REVISTA INVESTIGACIÓN OPERACIONAL Vol. 46, No. 1, 1-15, 2025

OBESITY: THE DYNAMICAL IMPACT OF MEDIA COVERAGE ON OVERWEIGHT INDIVIDUALS

Séverine Bernard, Ténissia Cesar and Alain Piétrus*
Laboratoire LAMIA, Université des Antilles, France

ABSTRACT

In this paper, we introduce a three dimensional deterministic compartmental model to study obesity dynamic. Contrary to other works on this subject we explore it in a new way: by analizing the influence of media on the spread of obesity in a constant population, considering only its impact on the overweight individuals. A stability analysis of the model shows that the disease-free equilibrium point is globally asymptotically stable if a certain threshold, the basic reproduction number is less than unity. We also show that if the basic reproduction number is bigger than unity, the unique endemic equilibrium point is globally asymptotically stable under some conditions.

KEYWORDS: Overweight, Obesity, media equilibrium, stability.

MSC: 92B99, 37N25

RESUMEN

En este artículo, se presenta un modelo compartimental determinista tridimensional para el estudio de la dinámica de la obesidad. Al contrario de otros trabajos sobre este tema, aquí se explora de una nueva manera: analizando la influencia de los medios de comunicación sobre la propagación de la obesidad en una población constante, teniendo en cuenta solo su impacto en las personas con sobrepeso. Un análisis de estabilidad del modelo muestra que el punto de equilibrio sin enfermedad es globalmente asintomáticamente estable cuando el número básico de reproducción es menor que la unidad. También mostramos que si el número básico de reproducción es mayor que la unidad, el único punto de equilibrio endémico es globalmente asintomáticamente estable bajo algunas condiciones.

PALABRAS CLAVE: Sobrepeso, Obesidad, medios de comunicación, equilibrio, estabilidad.

1. INTRODUCTION

Overweight, obesity and the associated diseases are growing health problems worldwide. An usual tool to classify individuals according to their weight is using the BMI (Body Mass Index) which is

*severine.bernard@univ-antilles.fr, tisci@live.fr, alain.pietrus@univ-antilles.fr

obtained by dividing the body mass over the square of the body height. If the BMI is less than 18.5 the person is classified underweight. If this measure is between 18.5 and 24.9, the weight is normal and if it is between 25 and 30 the person is overweight. Once the BMI score is greater than 30 the person is considered as obese [34]. We remark that the incidence of obesity increased dramatically [26, 29] over the last decades and this phenomenon is particularly serious in the US but is also observed globally [32]. In France more than 17 percent of the population is obese.

Overweight and obesity often occur when people regularly take in more calories that it burns, in other words, when there is an energy imbalance between consumed calories and expended calories. Other factors are known such as sedentary lifestyles, genetic predispositions, cultural, social and economic conditions of the environment in which people develop. Obesity entails substantial health costs. For example, obese individuals are more likely to suffer from diseases such as type 2 diabetes, high blood pressure, cardiovascular diseases, metabolic syndrome, cancer, and many more [4, 14, 15, 30]. Obesity is not only a serious health concern, but it also has a notable impact on the economy [16, 17].
Nowadays in many countries, obesity has become a prevalent problem which is mostly overlooked at the beginning. In some countries this phenomenon is important and captured more than 70 percent of the total population [8]. Considered as the "Pandemia of the 21st Century", obesity is all over the world and some preventive actions are needed. Obesity is often identified as a contagious problem which is spreading with contacts over social networks [4, 5, 18, 19, 21, 33] and has been studied theoretically using epidemic models [1, 19, 25]. With a SIR model introduced by Ejima in [13], it is shown that social contagion is the significant factor for the increase of obesity, and its spread has shown the pattern of infectious disease. In [27] Paudel proposed a SIR model for the dynamics of obesity in the southeastern region of the United States, and discussed the effect of social network on the spread of obesity among friends and family members. In [33] the authors claimed that prevention strategies are more effective by analyzing numerically a variant of a SIS epidemic model. In [20] the authors introduced a system of SIS difference equations model and drew a similar conclusion. In [23] a new model is introduced for taking into account both social and nonsocial contagion risks of obesity. A way to complete the preventive actions is to consider the role of the media that could be important. This fact has been underlined in [22] and [31] for the AIDS epidemic and in [6] and the references therein for the SARS coronavirus. To emphasize the action of media on diseases, the authors used in their model an incidence function with a key role in the qualitative description of the spreading.

This present work is a continuation of the recent study made in [3]. The paper is organized as follows. In Section 2 we introduce a new model for the spread of obesity taking into account the importance of media coverage in order to make prevention. The media is characterized in this context by a reasonable incidence function acting only on overweight individuals. In Section 3, we study the existence of the equilibrium points and show that the existence of an endemic point for obesity depends on the properties of the incidence function and the reproduction number $\mathcal{R}_0'$. In Section 4 we shall study the local and global stability of the disease-free equilibrium point and the unique endemic-point when it exists. We finish the paper with some numerical illustrations, a comparison with the results obtained in [3] and some comments.

2. THE MODEL

The compartmental model introduced in this paper is for a population with a normalized constant size. Throughout the paper, the population at time t is divided into three separate classes of individuals: the proportion of normal weight individuals, $S(t)$; the proportion of overweight individuals, $O_w(t)$ and the proportion of obese individuals, $O_b(t)$. We assume that the changes from the normal weight compartment to the others are essentially made by contact, social pressure or unhealthy lifestyle. Contrary to the model studied in [3] where the arrival in the compartment of the obeses is attenuated thanks to the media coverage, as it has been done in [6, 7] for epidemic models, in the present one, only the arrival in the compartment of the overweight is attenuated thanks to the media coverage with the help of an incidence function. The reason of this consideration is because we think that the influence of the media could also be important on this class. We make the assumption that when you are overweight the actions to help you to change bad behaviors are as efficient than the same actions on obese individuals.

Let us denote by β_{11} (resp. β_2) the transmission rate by social pressure to adopt an unhealthy lifestyle and contact with the O_w (resp. O_b) group. Moreover, β_{12} is the maximum reduced contact (and unhealthy lifestyle) rate due to the presence of the media and γ is the rate at which overweight individuals become obese individuals due to an unhealthy lifestyle. Finally, δ denotes the rate at which overweight individuals move to the S group of normal weight individuals and η is the rate at which obese individuals with a healthy lifestyle move to the group of overweight individuals. This transmission is described by Figure 1.

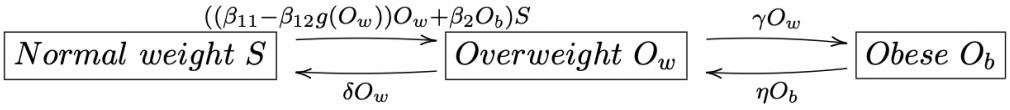

Figure 1: Diagram of the model

And we obtain the following system:

$$\begin{cases} \frac{dS}{dt} = \delta O_w - (\beta_{11} - \beta_{12} g(O_w)) O_w S - \beta_2 O_b S, \\ \frac{dO_w}{dt} = (\beta_{11} - \beta_{12} g(O_w)) O_w S + \beta_2 O_b S - (\gamma + \delta) O_w + \eta O_b, \\ \frac{dO_b}{dt} = \gamma O_w - \eta O_b. \end{cases} \tag{2.1}$$

Since $\frac{dS}{dt} + \frac{dO_w}{dt} + \frac{dO_b}{dt} = 0$, the population has a constant size and in the rest of the paper we suppose

the normalization condition $S + O_w + O_b = 1$.

In what follows, we ask to the incidence function $g : [0,1] \longrightarrow [0,1]$ to satisfy

$$g(0) = 0, \quad g'(O_w) \geq 0, \quad g(O_w) \leq 1, \tag{2.2}$$

and we suppose that $\beta_{11} > \beta_{12}$.

The case $\beta_{12} = 0$ meaning the absence of media has been considered in [25] ($\beta_{11} = \beta_1$) and in this case the system has two equilibrium points. The first one ($S_0^* = 1, O_{w0}^* = 0, O_{b0}^* = 0$) corresponds to a population with no obese people. This disease free equilibrium point is locally asymptotically stable if $\beta_1 + \beta_2 \frac{\gamma}{\eta} < \delta$ and unstable otherwise (see [25]). The second equilibrium point (endemic-equilibrium point) is:

$$\left(S_1^* = \frac{\delta(\gamma+\eta)}{\eta(1+\frac{\gamma}{\eta})(\beta_1+\beta_2\frac{\gamma}{\eta})}, O_{w1}^* = \frac{(\beta_1+\beta_2\frac{\gamma}{\eta})-\delta}{(1+\frac{\gamma}{\eta})(\beta_1+\beta_2\frac{\gamma}{\eta})}, O_{b1}^* = \frac{\gamma}{\eta}\frac{(\beta_1+\beta_2\frac{\gamma}{\eta})-\delta}{(1+\frac{\gamma}{\eta})(\beta_1+\beta_2\frac{\gamma}{\eta})} \right)$$

and corresponds to the case where there is a significant group of obese and overweight individuals. When $-\beta_2\frac{\gamma}{\eta} < \beta_1 - \delta < \gamma + \eta$, this endemic equilibrium point is locally asymptotically stable (see [25]).

The basic reproduction number $\mathcal{R}_0$, firstly introduced by MacDonald [24], appears in an important number of contributions in epidemiology, see [9] for a suitable definition from Diekmann and Heersterbeek and the monograph [2] from Brauer and Castillo-Chavez.
In [25], one has $\mathcal{R}_0 = \dfrac{\beta_1\eta + \beta_2\gamma}{\eta\delta}$ and it is easy to see that the endemic equilibrium point can be rewritten

$$\left(S_1^* = \frac{\delta(\gamma+\eta)}{\eta(1+\frac{\gamma}{\eta})(\beta_1+\beta_2\frac{\gamma}{\eta})}, O_{w1}^* = \frac{\delta(\mathcal{R}_0-1)}{(1+\frac{\gamma}{\eta})(\beta_1+\beta_2\frac{\gamma}{\eta})}, O_{b1}^* = \frac{\gamma}{\eta}\frac{\delta(\mathcal{R}_0-1)}{(1+\frac{\gamma}{\eta})(\beta_1+\beta_2\frac{\gamma}{\eta})} \right).$$

3. EXISTENCE OF EQUILIBRIUM POINTS

This section is devoted to the study of existence of equilibrium points for our new model (2.1). Since $S(t) = 1 - O_b(t) - O_w(t)$, System (2.1) is reduced to

$$\begin{cases} \frac{dO_w}{dt} = -\beta_2 O_b^2 + \Big(\beta_2 + \eta - \big(\beta_2 + \beta_{11} - \beta_{12} g(O_w)\big)O_w\Big)O_b + \\ \big(\beta_{11} - (\gamma+\delta) - \beta_{12} g(O_w)\big)O_w - \big(\beta_{11} - \beta_{12} g(O_w)\big)O_w^2, \\ \\ \frac{dO_b}{dt} = \gamma O_w - \eta O_b. \end{cases} \tag{3.1}$$

An equilibrium point (O_w^*, O_b^*) satisfies the equality $O_b^* = \frac{\gamma}{\eta} O_w^*$ and then O_w^* becomes a solution of the equation

$$\begin{aligned} &-\beta_2 \tfrac{\gamma^2}{\eta^2} O_w^2 + \tfrac{\gamma}{\eta}\Big(\beta_2 + \eta - \big(\beta_2 + \beta_{11} - \beta_{12} g(O_w)\big)O_w\Big)O_w \\ &+\big(\beta_{11} - (\gamma+\delta) - \beta_{12} g(O_w)\big)O_w + \big(\beta_{12} g(O_w) - \beta_{11}\big)O_w^2 = 0. \end{aligned} \tag{3.2}$$

The disease free equilibrium point $E_0 = (0, 0)$ is a solution of the last equation and the other is not always easy to be obtained because we have no more informations about the incidence function g which could be non linear. Some conditions will be added in the sequel in order to obtain existence and uniqueness of an endemic equilibrium point.

The basic reproduction number associated to System (2.1) could be calculated using the next generation method (see [10, 11, 12]). So for our model (2.1), the basic reproduction number is $\mathcal{R}'_0 = \dfrac{\beta_{11}\eta + \beta_2\gamma}{\eta\delta}$.

Theorem 1. *We suppose that the incidence function g satisfies the assumptions* (2.2) *and its derivative satisfies* $\sup\limits_{x\in[0,1]} g'(x) \leq \dfrac{\beta_2}{\beta_{12}}\left(1 + \dfrac{\gamma}{\eta}\right)$. *If* $\mathcal{R}'_0 \leq 1$ *then System* (2.1) *has no endemic equilibria and if* $\mathcal{R}'_0 > 1$ *then there exists an unique endemic equilibrium point.*

Proof. The equilibrium equation (3.2) gives us the indication to consider the function Φ defined by

$$\begin{aligned}\Phi(O_w) = &\left(\beta_{11} + \big(\beta_2 + \beta_{11}\big)\tfrac{\gamma}{\eta} + \beta_2\tfrac{\gamma^2}{\eta^2} - \beta_{12}(1 + \tfrac{\gamma}{\eta})g(O_w)\right)O_w \\ &-\left(\beta_2\tfrac{\gamma}{\eta} - \delta + \beta_{11} - \beta_{12}g(O_w)\right).\end{aligned} \tag{3.3}$$

According to (2.2), we have

$$\Phi(0) = -\beta_2\frac{\gamma}{\eta} - \beta_{11} + \delta = -\Big(1 - \frac{1}{\mathcal{R}'_0}\Big)\Big(\beta_2\frac{\gamma}{\eta} + \beta_{11}\Big). \tag{3.4}$$

It is clear that $\Phi(0) < 0$ when $\mathcal{R}'_0 > 1$.

We also have

$$\Phi(1) = \beta_2\frac{\gamma^2}{\eta^2} + \big(\beta_{11} - \beta_{12}g(1)\big)\frac{\gamma}{\eta} + \delta. \tag{3.5}$$

Using the fact that $\beta_{11} - \beta_{12} > 0$ and assumptions (2.2), this implies that $\Phi(1) > 0$.

The function Φ is clearly derivable and we have

$$\begin{aligned}\Phi'(O_w) = &-\beta_{12}g'(O_w)(1 + \tfrac{\gamma}{\eta})O_w + \beta_{12}g'(O_w) + \beta_{11} \\ &+(\beta_2 + \beta_{11})\tfrac{\gamma}{\eta} + \beta_2\tfrac{\gamma^2}{\eta^2} - \beta_{12}(1 + \tfrac{\gamma}{\eta})g(O_w).\end{aligned} \tag{3.6}$$

The assumptions $\sup\limits_{x\in[0,1]} g'(x) \leq \dfrac{\beta_2}{\beta_{12}}\left(1 + \dfrac{\gamma}{\eta}\right)$ and (2.2) imply the positivity of Φ'.

Consequently, we have proved that $\Phi(1) > 0$, $\Phi(0) < 0$ if $\mathcal{R}'_0 > 1$ and Φ is monotonically increasing. Thus Ψ has an unique positive root denoted by O^*_w in the interval $(0, 1)$. The unique endemic point is given by $E^* = (O^*_w, O^*_b)$ with $O^*_b = \dfrac{\gamma}{\eta}O^*_w$.

Remark. An example of function that fulfills the conditions of this previous theorem is the one taken for the numerical experiments in Section 5.

4. STABILITY ANALYSIS OF THE EQUILIBRIUM POINTS

Our aim in this section is to give local and global stability results for the disease-free equilibrium point E_0 and the endemic equilibrium point E^* .

4.1. Local Stability

The first result is about local stability for E_0, the disease-free equilibrium point.

Theorem 2. *The disease-free equilibrium point E_0 is locally asymptotically stable if $\mathcal{R}'_0 < 1$ and unstable if $\mathcal{R}'_0 > 1$.*

Proof. The Jacobian matrix $J = (J_{ij})$ of System (3.1) is a 2×2 matrix whose components are

$$\begin{aligned} J_{11} = &-\big(\beta_2 + \beta_{11} - \beta_{12} g(O_w)\big) O_b - \beta_{12} g'(O_w) O_w (1 - O_w - O_b) \\ &+\beta_{11} - \beta_{12} g(O_w) - (\gamma + \delta) - 2\big(\beta_{11} - \beta_{12} g(O_w)\big) O_w, \end{aligned}$$

$$J_{12} = -2\beta_2 O_b + \beta_2 + \eta - \big(\beta_2 + \beta_{11} - \beta_{12} g(O_w)\big) O_w \ , \ J_{21} = \gamma \ \text{ and } \ \ J_{22} = -\eta.$$

Since $g(0) = 0$, the characteristic polynomial of J evaluated at $E_0 = (0, 0)$ is

$$\lambda^2 + (\eta - \beta_{11} + \gamma + \delta)\lambda + \eta(\delta - \beta_{11}) - \gamma\beta_2.$$

Using the Routh-Hurwitz criterion, it is easy to see that this polynomial is stable if and only if

$$\eta - \beta_{11} + \gamma + \delta > 0 \ \text{ and } \ \eta\Big(\delta - (\beta_{11} + \beta_2 \frac{\gamma}{\eta})\Big) > 0.$$

It is obvious that the two last conditions reduce to $\beta_{11} + \beta_2 \frac{\gamma}{\eta} < \delta$ which is equivalent to $\mathcal{R}'_0 < 1$.

Noting that the stability of the characteristic polynomial is equivalent to the fact that the two eigenvalues of J evaluated at E_0 have negative real parts, this means that E_0 is locally asymptotically stable.

□

Now we study the local asymptotical stability of System (3.1) at the endemic equilibrium point E^* when $\mathcal{R}'_0 > 1$. Since the computation of the endemic-point is not obvious, we can't as above apply the Routh-Hurwitz criterion to conclude.

Theorem 3. *We suppose that $\mathcal{R}'_0 > 1$, $\gamma \leq \eta$, $\delta < \beta_{11}$ and $\beta_{11} < \gamma + \eta + \delta$. Then the endemic-equilibrium point E^* is locally asymptotically stable.*

Proof. It is well-known that E^* is locally asymptotically stable if

$$Tr(J(E^*)) < 0 \quad \text{and} \quad det(J(E^*)) > 0,$$

where $J(E^*)$ is the Jacobian matrix associated to System (3.1) and evaluated at E^*.

It is easy to see that the preliminary assumptions and the fact $\beta_{11} < \gamma+\eta+\delta$ imply that $Tr(J(E^*)) < 0$.

Moreover, we have

$$\begin{aligned} det(J(E^*)) = \eta\Big(&\big(\beta_2 + \beta_{11} - \beta_{12}g(O_w^*)\big)O_b^* + \beta_{12}g'(O_w^*)O_w^*(1 - O_w^* - O_b^*) \\ &-\beta_{11} + \beta_{12}g(O_w^*) + (\gamma+\delta) + 2\big(\beta_{11} - \beta_{12}g(O_w^*)\big)O_w^*\Big) \\ &-\gamma\Big(- 2\beta_2 O_b^* + \beta_2 + \eta - \big(\beta_2 + \beta_{11} - \beta_{12}g(O_w^*)\big)O_w^*\Big). \end{aligned} \tag{4.1}$$

The fact that $O_b^* = \frac{\gamma}{\eta}O_w^*$ yields

$$\begin{aligned} det(J(E^*)) = \eta\Big(\delta - \big(\beta_{11} + \beta_2\frac{\gamma}{\eta}\Big) &+ 2\gamma\big(\beta_2 + \beta_{11} - \beta_{12}g(O_w^*)\big)O_w^* \\ &+2\eta(\beta_{11} - \beta_{12}g(O_w^*))O_w^* + \eta\beta_{12}g(O_w^*) \\ &+2\beta_2\frac{\gamma^2}{\eta}O_w^* + \eta\beta_{12}g'(O_w^*)O_w^*(1 - O_b^* - O_w^*). \end{aligned} \tag{4.2}$$

We remark that all the terms of $det(J(E^*))$ are positive except $\eta\Big(\delta - \big(\beta_{11} + \beta_2\frac{\gamma}{\eta}\Big)$ because $\mathcal{R}_0' > 1$. For this reason it is not possible to conclude directly.

But the fact that O_w^* is a root of Φ allows us to write

$$\left(1 + \frac{\gamma}{\eta}\right)(\beta_{11} - \beta_{12}g(O_w^*))O_w^* = \beta_2\frac{\gamma}{\eta} - \delta + \beta_{11} - \beta_{12}g(O_w^*) - \beta_2\frac{\gamma}{\eta}\left(1 + \frac{\gamma}{\eta}\right)O_w^*$$

and consequently

$$\begin{aligned} det(J(E^*)) = \eta\Big(\delta - \beta_{11} - \beta_2\frac{\gamma}{\eta}\Big) &+ 2\beta_2\frac{\gamma^2}{\eta}O_w^* + 2\gamma\beta_2 O_w^* + \eta\beta_{12}g(O_w^*) \\ &+\frac{2\eta\gamma}{\gamma+\eta}\Big(\beta_2\frac{\gamma}{\eta} - \delta + \beta_{11} - \beta_{12}gO_w^*) - \beta_2\frac{\gamma}{\eta}\big(1 + \frac{\gamma}{\eta}\big)O_w^*\Big) \\ &+2\eta(\beta_{11} - \beta_{12}g(O_w^*))O_w^* + \eta\beta_{12}g'(O_w^*)O_w^*(1 - O_b^* - O_w^*), \end{aligned} \tag{4.3}$$

which can be rewritten

$$\begin{aligned} det(J(E^*)) = \eta\Big(\delta - \beta_{11} - \beta_2\frac{\gamma}{\eta}\Big) &+ 2\gamma\beta_2 O_w^* + \frac{2\gamma^2}{\gamma+\eta}\beta_2 \\ &-\frac{2\eta\gamma}{\gamma+\eta}\delta + \frac{2\eta\gamma}{\gamma+\eta}\beta_{11} + \eta\beta_{12}g(O_w^*)\Big(1 - \frac{2\gamma}{\gamma+\eta}\Big) \\ &+2\eta(\beta_{11} - \beta_{12}g(O_w^*))O_w^* + \eta\beta_{12}g'(O_w^*)O_w^*(1 - O_b^* - O_w^*). \end{aligned} \tag{4.4}$$

With assumptions (2.2) and the fact that $\beta_{11} > \beta_{12}$, we obtain

$$
\begin{aligned}
det(J(E^*)) > \eta\Big(\delta - \beta_{11} - \beta_2\frac{\gamma}{\eta}\Big) + 2\gamma\beta_2 O_w^* + \frac{2\gamma^2}{\gamma+\eta}\beta_2 \\
-\frac{2\eta\gamma}{\gamma+\eta}\delta + \frac{2\eta\gamma}{\gamma+\eta}\beta_{11} + \eta\beta_{12}g(O_w^*)\Big(1 - \frac{2\gamma}{\gamma+\eta}\Big),
\end{aligned}
\tag{4.5}
$$

which implies

$$
\begin{aligned}
det(J(E^*)) > \eta\Big[\delta\big(1 - \frac{2\gamma}{\gamma+\eta}\big) + \beta_{11}\big(\frac{2\gamma}{\gamma+\eta} - 1\big) \\
+\beta_2\frac{\gamma}{\eta}\big(\frac{2\gamma}{\gamma+\eta} - 1\big) + \beta_{12}g(O_w^*)\big(1 - \frac{2\gamma}{\gamma+\eta}\big)\Big].
\end{aligned}
\tag{4.6}
$$

The last inequality can be rewritten

$$
det(J(E^*)) > \eta\left(1 - \frac{2\gamma}{\gamma+\eta}\right)\left[\delta - \beta_{11} - \beta_2\frac{\gamma}{\eta} + \beta_{12}g(O_w^*)\right]. \tag{4.7}
$$

Assumptions (2.2) and the fact that $\gamma \leq \eta$, $\delta < \beta_{11}$ imply that the right side of the previous inequality is positive and the proof is complete.

□

4.2. Global Stability

We study in this subsection the global behavior of the equilibria points of System (3.1). However the techniques are different according the equilibrium point. In the theorem below, we obtain the global stability property of the disease-free equilibrium point E_0 with simple assumptions on some coefficients of the system.

Theorem 4. *The disease-free equilibrium point E_0 is globally asymptotically stable whenever $\beta_{11} + \gamma - \delta < 0$ and $\beta_2 - \eta < 0$.*

Proof. We consider the function L defined by $L(t) = O_w(t) + 2O_b(t)$ and we have

$$
\begin{aligned}
\dot{L} = -\beta_1 O_b^2 + \Big(\beta_2 - \eta - \big(\beta_2 + \beta_{11} - \beta_{12}g(O_w)\big)O_w\Big)O_b + \\
\big(\beta_{11} - \delta + \gamma - \beta_{12}g(O_w)\big)O_w - \big(\beta_{11} - \beta_{12}g(O_w)\big)O_w^2.
\end{aligned}
\tag{4.8}
$$

The fact that $\beta_{11} - \beta_{12} > 0$ combined with assumptions (2.2) and the one taken in the theorem imply that $\dot{L} \leq 0$. With these assumptions, we remark that $\dot{L}$ is null only at point E_0 and we can conclude that L is a Lyapunov function.

Moreover, with the assumptions given in the theorem, we also have

$$
\mathcal{R}'_0 = \frac{\beta_{11}\eta + \beta_2\gamma}{\eta\delta} < \frac{\beta_{11} + \gamma}{\delta} < 1.
$$

Thus E_0 is the only equilibrium point and the LaSalle's invariance principle implies that E_0 is globally asymptotically stable. □

In the next theorem we obtain the global stability of the endemic-equilibrium point in a different way and without additional conditions. The main tool is the use of a Dulac function and the Dulac criterion given in [28].

Theorem 5. *Under the assumptions of Theorem 3, the endemic-equilibrium point E^* is globally asymptotically stable.*

Proof. Let us set

$$\begin{aligned} T = &-\beta_2 O_b^2 + \Big(\beta_2 + \eta - \big(\beta_2 + \beta_{11} - \beta_{12} g(O_w)\big) O_w\Big) O_b \\ &+ \big(\beta_{11} - (\gamma + \delta) - \beta_{12} g(O_w)\big) O_w - \big(\beta_{11} - \beta_{12} g(O_w)\big) O_w^2 \end{aligned} \tag{4.9}$$

and

$$V = \gamma O_w - \eta O_b.$$

Note that T and V are in fact the right side of the first and second line of System (3.1) respectively.

Let us now consider the Dulac function $D = \dfrac{1}{O_b.O_w}$. We have

$$\frac{\partial(DT)}{\partial O_w} = -\frac{\beta_2(1 - O_b) + \eta}{O_w^2} - \frac{\beta_{11} - \beta_{12} g(O_w)}{O_b} - \beta_{12} \frac{g'(O_w)}{O_b}\big(1 - O_b - 0_w\big) \tag{4.10}$$

and

$$\frac{\partial(DV)}{\partial O_b} = -\frac{\gamma}{O_b^2}.$$

We remark that $\dfrac{\partial(DT)}{\partial O_w}$ and $\dfrac{\partial(DV)}{\partial O_b}$ are both strictly negative in the domain on which we work with our preliminary assumptions, thus

$$\frac{\partial(DT)}{\partial O_w} + \frac{\partial(DV)}{\partial O_b} < 0.$$

The Dulac criterion can be applied and implies the nonexistence of closed orbit and this allows us to conclude that the endemic equilibrium point E^* is globally asymptotically stable.

□

5. NUMERICAL EXPERIMENTS

In this section, we will study different numerical simulations in order to see the effects of media coverage on overweight individuals. Then we will compare these results with those obtained in the case of media coverage on obeses in [3]. All the experiments are implemented in Mapple 2021.2 and performed on Apple Mc Book Air 10.1 with Apple M1 chip (8-core CPU with 4 high-performance cores and 4 energy-efficient cores, GPU up to 8 cores, Neural Engine 16 cores) and RAM 8.00 GB.
For the variable at the origin, we take

$$S(0) = 0.8, \quad O_w(0) = 0.15, \quad O_b(0) = 0.05,$$

and for the incidence function, a good choice inspired from [7] is $g(O_w) = \dfrac{O_w}{0.5 + O_w}$.

In the figures 2 to 5, S is in red, O_w in blue and O_b in green. For the first figure 2, we take $\beta_{11} = 0.001$, $\beta_2 = 0.007$, $\delta = 0.002$, $\gamma = 0.0015$ and $\eta = 0.1$ in order to be in the case $\mathcal{R}'_0 < 1$. We know that in this case $\mathcal{R}'_0 < 1$, there is only one equilibrium state which is stable and for which $S = 1$, $O_w = O_b = 0$ and this is what we obtain with Figure 2, whatever the value of β_{12}.

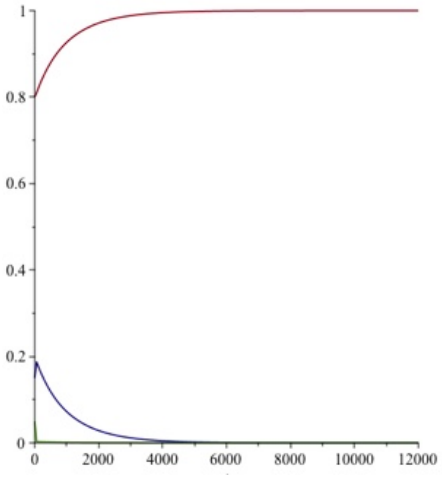

Figure 2: $\mathcal{R}'_0 < 1$ and $\beta_{12} = 0$ or 0.0006

In the three following figures 3, 4 and 5, we take $\beta_2 = 0.0007$, $\delta = 0.00035$ and $\gamma = \eta = 0.00028$ and have $\mathcal{R}'_0 > 1$. By comparing Figures 3, 4 and 5, we can notice that when β_{12} increases, that is when you have more media coverage, the number of normal weight individuals S decreases less, that is the number of overweight and obese individuals increases less. Therefore these numerical experiments boost our theoretical study and highligth the influence of media coverage on the dynamics of our model.

In the last figure 6, we take $\beta_2 = 0.0007$, $\delta = 0.00035$ and $\gamma = \eta = 0.00028$ and have $\mathcal{R}'_0 > 1$. On the same picture,we present the results obtained in [3] and those obtained with our model. O_b is in red and O_w in dark blue with media coverage on the obeses and the coefficient (β_{22}) which indicate this is equal to 0.0006 (see [3]). For our model, O_b is in green and O_w in light blue with media coverage on the overweight individuals and β_{12} which indicates this, is also equal to 0.0006. The picture seems to emphasize that the impact of media coverage is better on obese than on overweight individuals.

6. CONCLUSION AND COMMENTS

In this paper, we proposed and studied a new model of obesity with media coverage on overweight individuals, instead of obese as proposed in [3]. After its presentation, we showed the existence of equilibrium states to analyse then their stability. Some numerical simulations are performed in the following to highlight the effect of media coverage on overweight individuals and compare with our

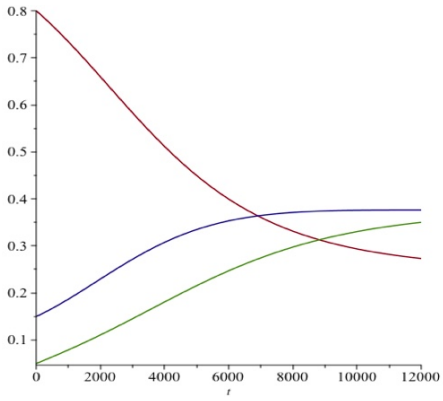

Figure 3: $\mathcal{R}'_0 > 1$ and $\beta_{12} = 0$

previous work [3] to remark that media coverage have better effects on obese than overweight individuals in the dynamic of obesity.
In future works, it will be interesting to construct a new model with media coverage on obese and overweight individuals simultaneously and compare it with our previous studies. Once all done, it could be an interesting strategy that authorities could use to fight this global scourge of obesity.

Acknowledgments. We would like to thanks the three anonymous referees for their careful review and constructive comments on the manuscript which led us to a better presentation of it.

Conflict of interests. The authors declare that there is no conflict of interests regarding the publication of this paper.

RECEIVED: APRIL, 2023.
REVISED: FEBRUARY, 2024.

REFERENCES

[1] ALDILA, D., RARASATI N., NURAINI N. and SOEWONO E. (2014): Optimal control problem of treatment for obesity in a closed population. **International Journal of Mathematics and Mathematical Sciences**, Article ID 273037, 7 pages.

[2] BRAUER F. and CASTILLO-CHAVEZ C. (2012): **Mathematical Models in Population Biology and Epidemiology**, Volume 40 of **Texts in Applied Mathematics**. Springer, New-York, second edition.

[3] BERNARD S., CESAR T. and PIETRUS A. (2022): The impact of media on Obesity. **Contemporary Mathematics**, 3(1), 93-103.

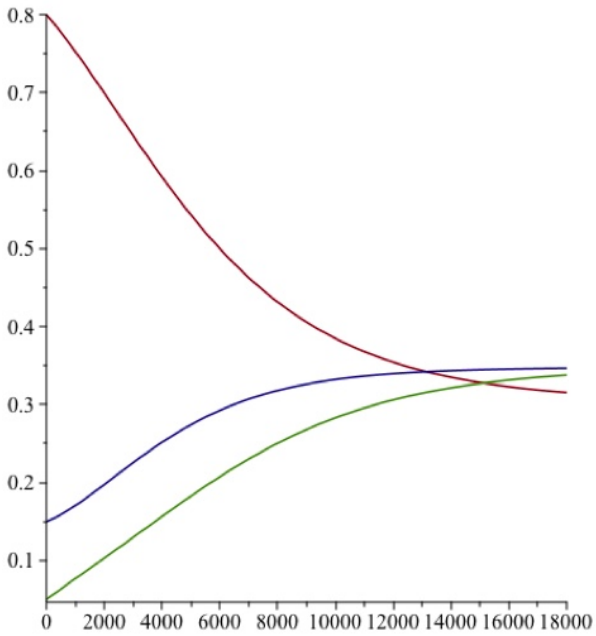

Figure 4: $\mathcal{R}'_0 > 1$ and $\beta_{12} = 0.0006$

[4] Bray G.A., Kim K.K., Wilding J.P.H. (2017): Obesity: A chronic relapsing progressive disease process. A position statement of the World Obesity Federation. **Obesity Reviews**, 18, 715-723.

[5] CHRISTAKIS N.A. and FOWLER J.H. (2007): E-spread of obesity in a large social network over 32 years. **e-New England Journal of Medicine**, 357(4), 370-379.

[6] CUI J., SUN Y. and ZHU H. (2008): The impact of media on the control of infections diseases. **J. Dynamics Differential Equations**, 20, 31-53.

[7] CUI J.A., TAO X. and ZHU H. (2008): An SIS infection model incorporating media coverage. **Rocky-Mountain Journal of Mathematics**, 38(5), 1323-1334.

[8] DANIEL J. (2013): How Mexico got so fat and is now more obese than America, available at http://www.dailymail.co.uk/news/article- 2358472/How-Mexico-got-fat-obese-America.html, last consultation date: february 2024.

[9] DIEKMANN O. and HEERSTERBEEK J.A.P. (2000): **Mathematical Epidemiology of Infectious Diseases: Model Building. Analysis and Interpretation**. Wiley, New-York.

[10] DIEKMANN O., HEESTERBEEK J.A.P and METZ J.A.J. (1990): On the definition and the computation of the basic reproduction ratio R_0 in model for infectious diseases in heterogeneous populations, **J. Math. Biol.**, 28, 365-382.

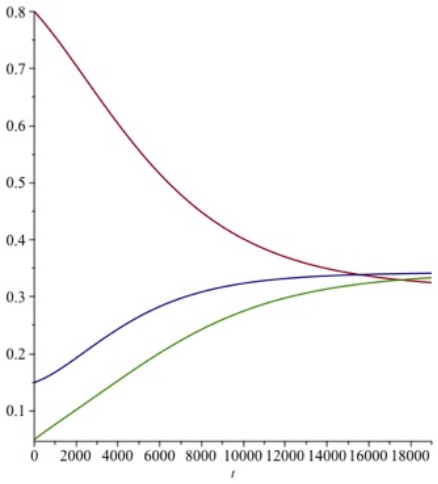

Figure 5: $\mathcal{R}'_0 > 1$ and $\beta_{12} = 0.0006999$

[11] DIEKMANN O., HEESTERBEEK J.A.P. and ROBERTS M.G. (2010): The construction of next-generation matrices for compartmental epidemical models. **J. Royal Society Interface**, 7, 873-885.

[12] DRIESSCHE P.V. and WATMOUGH J. (2002): Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. **Mathematical Biosciences**, 180(1-2), 29-48.

[13] EJIMA K., AIHARA K. AND NISHIURA H. (2013): Modeling the obesity epidemic: social contagion and its implications for control. **Theoretical Biology and Medical Modelling**, 10-17.

[14] FIELD A.E., COAKLEY E.H., MUST A., SPADANO J.L., LAIRD N., DIETZ W.H., RIMM E. and COLDITZ G.A. (2001): Impact of overweight on the risk of developing common chronic diseases during a 10-year period. **Arch. Intern. Med.**, 161(13), 1581-1586.

[15] FLEGAL K.M., GRAUBARD B.I., WILLIAMSON D.F. and GAIL M.H. (2005): Excess deaths associated with underweight, overweight, and obesity. **Jama**, 293(15), 1861-1867.

[16] FINKELSTEIN E.A., TROGDON J.G., COHEN J.W. and DIETZ W. (2009): Annual medical spending attributable to obesity: payer-and service-specific estimates. **Health Affairs**, 28(5), w822-w831.

[17] HAMMOND R.A. and LEVINE R. (2010): The economic impact of obesity in the United States, Diabetes, Metabolic Syndrome and Obesity. **Targets and Therapy**, 3, 285-295.

[18] JAMES P.T. (2004): Obesity: the worldwide epidemic. **Clin. Dermatol.**, 22(4), 276-280.

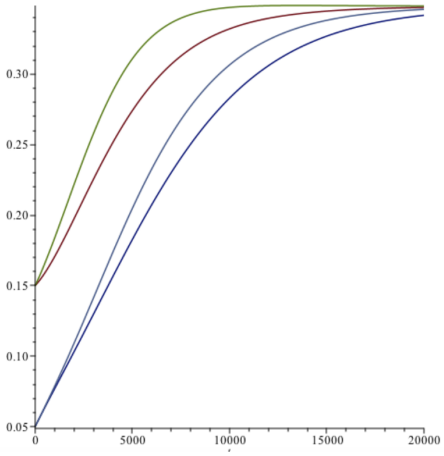

Figure 6: $\mathcal{R}'_0 > 1$ and $\beta_{12} = \beta_{22} = 0.0006$

[19] JODAR L., SANTONJA F.J. and GONZALEZ-PARRA G. (2008): Modeling dynamic of infant obesity in the region of Valencia, Spain. **Computer and Mathematics with Applications**, 56, 679-689.

[20] KIM M.S., CHU C. and KIM Y. (2011): A note on obesity as epidemic in Korea. **Osong Public Health and Research Perspectives**, 2(2), 135-140.

[21] KIM B.N., MASUD M.A. and KIM Y. (2014): Optimal implementation of intervention to control the self-harm epidemic. **Osong Public Health and Research Perspectives**, 5(6), 315-323.

[22] KHAN M.A., RAHMAN M., KHANAM P.A., KHUDA B.E., KANE T.T. and ASHRAF A. (1997): Awareness of sexually transmitted diseases among woman and service providers in rural Bangladesh. **International J. STD AIDS**, 8, 688-696.

[23] LOZANO-OCHOA E., CAMACHO J.F. and VARGA-DE-LEON C. (2017): Qualitative stability analysis of an obesity epidemic model with social contagion. **Discrete Dynamics in Nature and Society**, 12 pages. https://doi.org/101155/2017/1084769.

[24] MAC-DONALD G. The analysis of equilibrium in malaria. (1952): **Tropical Diseases Bulletin**, 49(9), 813-819.

[25] OH C. (2014): A note on the Obesity as epidemic. **Honam Mathematical J.** 36(1), 131-139.

[26] OGDEN C. and CARROLL M. (2010): Prevalence of overweight, obesity and extreme obesity among adults: United States, trends 1976-1980 through 2007-2008. NCHS Health Stats .

[27] PAUDEL L.P. (2019): Mathematical modeling on the obesity dynamics in the southeastern region and the effect of intervention. **Universal Journal of Applied Mathematics**, 7(3), 41-52.

[28] PERKO L. (1996): **Differential equations and dynamic systems**. Springer, New-York.

[29] VON RUESTEN A., STEFFEN A., FLOEGEL A., MASALA G., TJONNELAND A., HALKJAER J., PALLI D., WAREHAM N., LOOS R., SORENSEN T. et al. (2011): Trend in obesity prevalence in european adult cohort populations during follow-up since 1996 and their predictions to 2015, **PLoS One**, 6(11) e27455 doi: 10.1371/journal.pone.0027455.

[30] National Institutes of Health (NIH) (2022): What are overweight and obesity, available at http://www.nhlbi.nih.gov/health/health-topics/topics/obe/, last consultation date: february 2024.

[31] RAHMAN M.S. and RAHMAN M.L. (2007): Media and education play a tremendous role in mounting AIDS awareness among married couples in Bangladesh. **AIDS Research Terapy**, 4:10 doi: 10.1186/1742-6405-4-10.

[32] SASSI F. (2010): Obesity and the Economics of Prevention, **OECD publications**.

[33] SANTONJA F.J., VILLANUEVA R.J., JODAR L. and GONZALEZ-PARRA G. (2010): Mathematical Modelling dynamic of social obesity in the region of Valencia, Spain. **Math and Computer Modelling of Dynamical Systems**, 6(1), 23-34.

[34] World Health Organization (WHO): Health topics, Obesity, available at http://www.who.int/topics/ obesity/en/, last consultation date: february 2024.