understand the effects of control strategies. Three scenarios of intervention programs can be introduced; (1) awareness programs to promote healthy diet and active lifestyle for the overweight population, (2) treatment programs for the obese population, and (3) weight loss maintenance programs such as follow-ups for the ex-overweight population. Despite future work, the proposed model successfully simplified the dynamics of overweight and obesity prevalence, which is crucial for effective intervention and prevention control programs.



(a)



(b)



(c)

Figure 3. Time series of unhealthy and healthy weight individuals when (a) $m_1 = 0.02$, (b) $m_1 = 0.035$, solid lines with initial conditions $(W(0), B(0), R(0)) = (0.04, 0.001, 0.001)$ and dashed lines with initial conditions $(W(0), B(0), R(0)) = (0.01, 0.001, 0.001)$ and (c) $m_1 = 0.04$.

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