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Long Time Bihavior of Some Epidemic Models

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THESIS

Dedication

***To** The memory of **M**y dear **F**ather*

***To** **M**y dearest **M**other, my biggest supporter*

***To** **D**r. **A**bdelheq **M**ezouaghi*

***To** **D**r. **S**alih **D**jilali*

***To** **D**r. **A**bdelkader **B**enali*

***To** **M**y dear **B**rother, and **M**y dear **S**isters*

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Résumé

Cette thèse étudie un modèle endémique d'obésité et de problèmes de santé associés, en prenant en compte les facteurs sociaux et les liens sociaux. Le but essentiel de notre modèle proposé, est de comprendre et par la suite contrôler dans la mesure du possible l'évolution de chaque maladie. L'obésité et les problèmes de santé associés peuvent être transmis par la pression des pairs pour manger de la malbouffe et les habitudes malsaines apprises de la famille et des amis. Dans notre modèle, l'impact de ces facteurs sociaux est représenté par les paramètres β_i. Nous fournissons une démonstration théorique de la stabilité asymptotique des équilibres sans maladie et endémique, ainsi que de l'existence et de l'unicité de la solution, pour le modèle d'obésité et ses complications associées. Afin de corroborer les résultats théoriques du modèle par des simulations numériques, étudier les effets (impact) des changements de facteurs sociaux sur la dynamique de propagation de la maladie.

Mots clés : Obésité; diabète de type 2; maladie cardiovasculaire; hypertension; stabilité locale; équilibre endémique; nombre de reproduction de base.

Abstract

This thesis investigates an endemic model of obesity and related health issues, considering social factors and social connections. The essential goal of our proposed model is to understand and subsequently control as far as possible the evolution of each disease. Obesity and its related health issues can be transmitted through peer pressure to eat fast food and unhealthy habits learned from family and friends. In our model, the impact of these social factors is represented by the parameters β_i. We provide a theoretical demonstration of the asymptotic stability of both the disease-free and endemic equilibria, as well as the solution's existence and uniqueness, for the model of obesity and its related issues. In order to corroborate the model's theoretical findings through numerical simulations, to study the effects (impact)of changes in social factors on disease spread dynamics.

Keywords: Obesity disease; type 2 diabetes; cardiovascular diszase; hypertension; local stability; endemic equilibrium; basic reproduction number.

ملخص:

هذه الأطروحة تبحث في نموذج وبائي للسمنة والقضايا الصحية المرتبطة بها، مع الأخذ بعين الاعتبار العوامل والروابط الاجتماعية. يمكن أن تنتقل السمنة ومشاكلها الصحية المرتبطة بها من خلال الضغط الاجتماعي والعادات غير الصحية المكتسبة من الأسرة والأصدقاء كتناول الوجبات السريعة. في نموذجنا، يتم تمثيل تأثير هذه العوامل الاجتماعية بواسطة المعاملات β_i، نقدم إثباتًا نظريًا للاستقرار أو السلوك بجوارالمالانهاية لكل من التوازن الخالي من المرض والتوازن المتوطن، بالإضافة إلى وجود ووحدانية الحل للنموذج الخاص بالسمنة ومضاعفاته المرتبطة به. من أجل دعم النتائج النظرية للنموذج يتم ذلك من خلال المحاكاة العددية، ودراسة تأثير التغيرات في العوامل الاجتماعية على انتشار المرض.

الكلمات المفتاحية :مرض السمنة؛ السكري من النوع الثاني؛ أمراض القلب والأوعية الدموية؛ ارتفاع ضغط الدم؛ الاستقرار المحلي؛ التوازن المتوطن؛ رقم التكاثر الأساسي.

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General introduction

Obesity is a complicated medical disease that goes beyond appearances; it is defined by an excessive quantity of body fat. It significantly increases the risk of developing a variety of other diseases, such as diabetes, high blood pressure, heart disease, liver disease, cholesterol issues, and sleep apnoea, as well as some cancers. An individual is considered overweight if their body mass index (BMI) is 25 or higher, and obese if it is 30 or higher [41]. In 2019, according to estimates having a body mass index (BMI) outside of the healthy range contributed to around 5 million deaths from non-communicable diseases (NCDs). Obesity and overweight are becoming more common in both adults and children. Between 1990 and 2022, the percentage of obese children and adolescents (ages 5 to 19) globally increased from 2% to 8%, a quadruple, while adults above the age of 18 had an exponentially higher prevalence of obesity from 7% to 16%. One aspect of the double burden of malnutrition is obesity, and obesity now exceeds underweight in all regions, with the exception of Southeast Asia. Once thought to be a problem only in wealthy nations, Today, overweight and obesity prevalence is among the highest worldwide in a middle-income countries [42].

Mathematical models of obesity are simplified representations of a complex phenomenon or system, such as the human body and its processes, using the language of mathematics. In the context of obesity, these models attempt to understand the complex relationship between different factors that influence weight, such as:

Food intake: food type, quantity, timing.

Physical activity: exercise type, duration, intensity.

Genetic factors: genes that influence weight.

Environmental factors: social, economic, cultural conditions.

The study of mathematical models of obesity contributes significantly to the fight against obesity through a deeper understanding of the mechanisms of obesity, which helps in developing nutritional and exercise programs tailored to each individual, evaluating the effectiveness of different interventions, and predicting weight development, thus enabling individuals to make informed health decisions and prevent obesity and its associated diseases. Mathematical models have been extensively used to describe the spread of infectious diseases (Smith et al., 2004) in [74]. Although obesity is not a contagious disease, its propagation within a population may exhibit analogous patterns. Researchers have developed epidemiological models to simulate the spread of obesity. Santonja et al. (2010) examined the prevalence of excess weight among adults in Valencia, Spain, and explored the potential of healthy advertising campaigns to mitigate the rising obesity rates [75]. Delavani et al. (2021) formulated a differential equation model to examine the propagation of obesity within human populations and the influence of media campaigns. However, the model was not validated against empirical data [11]. In [80] Thomas et al. (2014) aimed to develop a model to predict BMI trajectories and identify the factors associated with a stabilization of obesity prevalence. Their model [80], which considered both social and non-social factors influencing weight gain, was based on data from the U.S. and U.K. between 1988 and 1998. Levine (2011) proposes that poverty and obesity are interrelated, suggesting that limited access to fresh food in impoverished neighborhoods, often termed food deserts, may contribute to higher rates of obesity. Their study [63] revealed a positive correlation between food insecurity, reduced physical activity, and increased prevalence of obesity. In [76] Smith et al. (2020) demonstrated that social connections significantly influence obesity, weight, and dietary behaviors. Potential mechanisms for this occurrence include social support, social norms, social comparison, and behavioral modeling.

Previous studies have employed **SIR-type** models to simulate the spread of obesity within populations [75, 11, 80]. The following model developed in this study was a three-compartment differential equation model that accounts for societal factors. The authors attempted to analyze the impact of factors such as declining poverty rates on obesity trends. These trends are divided into three categories: healthy weight, overweight, and obesity. The model is defined

by the model of differential equations as follows:

$$\begin{cases} s^{'}(t) = -\beta_1 * s(t) * o_v(t) - \beta_2 * s(t) * o_b(t) + r_1 * o_v(t), \\ o^{'}(t) = \beta_1 * s(t) * o_v(t) + \beta_2 * s(t) * o_b(t) - k * o_v(t) + r_2 * o_b(t) - r_1 * o_v(t), \\ o_b^{'}(t) = k * o_v(t) - r_2 * o_b(t), \end{cases} \tag{0.0.1}$$

such that s, o_v, and o_b denotes the healthy weight, overweight, and obese categories, respectively. This model represents a simplification of the models proposed by Delavani et al. (2021) and Thomas et al. (2014), which consisted of systems of four and six equations, respectively. Authors S. Kim and S.Y. Kim in 2018 in [58] created a mathematical model for the dynamics of obesity to investigate the long-term trend obesity Considering social and psychological factors since obesity is becoming more commonplace worldwide. The model SIR that depicts the dynamics of obesity in the Southeast region of the United States was created by the authors Laxmi et al. in [37]. They talk about how social media might spread obesity among friends and family. Using the mathematical model, they are discussing the efficacy of current obesity intervention strategies. They also suggest constructive measures that could reduce or even reverse the obesity trend to the community, the urban planning authority, and public health decision-makers.

This thesis aims to develop an advanced dynamic mathematical model to study the intricate connections between obesity, cardiac disease, hypertension disease and type 2 diabetes mellitus (T2DM). The proposed model is based on integrating a wide range of influencing factors, including behavioral and social factors, to provide a deeper understanding of the physiological mechanisms linking these diseases. And to be aware of a successful plan for getting rid of any or all of these disorders. For disease control, we use numerical simulations to examine the variables influencing disease spread, leading to a more targeted perspective.

The structure of this manuscript is as follows:

In the first chapter; we presented fundamental concepts of dynamic systems, defining equilibrium points and introducing the notion of stability. We also discussed various methods used for proving system stability. In the second part of this chapter, we explored mathematical models of epidemics, examining the historical progression of epidemic modeling techniques by researchers over time.

In the second chapter; firstly we provide a biological view of the phenomenon of obesity as an

epidemic, exploring the complex interplay of genetic, Psychological, and social factors that contribute to excessive weight gain and its associated health risks, Presenting strategies to prevent and treat obesity effectively. Secondly, we delved into a historical developpement of most prominent and important mathematical models used to investigate obesity and its co-morbidities.

In the third chapter; we provide a mathematical model formulated by ordinary differential equations that study the interactions between obesity and its complications, type 2 diabetes, cardiovascular disease (heart disease), and hypertension disease (high blood pressure) to understand how obesity contributes to the development of these diseases. This is due to peer pressure to follow bad habits on individuals in the population. We demonstrate theoretically the asymptotic local stability of disease-free equilibrium and the asymptotic local stability of endemic equilibrium, proving the existence and uniqueness of the solution to the obesity model and its complications. To confirm the model's theoretical results, we conduct numerical simulations to study the impact of variation in social factor parameters on the diseases studied.

finally; we end this work with a conclusion and by giving perspectives of our mathematical model.

CHAPTER 1

Mathematical modeling of epidemic dynamics

In this chapter, we conducted a comprehensive examination of mathematical models of epidemics, analyzing the historical progression of modeling techniques employed by researchers.

1 Mathematical tools

This section is devoted to the presentation of the mathematical definitions and tools used in our work.

1.1 Generalities on dynamic systems

Definition 1.1.1 ***(Autonomous, non-autonomous systems)***

Suppose that X is a subset of $\mathbb{R}^n$ and T is a subset of $\mathbb{R}$.

A differential system of the following form:

$$\frac{dx}{dt} = f(x,t); \quad x \in X, \quad t \in T. \tag{1.1.1}$$

is said to be an autonomous system if the function f depends only on the vector variable x.

Otherwise, it is non-autonomous.

An autonomous system is written as follows:

$$\frac{dx}{dt} = f(x); \quad x \in X. \tag{1.1.2}$$

where f is a function defined on a connected open set X of $\mathbb{R}^n$, with value in $\mathbb{R}$. We assume that $f \in C^1(X)$.

Definition 1.1.2 *Let $f : X \longrightarrow \mathbb{R}^n$ be a continuous function and let X is an open set of $\mathbb{R}^n \times \mathbb{R}$. We say that f is locally Lipschitzian at x for any closed bounded (i.e. compact) set in $\mathbb{R}^n$, if a constant $L \geq 0$ exists where:*

$$\|f(t,x_1) - f(t,x_2)\| \leq L|x_1 - x_2|, \qquad \forall (t,x_1), \quad (t,x_2) \in X. \tag{1.1.3}$$

Definition 1.1.3 *The Cauchy problem is the solution of a system of differential equations (1.1.1) with the function f is defined on the domain $X \times T$, and of a starting point $(t_0, x_0) \in R \times \mathbb{R}^n$ called the initial condition of the problem (1.1.1). We denote it by*

$$\begin{cases} \dfrac{dx}{dt} & = f(x,t); \qquad x \in X, \quad t \in T, \\ x(t_0) & = x_0. \end{cases} \tag{1.1.4}$$

The Cauchy-Lipschitz theorem provides us the conditions that guarantee the existence and uniqueness of the solution of the system (1.1.4).

In all that follows, we are only interested in autonomous type systems where the function f implicitly depends on t, where x will be the state variable and t represents time.

1.1.1 Dynamical properties

Definition 1.1.4 ***(Float)***

We call the float of the differential system (1.1.2) with initial condition $x(t_0) = x_0$ the application

$$\begin{cases} \Phi_t : (T,X) & \longrightarrow \mathbb{R}^n \\ (t,x_0) & \longrightarrow \Phi_t = \Phi(x,t), \end{cases} \tag{1.1.5}$$

A semi-float on X is a continuous function $\Phi : X \times \mathbb{R}^+ \longrightarrow X$ satisfying:

(i) $\Phi_0 = Id_X$ such that Id_X represents the identity of X.

(ii) $\Phi_t \circ \Phi_s = \Phi_{t+s}$ for $t, s \geq 0$.

With $\Phi_t(x) \equiv \Phi(x,t)$ for $x \in X$ and $t \in \mathbb{R}^+$.

We indicate the solution of the differential system (1.1.2) by $\Phi_t(x)$.

Definition 1.1.5 ***(Trajectory, semi-orbit, orbit)***

- *The trajectory of a point x of X is called the application $\Phi_x : t \longrightarrow \Phi_t(x)$.*
- *$\gamma^+ = \Phi_t(x) :\ t \in \mathbb{R}^+$ (resp. $\gamma^- = \Phi_t(x) :\ t \in \mathbb{R}^-$) is the positive semi-orbit of x (resp. is the negative semi-orbit of x).*
- *$\gamma(x) = \Phi_t(x) :\ t \in \mathbb{R}$ is the orbit of x.*

Definition 1.1.6 ***(Limit sets)***

The assumptions and notations are those of the previous definition, we assume that X is a separated topological space. let x be a point of X;
let $I_x = \Phi_t(x)$ is defined;
We assume that I_x is bounded on the right.
We call the set ω limit of variable x, and the set of adhesion values of the trajectory $t \longrightarrow \Phi_t(x)$ of x is represented by $\omega(x)$, when t tends towards $+\infty$.
we assume that I_x is not left-bounded. We call the α limit set of x and the set of adhesion values of the trajectory $t \longmapsto \Phi_t(x)$ of x is denoted by $\alpha(x)$, when t tends towards $-\infty$.

Definition 1.1.7 ***(Absorbent set)***

Suppose that the system (1.1.2) in which X is an open set of $\mathbb{R}^n$ and that Φ is of class C^1. Suppose further that this equation admits admits solutions whatever $t \geq 0$. A subset D of X is said to be absorbent according to (1.1.2) if every bounded subset Γ of satisfies $x(t, \Gamma) \subset D$ for all sufficiently large time t. Similarly, D is said to be absorbent when for any initial condition x_0, there exists $t > 0$ such that $\Phi_t(x_0) \in D$.

Definition 1.1.8 ***(Invariant set)***

- *A subset Γ of X is said to be positively invariant if $\Phi_t \Gamma \subset \Gamma$ for all $t \geq 0$.*
- *A subset Γ of X is said to be negatively invariant if $\Phi_t \Gamma \subset \Gamma$ for all $t \leq 0$.*
- *A set Γ of X is said to be invariant if $\Phi_t \Gamma \subset \Gamma$ for all $t \in \mathbb{R}$.*

The sets α-limit and ω-limit are examples of invariant and absorbant sets.

Definition 1.1.9 *Let x^* be an equilibrium point for the system (1.1.2).*

- *The set of elements $x \in X$ such that for all $t \in \mathbb{R}^+$, $\Phi(t,x)$ is defined and that*

$$\lim_{t \longrightarrow \infty} \Phi_t(x) = x^*.$$

this is called the basin of attraction of a point x^*.

We call the repulsion basin of a point x^* the set of elements $x \in X$ such that for all $t \in \mathbb{R}^+$, $\Phi(t,x)$ is defined and that

$$\lim_{t \longrightarrow -\infty} \Phi_t(x) = x^*.$$

Let X be the phase space associated with $\Phi(t,x)$ such that $\Phi(t,x) : \mathbb{R}^+ \times X \longrightarrow X$.

1.1.2 Concept of stability and equilibrium point

Definition 1.1.10 ***(Equilibrium point)***

We define equilibrium point (also known as critical point or stationary point) of the system (1.1.2) any point $x^* \in X$ such that:

$$\forall x \in X,\ f(x^*) = 0.$$

The equilibrium points are trajectories of the system (1.1.2), they correspond to the constant solutions.

Definition 1.1.11 ***(Concept of stability)***

Let the system (1.1.2) be $x^* \in X \subset \mathbb{R}^n$ at an equilibrium point and $\|.\|$ a norm of $\mathbb{R}^n$.

- *We say that x^* is a stable equilibrium point if*

$$\forall \varepsilon > 0,\ \exists \delta > 0 \text{ such that } \|x_0 - x^*\| < \delta \Longrightarrow \|\Phi_t(x_0) - x^*\| < \varepsilon,\ \forall t \geq t_0.$$

- *The equilibrium point x^* of the system (1.1.2) is said to be unstable if it is not stable.*

Definition 1.1.12 ***(Asymptotically stable equilibrium point)***

If an equilibrium point x^* of the system of differential equations (1.1.2) is stable and if,

$$\exists \delta_0 > 0 \text{ such that } \|x_0 - x^*\| \leq \delta_0 \Longrightarrow \lim_{t \longmapsto +\infty} \|\Phi_t(x_0) - x^*\| = 0.$$

Then, the equilibrium x^* is asymptotically stable.

Definition 1.1.13 ***(Attractive equilibrium point)***

The equilibrium point x^* is considered attractive if there is $\rho > 0$ where

$$\|x_0 - x^*\| \leq \rho \Longrightarrow \lim_{t \longmapsto +\infty} \|\Phi_t(x_0) - x^*\| = 0.$$

Attractiveness means that if the state is initialized in a certain neighborhood of the equilibrium state, then the trajectory resulting from this initial state will converge towards the equilibrium state after a sufficient time (even infinite).

Definition 1.1.14 ***(Globally asymptotically stable equilibrium point)***

The equilibrium point is globally asymptotically stable if the asymptotic stability defined in Definition (1.1.2) is valid for any initial state in $\mathbb{R}^n$.

1.2 Stability criteria

We consider the system of differential equations given by equation (1.1.2), depending on the function f we distinguish two cases as follows:

1.2.1 linear case

In this case the differential system (1.1.2) is written

$$\dot{x} = Ax, \tag{1.1.6}$$

where A is a matrix of order n, $(A \in M_n(\mathbb{R}))$.

Theorem 1.1.1

- *The equilibrium point $x^* = 0$ of (1.1.6) is said to be asymptotically stable if and only if all the eigenvalues of the matrix A have strictly negative real parts.*
  The equilibrium is also said to be exponentially stable (i.e. there exists $C, > 0$ such that $\|e^{tA}\| \leq Ce^{-\gamma t}, \forall t > 0$.)
- *The equilibrium state x^* is said to be stable if and only if all the eigenvalues of the matrix A have negative or zero real parts and those with zero real parts are semi-simple.*
  The semi-flow is uniformly bounded: if there exists $C > 0$ such that $\|e^{tA}\| \leq C., \forall t > 0$.

Theorem 1.1.2 *The following properties are equivalent:*

- *An eigenvalue λ is semi-simple (or non-defective).*
- *The multiplicity of λ in the minimal polynomial is 1.*
- *The algebraic and geometric multiplicities of λ coincide.*

1.2.2 Non-linear case

Definition 1.1.15 *We represent by the linearized system of the system (1.1.2) in x^*, the system which is formulatted as follows:*

$$\dot{x} = J(x^*)(x - x^*) + f(x^*) \tag{1.1.7}$$

such that $J = (Df)(x^*)$ is the derivative of f at x^* and is called the Jacobian matrix.

Before presenting the theorem which gives us the relation between the stability of systems (1.1.7) and (1.1.2).
The following results are used:

Definition 1.1.16 *Let x and y be two vector fields on a manifold M. They are said to be topologically equivalent if there exists a homeomorphism $h : M \longrightarrow M$ that maps the trajectories of x onto those of y and that respects the direction of time. If, moreover, h is a C^k-diffeomorphism, x and y are k-equivalent.*

Theorem 1.1.3 ***(Hartman-Grobman, [19])***
Let x^* be a hyperbolic equilibrium point of a regular vector field x, there exists a neighborhood U of x^* such that the vector fields x and $J(x^*)$ are topologically equivalent on U.

Theorem 1.1.4

- *The Jacobian matrix J is a Hurwitz matrix (i.e. all eigenvalues have negative real parts) so the equilibrium x^* is locally asymptotically stable.*
- *Suppose that $J(x^*)$ has at least one eigenvalue with a strictly positive real part, then x^* is unstable.*

Finally, it is clear that the stability condition of an equilibrium point requires a verification that all the eigenvalues of the Jacobian matrix have a negative real part, something that is

not always easy and obvious because the calculation of the eigenvalues of the Jacobian is not always easy. However, there are criteria that allow us to conclude on the local stability or instability of an equilibrium point without calculating the eigenvalues of the Jacobian matrix associated with the system.

We start with the following Routh-Hurwitz criterion:

1.2.3 Routh-Hurwitz criteria [38]

The Routh-Hurwitz criterion is an algebraic criterion for evaluating the stability of a system and determining a necessary and sufficient condition for a polynomial to admit all its roots in the open left half-plane.

Soit le polynôme P, défini par l'expression ci-dessous,

$$P(\lambda)=\alpha_n\lambda^n+\alpha_{n-1}\lambda_{n-1}+...+\alpha_1\lambda+\alpha_0, \quad \alpha_0>0. \tag{1.1.8}$$

Let H be a so-called Hurwitz matrix, H is a square matrix of order n defined as follows:

$$H=\begin{pmatrix} \alpha_{n-1} & \alpha_{n-3} & \alpha_{n-5} & \alpha_{n-7} & \dots \\ \alpha_n & \alpha_{n-2} & \alpha_{n-4} & \alpha_{n-6} & \dots \\ 0 & \alpha_{n-1} & \alpha_{n-3} & \alpha_{n-5} & \dots \\ 0 & \alpha_n & \alpha_{n-2} & \alpha_{n-4} & \dots \\ 0 & 0 & \alpha_{n-1} & \alpha_{n-3} & \dots \\ \vdots & \vdots & \vdots & \vdots & \vdots \end{pmatrix}$$

Let us denote by H_k the minor of order k of the matrix H.

$$\begin{aligned} H_1 &= \alpha_{n-1}, \\ H_2 &= \begin{vmatrix} \alpha_{n-1} & \alpha_{n-3} \\ \alpha_n & \alpha_{n-2} \end{vmatrix}, \\ H_3 &= \begin{vmatrix} \alpha_{n-1} & \alpha_{n-3} & \alpha_{n-5} \\ \alpha_n & \alpha_{n-2} & \alpha_{n-4} \\ 0 & \alpha_{n-1} & \alpha_{n-3} \end{vmatrix}. \end{aligned}$$

Theorem 1.1.5 *All roots of P have a negative real part $\Longleftrightarrow H_k>0, \forall k\in[1,n]$.*

In our study the roots of P are the eigenvalues of the characteristic equation $\det(J\lambda I)=0$, which implies the following result: The equilibrium point is asymptotically stable $\Longleftrightarrow \mathrm{H}_k > 0$, $\forall k \in [1,n]$.

- For $n=1$, we clearly have $H_1=\alpha_0>0$ implies that the root of $P(\lambda)$ is $-\dfrac{\alpha_0}{\alpha_1}<0$.
- For $n=2$, the Hurwitz conditions are $H_1>0$ and $H_2>0$:

$$H_1=|\alpha_{n-1}|, \quad H_2=\begin{vmatrix}\alpha_1 & 0\\ \alpha_2 & \alpha_0\end{vmatrix}=\alpha_0\alpha_1.$$

The Routh-Hurwitz criteria $H_1>0$ and $H_2>0$ reduce to

$$\alpha_0>0 \text{ et } \alpha_0\alpha_1>0.$$

These conditions are equivalent to $Tr(A)<0$ and $\det(A)>0$ where A is a matrix of dimension 2 of which P is its characteristic polynomial.

1.2.4 Descartes' Rule [12, 62]

Let P be a polynomial of order n defined by:

$$P(x)=a_0+a_1x+a_2x^2+\ldots+a_nx_n,\ a_n\neq 0.$$

Proposition 1.1.0 *If all coefficients of P are positive, then P has no positive roots.*

Corollary 1.1.1 *If all the coefficients of P are non-zero and of alternating signs then P does not admit a negative root.*

Proposition 1.1.0 *If there is only one sign change in the coefficients of P, then there is exactly one positive root.*

Theorem 1.1.6 ***(Descartes' rule of signs)***
The number of positive roots of a polynomial P with real coefficients does not exceed the number of sign changes of its ordered coefficients (ordered by the value of their index). A zero coefficient is not counted as a sign change.

1.2.5 Vidyasagar Theorem

We recall the statement of Vidyasaga's theorem [77].

Theorem 1.1.7 *A class C^1 system is considered as follows:*

$$\begin{cases} x_1^{'} = f_1(x_1), \\ x_2^{'} = f_2(x_1, x_2). \end{cases} \tag{1.1.9}$$

Such that the origin of $\mathbb{R}^n$ is globally asymptotically stable for the isolated system $x_1^{'} = f_1(x_1)$ on $\mathbb{R}^n$ and such that the origin of $\mathbb{R}^m$ is globally asymptotically stable for $x_2^{'} = f_2(0, x_2)$.
Then the origin is asymptotically stable for the system (1.1.9).
Si toutes les trajectoires sont bornées alors l'origine est globalement asymptotiquement stable sur $\mathbb{R}^n \times \mathbb{R}^m$.

1.2.6 Lienard-Chipart (LC) Criteria [27]

The LC criteria is a standard tool to understand the Hurwitz stability problem, which in turn has important consequences on the dynamics of some systems of differential equations. According to LC theorem, the system having the characteristic polynomial $P(\lambda) = C_n + C_{n-1}\lambda + ... + \lambda_n = 0$, $C_n > 0$ is stable if any of the following four sets of conditions is satisfied:

1. Both the even order Hurwitz determinants and the even coefficients are positive.
2. Both the odd order Hurwitz determinants and the even coefficients are positive.
3. Both the even order Hurwitz determinants and the odd coefficients are positive.
4. Both the odd order Hurwitz determinants and the odd coefficients are positive.

The French mathematicians Lienard and Chipart developed the aforementioned stability requirements in 1914 after becoming concerned about the redundancy of the RH stability conditions.

1.3 Lyapunov Methods

Lyapunov functions play an important role in the study of the stability of dynamical systems. This section is devoted to some results due to Lyapunov. Let $V : \Omega \subset \mathbb{R}^n \longrightarrow \mathbb{R}$ be a continuous function;

Definition 1.1.17

- *If $V(x_0) = 0$ and $V(x) > 0$ in a neighborhood 0 of x_0 for all $x \neq x_0$ in this neighborhood. Hence, The function V is said to be positive definite;*
- *If V is positive definite. Then, the function V is said to be negative definite;*
- *If $V(x_0) = 0$ and $V(x) \geq 0$ in a neighborhood Ω_0 of x_0. So, the function V is said to be semi-positive.*

Definition 1.1.18 ***(Lyapunov Function)***

A function $V : \Omega \longrightarrow \mathbb{R}$ is a Lyapunov function for the system (1.1.2) if it decreases along the trajectories of the system. If V is of class C^1, this amounts to saying that its derivative $\dot{V}$ with respect to the system (1.1.2) is negative on Ω, i.e., $\dot{V}(x) \leq 0$ for all $x \in \Omega$.

Theorem 1.1.8 ***(Lyapunov Theorem)***

- *If the function V is positive definite and $\dot{V}$ negative semi-definite on Ω, then the equilibrium point x_0 is stable for system (1.1.2).*
- *If the function V is positive definite and $\dot{V}$ is negative definite on Ω, then x_0 is an asymptotically stable equilibrium point for the system (1.1.2).*

This theorem states that to show that an equilibrium point x_0 is stable, it suffices to find a Lyapunov function at this point. Moreover, to use the original Lyapunov theorem to show the asymptotic stability of a given system, we must determine a positive definite function V whose derivative $\dot{V}$ is negative definite. In the general case, this is not obvious. The condition on the derivative $\dot{V}$ can be lightened by using La Salle's principle which will be stated in the next part.

Theorem 1.1.9 ***(LaSalle's invariance principle [60], [61])***

Let Ω be a subset of $\mathbb{R}^n$; suppose that is a positively invariant open set for the system (1.1.2) at x_0. Suppose that $V : \Omega \longrightarrow \mathbb{R}$ is a function of class C^1 for the system (1.1.2) at x_0 where:

- $\dot{V} \leq 0$ *on* Ω.
- *We consider* $E = \{x \in \Omega | \dot{V}(x) = 0\}$ *and* L *be the largest set invariant by* X *and contained in* E.

Then, any bounded solution starting in tends to the set L *as time tends to infinity.*

This theorem is a very important tool for systems analysis; unlike Lyapunov, it does not require the function V to be positive definite, nor its derivative $\dot{V}$ to be negative. However, it only provides information about the attractiveness of the system considered at the equilibrium point x_0. For example, it can be used to prove that solutions tend to an equilibrium point only when the set L is reduced to this equilibrium point. It does not indicate whether this equilibrium point is stable or not. When we want to establish the asymptotic stability of an equilibrium point x_0 of, we use the following corollary which is a consequence of the LaSalle invariance principle.

Corollary 1.1.2 ***(Lasalle [60])*** *Suppose* $\Omega \subset \mathbb{R}^n$ *is a connected open set such that* $x_0 \in \Omega$.
Let $V : \mathbb{U} \longrightarrow \mathbb{R}$ *be a positive definite function of class* C^1 *such that* $\dot{V} \leq 0$ *on* $\mathbb{R}$. *Let* $E = \{x \in \mathbb{U} | \dot{V}(x) = 0\}$*; suppose that the largest positively invariant set contained in* E *is reduced to the point* x_0.
Therefore x_0 *is an asymptotically stable equilibrium point for the system (1.1.2).*
If these conditions are satisfied for $\mathbb{U} = \Omega$ *if moreover* V *is proper on* Ω, *that is to say if* $\lim V(x) = +\infty$ *when* $d\left(x, \frac{\partial}{\partial x}\Omega + \|x\| \longrightarrow +\infty\right)$, *then all trajectories are bounded for all* $t \geq 0$ *and* x_0 *is a globally stable equilibrium point for the system (1.1.2).*

Corollary 1.1.3 *Under the assumptions of the previous theorem, if the set* L *is reduced to the point* $x_0 \in \Omega$, *then* x_0 *is a globally asymptotically stable equilibrium point for the system (1.1.2) defined on* Ω.

1.4 The basic reproduction number

Mathematical models in epidemiology play a very important role in eradicating infectious diseases.

The $\mathbf{R}_0$ which has several names such as contagion index, original reproduction number, basic reproduction rate ..., that is to say the number of individuals who are infected on average by a single contaminated individual.

1.4.1 The concept of $\mathbf{R}_0$ in epidemiology [28]

$\mathbf{R}_0$ is defined epidemiologically as the average number of secondary infections, wich are infections that occur during or after treatment of another infection, produced when an infected person is brought into contact with a population of susceptible individuals, or an infected individual has contracted the disease, and susceptible individuals are healthy but may contract the disease.

1.4.2 Mathematical definition of $\mathbf{R}_0$ [28]

The correct mathematical definition of $\mathbf{R}_0$ was presented by O. Dikemann, J.A.P. Heers-terbeek and J.A.J. Metz in 1990 [28], addressed the dominant eigenvalue of what is known as the "next generation operator". The authors in this definition concentrate on a specific equilibrium point of the epidemiological dynamic system, often referred to as the disease-free equilibrium DFE, corresponds to the state in which, in the absence of the infectious agent, the population is in equilibrium and is defined by a constant susceptible population. The $\mathbf{R}_0$ value, i.e. the dominant eigenvalue of the next generation operator, aims to ensure the stability [29] ($\mathbf{R}_0 < 1$) if not the instability ($\mathbf{R}_0 > 1$) of the DFE.
It is calculated using a simple equation:

$$\mathbf{R}_0 = \beta . c . d$$

- β: representing the probability of transmission.
- c: the contact rate (or number of contacts per unit of time).
- d: the duration of contagiousness.

1.5 Next generation matrix method

We employ the next generation matrix method to calculate the $\mathbf{R}_0$.
This method, proposed by P. Van Den Driessche and J. Watmough to determine the main

eigenvalues of the "new generation matrix" which specificities epidemic EDO. This method is called the Next generation matrix [30]. By separating the state variables and flows associated with the infectious process from the others, the authors suggest creating the EDO epidemic system. We define X as the set of all disease-free states. That is, $X = \{x_i > 0 | x_i = 0, 1, ..., n`\}$. The dynamics is then expressed as the compartmentalised system shown below:

$$x_i^{'}(t) = f_i(x(t)) = \mathscr{F}_i(x) - \mathscr{V}_i(x), \quad i = 1, ...n,$$

with $\mathscr{V}_i = \mathscr{V}_i^- + \mathscr{V}_i^+$.
Où $\mathscr{F}_i$, $\mathscr{V}_i^-$ and $\mathscr{V}_i^+$ are positive functions such that:
$\mathscr{V}_i^-$: The rate of occurrence of new infections in compartment i.
$\mathscr{V}_i^+$: The rate of transfer of individuals into compartment i.
The functions satisfy the following hypotheses (H1)(H5):

- $(H_1) : if$ x≥ 0, then $\mathscr{F}_i$, $\mathscr{V}_i^-$; $\mathscr{V}_i^+ \geq 0$, $i = 1....n$, note that each function presents a directed transfer of individuals, they are all non-negative.
- $(H_2) : if$ $x_i = 0$, then $\mathscr{V}_i^- = 0$ if there is nothing in the compartment, nothing can come out.
- (H_3) :$F_i = 0$ if $i < m$ this means that infected people cannot enter the uninfected compartments.
- $(H_4) : if$ x$\in X$ then $\mathscr{F}_i(x) = 0$ and $\mathscr{V}_i(x) = 0$ for $i = 1....n$ if there is no immigration for infection, and the population is disease free, they will remain disease free.
- $(H_5) : The eigenvalues of the Jacobian matrix of$ Df(x_0) have negative real parts, if $F \equiv 0$ for all x.

With x_0 is the DFE and $Df(x_0)$ is the Jacobian matrix.

Lemma 1.1.1 *If x_0 is a DFE, with hypotheses (H1)-(H5), then the derivatives $\mathscr{F}(x_0)$ and $\mathscr{V}(x_O)$ are partitioned as:*

$$D\mathscr{F}(x_0)\begin{pmatrix} F & 0 \\ 0 & 0 \end{pmatrix}$$

$$D\mathcal{V}(x_0)\begin{pmatrix} V & 0 \\ J_1 & J_2 \end{pmatrix}$$

Where F and V are m-dimensional square matrices defined by:

$$F = [\frac{\partial \mathcal{F}_i}{\partial x_i}(x_{0)}]$$

and

$$V = [\frac{\partial \mathcal{V}_i}{\partial x_i}(x_{0)}]$$

Additionally, V is a non-singular Matzler matrix, F is non-negative, and every eigenvalue of J_2 has a positive real part.

Definition 1.1.19 ***(spectral radius)***

We define the spectral radius of a square matrix A of dimension m as the maximum value of the modulus of its eigenvalues.

$$\rho(A) = \max|\lambda_i|.$$

Definition 1.1.20 *[78] We denote the basic reproduction number related to the DFE of a dynamic system, by $\mathcal{R}_0$ and is defined as follows:*

$$\mathcal{R}_0 = \rho(-FV^{-1}).$$

Such that:

FV^{-1}: The Next generation matrix for the model.

$\rho(-FV^{-1})$: The spectral radius of the FV^{-1} matrix.

2 History of epidemiological models

The use of modeling is frequently employed to simplify the evaluation of disease management actions. Epidemiological models are valuable because they can be used to investigate hypothetical scenarios and to provide decision-makers the instruments they need to predict the effects of disease outbreaks and the efficacy of intervention strategies. A thorough assessment of epidemiological models is necessary to increase confidence in their forecasts. International cooperation can support the models' validation and increase their applicability in

the handling of health crises. It is important to note that the majority of nations that make up the Organization mondiale de la santé animale (OIE) acknowledge the critical role that the OIE plays in developing guidelines for the development, verification, validation, and use of models. It is imperative to precisely define what an epidemiological model is. However, it appears that there isn't a clear definition of this phrase in the veterinary field. Generally speaking, an epidemiological model is defined as a mathematical and/or logical representation of the epidemiology of disease transmission and the processes involved. These quantitative models provide a representation of the spatiotemporal dynamics of disease transmission among individual animals or among groups of animals. Therefore, an epidemiological model makes it easier to evaluate the possible efficacy of intervention measures and offers an approximation of anticipated geographic range of an outbreak in the event that particular preventive measures are implemented. In terms of managing animal illnesses, epidemiological models may be defined more broadly and comprise a range of statistical and mathematical models that aren't always restricted to defining the disease's severity [17].

A mathematical model is a description of a system or process (epidemiology being no exception) using mathematical tools and language. The process of developing mathematical models is called a mathematical model. We are going to be interested in modeling the spread of infectious diseases. Typically, mathematical models are made up of variables and parameters connected by exact and careful formulations. The modeling process requires the translation of a given scenario (biological or epidemiological for example) into a mathematical problem. After comprehending the model, it is important to evaluate its findings in the context of the scenario under consideration and perhaps look for the initial question's response [1].

Early in the 20th century, the first ideas for epidemiological models were put forth. Mathematical epidemiology's recent history most likely starts on April 16, 1760, at the Royal Academy of Sciences of Paris which presents for public reading a work by Daniel Bernoulli I relating to an analysis of mortality caused by smallpox, and the advantages of inoculation [54] to prevent it.

Through this first mathematical model in the history of epidemiology, which is not a dynamic model, Bernoulli weighs the pros and cons of variolation and predicts that overall variolation applied on the scale of an entire population would increase life expectancy. If the first

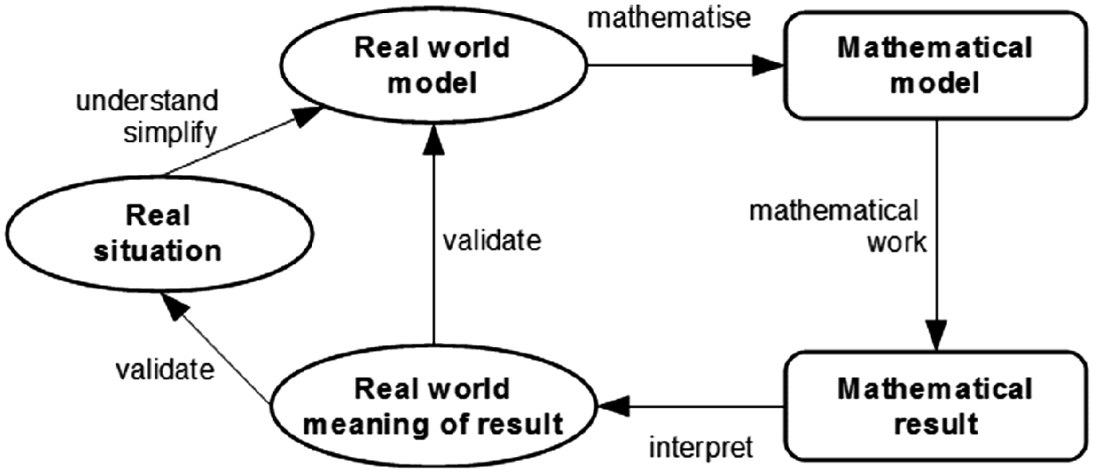

Figure 1: Schematic Summary of Modeling and Synthesis

epidemiological model dates from the 18th century, it was only from the beginning of the 20th century that mathematical epidemiology really developed with models involving dynamic processes. In 1906, by studying the recurrence of measles epidemics, William HAMER constructed a discrete model in which he postulated that the number of new cases during an epidemic is proportional to the product of the number of susceptible individuals in the population and the number of infected patients determines the number of new cases during an epidemic: this is the application of the so-called "mass action". In 1911, Sir Ronald ROSS published a book on the prevention of malaria, and he attempted to formulate the first of many mathematical models for this infection. Most of his work aimed to study the relationship between the incidence of malaria and the proliferation of vectors. Following the work they carried out on the great plague epidemic which appeared in London between 1665 and 1666, William Ogilvy KERMACK and Anderson Gray McKENDRICK published models between 1927 and 1939, in London and Cambridge, which remained famous. Stochastic epidemiological models were developed at the beginning of the twentieth century. Sensed by McKENDRICK in 1926, the stochastic approach appeared for the first time with the publication in Cambridge of the work of Major GREENWOOD. But it was Norman T. J. BAILEY who systematized the stochastic approach with the publication in 1957 of a work entitled "Mathematical Theory of Epidemics". In 1993 the Isaac Newton Institute in Cambridge hosted a semester of work on the question of stochastic epidemiological models [55].

3 The fundamental models of epidemiology

In this section we recall some basic models, which are still used today.

3.1 First mathematical model in epidemiology of Daniel Bernoulli

Daniel Bernoulli, Swiss mathematician and physician, 1700-1782.

Figure 2: Daniel Bernoulli (1700-1782).

The intervention of models in epidemiology has took place on April 30, 1760, dans un mémoire de l'Académie de Sciences de Paris. D. Bernoulli presents a model and his calculations concerning the smallpox epidemic, creating a blueprint for what we today call "biomathematics". Bernoulli's research is based on the principle of proving whether inoculation of the disease is more beneficial than risky for the target population of this epidemic. Smallpox unfortunately presents a nightmare that threatens human peace because it poses a problem in the way of voluntary eradication by humans [55]. D. Bernoulli adopted the following hypotheses:

- an individual infected for the first time with smallpox has a probability p of dying and a probability $1-p$ of surviving, independently of his age;
- an individual has a probability q of being infected in the year, independently of their age (i.e. the probability that an individual is infected during the small time interval dx between age x and age $x+dx$ is $q.dx$);
- when an individual survives after being infected with smallpox, they are immune for the rest of their life.

Let $m(x)$ denote the natural mortality at age x, then the probability that an individual dies in a small time interval dx between age x and age $x+dx$ is $m(x).dx$. Considering a group of P_0 individuals born in the same year, we note $S(x)$ is the number of individuals who are still alive at age x without having been infected (and who are therefore still likely to be infected). $R(x)$ is the number of individuals who are still alive at age x and immune. $P(x)=S(x)+R(x)$ the total number of individuals still alive at age x. Consider the following model

$$\begin{cases} \dfrac{dS}{dx} = -qS(x) - m(x)S(x), \\ \dfrac{dR}{dx} = q(1-p)S(x) - m(x)R(x), \end{cases} \tag{1.3.10}$$

with $q(1-p)S(x)$ the number of individuals who remain alive and who have acquired the character of being immune. So the total population verified by the following equation

$$\frac{dP}{dx} = -pqS - m(x)P(x). \tag{1.3.11}$$

According to Bernoulli's model we can prove that the fraction of individuals who at age a is still likely to catch smallpox is,

$$\frac{S(x)}{P(x)} = \frac{1}{(1-p)e^{qx}+p}. \tag{1.3.12}$$

Then Bernoulli took charge of the case where smallpox would be inoculated in a gentle, non-harmful way to the entire population from birth. The infectious source of smallpox would be destroyed and the question that arises is to know what success would be in terms of life expectancy [72].

3.2 SI model

The first dynamic model was published in 1906 by W. H. Hamer. This is a simple epidemic model, considering that the population N under study can be divided into two groups, individuals who are susceptible to infections (S), and infected individuals (I). The infection is spread by direct contact of one of the susceptible S with one of the infected I, with a factor β expresses the infection rate. We assume that $N = S + I$ for all $t \in \mathbb{R}^+$, where N is constant and corresponds to the size of the total population. Figure (3) shows the disease transmission diagram.

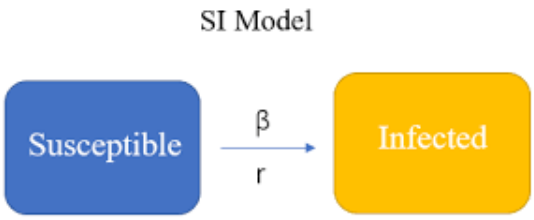

Figure 3: The SI model's transmission schematic.

Hamer's model is described by the following differential system

$$\begin{cases} \frac{dS}{dt} = -\beta SI, \\ \frac{dI}{dt} = \beta SI, \end{cases} \tag{1.3.13}$$

where $f(S,I) = \beta SI$ is the incidence of the disease, i.e. the rate at which infection occurs [20].

3.3 Illustration of mathematical épidemiological models SI

The SI model is the simplest form of all disease models. Individuals are born into the simulation with no immunity (susceptible). Once infected and with no treatment, individuals stay infected and infectious throughout their life, and remain in contact with the susceptible population. Among the mathematical models of SI-type epidemics:

Ronald Ross Model 1911

In 1911, Sir Ronald Ross proposed a model which took into account the Anopheles and human populations. This model is certainly the starting point for vector-host models. Ross divides hosts (humans) and vectors (Anopheles) into two classes of susceptible and infected respectively. Let S_H be the population of susceptible humans and I_H the population of infected humans. Ross assumes that there is no latency period and therefore an infected person is automatically infectious. Similarly, let S_V be the population of susceptible anopheles and I_V the population of infected anopheles. In his model, Ross assumes that the population of humans as well as that of Anopheles mosquitoes is constant and that a mosquito bites k humans per unit time where k is constant. We have graph (4) [22] .

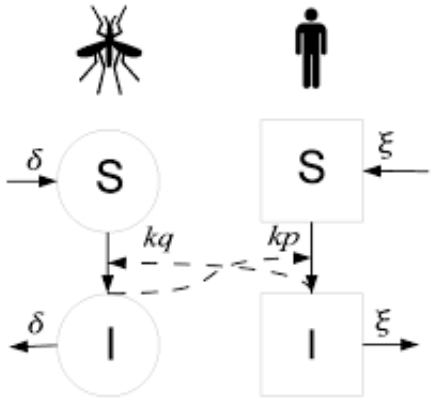

Figure 4: Ross's compartmental model.

The mathematical model is given as follows [84]:

$$\begin{cases} \dfrac{dS_h}{dt} = \xi H - \xi S_H - kp\dfrac{I_V}{H}S_H + \gamma_H I_H, \\ \dfrac{dI_h}{dt} = kp\dfrac{I_V}{H}S_H - (\xi+\gamma_H)I_H, \\ \dfrac{dS_V}{dt} = \delta V - \delta S_V - kq\dfrac{I_H}{H}S_V, \\ \dfrac{dI_V}{dt} = kq\dfrac{I_H}{H}S_V - \delta I_V, \end{cases} \tag{1.3.14}$$

The parameters are given by the following table:

Parameters	Description
k	rate of human bites per unit of time.
p	proportion of bites producing infection in humans.
q	proportion proportion of bites by which a susceptible mosquito becomes infected.
γ_H	proportion The rate of recovery for humans.
ξ	proportion The rate of mortality for humans.
δ	proportion The rate of mortality for mosquito.
H	proportion The total human population (cst).
V	proportion The total mosquito population (cst).

This model of Ross was the point starting from a rich and varied literature on vector-host mathematical models [2, 52, 64] and the references that are there. An excellent review was recently done by Mandal et al. in [65]. The mathematical analysis of the Ross model (1.3.14)

was fully studied by Lotka [65] in 1923. Ross's simple model did not take into account this period of parasite latency in mosquitoes and their survival during the latter [21].

Anderson and May Model

Anderson and May [5] built an extension of R. Ross and Macdonald's models, introducing the class of exposures into the human population. This divided the host population into three compartments (S_H, E_H, I_H), same for the mosquito population (S_V, E_V, I_V).

Their model consists of four ordinary differential equations describing the evolution, in relation to time, of exposed and infected classes [22],

$$\begin{cases} \frac{dE_H}{dt} = abmI_V(t)(1-E_H(t)-I_H(t))-\gamma E_H(t)-\mu_H E_H(t) \\ \qquad -abmI_V(t-\tau_H)[1-E_H(t-\tau_H)-I_H(t-\tau_H)]e^{-(\gamma+\mu_H)\tau_H}, \\ \frac{dI_H}{dt} = abmI_V(t-\tau_H)[1-E_H(t-\tau_H)-I_H(t-\tau_H)]e^{-(\gamma+\mu_H)\tau_H}-\gamma I_H(t)-\mu_H I_H(t), \\ \frac{dE_V}{dt} = acI_H(t)[1-E_V(t)-I_V(t), \\ \qquad -acI_H(t-\tau_V)[1-E_V(t-\tau_V)-I_V(t-\tau_V)]e^{-\mu_V\tau_V}-\mu_V E_V(t), \\ \frac{dI_V}{dt} = acI_H(t-\tau_V)[1-E_V(t-\tau_V)-I_V(t-\tau_V)]e^{-\mu_V\tau_V}-\mu_V I_V(t), \end{cases} \tag{1.3.15}$$

The model parameters are given in the following table:

Parameters	Description
a	Number of bites per mosquito and time unit (time^{-1})
b	Probability of infection from infectious mosquitoes to humans by bites.
c	Probability of transmission of infection from an infected human to a mosquito by bite.
γ	Average recovery rate for humans.
m	Nnumber of mosquitoes per human (host)(time^{-1}).
μ_H	Natural mortality rate of hosts (time^{-1}).
μ_V	Mosquito mortality rate (time^{-1}).
τ_V	The mosquito latency period (estimated between 5 and 15 days).
τ_H	The latency period of humans, estimated at between 10 and 100 days.

This turning point, initiated by the work of Anderson and May,is linked to the interest that ecologists have begun to focus on host-parasite systems (Anderson and May 1991).

Gaff and Gross model in 2007

In 2007, Holly D. Dry and Louis J. Gross was considered a model of tick-borne infection dynamics in the case of one host, one pathogen, and one life stage. The following table represents variable and parameters used in the model [39].

Parameters	Description
β	Growth rate for hosts.
$\tilde{\beta}$	Growth rate for ticks.
K	Carrying capacity for hosts per m^2.
M	Maximum number of ticks per host.
b	External death rate of hosts.
$\tilde{b}$	External death rate of ticks.
A	Transmission rate from hosts to ticks.
$\tilde{A}$	Transmission rate from ticks to hosts.
v	Recovery rate of hosts.

Where, the density of the host population N and ticks V as well as the density of individuals in each group infected with the disease (Y and X, respectively for hosts and ticks). The model is descripted as follows:

$$\begin{cases} \frac{dN}{dt} = \beta\left(\frac{K-N}{K}\right)N - bN, \\ \frac{dV}{dt} = \tilde{\beta}V\left(\frac{MN-V}{MN}\right) - \tilde{b}V, \\ \frac{dY}{dt} = A\left(\frac{N-Y}{N}\right)X - \beta\frac{NY}{K} - (b+v)Y, \\ \frac{dX}{dt} = \tilde{A}\left(\frac{Y}{N}\right)(V-X) - \tilde{b}\frac{VX}{MN} - \tilde{b}X, \end{cases} \tag{1.3.16}$$

The disease model presented here incorporates non-constant population sizes and spatial heterogeneity utilizing a system of differential equations that may be applied to a variety of spatial patches.

Aqeel Ahmad et al in 2020

In this 2020 work the authors designed the analysis and modeling of bovine papesia disease using fractional calculation. The solution to the fractional arrangement system for bovine papesia and tick populations was determined using the fractional derivatives of Cabuto and

Atangana-Paleano-Caputo (ABC). By applying the method of symmetry analysis and Laplace conversion with polynomial symmetry, the analytical solution of bovine babysia was obtained [85].

Taking the same assumptions as in Aranda et al. model in 2012, and considering the following proportions:

$$U=\frac{S_B}{N_B},\quad V=\frac{I_B}{N_B},\quad W=\frac{Z_B}{N_B},\quad X=\frac{S_T}{N_T},\quad Z=\frac{I_T}{N_T}.$$

we have the following fractional order model in time t is given by:

$$\begin{cases} D^{\beta}U=(\mu_B+\alpha)(1-U-V)-\beta_B UZ, \\ D^{\beta}V=\beta_B UZ-\lambda_B V, \\ D^{\beta}Z=\beta_T(1-Z)V-\mu_T pZ, \end{cases} \tag{1.3.17}$$

With initial conditions $U(0)>0$, $V(0)>0$ and $Z(0)>0$.

The fractional order system was subjected to a qualitative analysis, and the uniqueness of the associated fractional order model was established. The fractional order model, employing the Atangana-Baleanu (ABC) derivative, effectively captures the dynamics of bovine babesiosis and tick populations.

3.4 SIS model

SIS stands for susceptible, infected, and susceptible. The total population size, denoted by N, stays constant. The probability of contact is denoted by β. Once meeting the infected, one is to be infectious, Every infected individual can recover with a deterministic probability. The probability of infection again after recovering, set as γ. Figure (5) shows the compartmental diagram for the SIS model [50].

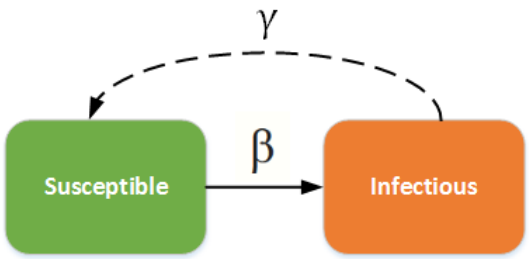

Figure 5: The SIS model's transmission schematic.

Thus model is given by the following

$$\begin{cases} \dfrac{dS}{dt} = -\beta SI + \gamma I, \\ \dfrac{dI}{dt} = \beta SI - \gamma I, \end{cases} \tag{1.3.18}$$

with initial conditions $S(0) = S_0 > 0$, $I(0) = I_0 > 0$, $R(0) = R_0 > 0$.

Let us verify that such a system of equations admits one and only one solution with fixed initial conditions, and that this solution has a meaning in terms of population [55].

3.5 Illustration of mathematical épidemiological models SIS

The Susceptible–Infected–Susceptible (SIS) epidemic model is a widely used mathematical framework in epidemiology for analyzing the spread of infectious diseases within a population. We present mathematical models of the SIS form that are applicable to a some of epidemic diseases:

Ross-Macdonald model in 1926

Macdonald's model is based on the Ross model, where the disease-causing parasite spends approximately 10 days in a mosquito during its life cycle [84]. Ross's simple model did not take into account this period of parasite latency in mosquitoes and their survival during the latter. The total human population H is split into susceptible humans $S_h = H - I_h$ and infectious humans I_h. The vector population is divided as follows: S_v mosquitoes susceptible, E_v exposed mosquitoes, and I_v infected mosquitoes.

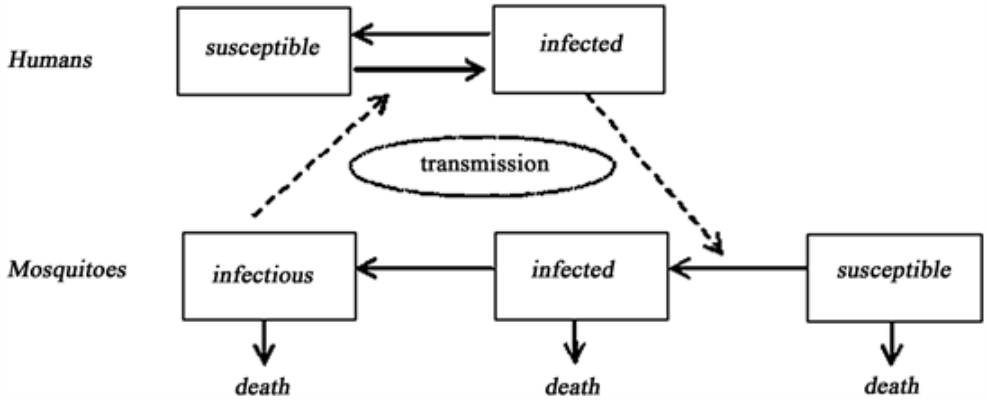

Figure 6: Ross-Macdonald compartmental model.

Taking into account normalized variables,

$$x = \frac{I_h}{H}, \quad y = \frac{E_v}{V}, \quad z = \frac{I_v}{V}$$

The model takes the following form

$$\begin{cases} \frac{dx}{dt} = mabz(t)(1-x(t)) - \gamma x(t), \\ \frac{dy}{dt} = acx(t)(1-y(t)-z(t)) - \mu y(t) - acx(t-\tau)(1-y(t-\tau)-z(t-\tau))e^{-\mu\tau}, \\ \frac{dz}{dt} = acx(t-\tau)(1-y(t-\tau)-z(t-\tau))e^{-\mu\tau} - \mu z(t), \end{cases} \tag{1.3.19}$$

Where the parameters are represented by the following table:

Parameters	Description
m	Number of mosquitoes per human (host).
a	Number of mosquito bites per unit of time (time^{-1}).
b	Probability of transmitting the infectious mosquito infection to humans by a needle.
c	Probability of transmitting infection from an infected man to a mosquito through a sting.
γ	The rate of recovery for humans (time^{-1}).
μ	The rate of mortality for mosquito (time^{-1}).
τ	The mosquito latency period (estimated between 5 and 15 days).

The time spent in the class is represented by an exponential distribution.

Using the delayed differential equations we can model the incubation period (the time it takes an infected mosquito to become infectious) as a fixed duration. The same assumptions are made about human population dynamics as in the Ross model.

The Mcdonald-Ross model requires a theory of delayed differential equations, that it can be simplified to a system of ordinary differential equations (as in the article Nakul Chitnis [18] where the mathematical study is well detailed) [22].

3.6 SIR model

At the beginning of the 20th century, W.O. Kermack (public health doctor) and A.G. Mc Kendrick (biochemist) publish a simple model of the spread of epidemics by direct contact. At the time, they compared their model with real data on the spread of the Bombay plague between 1905

and 1906. The population is divided into three categories: susceptible individuals (S), infected individuals (I), and Recovered individuals individuals (R). The model contains two parameters: β infection rate and γ recovery rate. The total population N is assumed to be constant [55]:

$$N = S + I + R$$

The Kermack-Kendrick model is represented by the following diagram.

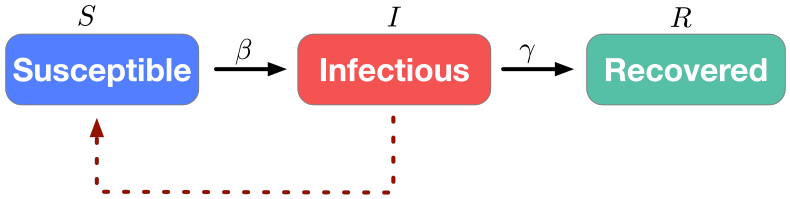

Figure 7: The SIR model's transmission schematic.

The SIR model is formulated by the following differential system

$$\begin{cases} \dfrac{dS}{dt} = -\beta SI, \\ \dfrac{dI}{dt} = \beta SI - \gamma I, \\ \dfrac{dR}{dt} = \gamma I, \end{cases} \tag{1.3.20}$$

The basic reproduction number for the SIR compartmental model (3.3.11) is given by

$$R_0 = \frac{\beta N}{\gamma}$$

The most crucial factor to take into account when examining any epidemic model for an infectious disease is without a doubt the number R_0, often known as the fundamental reproduction number. In particular, R_0 determines whether an epidemic can occur at all; to see this for the basic SIR model, note in two equations of model (3.3.11) that I can never increase unless $R_0 > 1$. This makes intuitive sense, since if each individual transmits the infection to an average of less than one individual then the number of cases must decrease with time [35]. If we expand the SIR model to include B births per unit time and a natural mortality rate (per

capita) then our equations become [35].

$$\begin{cases} \frac{dS}{dt} = B - \beta SI - \mu S, \\ \frac{dI}{dt} = \beta SI - \gamma I - \mu I, \\ \frac{dR}{dt} = \gamma I - \mu R, \end{cases} \tag{1.3.21}$$

We define the basic reproduction rate as follows [72],

$$R_0 = \frac{\beta}{\gamma + \mu}.$$

3.7 Illustration of mathematical épidemiological models SIR

Many human diseases are studied using SIR mathematical models. Our major goals are to examine the structure of these models, discuss what useful information can be derived from them, and indicate how they may be used to make general predictions on the possible courses of the associated diseases when particular types of actions are taken. We conclude that the simplest SIR models are valuable as tools for deriving critical qualitative features of the spread of disease. Various issues are also considered relative to the successes and failings of these models. We introduce SIR models to study various epidemics:

Tumwiine and Mugisha model

This model is type SIRS for hosts and SI for vectors, the authors assume that individuals of all classes have the same mortality rate. They exclude mosquitoes in their model from the moment they no longer participate in the infection cycle.

Their model is given as follows [79].

The schema that represents the model is

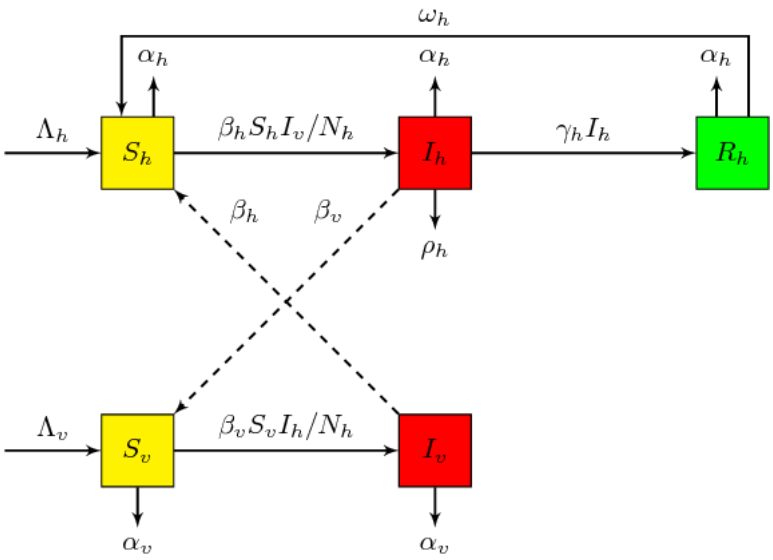

Figure 8: Tumwiine and Mugisha compartmental model.

With mathematical representation by a system of differential equations following.

$$\begin{cases} \dfrac{dS_H}{dt} = \lambda_H N_H - \dfrac{ab}{N_H} S_H I_V + \nu I_H + \gamma R_H - \mu_H S_H, \\ \dfrac{dI_H}{dt} = \dfrac{ab}{N_H} S_H I_V - (\nu + r + \delta + \mu_H) I_H, \\ \dfrac{dR_H}{dt} = r I_H - \gamma R_H - \mu_H R_H, \\ \dfrac{dS_V}{dt} = \lambda_V N_V - \dfrac{ac}{N_H} S_V I_H - \mu_V S_V, \\ \dfrac{dI_V}{dt} = \dfrac{ac}{N_H} S_V I_H - \mu_V I_V, \end{cases} \tag{1.3.22}$$

The basic reproduction number is then calculated and given by [31]:

$$R_0 = \frac{a^2 b_1 b_2 m}{\lambda_m (r_h + \gamma + \lambda_h + \delta}.$$

The models we have presented so far only take into account the interaction between the main actors involved in the malaria transmission process. The increasing observation of the seasonality of malaria epidemics has led authors to propose mathematical models of the dynamics of malaria transmission by integrating the influence of temperature on the life cycle of mosquitoes.

Model of Aranda in 2012

Aranda et al. (2012) in [6] used a system of ordinary differential equations to present a mathematical model for bovine babesiosis. The Babesiosis disease model will be constructed based

on the following fundamental hypotheses:

The bovine population $N_B(t)$ is categorized into three groups: susceptible $S_B(t)$, infected $I_B(t)$, and controlled $C_B(t)$.

The parameter μ_B is the birth rate of the bovines. The birth rate μ_B is assumed equal to the natural death.

The total tick population $N_T(t)$ is composed of two groups: ticks susceptible to infection $S_T(t)$ and ticks infected with Babesia $I_T(t)$.

The parameter μ_T is the birth rate of the bovines and it is assumed equal to the natural death.

A susceptible bovine can infect a portion of the diseased population I_B through the efficient transmission of a pathogen by an infected tick, occurring at a rate of β_B.

A susceptible tick can be infected if there exists an effective transmission when it stings an infected bovine, at a rate β_T .

A hundred percent vertical transmission in the bovine populations μ_B and ticks populations it occurs with probability $(1-p)$ where p is the probability that a susceptible tick was born from an infected one.

A fraction λ_B of the infected bovines are controlled, that is, treated against Babesia parasite.

A fraction α_B of the controlled bovine may return to susceptible state.

Homogeneous mixing is assumed, that is, all susceptible bovines have the same probability to be infected and all susceptible ticks have the same probability to be infected.

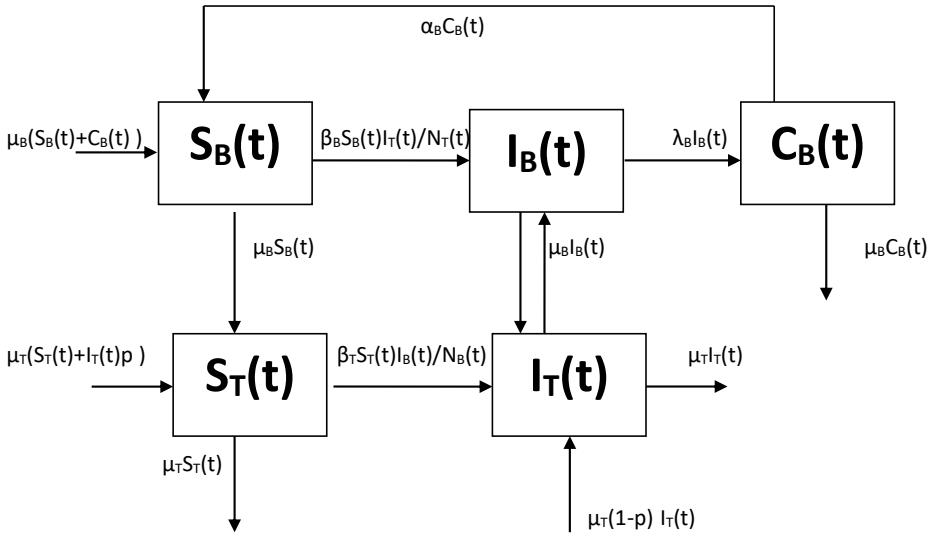

Figure 9: Dynamics of Babesiosis disease in bovine and tick populations.

The following system of equations is used to model the dynamic transmission of Babesiosis between bovine and ticks.

$$\begin{cases} S_B^{'}(t) = \beta_B S_B(t)\dfrac{I_T(t)}{N_T(t)} + (\mu_B + \alpha_B)C_B(t), \\ I_B^{'}(t) = \beta_B S_B(t)\dfrac{I_T(t)}{N_T(t)} - \lambda_B I_B(t), \\ C_B^{'}(t) = \lambda_B I_B(t) - (\mu_B + \alpha_B)C_B(t), \\ S_T^{'}(t) = -\beta_T S_T(t)\dfrac{I_B(t)}{N_B(t)} + \rho\mu_T I_T(t), \\ I_T^{'}(t) = \beta_T S_T(t)\dfrac{I_B(t)}{N_B(t)} - \rho\mu_T I_T(t), \end{cases} \tag{1.3.23}$$

Aranda and other authors have proven that the basic reproduction number R_0 is the value of a threshold that determines global dynamics and their outcomes are satisfactory. If the threshold parameter is $R_0 < 1$, the solution converges to a disease-free balance point. On the other hand, if $R_0 > 1$, the solution converges to the endemic balance point.

Bouzid and Belhamiti model in 2017

Bouzid, L. et Belhamiti,.O., in 2017 investigated the impact of seasonal fluctuations on Babesiosis transmission in cattle and ticks, finding that infestation rates significantly influence infection dynamics. A representation of the model is provided in the following diagram [13].

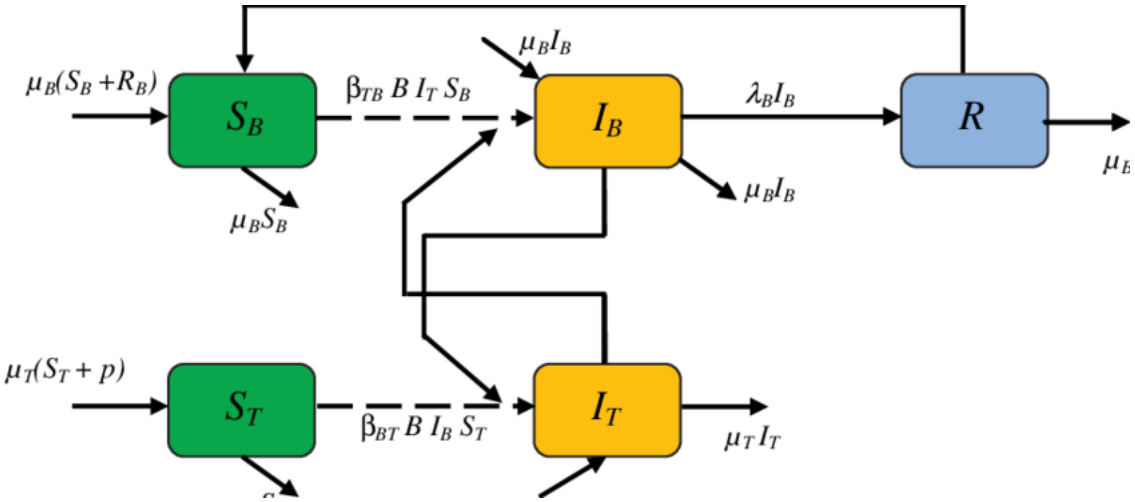

Figure 10: Illustrative diagram SIRSI of the babesiosis model for bovines and ticks.

The model is described by the following system of nonlinear differential equations,

$$\begin{cases} S_B'(t) = \beta_{TB} B(t) S_B I_T(t) + (\mu_B + \alpha_B) R_B(t), \\ I_B'(t) = \beta_{TB} B(t) S_B(t) I_T(t) - \lambda_B I_B(t), \\ R_B'(t) = \lambda_B I_B(t) - (\mu_B + \alpha_B) R_B(t), \\ S_T'(t) = -\beta_{BT} B(t) S_T(t) I_B(t) + \rho \mu_T I_T(t), \\ I_T'(t) = \beta_{BT} B(t) S_T(t) I_B(t) - \rho \mu_T I_T(t), \end{cases} \tag{1.3.24}$$

with parameters β_{BT}, β_{TB}, α_B, λ_B, μ_B, μ_T and p are defined as in the model of Aranda et al., $B(t)$ is the infestation rate as a function of time.

The seasonal fluctuation in babesiosis prevalence might be correlated with the incidence rate, but there are likely additional factors that were not accounted for in this study. The forecasts generated by the proposed model appear quite promising.

Model by G. Giordano et al. in March 2020

Another model introduced by G. Giordano et al. [26], which assumes that the total population $N(t)$ is subdivided into eight subpopulations: susceptible class (S), asymptomatic infected (undetected) class (I), asymptomatic infected detected class (D), symptomatic infected undetected class (A), symptomatic infected detected class (R), threatened class i.e. infected with potentially fatal symptoms detected (T), resistant class (H) and the death class (E). In this case, the distinction between diagnosed and undiagnosed people is very important because undiagnosed people are more likely to spread the infection than diagnosed people, as the

latter are usually isolated and can explain the misperceptions of the case fatality rate and severity of cases. It is also important to predict the number of patients with life-threatening symptoms, because in this situation the disease requires hospitalization and challenges the capacity of the health system.

The SIDARTHE dynamical system consists of eight ordinary differential equations:

$$\begin{cases} \frac{dS(t)}{dt} = -S(t)(\alpha I(t) + \beta D(t) + \gamma A(t) + \delta R(t)), \\ \frac{dI(t)}{dt} = S(t)(\alpha I(t) + \beta D(t) + \gamma A(t) + \delta R(t)) - (\epsilon + \zeta + \lambda) I(t), \\ \frac{dD}{dt} = \epsilon I(t) - (\eta + \rho) D(t), \\ \frac{dA(t)}{dt} = \zeta I(t) - (\theta + \mu + k) A(t), \\ \frac{dR}{dt} = \eta D(t) + \theta A(t) - (\upsilon + \xi) R(t), \\ \frac{dT}{dt} = \mu A(t) + \upsilon R(t) - (\sigma + \tau) T(t), \\ \frac{dH}{dt} = \lambda I(t) + \rho D(t) + k A(t) + \xi R(t) + \sigma T(t), \\ \frac{dE}{dt} = \tau T(t), \end{cases} \tag{1.3.25}$$

with,

- ϵ and θ represent the detection probabilities relative to asymptomatic and mildly symptomatic cases respectively.
- α, β, γ and δ represent respectively the transmission rate due to contacts between susceptible and infected (class of undetected asymptomatic infected, class of detected asymptomatic infected, class of undetected symptomatic infected, class of detected symptomatic infected).
- ζ, η represent the rates at which an infected person, respectively unconscious and conscious of being infected.
- τ The mortality rate.
- μ and À are respectively the rates at which infected individuals with undetected and detected symptoms become infected with potentially fatal symptoms detected.

- λ, k, ξ, ρ and σ represent the recovery rates of the five classes of infected.

Model by S. Djilali et al. in March 2020

The authors in [23] were interested in highlighting the important role of people's age in COVID-19 deaths. They have adopted age-structured models in recent years to understand the spread of infectious diseases. Thus, based on the biological statistics and background of age-structured models, they proposed the following age-structured model:

$$\begin{cases} \frac{\partial S(t,a)}{\partial t} + \frac{\partial S(t,a)}{\partial a} = -\beta S(t,a) \int_0^{+\infty} I(t,\theta) d\theta, \\ \frac{\partial I(t,a)}{\partial t} + \frac{\partial I(t,a)}{\partial a} = \beta S(t,a) \int_0^{+\infty} I(t,\theta) d\theta - (\gamma + \delta(a)) I(t,a), \\ \frac{\partial D(t)}{\partial t} = \int_0^{+\infty} \delta(a) I(t,a) da, \\ \frac{\partial R(t)}{\partial t} = \gamma \int_0^{+\infty} I(t,a) da, \end{cases} \tag{1.3.26}$$

With the initial conditions:

$S(t,0) = 0$, $I(t,0) = 0$, $S(0,a) = S_0(a) \in L^1(0,+\infty)$, and $I(0,a) = I_0(a) \in L^1(0,+\infty)$.

In this population model, $S(t,a)$ and $I(t,a)$ represent the fractions of the population that are susceptible and infected at time t and age a. These values are constrained between 0 and 1. $D(t)$ represents the number of reported coronavirus deaths at time t. $R(t)$ is the count of individuals who have recovered or been removed from the infected population at time t. The variable a signifies the age of the individuals, measured in years. The time t is measured in days. b is the transmission rate. γ represents the removal rate. $\frac{1}{\gamma}$ is the average of the infection period which is estimated for COVID-19 as 10 to 14 days. Considering that $\gamma = 0.1$, which means that the average of the infection period is 10 days. $\int_0^{+\infty} I(t,a) da$ stands for the total fraction of the infected individuals at time t.

Arti Awasthi model in 2023

Arti Awasthi in [10] proposed a mathematical model of COVID-19 to study the dynamics of transmission by taking into account the role of asymptomatic and asymptomatic infected individuals while focusing on the impact of non-pharmacological interventions in controlling the spread of the virus. In their deterministic COVID-19 mathematical model assumed that: The total host population, $N(t)$, is divided into four classes: susceptible $S(t)$, asymptomatic

infected $I_a(t)$, symptomatic infected $I_s(t)$, and recovered R.

$$\begin{cases} \frac{dS}{dt} = \omega - \beta_1 S I_a - \beta_2 S I_s - \alpha S - \mu S, \\ \frac{dI_a}{dt} = \beta_1 S I_a - (\beta_3 + k + \mu) I_a, \\ \frac{dI_s}{dt} = \beta_2 S I_s + \beta_3 I_a - \gamma I_s - \mu I_s - \mu_1 I_s, \\ \frac{dR}{dt} = \gamma I_s - \mu R + \alpha S + k I_a. \end{cases} \tag{1.3.27}$$

where $S(0) > 0$, $I_a(0) > 0$, $I_s(0) > 0$, $R(0) \geq 0$ and all other parameters are taken positive.

The parameters are defined in the following table:

Parameters	Description
ω	Rate at which susceptible individuals are recruited.
β_1	Rate of asymptomatic infection among susceptibles.
β_2	Rate of symptomatic infection among susceptibles.
β_3	Rate at which asymptomatic infected people are getting symptomatically infected.
μ	Natural death rate for individuals of all population classes.
μ_1	Disease induced death rate for symptomatic infected individuals.
α	Recovery rate of susceptible individuals through non-pharmaceutical interventions.
γ	Rate of recovery for symptomatically infected individuals.
k	Rate of recovery for asymptomatically infected individuals.

The model is represented in the following schematic diagram:

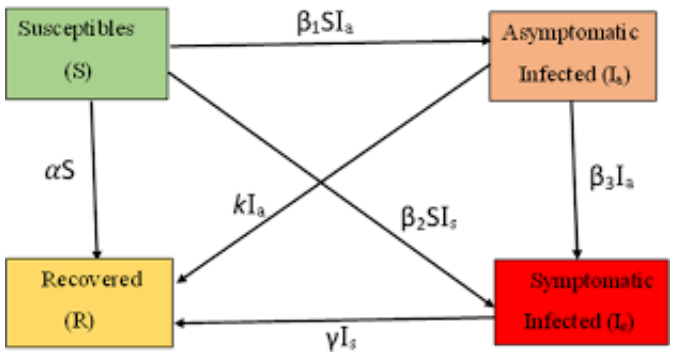

Figure 11: COVID-19 transmission model schematic.

A nonlinear model has been developed in this study to examine the transmission patterns of

COVID-19 infection using both deterministic and stochastic approaches.

3.8 SEIR model

When the infection affects a susceptible individual, a time interval is essential before the signs and symptoms set in and with them the term contagion between susceptible individuals. We have taken the help of individuals who have been infected by the germ (pathogen) of the disease but who are healthy carriers, that is to say, not having the power of transmission, by defining that there is a period which takes the name of latency during which the developing pathogens responsible for the infection are quiescent or potential. During this period of time, the infections caused by latent or exposed symptoms are no apparent symptoms, so don't be able to transmit the malady to the other person because our new type of exposure "E" In all of our places all of the individuals that are likely to cause severe infections in a single attack [55].

We will focus on a SEIRS model. In this case, the population is divided into four compartments: the susceptible S, the exposed E, the infected I and the recovered R. Let S, E, I and R be the respective sizes of the different compartments. So, the model can be described by the following illustrative diagram:

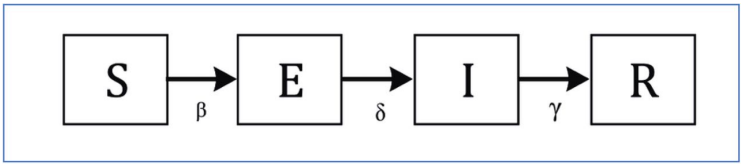

Figure 12: The SEIR model's transmission schematic.

The system of differential equations is:

$$\begin{cases} \frac{dS}{dt} = -\beta SI, \\ \frac{dE}{dt} = \beta SI - \delta E, \\ \frac{dI}{dt} = \delta E - \gamma I, \\ \frac{dR}{dt} = \gamma I, \end{cases} \tag{1.3.28}$$

with β is the disease transmission rate, δ is the infection rate, and γ represents the recovery rate.

The basic reproduction number for the SEIR compartmental model (1.3.28) is given by

$$R_0 = \frac{\beta N}{\gamma}.$$

3.9 Illustration of mathematical épidemiological models SEIR

The SEIR model is presented where there is an exposed period between being infected and becoming infective. It provides a framework for understanding how infectious diseases spread through a population over time. We propose SEIR models as mathematical representations of certain epidemics:

Model by A.Mezouaghi et al. in 2019

In 2019, A.Mezouaghi et al. proposed a spatio-temporal model of the transmission of babesiosis in both cattle and tick populations where the spatial effect was integrated using a diffusion term. The study considered that the study space is a square of length L, so a SEIR-SEI compartmentalized EDP system was used. The model took into account two important factors in the spread of the epidemic: the first is the mobility of infected bovines and infected ticks, and the second is the nearest neighbor infection mechanism. The reaction and propagation model that describes the development of babesiosis is given by the following figure [13].

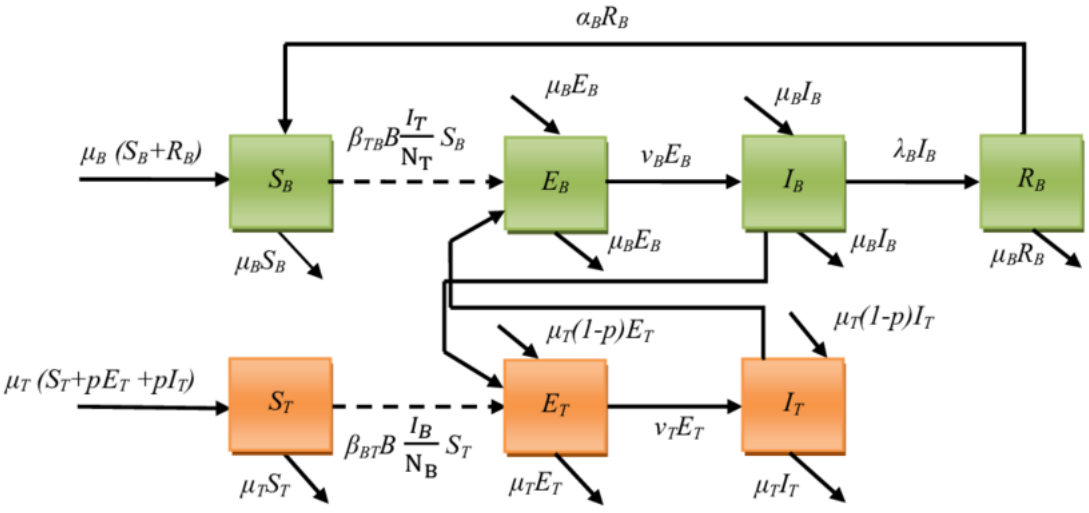

Figure 13: Illustrative diagram of the model.

The transmission dynamics of babesiosis for bovines and tick populations were modeled by the following nonlinear standardized differential equation system,

$$\begin{cases} \frac{\partial S_B(t,x,y)}{\partial t} = -\beta_{TB} B S_B(t,x,y) I_T(t,x,y) - K_{TB} S_B(t,x,y) \nabla^2 I_T(t,x,y) + (\mu_B + \alpha_B) R_B(t,x,y), \\ \frac{\partial E_B(t,x,y)}{\partial t} = \beta_{TB} B S_B(t,x,y) I_T(t,x,y) + K_{TB} S_B(t,x,y) \nabla^2 I_T(t,x,y) - \upsilon_B E_B(t,x,y), \\ \frac{\partial I_B(t,x,y)}{\partial t} = D_B \nabla^2 I_B(t,x,y) + \upsilon_B E_B(t,x,y) - \lambda_B I_B(t,x,y), \\ \frac{\partial R_B(t,x,y)}{\partial t} = \lambda_B I_B(t,x,y) - (\mu_B + \alpha_B) R_B(t,x,y), \\ \frac{\partial S_T(t,x,y)}{\partial t} = -\beta_{BT} B S_T(t,x,y) I_B(t,x,y) - K_{BT} S_T(t,x,y) \nabla^2 I_B(t,x,y) + p\mu_T (I_T(t,x,y) + E_T(t,x,y)), \\ \frac{\partial E_T(t,x,y)}{\partial t} = \beta_{BT} B S_T(t,x,y) I_B(t,x,y) + K_{BT} S_T(t,x,y) \nabla^2 I_B(t,x,y) - (p\mu_T + \upsilon_T) E_T(t,x,y), \\ \frac{\partial I_T(t,x,y)}{\partial t} = D_T \nabla^2 I_T(t,x,y) - p\mu_T I_T(t,x,y) + \upsilon_T E_T(t,x,y). \end{cases} \tag{1.3.29}$$

with parameters β_{BT}, β_{TB}, α_B, λ_B, μ_B, μ_T and p are defined as in the model of Aranda et al., and υ_B is the daily rate at which exposed cattle become infected. υ_T is the daily rate at which the exposed tick becomes infected.

The model showed that the mobility of infected agents is the main driver of long-distance transmission of infectious diseases and that its control can significantly reduce the spread and speed of the disease.

Model by A. Zeb et al. in 2020

A. Zeb et al. [85] introduced a mathematical model where they considered that human contact is the primary cause of the spread of COVID-19. Therefore, isolating infected people as a whole can reduce the risk of transmission of the virus. To do this, they divided the total population into five compartments: susceptible (S), exposed (E), infected, quarantine (Q) and resistant (R).

The mathematical model is given by:

$$\begin{cases} \frac{dS}{dt} = A-\mu S(t)-\beta\frac{S(t)}{N}(E(t)+I(t)), \\ \frac{dE}{dt} = \beta\frac{S(t)}{N}(E(t)+I(t))-\pi E(t)-(\mu+\gamma)E(t), \\ \frac{dI}{dt} = \pi E(t)-\delta I(t)-\mu I(t), \\ \frac{dQ}{dt} = \gamma E(t)+\delta I(t)-\theta Q(t)-\mu Q(t), \\ \frac{dR}{dt} = \theta Q(t)-\mu R(t), \end{cases} \tag{1.3.30}$$

with,

- A: The birth rate.
- β: The rate at which susceptible people move to the infected and exposed class.
- π: The rate at which the exposed population moves to the infected population.
- γ: The rate at which exposed people take their place as isolated.
- δ: The rate at which infected people have been moved to the isolated compartment.
- θ: The recovery rate of isolated people.
- μ: The natural mortality rate plus the disease-related mortality rate.

This work shows that isolating infected humans from the population can reduce the risk of COVID-19 spreading.

CHAPTER 2

Mathematical models of the spread of obesity and its complications

In this chapter, we talk about the biological disease of obesity and the importance of mathematical modeling to control its spread.

1 Biology of obesity and its complications

1.1 Introduction

Obesity has been recognized as a chronic disease by the World Health Organization since 1997. Although not contagious, it is now a pandemic. Obesity is a major public health problem in many countries. It has very serious health consequences, as it causes diabetes, cardiovascular disease, reduced life expectancy, etc. By 2025, estimates predict that obesity will affect half of men and one third of women. According to the WHO, "overweight and obesity are defined as an abnormal or excessive accumulation of body fat that can be harmful to health. Body mass index (BMI) is a simple measure of weight versus height commonly used to estimate overweight and obesity in adults. It is the weight divided by the square of the size, expressed in kg/m^2. Thus, a person is considered overweight when their BMI is 25 or higher, and obesity when their BMI is 30 or higher. To fight obesity, you need to understand the cause of your weight gain. For this, consulting a doctor is essential to set up a personalized diet and receive appropriate advice. However, in general terms, a diet to combat or reduce obesity

is based on basic dietary principles: a balanced diet (fish, meat, dairy products, vegetables, fruits, etc.) and a healthy lifestyle (regular sporting activity, no nibbling). When the number of calories consumed in a day is significantly higher than the number of calories spent, the human body stores an excess of fat. It is this imbalance that partly defines obesity. This definition is then supplemented by the many risk factors that accentuate weight gain, such as junk food, sedentary lifestyle, heredity, hormonal imbalance, smoking cessation. There are also different types (generalized, android, gynoid) and stages of obesity (moderate, severe and morbid) [70]. Obesity is a significant economic and health burden as it is associated with other comorbidities such as diabetes, cardiovascular disease (CVD), certain cancers, gynecological problems, osteoarthritis and premature mortality

1.1.1 Limits of BMI

When calculating BMI in children, physiological changes in fat mass during growth should be taken into account. There are BMI curves in the health record according to age and sex.

Figure 14: Limits of BMI.

For adults, it is also necessary to know how to weight the classification of different types of obesity by BMI taking into account other parameters such as body composition, adipose tissue distribution or the age of obesity. Moreover, for the same BMI, high-performance athletes have more muscle mass and less fat than most subjects [36].

1.1.2 Obesity in figures

Today, the number of overweight people has exceeded the number of undernourished. According to the WHO, at global level, one billion of people are overweight, while 800 million people do not eat enough. Thus, as Coluche said: "I would like to reassure the peoples who are dying of hunger in the world: here we eat for you" [36].

Obesity in France

Since 1997, the Roche Institute, in partnership with TNS Healthcare SOFRES, has been conducting a three-year survey called ObEpi (Obesity Epidemiology) on the frequency of overweight and obesity in a representative sample of the adult French population. It is noted that in 12 years, the average weight of the French has increased by 3.1 kg (72 kg in 2009) and that the BMI has increased by 1 kg/m^2 (25.3 kg/m^2 in 2009). There is also a clear increase in overweight among young adults and the ObEpi series of surveys reveals that obesity in France occurs earlier and earlier. The prevalence of obesity is increasing in all socio-occupational categories and in all urban areas. Today, 31.9% of French people are overweight and 14.5% are obese. This increase is steady with a rate of 0.5% per year. Figure 1 shows the evolution of overweight and obesity in France compared to a normal BMI between 1997 and 2009. Obesity is increasing sharply (from 8.2% in 1997 to 13.4% in 2009). At the same time, prevalence of normal weight increased from 57.5% to 50% [44].

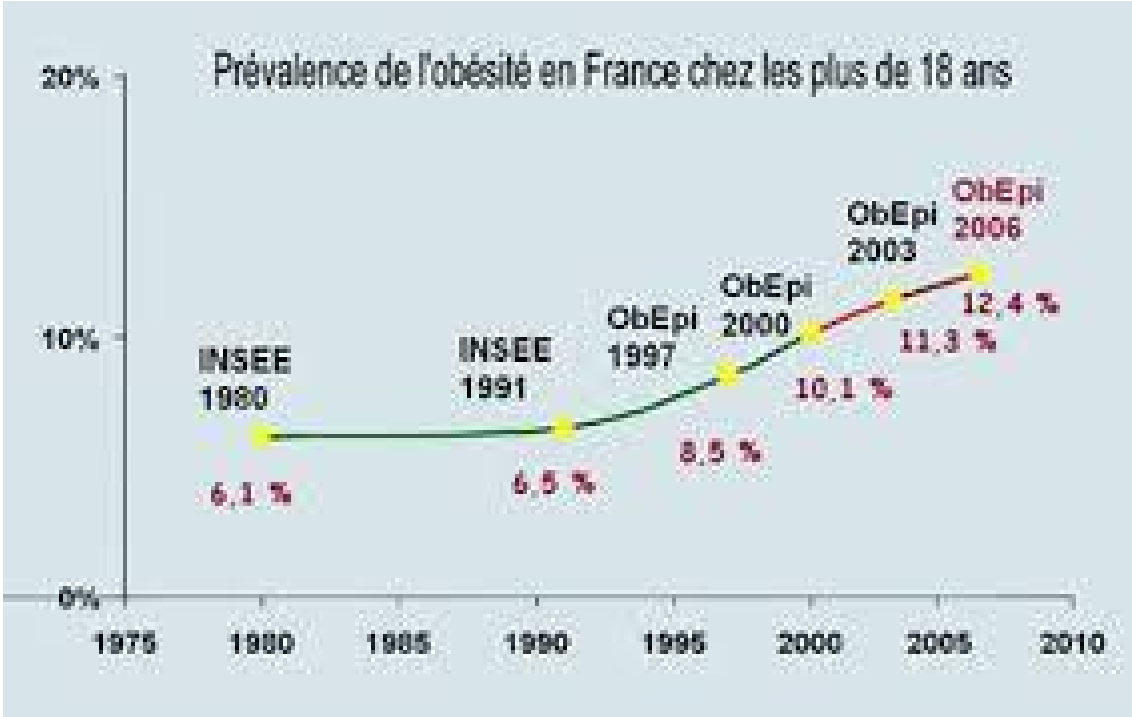

Figure 15: Prevalence of overweight and obesity in France (ObEpi surveys 1997 to 2009).

ObEp showed that the prevalence of overweight (including overweight and obesity) was 47.3%, with 17% of the subjects being obese. At first sight, these figures are not very different from the latest estimates in the 2012 Obépi-Roche study. However, if we look at the trends since 1997 and look at overweight on one side and obesity on the other, the results are more mixed. Since 1997, the prevalence of overweight has fluctuated around 30%, while the prevalence of obesity continues to increase at a rapid rate. It rose from 8.5% in 1997 to 15% in 2012 and 17% in 2020. The increase is even more pronounced in younger age groups and for morbid obesity, whose prevalence has increased almost sevenfold over the period [45].

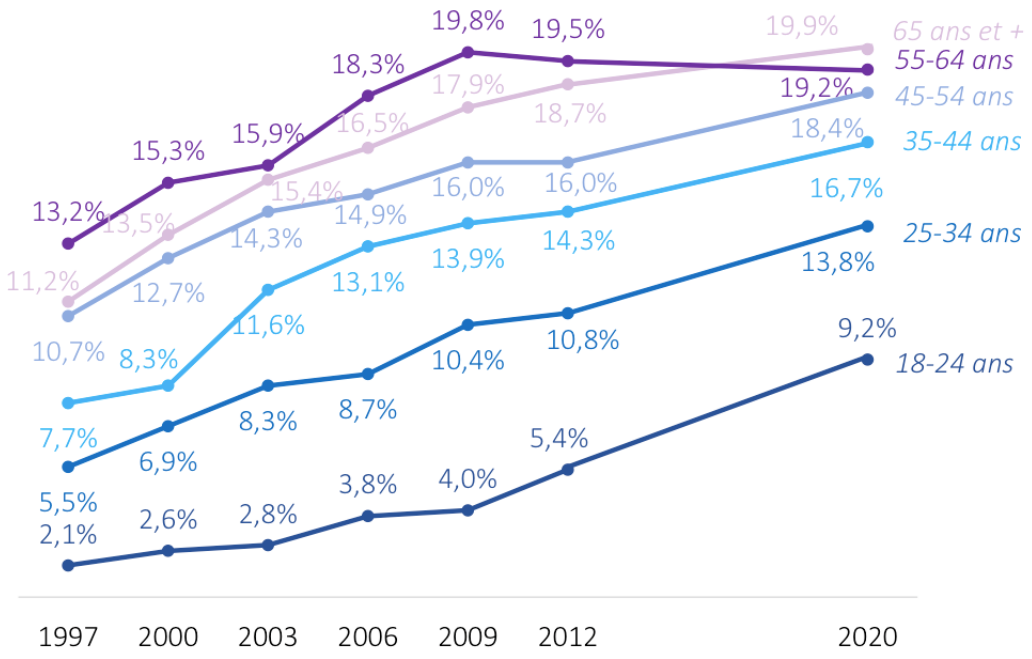

Figure 16: Evolution of prevalence of obesity by age between the 1997-2012 Obépi-Roche surveys and the 2020 Obépi survey.

Obesity in the world

In 2022, 2.5 billion adults aged 18 and over were overweight and of this total, more than 890 million were obese, representing 43% of adults aged 18 and over who are overweight (43% men and 44% women); this proportion has increased compared to 1990, where it was 25%. The prevalence of overweight varies by region, ranging from 31% in the WHO Southeast Asia and Africa Region to 67% in the Americas Region. In 2022, the number of overweight children under 5 was estimated at 37 million. Once considered a high-income problem, overweight is on the rise in low- and middle-income countries. In Africa, the number of children under 5 years old who are overweight has increased by almost 23% since 2000. In 2022, nearly half of all children under 5 years old who were overweight or obese lived in Asia [56].

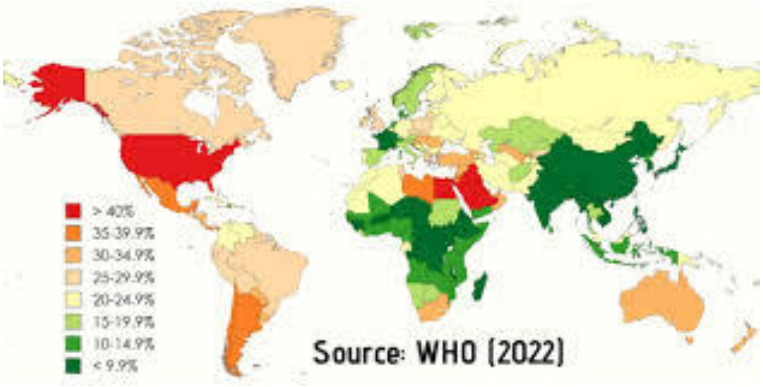

Figure 17: Global prevalence of obesity 2022.

1.2 Causes of obesity

Obesity and overweight stem from a variety of causes. There are many factors that can explain it. Lifestyle has changed significantly, especially diet and physical activity [46].

- **One of the causes of obesity:**

  everyday habits: Diet is the leading cause of obesity in adults. Indeed, a poor diet can have very harmful consequences on health. It is enough to have too great an imbalance between what we eat and what we spend in energy, and this, for a long time, so that the weight gradually increases. This is due to the lack of physical activities on a daily basis and excessive sedentary (computer, television...). Also, eating fat, salty, sweet, in large quantities and without respecting meal times can lead to weight gain [48].

- **Other causes of obesity:**

  - **Heredity:** a person with a parent who also has obesity or overweight sees the risk of being obese increased. Indeed, a study has shown that 70% of obese people have at least one person in their family who suffers from obesity.

  - **Psychological causes**: very common, people in situations of depression or stress, turn to food to comfort themselves [48].

  - **Lack of sleep:** French adults (18-55 years) sleep an average of 7 hours per night during the week. With more than a third sleeping only 6 hours per night. Moreover,

half of the teenagers sleep less than 8 hours per night, unlike the recommended 8:30/9:15. Several studies have shown a correlation between short sleep time and high BMI. The risk of obesity increases by 60% when you sleep only 5 hours per night. This is due to a reduction in leptin and an increase in ghrelin (appetite-stimulating hormone) [48].

- **Drugs:** some treatments cause a change in appetite. To avoid weight gain due to medication, it is advisable to pay attention to your diet [48].

1.3 Consequences of obesity

Obesity is a multifactorial disease that has serious consequences for the physical health of the individual, but also consequences for his or her mental health, an area in which a true vicious circle can develop [50]. The consequences of obesity are numerous, whether physical and/or psychological. The difficulty of treatment is often related to the denial of people with the disease [48].

- **Diabetes:** Type 1 diabetes is insulin-dependent diabetes. As the name suggests, people with insulin are insulin-dependent because their bodies do not make insulin. It is a genetic disease that accounts for 10% of cases. Type 2 diabetes, which accounts for 90% of cases, is caused by obesity, sedentary lifestyle and old age. It corresponds to a hyperglycemia, that is a level of sugar too high in the blood. The epidemic is so widespread that it is known as “diabetic”. This is one of the most serious consequences of obesity. It is responsible for many cases of amputation, cardiovascular disorders and can lead to vision loss. In addition to these serious complications, obesity-related diabetes causes more than 4 million deaths worldwide every year. Unlike type 1 diabetes, it can be reduced and even treated. For this, the patient must lose weight, cure his hypertension, have a cholesterol and triglyceride levels in the norms. To achieve these goals, he has several options: diet, sport, spa treatment, stay in a centre for obese people, bariatric surgery [48].

- **Cardiovascular disease:** An obese person is often affected by one or more cardiovascular diseases, including high blood pressure, venous insufficiency, sleep apnea syn-

drome, heart failure, dyslipidemia (hyper-triglyceridemia). High blood pressure is a cardiovascular disease. This can affect anyone who is obese: children, adolescents, adults and seniors. We talk about high blood pressure when the pressure is greater than 14/9. The first value is systole, which measures the resting heart muscle pressure; the second value is diastole, which measures the pressure of the working heart muscle. An obese person is six times more likely to have high blood pressure. Venous insufficiency is due to overweight and lack of physical activity. It is a poor circulation of blood that causes a feeling of heavy legs, varicose veins and in the most severe cases ulcers. On the other hand, sleep apnea includes both apnea syndrome and pathological snoring. It is not uncommon for an obese person to be on oxygen to sleep. In addition, obesity and overweight can cause fat to accumulate in the bloodstream. This causes blood circulation to be disturbed and causes the patient to have heavy legs, varicose veins, edema, phlebitis and, more generally, severe pain. Among the cardiovascular diseases related to obesity, heart failure is the most serious. It is manifested when the obese person makes a physical effort and is clearly accentuated by diabetes, hypertension, respiratory failure. These various disorders are compounded by the risk of heart attack and myocardial infarction, sudden death in adults, angina pectoris, decreased blood flow and venous disorders, necrosis and strokes [48].

- **High blood pressure (hypertension):** High blood pressure (HTA) is an abnormal rise in resting blood pressure (or blood pressure). Blood pressure is the pressure exerted by blood on the walls of arteries. It is expressed in two digits:
  The systolic voltage (maximum value),
  diastolic pressure (minimum value).
  Hypertension is referred to in adults when the systolic pressure is usually greater than or equal to 140 mmHg and/ or diastolic pressure is equal to or greater than 90 mmHg. In many cases, it is not clear from where it originated. However, age and family history are factors associated with AHT. Smoking, obesity, excessive salt or alcohol consumption, stress, diabetes are some of the factors that aggravate AHT. Excess weight (especially abdominal obesity) promotes hypertension. Conversely, even a small weight loss can lower blood pressure. There is three times more arterial hypertension in obese subjects

than in non-obese subjects. Between 30 and 40% of obese people are hypertensive (the main cause being abdominal obesity). The frequency is particularly high in obese subjects after 45 years [49].

- **Psychological consequences:** Depression is both a cause and a consequence of obesity. It is a difficult disease to fight and leaves visible but also invisible marks, such as depression. That is why psychological support is included in the treatment. The depression of the obese person can also be caused by rejection of his body, misunderstanding of his illness by his surroundings and by himself, disability caused by overweight, pain and discrimination. Discrimination is also one of the consequences of obesity. Indeed, an obese person is often misregarded by his entourage and perceived as sick, lazy and with a general hygiene that leaves something to be desired. The discrimination of obese people is present both in the family circle and in the professional environment [70].

1.4 Treatment and prevention

Treatment of obesity:

Obesity management and treatment aims to reduce health risks and improve quality of life. An appropriate weight management program usually combines physical activity with a healthy diet and changing daily habits. Other programs may also include psychological counselling and, in some cases, drug treatment. Losing weight and not taking it back is an ambitious goal that requires lifestyle and behaviour changes. What matters is a healthy and balanced diet. Fashionable diets and draconian diets are rarely successful and can be dangerous. The diet must provide the body with a minimum amount of energy to function normally. No diet with less than 1,200 calories should be followed without medical supervision. Draconian diets are never a good long-term solution because weight loss usually comes back when they are no longer followed [19].

- **Weight loss through healthy eating:** It is the primary means of combating overweight and obesity. Weight loss must be done in a reasoned way; a balanced diet and regular sports practice allow to lose weight gradually without putting the person concerned in physical and mental danger. It is advisable to have a suitable therapeutic follow-up.

- **The treatment of obesity by surgery:** The treatment of obesity by surgery (or bariatric surgery), is intended for adults with so-called "massive" or "morbid" (BMI greater than or equal to 40) or "severe" (BMI greater than or equal to 35) obesity combined with a disease. These people must demonstrate multiple failed attempts at weight loss and must not have medical contraindications or psychological support. a stomach surgery is not without risks and the patient should have regular post-operative follow-up. Surgery will only be effective if the patient adopts good eating habits and changes their lifestyle. It is important to do psychological work on yourself in order to learn how to live with a new image.

- **The treatment of obesity with drugs:** To date, there are no miracle drug treatments for obesity. However, some medications help with weight loss and limit the body's absorption of dietary fats. They must be prescribed by a doctor, under very specific indications. In addition, their intake must be combined with a balanced diet [32].

The prevention:

Preventing obesity is a public health priority. A "National Nutrition Health Plan" has been in place for some years to prevent obesity. In everyday life, it is recommended to adopt certain gestures [32]:

- Exercise regularly: 30 minutes of exercise a day can reduce the risk of cardiovascular disease and diabetes. Increased physical activity may be needed if you are overweight. For children, activity time should be at least 1 hour per day;

- Have a diversified and balanced diet;

- Limit consumption of saturated fats;

- Limit consumption of sugars and salt;

- Eat fruits and vegetables daily;

Obesity prevention is especially targeted at people who have been affected by childhood obesity or have at least one obese parent. But it also affects individuals who have stopped smoking or playing sports, are taking medication at risk, or have recently gained a lot of weight

(more than 5% of their body weight). Finally, it is aimed at people suffering from bulimia, as well as pregnant women or menopausal women [70].

2 Evolution of obesity models

Mathematical modeling is a very important tool in the study of obesity and its complications, as it contributes to understanding the dynamics of the disease spread, predicting incidence rates, and evaluating the effectiveness of therapeutic and preventive interventions. By representing the factors affecting obesity (such as genetics, environment, and behavior) with mathematical equations, their interactions and their impact on the spread of the disease can be analyzed. Mathematical models also help in predicting the future spread of obesity, which enables the development of proactive health plans. In addition, these models are used to evaluate the effectiveness of different interventions such as nutrition and physical activity programs, and to identify major risk factors such as high blood pressure, heart disease, and diabetes. As these models develop, they can be used to develop other treatment strategies [16].

To investigate the mechanisms of obesity, there are some works based on compartment models which are widely used in epidemiology, among these contributions, we cite:

Model by Evangelista et al. 2004

Evangelista et al. [32] in 2004 suggested a model to investigate the impact of fast food on personal weight. They developed a mathematical model with particular scenarios to investigate the rates of development of normal N, overweight O_1, and obese O_2 people. Based on their BMI, people are classified as N, O_1, and O_2. Peer pressure, which causes people to start dining out, is used to gauge the rate at which normal persons become overweight. People begin dining at fast food restaurants not only because others invite them, but also because of their financial standing, ease of access, and convenience. They use the system of non-linear differential equations to examine the consequences of this pressure to quit:

$$\begin{cases} \frac{dN}{dt} = \mu P - \beta N \frac{(O_1+O_2)}{P} - \mu N, \\ \frac{dO_1}{dt} = \beta N \frac{(O_1+O_2)}{P} + \phi_1 Q_1 - (\gamma+\mu)O_1 - \left(\alpha_1 + \frac{\alpha_0 O_2}{L+O_2}\right), \\ \frac{dO_2}{dt} = \gamma O_1 + \phi_2 Q_2 - \mu O_2 - \left(\alpha_2 + \frac{\alpha_0 O_2}{L+O_2}\right)O_2, \\ \frac{dQ_1}{dt} = \left(\alpha_1 + \frac{\alpha_0 O_2}{L+O_2}\right) - (\phi+\mu)Q_1, \\ \frac{dQ_2}{dt} = \left(\alpha_2 + \frac{\alpha_0 O_2}{L+O_2}\right)O_2 - (\phi_2+\mu)Q_2, \\ P = N + O_1 + O_2 + Q_1 + Q_2. \end{cases} \tag{2.2.1}$$

The diagram below shows the model (2.2.1):

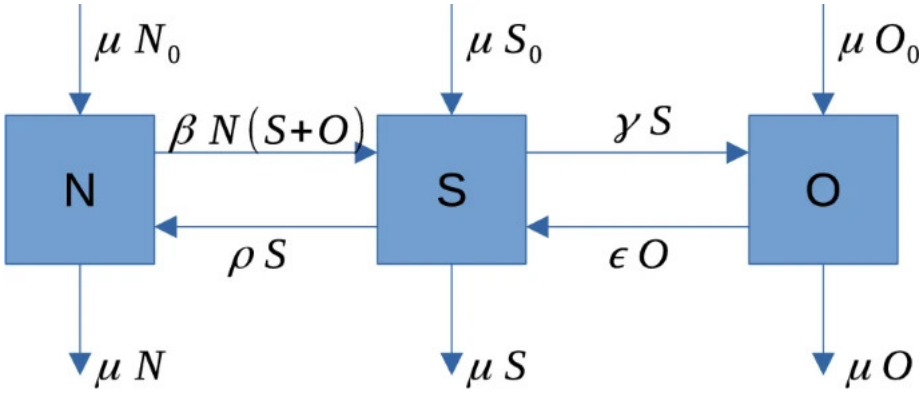

Figure 18: The schematic of model 2.2.1.

The model is defined by the following parameters:

Parameters	Description
β	Peer-pressure rate to start eating fast food (media, economic factor, etc.)
μ	The death rate.
γ	Rate of transition from overweight to obese due to fast food consumption.
α_i	rate of quitting fast food due to family or healthcare advice, $i = 1,2$
α_0	The highest rate at which obese individuals quit.
L	the obesity level at which the quit rate attains half its maximum value, α_0.
ϕ_i	Relapse rate, for $i = 1,2$

Model by Jodar et al. 2008

In [53], Jodar et al. (2008) presented a model of infantile obesity, to study the evolution of this epidemic in Valencia, Spain. After field surveys, they found that sociocultural characteristics determine unhealthy eating habits in children. This analysis allowed them to build an epidemiological mathematical model to study the evolution of childhood obesity and is given by a system of non linear differential equations:

$$\begin{cases} N'(t) = \mu + \varepsilon D_S(t) - \mu N(t) - \beta N(t)\Big(L(t)+S(t)+O(t)\Big), \\ L'(t) = \beta N(t)\Big(L(t)+S(t)+O(t)\Big) - \Big(\mu+\gamma_L\Big)L(t), \\ S'(t) = \gamma_L L(t) + \varphi D_S(t) - \Big(\mu+\gamma_S+\alpha\Big)S(t), \\ O'(t) = \gamma_S S(t) + \delta D_o(t) - \Big(\mu+\sigma\Big)O(t), \\ D_S'(t) = \gamma_D D_O(t) + \alpha S(t) - \Big(\mu+\varepsilon+\varphi\Big)D_S(t), \\ D_O'(t) = \sigma O(t) - \Big(\mu+\gamma_D+\delta\Big), \end{cases} \tag{2.2.2}$$

The childhood population is classified into six groups: normal weight $N(t)$, latent $L(t)$, overweight $S(t)$, obese $O(t)$, overweight on diet $D_S(t)$, and obese on diet $D_O(t)$.

The following diagram provides a representation of the model (2.2.2):

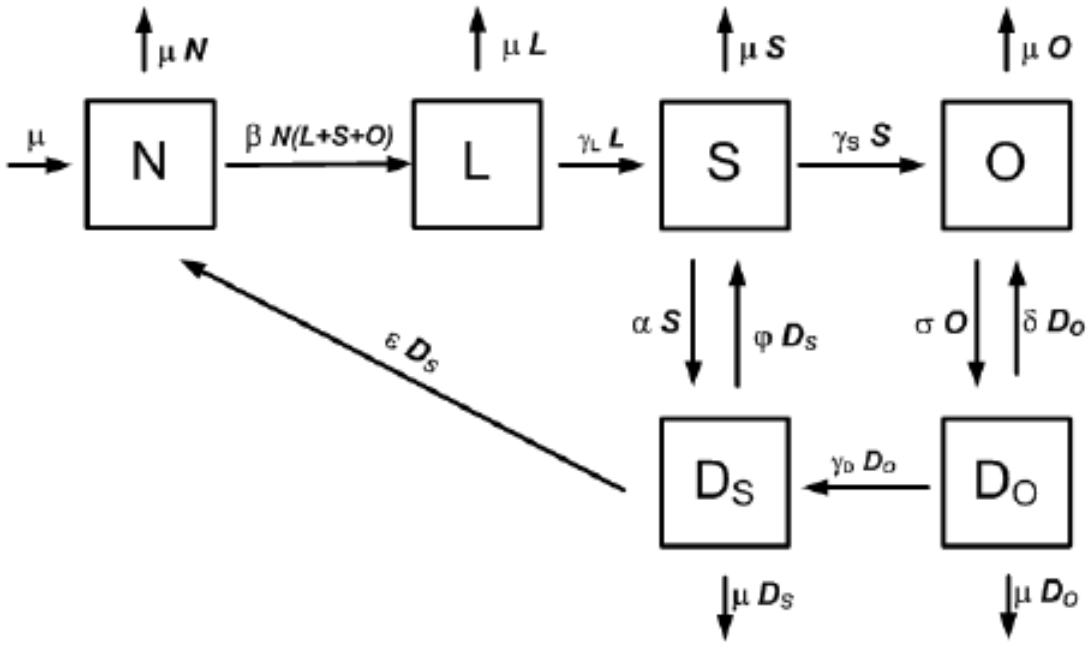

Figure 19: Diagram of the obesity model for 3-5 year-old Valencian children.

The model parameters are:

Parameters	Description
β	Rate of BFS consumption transmission attributed to social pressure (family, friends, marketing, TV, etc.)
μ	inversely related to the time 3-5-year-olds spend in the system.
γ_L	Rate at which latent individuals progress to the overweight state.
γ_S	Rate at which overweight individuals progress to obesity via continuous BFS consumption.
ε	Rate at which overweight individuals achieve a normal weight through dieting.
α	Rate at which overweight individuals undergoing dietary intervention reduce or eliminate fast food consumption.
ϕ	Rate of diet failure among overweight individuals.
σ	Rate at which obese individuals discontinue or decrease their consumption of BFS.
δ	Failure rate of diets in obese individuals.
γ_D	Rate at which obese individuals on diet transition to overweight status.

Model by González et al. 2010

In [40], González et al. modelled and studied the correlation between the development of obesity in an age structured population, they have treated different scenarios in Valencia, Spain. In their work, they considered that the overweight population and the obese population, but using a model with age structure. This model has the advantage of giving the prevalence of obesity for each age group. The authors developed a partial differential equation model according to age is constructed from partial differential equations:

$$\begin{cases} \dfrac{\partial S}{\partial a}+\dfrac{\partial S}{\partial t}=I+\varphi O-\gamma S-\mu S, \\ \dfrac{\partial S}{\partial a}+\dfrac{\partial S}{\partial t}=I+\varphi O-\gamma S-\mu S, \end{cases} \tag{2.2.3}$$

A graphical representation of the model is presented in the diagram below:

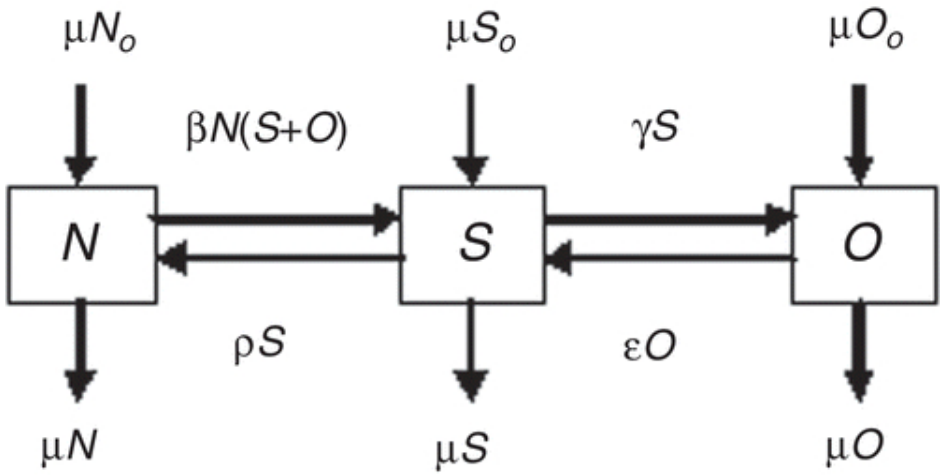

Figure 20: Diagram of an age-structured model for obesity dynamics in overweight individuals.

The following parameters characterize the model:

Parameters	Description
$S(a,t)$	The number of overweight people aged a at time t.
$O(a,t)$	The number of obese people aged a at time t.
$I(a,t)$	Individuals progress from a normal weight state to an overweight state.
$\varphi(a,t)O(a,t)$	The number of overweight individuals increased by $\varphi(a,t)O(a,t)$.
$\gamma(a,t)S(a,t)$	The number of overweight individuals decreased by $\gamma(a,t)S(a,t)$.
$\mu(a,t)$	The natural mortality rate.
$\sigma(a,t)$	Rate at which individuals become overweight.
$r(a,t)$	Obesity prevalence in overweight individuals at age a and time t.

Where $n(a,t)=S(a,t)+O(a,t)$.

Model by Ejima et al. 2013

In [33], a three-compartment model (RIS) comprising susceptible (never obese), infected (obese), and resistant (ex-obesity) was proposed by Ejima et al. The purpose of this study is to characterise the epidemiological process of obesity in a population that is randomly mixed, In a basic manner, it explains the implications of obesity transmission from person to person for public health monitoring and highlights the critical gaps that require immediate attention in sempiric observatories. The mathematical model takes into account two hypotheses, the

contagion and the non contagion of obesity, which is given by a system of ordinary differential equations:

$$\begin{cases} \frac{dS}{dt} = \mu N - \big(\beta I(t) + \varepsilon\big) S(t) - \mu S(t), \\ \frac{dI}{dt} = \big(\beta I(t) + \varepsilon\big) S(t) + \sigma\big(\beta I(t) + \varepsilon\big) R(t) - (\mu + \gamma) I(t), \\ \frac{dR}{dt} = \gamma I(t) - \sigma\big(\beta I(t) + \varepsilon\big) R(t) - \mu R(t), \end{cases} \quad (2.2.4)$$

such that N denotes the total population, and $N(t) = S(t) + I(t) + R(t)$.

A schematic representation of the model is presented in the diagram that follows:

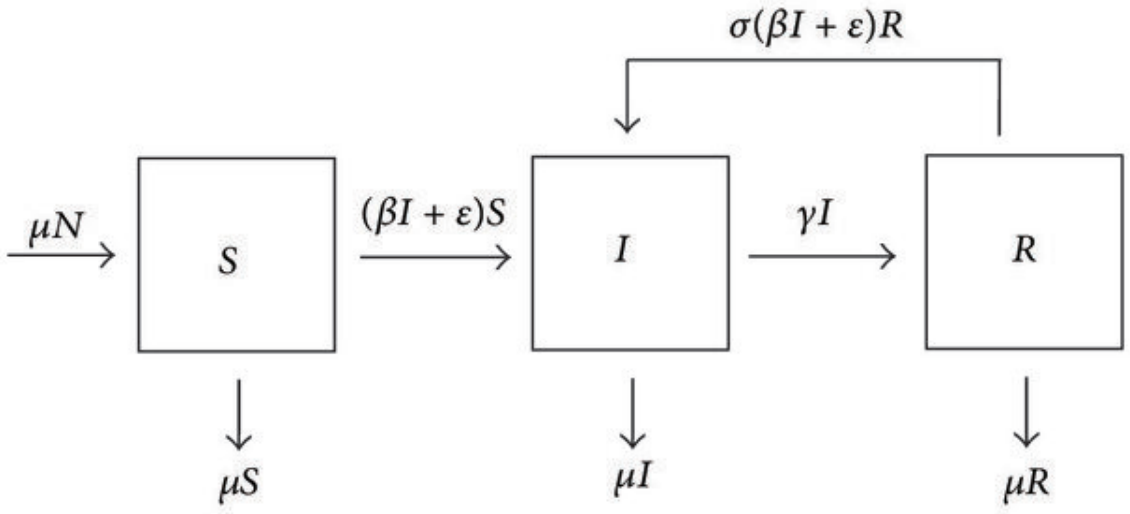

Figure 21: Diagram of the mathematical model by Ejima et al.

The model is configured with the parameters described below:

Parameters	Description
μ	Rate of human birth and death.
β	The transmission rate.
ε	The hazard of obesity arising from non-contagious causes.
γ	The natural recovery rate.
σ	A measure of the likelihood that a former obese individual will regain weight, which is typically higher than the general population due to the tendency to return to previous weight patterns

Model by Boutayeb et al. 2015

Boutayeb et al. (2015) in [14] studied the effect of obesity on type 2 diabetes in people with a genetic predisposition to diabetes development and they represented the hypotheses of the model by system of ordinary differential equations:

$$\begin{cases} \frac{dP}{dt} = n - \left(I_1 + I_2 + I_3 + \mu\right)P + \gamma_1 E, \\ \frac{dE}{dt} = I_1 P - \left(\gamma_1 + \beta_1 + \beta_3 + \mu\right)E, \\ \frac{dD}{dt} = I_2 P + \beta_1 E + \gamma_2 C - \left(\beta_2 + \mu\right)D, \\ \frac{dC}{dt} = I_3 P + \beta_2 D + \beta_3 E - \left(\gamma_2 + \mu + \delta\right)C. \end{cases} \tag{2.2.5}$$

The diagram below illustrates the structure and components of the model:

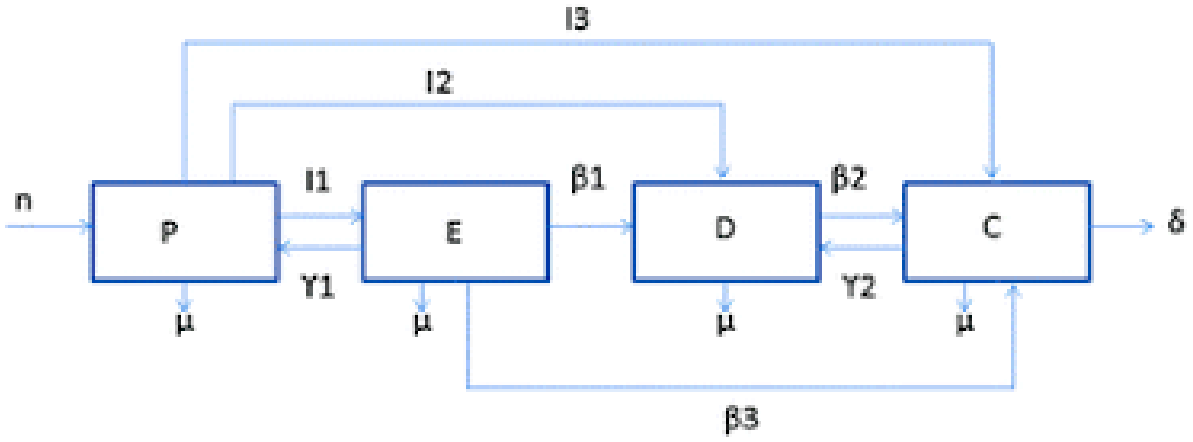

Figure 22: Dynamics of healthy, pre-diabetic, and diabetic individuals with and without complications.

The model's parameters are specified as follows:

Parameters	Description
P	The number of healthy people.
E	The number of pre-diabetic people.
D	The number of diabetics people with complications.
C	The number of diabetics people without complications.
n	The incidence rate among adults.
I_1	The rate at which healthy individuals progress to pre-diabetes.
I_2	The rate at which healthy individuals progress to diabetic.
I_3	the incidence of complications among healthy people.
μ	The natural death rate.

Model by Hakiki et al. 2018

In this work, the authors present a new mathematical model investigating obesity-related complications. They assume an obese population of size N, divided into two subgroups: The population of obese individuals without complications is denoted by $O(t)$, for $t > 0$. The population of obese individuals with complications is divided into two compartments: $C_1(t)$ for those with acute complications and $C_2(t)$ for those with chronic complications.
The diagram in Figure (34) describes the complications of obesity:

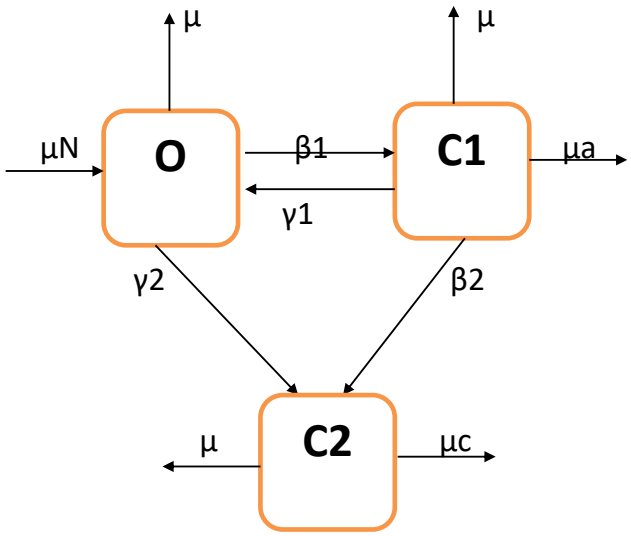

Figure 23: The model diagram of obesity complications.

This diagram can be formulated as the following system:

$$\begin{cases} \frac{dO}{dt} = I + \beta_1 C_1(t) - \mu O(t) - \gamma_1 \frac{C_1(t)}{N} O(t) - \gamma_2 \frac{C_2(t)}{N} O(t), \\ \frac{dC_1}{dt} = \gamma_1 \frac{C_1(t)}{N} O(t) - (\mu + \mu_a + \beta_1 + \beta_2) C_1(t), \\ \frac{dC_2}{dt} = \gamma_2 \frac{C_2(t)}{N} O(t) + \beta_2 C_1(t) - (\mu + \mu_c) C_2(t), \end{cases} \tag{2.2.6}$$

We define the model parameters as:

Parameters	Description
β_1	The rate at which obese individuals with acute complications recover and return to the obese state.
β_2	rate at which obese individuals with acute complications develop chronic complications.
γ_1	The probability that an obese individual will develop an acute disease.
γ_2	The probability that an obese individual will develop an chronic disease
μ	Natural mortality rate.
μ_c	The death rate due to chronic complications.
μ_a	The death rate due to aigue complications.

Model by Wendi Wang. 2020

In [83], a mathematical model that incorporates individual variation and overeating behaviours is proposed by Wang to predict the dynamics of societal obesity. A calculation of the disease's fundamental breeding number reveals that it is an invasion threshold. The Lyapunov functions provide enough requirements for an endemic equilibrium's overall stability. Numerical simulations are presented to demonstrate how dietary behaviour modification and sensitive person education can slow the progression of disease. The population was divided into four groups: The susceptible group S of individuals with normal eating habits, the group I in which every individual engages in excessive food consumption, The group T that receives treatment to become part of the group R, where individuals are immune to overeating behaviors. Members of groups S and I were further classified into two subgroups based on their body mass index (BMI). Let $S_N(t)$ and $S_O(t)$ denote the number of normal-weight and overweight individuals in group S at time t, respectively, $I_N(t)$ and $I_O(t)$ represent the number of individuals in group I with normal and higher weights, respectively, at time t, $T(t)$ and

$R(t)$ represent the number of individuals in groups T and R, respectively, at time t.

where $S(t) = S_N(t) + S_O(t)$ and $I(t) = I_N(t) + I_O(t)$.

The diagram below shows the model:

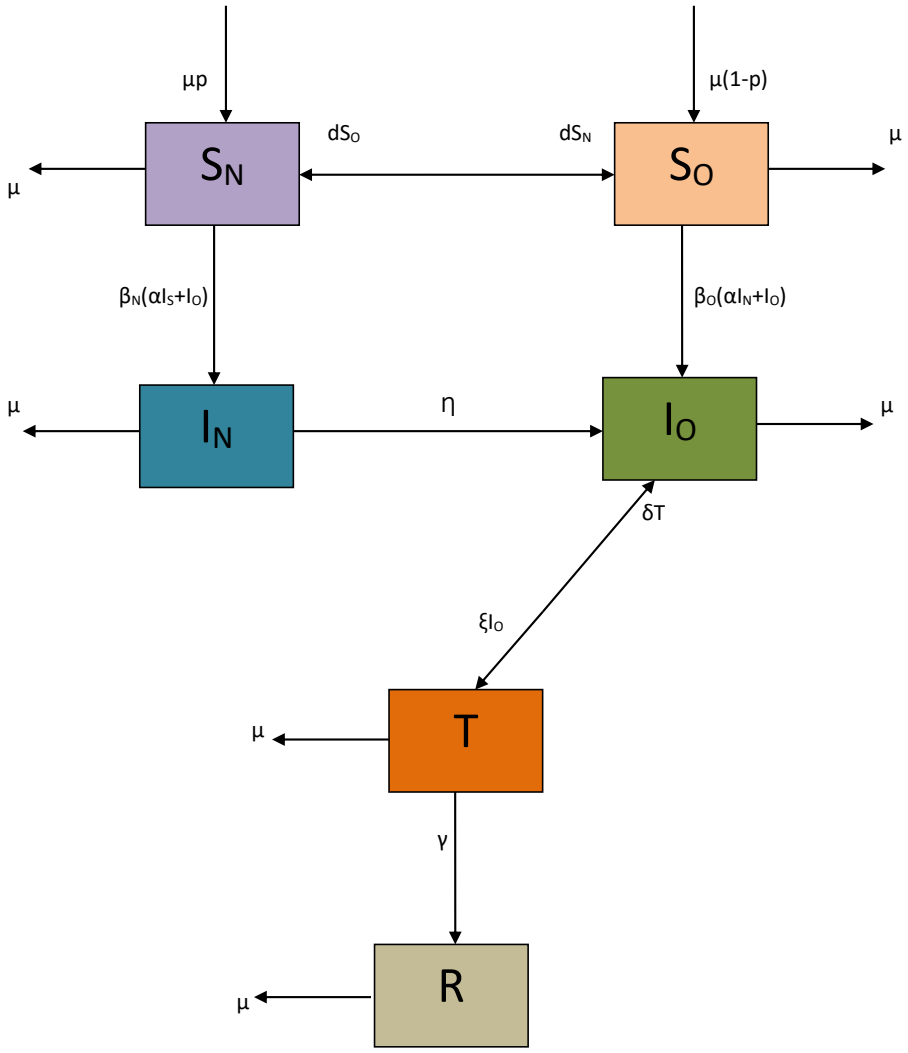

Figure 24: Diagram of the mathematical model by Wendi Wang.

The model is represented by the following system:

$$\begin{cases} \frac{dS_N}{dt} = \mu p - \mu S_N - \beta_N\left(\alpha I_N + I_O\right)S_N + d\left(S_O - S_N\right), \\ \frac{dS_O}{dt} = \mu(1-p) - \mu S_O - \beta_O\left(\alpha I_N + I_O\right)S_O + d\left(S_N - S_O\right), \\ \frac{dI_N}{dt} = \beta_N\left(\alpha I_N + I_O\right)S_N - \left(\mu + \eta\right)I_N, \\ \frac{dI_O}{dt} = \beta_O\left(\alpha I_N + I_O\right)S_O - \left(\mu + \xi\right)I_O + \eta I_N + \delta T, \\ \frac{dT}{dt} = \xi I_O - \left(\mu + \gamma + \delta\right)T. \end{cases} \tag{2.2.7}$$

The model is configured with the parameters described below:

Parameters	Description
μ	The recruitment rate of the population is eauivalent to the removing rate of individuals in all compartments.
p	The fraction to the category S_N attributed to genetic factors.
$1-p$	The fraction to the category S_O.
ξ	The rate at which individuals enter treatment.
γ	The recovery rate resulting from treatment.
δ	The relapse rate of treated individuals.
d	A random and unpredictable change in the state of individuals within group S, caused by random external factors, occurring at a rate determined by the coefficient d.
β_N	The transmission coefficients of overeating individuals to susceptible individuals with the normal BMI.
β_O	The transmission coefficients of overeating individuals to susceptible individuals with the higher BMI.

Model by Tuwairqi et al. 2021

In [9], the mathematical model is constructed to study how peer pressure can affect the temptation that individuals face to eat in fast food restaurants and consequently gain weight. The authors construct two models: model I studies the effect of peer pressure on obesity and fast food consumption; Model II is an extension of model I, where the training factor is included. The population is divided into four distinct classes of individuals: normal-weight individuals, $N(t)$; overweight people who eat fast food, $S(t)$; obese people who eat fast food meals, $O(t)$; and dropouts who no longer eat fast food, $Q(t)$.

The interactions between the subpopulations in the first model are presented in the following figure:

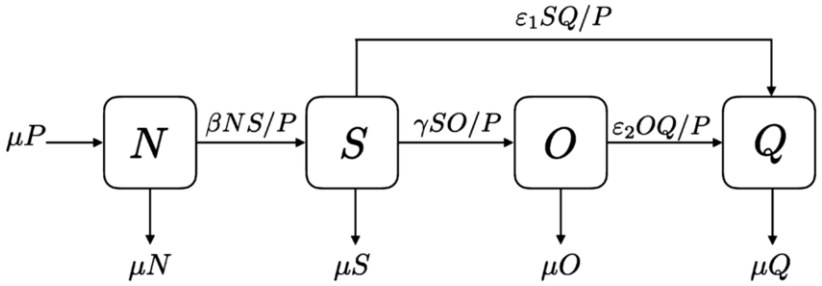

Figure 25: Diagram of the mathematical model by Tuwairqi.

Under a mathematical formulation of a system of differential equations:

$$\begin{cases} N'(t) = \mu P - \beta N\left(\frac{S}{P}\right) - \mu N, \\ S'(t) = \beta N\left(\frac{S}{P}\right) - \gamma S\left(\frac{O}{P}\right) - \varepsilon_1 S\left(\frac{Q}{P}\right) - \mu S, \\ O'(t) = \gamma S\left(\frac{O}{P}\right) - \varepsilon_2 O\left(\frac{Q}{P}\right) - \mu O, \\ Q'(t) = \varepsilon_1 S\left(\frac{Q}{P}\right) + \varepsilon_2\left(\frac{S}{P}\right) - \mu Q, \end{cases} \tag{2.2.8}$$

The model is defined by the following parameters:

Parameters	Description
β	Rate at which a normal-weight individuals enter compartment S due to peer pressure from overweight fast food consumers.
γ	Rate at which overweight individuals enter compartment O because of peer pressure from obese fast-food consumers.
ε_1	Rate of transition from the overweight individuals to compartment Q induced by the positive impact of avoiding fast food.
ε_2	Rate of transition from the obese indiciduals to compartment Q induced by the positive impact of avoiding fast food.
μ	Rate at which individuals enter and leave the model.

After adding the training factor, the first model was modified to take into account the individual exercise factor. This model aims to determine the effect of exercise on weight reduction in overweight and obese individuals who frequently eat at fast food restaurants and to examine the reserve effect of dropouts on obesity. The second mathematical model dynamics is given

by:

$$\begin{cases} N^{'}(t) = \mu P - \beta N\Big(\frac{S}{P}\Big) + \alpha_1 S + \alpha_3 Q - \mu N, \\ S^{'}(t) = \beta N\Big(\frac{S}{P}\Big) - \gamma S\Big(\frac{O}{P}\Big) - \varepsilon_1 S\Big(\frac{Q}{P}\Big) - \alpha_1 S + \alpha_2 O - \mu S, \\ O^{'}(t) = \gamma S\Big(\frac{O}{P}\Big) - \varepsilon_2 O\Big(\frac{Q}{P} - \alpha_2 O - \mu O, \\ Q^{t} = \varepsilon_1 S\Big(\frac{Q}{P}\Big) + \varepsilon_2 O\Big(\frac{Q}{P}\Big) - \alpha_3 Q -, \end{cases} \tag{2.2.9}$$

We have $P(t) = N(t) + S(t) + O(t) + Q(t)$.

Such that α_1 is the transition rate from the overweight compartment to the normal weight compartment due to exercise. α_2 is the rate at which obese individuals leave the obese compartment S due to exercise. and α_3 represents the rate at which individuals who have lost weight return to fast food consumption and re-enter the compartment N.

Model by F. Meghatria and O. Belhamiti. 2021

F. Meghatria and O. Belhamiti in their work [51], they investigate a predictive model that forecasts the likelihood of developing CVD and T2DM in obese populations, incorporating variables related to lifestyle and established risk factors. This model is described by a nonlinear system of ordinary differential equations:

$$\begin{cases} \frac{dO(t)}{dt} = I_O\Big((\mu + \phi_1 \frac{C(t)}{N(t)} + \phi_2 \frac{D(t)}{N(t)}\Big)O(t), \\ \frac{dC(t)}{dt} = \phi_1 \frac{C(t)}{N(t)} O(t) - (\delta + \mu + \mu_C)C(t), \\ \frac{dD(t)}{dt} = \phi_2 \frac{D(t)}{N(t)} O(t) - (\rho + \mu)D(t), \\ \frac{dG(t)}{dt} = \rho D(t) + \delta C(t) - (\mu + \mu_G)G(t), \end{cases} \tag{2.2.10}$$

The total obese population, $N(t)$, is divided into four subpopulations at time t: obese individuals without complications $O(t)$, obese individuals with cardiovascular disease (CVD), $C(t)$, obese individuals with type 2 diabetes mellitus (T2DM), $D(t)$, and obese individuals with both CVD and T2DM $G(t)$.

The model is presented in the following figure:

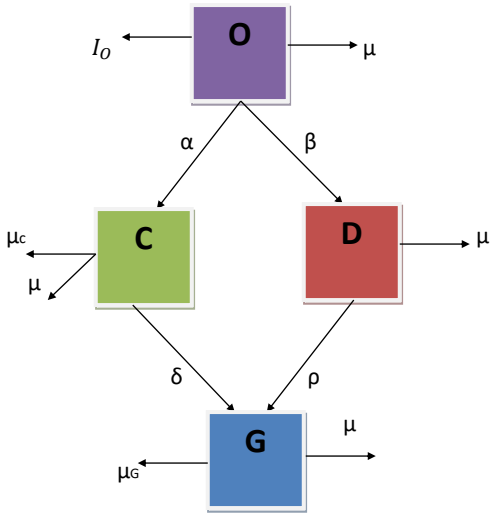

Figure 26: Graphical illustration of the mathematical model 2.2.10

The model is defined by the following parameters:

Parameters	Description
I_O	The recruitment rate.
μ	The natural mortality rate from each compartment.
μ_C, μ_G	The excess mortality rate due to complications.
ϕ_1, ϕ_2	Parameters related to the obese individual's education (diet, exercise, medicaltreatment, etc.).
δ	The rate at which cardiovascular disease progresses to diabetes.
ρ	The rate at which diabetes progresses to cardiovascular disease CVD.

Model by Mansouri et al. 2023

Mansouri et al. examined the detrimental effects of excess weight on diabetic quality of life using a mathematical model that replicates the relationship between rising obesity prevalence and diabetes. The maximum Pontryagin principle serves as the foundation for the method utilised to determine the best control techniques. Redundancy strategies are used to solve the resultant system [34].

The mathematical model formulated by ordinary differential equations:

$$\begin{cases} \frac{dS(t)}{dt} = \Lambda_1 - \mu S(t) - \beta \frac{S(t)}{N} W(t), \\ \frac{dW(t)}{dt} = \beta \frac{S(t)}{N} W(t) - \left(\tau + \sigma + \mu + \delta\right) W(t), \\ \frac{dR(t)}{dt} = \tau W(t) - \mu R(t), \\ \frac{dI(t)}{dt} = \delta W(t) - \left(\phi + \rho + \mu\right) I(t), \\ \frac{dE(t)}{dt} = \Lambda_2 - \left(\mu + \alpha\right) E(t), \\ \frac{dD(t)}{dt} = \sigma W(t) + \rho I(t) + \alpha E(t) - \left(\mu + \theta\right) D(t), \\ \frac{dC(t)}{dt} = \theta D(t) - \left(\mu + \lambda\right) C(t). \end{cases} \tag{2.2.11}$$

With initial conditions, $S(o) \geq 0$, $W(0) \geq 0$, $R(0) \geq 0$, $I(0) \geq 0$, $E(0) \geq 0$, $D(0) \geq 0$, and $C(0) \geq 0$. Figure 27 illustrates the model dynamics.

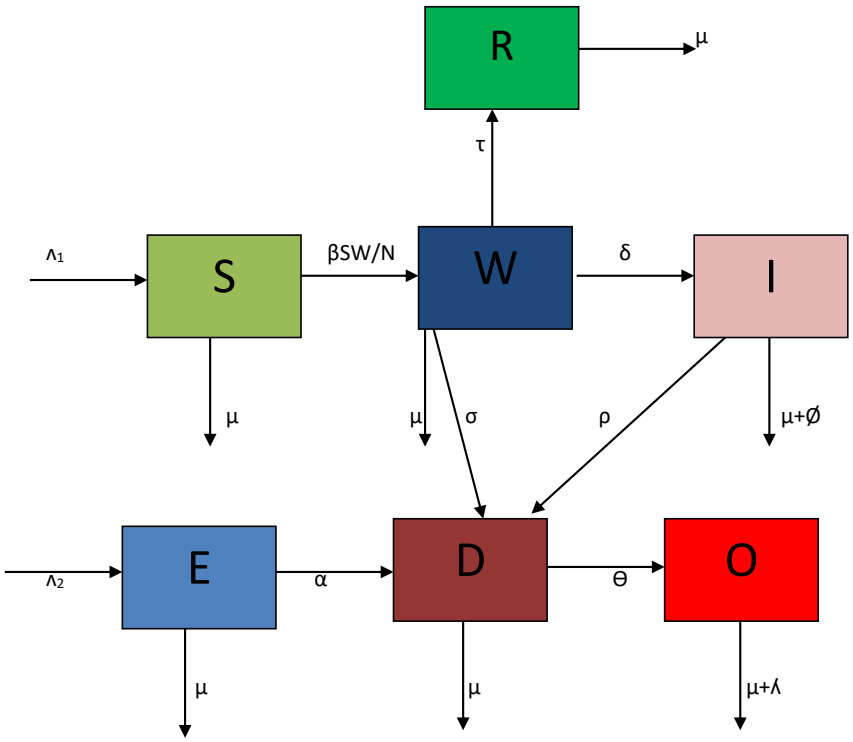

Figure 27: Schematic representation of model 2.2.11.

Let S, W, I, R, E, D, and C denote obesity-prone individuals, overweight or moderately obese people, obese people, recovered individuals, pre-diabetic people, complication-free diabet-

ics, and diabetics with significant health issues.

The model parameters are:

Parameters	Description
Λ_1	The rate of recruitment of individuals exposed to obesity.
μ	The natural mortality rate.
β	Rate of obesity development due to contact with infected individuals.
τ	The recovery rate of overweight people.
δ	The proportion of the overweight population that progressed to obesity.
λ	Rate of mortality caused by diabetes with complications.
ϕ	Rate of death caused by obesity.
ρ	Incidence of uncomplicated diabetes among the obese.
Λ_2	Rate of recruitment of pre-diabetic individuals without complications.
α	Rate of uncomplicated diabetes in pre-diabetic patients.
σ	The rate at which overweight individuals transition to a diabetic state without complications.
θ	The rate at which diabetics develop complications.

CHAPTER 3

The impact of obesity on the development of type 2 diabetes, cardiovascular disease and hypertension disease [3]

In this chapter, we introduce a new mathematical model utilizing ordinary differential equations to investigate the relationships among obesity, type 2 diabetes, cardiovascular disease, and hypertension, exploring the impact of peer pressure on the adoption of unhealthy behaviors that lead to these conditions. We conduct an analysis studty of our model.

1 Mathematical model

The model is represented in the following diagram:

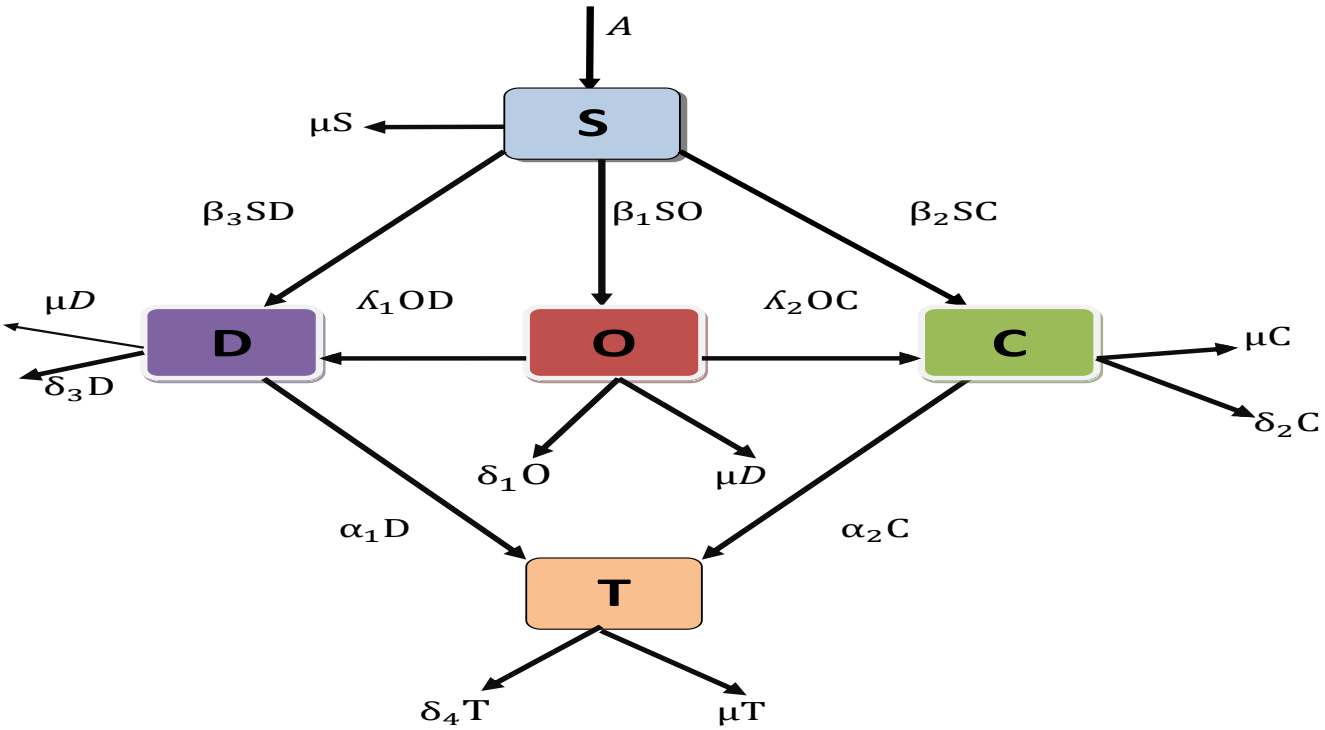

Figure 28: Diagram of obesity with its complications.

Using this model, we examine the influence of type 2 diabetic mellitus (T2DM), cardiovascular disease (CVD), and hypertension disease on the health evolution of obese populations. We analyse the dynamics of the obesity epidemic in a particular community by highlighting the negative consequences of being overweight on the onset of hypertension, cardiovascular disease, and type 2 diabetes.

N represents the total population, which is separated into five compartments so that **N=S+O+D+C+T** .

The different compartments are defined as follows:

S is the population of susceptible. O is the population of obese people. D is the population of people with type 2 diabetes mellitus (T2DM). C is The population of people with cardiovascular disease (CVD). And T is the population of people with hypertension disease.

We assume that: A is the birth rate. μ is the natural mortality rate. β_i, $i = 1,2,3$ are respectively the rate of transmission of obesity, cardiovascular disease, diabetes to a susceptible person due to the social effect (common unhealthy lifestyle, acquiring bad eating habits,...). λ_i, $i = 1,2$ are respectively the diabetes and cardiovascular developing rate of an obese person.

α_i, $i = 1,2$ are respectively the hypertension developing rate from diabetes and cardiovascular disease. δ_1 is the death rate due to obesity. δ_2 is the death rate due to cardiovascular disease. δ_3 is the death rate due to diabetes. δ_4 is the death rate due to hypertension disease. The following system describes the model:

$$\begin{cases} S'(t) = A - \Big(\mu + \beta_1 O(t) + \beta_2 C(t) + \beta_3 D(t)\Big) S(t), \\ O'(t) = \Big(\beta_1 S(t) - (\mu + \delta_1 + \lambda_1 D(t) + \lambda_2 C(t))\Big) O(t), \\ C'(t) = \Big(\beta_2 S(t) + \lambda_2 O(t) - (\mu + \delta_2 + \alpha_2)\Big) C(t), \\ D'(t) = \Big(\beta_3 S(t) + \lambda_1 O(t) - (\mu + \delta_3 + \alpha_1)\Big) D(t), \\ T'(t) = \alpha_1 D(t) + \alpha_2 C(t) - (\mu + \delta_4) T(t), \end{cases} \tag{3.1.1}$$

considering the initial conditions

$S(0) = S_0 \geq 0$, $O(0) = O_0 \geq 0$, $C(0) = C_0 \geq 0$, $D(0) = D_0 \geq 0$, and $T(0) = T_0 \geq 0$.

All model parameters are assumed to be strictly positive.

2 Positivity and boundedness of solutions

2.0.1 Solutions positivity

Theorem 3.2.1 *If $S(0) = S_0 \geq 0$, $O(0) = O_0 \geq 0$, $C(0) = C_0 \geq 0$, $D(0) = D_0 \geq 0$ and $T(0) = T_0 \geq 0$. Consequently, model (3.1.1) solutions S, O, C, D and T are all positive for any $t > 0$.*

Proof. Our assumption is that there exists $t_1 > 0$ where $S(t_1) = 0$, $S'(t_1) \leq 0$ and $S'(t_1 + \varepsilon) < 0$, for any $\varepsilon > 0$

According to the system's first equation (3.1.1), we get

$$S'(t_1) = A - \Big(\mu + \beta_1 O(t) + \beta_2 C(t) + \beta_3 D(t)\Big) S(t_1) = A > 0. \tag{3.2.2}$$

So, contradiction with our hypothesis. Since $S(0) = S_0 \geq 0$, then $S(t) \geq 0$ for all $t > 0$. We suppose that there exists $t_2 > 0$ where $O(t_2) = 0$, $O'(t_2) \leq 0$ and $O'(t_2 + \varepsilon) < 0$, for all $\varepsilon > 0$. From the second equation of system (3.1.1), we find

$$O'(t_2) = \Big(\beta_1 S(t_2) - (\mu + \delta_1 + \lambda_1 D(t_2) + \lambda_2 C(t_2))\Big) O(t_2) = 0, \tag{3.2.3}$$

hence

$$O(t)=O(t_2)exp\Big(\int_{t_2}^{t}\Big(\beta_1 S(s)-(\mu+\delta_1+\lambda_1 D(s)+\lambda_2 C(s))\Big)ds\Big),\quad \text{for all } t>t_2, \tag{3.2.4}$$

This results in

$$O(t)=0.\quad \text{for all } t>t_2.$$

As a result,

$$O(t_2+\varepsilon)=0.$$

It is contradictory with our assumption. Consequently, $O(t)\geq 0$ for all $t>0$. Similarly, we prove that $C(t)\geq 0$, and $D(t)\geq 0$, for all $t>0$. Assuming that there exists t_3 and t_4 are verified that

$$\begin{cases} C(t_3)=0, \quad C'(t_3)\leq 0 \quad \text{and } C(t_3+\varepsilon)<0, \text{ for all } \varepsilon>0\,, \\ D(t_4)=0, \quad D'(t_4)\leq 0 \quad \text{and } D(t_4+\varepsilon)<0, \text{ for all } \varepsilon>0. \end{cases}$$

Based on the third and fourth equations of system (3.1.1), we obtain

$$\begin{cases} C'(t_3)=\Big(\beta_2 S(t_3)+\lambda_2 O(t_3)-(\mu+\delta_2+\alpha_2)\Big)C(t_3)=0, \\ D'(t_4)=\Big(\beta_3 S(t_4)+\lambda_1 O(t_4)-(\mu+\delta_3+\alpha_1)\Big)D(t_4)=0. \end{cases}$$

Additionally, we have

$$\begin{cases} C(t)=C(t_3)exp\Big(\int_{t_3}^{t}\Big(\beta_2 S(s)+\lambda_2 O(s)-(\mu+\delta_2+\alpha_2)\Big)ds\Big), \quad \text{for all } t>t_3, \\ D(t)=D(t_4)exp\Big(\int_{t_4}^{t}\Big(\beta_3 S(s)+\lambda_1 O(s)-(\mu+\delta_3+\alpha_1)\Big)ds\Big), \quad \text{for all } t>t_4, \end{cases}$$

It provides

$$\begin{cases} C(t)=0, \quad \text{for all } t>t_3, \\ D(t)=0, \quad \text{for all } t>t_4. \end{cases}$$

Then,

$$\begin{cases} C(t_3+\varepsilon)=0, \\ D(t_4+\varepsilon)=0. \end{cases}$$

This is a contradiction with our considerations. Thus, we deduce that $C(t)\geq 0$ and $D(t)\geq 0$ for any $t>0$.

To prove that the solution T is positive, we suppose that there exists $t_2>0$ where $T(t_2)=0$, $T'(t_2)<0.$, $D(t_1)>0$, and $C(t_1)>0$. We obtain,

$$T'(t_2)=\alpha_1 D(t_2)+\alpha_2 C(t_2)>0. \tag{3.2.5}$$

That is contradiction. Therefore, $T(t)\geq 0$ for all $t>0$.

■

2.0.2 Invariant region

The next Lemma confirms the global existence of the solutions of model (3.1.1).

Lemma 3.2.1 *Let initial data be* $S(0)=S_0>0$, $O(0)=O_0>0$, $C(0)=C_0>0$, $D(0)=D_0>0$, *and* $T(0)=T_0>0$. *Then, there exists a domain* Γ *in which the solution set* (S,O,C,D,T) *is contained and bounded for the system (3.1.1), and satisfying*

$$\limsup_{t\longrightarrow\infty} N(t)\leq \tfrac{A}{\mu}.$$

Moreover, the solutions of model (3.1.1) exists for all $t>0$.

Proof. By adding the equations of the system (3.1.1), we get

$$\begin{aligned}\frac{dN(t)}{dt} &= A-\mu N-(\delta_1 O+\delta_2 C+\delta_3 D+\delta_4 T),\\ &\leq A-\mu N(t).\end{aligned} \tag{3.2.6}$$

Then, we get

$$N(t)\leq N(0)e^{-\mu t}+\frac{A}{\mu}, \tag{3.2.7}$$

hence,

$$\limsup_{t\longrightarrow\infty} N(t)\leq \frac{A}{\mu}.$$

This concludes the proof. ■

By Theorem 3.2.1 and the proof of Theorem 3.2.1, it is clear that the set $\Gamma=\{(S,O,C,D,T)\in \mathbb{R}^5_+ : N(t)\leq \frac{A}{\mu}\}$ is a positively invariant set for system (3.1.1) and is an attracted domain for initial condition belonging to Γ. Hence, global analysis of (3.1.1) will be focused in Γ.

Moreover, the solution of (3.1.1) is globally defined. Indeed, the local existence and uniqueness of the solution are demonstrated by the locally Lipschitz continuous right-hand side of model (3.1.1). The global existence of the solution follows from priori bound in Theorem 3.2.1.

3 Equilibrium points

Clearly, the system (3.1.1) admits a unique disease free equilibrium point $E_0=(\frac{A}{\mu},0,0,0,0)\in\Gamma$.

Proposition 3.3.0 *System (3.1.1) admits a cardiovascular free equilibrium point* $E_1^* = (S_1^*, O_1^*, 0, D_1^*, T_1^*)$ *with the following conditions of existence*

$$\begin{cases} \beta_1 > \beta_3, \quad \delta_3 > \delta_1, \\ \frac{(\mu+\delta_3+\alpha_1)}{\lambda_1} > \frac{\beta_3 A}{\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)}, \\ \frac{\beta_1 A}{\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)} > \frac{(\mu+\delta_1)}{\lambda_1}. \end{cases} \tag{3.3.8}$$

Proof. The cardiovascular free equilibrium denotes $E_1^* = (S_1^*, O_1^*, 0, D_1^*)$ is the constant solution of system (3.1.1) in the absence of the cardiovascular disease i.e $C_1^* = 0$. This equilibrium solves the system

$$\begin{cases} A-\mu S_1^*-\beta_1 O_1^* S_1^*-\beta_3 D_1^* S_1^* & = 0, \\ (\beta_1 S_1^*-(\mu+\delta_1+\lambda_1 D_1^*))O_1^* & = 0, \\ (\beta_3 S_1^*+\lambda_1 O_1^*-(\mu+\delta_3+\alpha_1))D_1^* & = 0, \\ \alpha_1 D_1^*-(\mu+\delta_4)T_1^* & = 0. \end{cases} \tag{3.3.9}$$

From the first equation of system (3.3.9), we find that

$$S_1^* = \frac{A}{\mu+\beta_1 O_1^*+\beta_3 D_1^*}. \tag{3.3.10}$$

From the second equation of system (3.3.9), we get

$$D_1^* = \frac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}. \tag{3.3.11}$$

The third equation of system (3.3.9) gives us

$$O_1^* = \frac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}. \tag{3.3.12}$$

We obtain from the fourth equation of system (3.3.9) that

$$T_1^* = \frac{\alpha_1 D_1^*}{\mu+\delta_4}. \tag{3.3.13}$$

Substituting (3.3.11) and (3.3.12) into (3.3.10), we find

$$S_1^* = \frac{\lambda_1 A}{\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)}. \tag{3.3.14}$$

Plugging (3.3.14) into (3.3.11) and (3.3.12), we get

$$D_1^* = \frac{\beta_1\lambda_1 A-(\mu+\delta_1)\Big(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)\Big)}{\lambda_1\Big(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)\Big)}, \tag{3.3.15}$$

and

$$O_1^* = \frac{(\mu+\delta_3+\alpha_1)\Big(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)\Big)-\beta_3\lambda_1 A}{\lambda_1(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1))}. \tag{3.3.16}$$

We substitute (3.3.15) in (3.3.13), we obtain

$$T_1^* = \frac{\alpha_1\Big(\beta_1\lambda_1 A-(\mu+\delta_1)(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1))\Big)}{\lambda_1(\mu+\delta_4)\Big(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)\Big)}. \tag{3.3.17}$$

Therefore, The equilibrium point $E_1^* = (S_1^*, O_1^*, 0, D_1^*)$ is given by

$$\begin{cases} S_1^* = \frac{\lambda_1 A}{\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)}, & O_1^* = \frac{(\mu+\delta_3+\alpha_1)\Big(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)\Big)-\beta_3\lambda_1 A}{\lambda_1(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1))}, \quad C_1^* = 0, \\ D_1^* = \frac{\beta_1\lambda_1 A-(\mu+\delta_1)\Big(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)\Big)}{\lambda_1\Big[\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)\Big]}, & T_1^* = \frac{\alpha_1\beta_1\lambda_1 A-(\mu+\delta_1)(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1))}{\lambda_1(\mu+\delta_4)\Big(\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)\Big)}, \end{cases}$$

E_1^* is positive under the following conditions

$$\begin{cases} \beta_1 > \beta_3, & \delta_3 > \delta_1, \\ \frac{(\mu+\delta_3+\alpha_1)}{\lambda_1} > \frac{\beta_3 A}{\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)}, & \frac{\beta_1 A}{\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)} > \frac{(\mu+\delta_1)}{\lambda_1}. \end{cases}$$

This completed the proof. ■

Proposition 3.3.0 *System (3.1.1) admits a diabetes-free equilibrium point $E_2^* = (S_2^*, O_2^*, C_2^*, 0, T_2^*)$ with the following conditions of existence*

$$\beta_1 > \beta_2, \quad \delta_2 > \delta_1, \quad \frac{(\mu+\delta_2+\alpha_2)}{\lambda_2} > \frac{\beta_2 A}{\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)}, \quad \frac{\beta_1 A}{\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)} > \frac{(\mu+\delta_1)}{\lambda_2}. \tag{3.3.18}$$

Proof. The diabetes free equilibrium $E_2^* = (S_2^*, O_2^*, C_2^*, 0, T_2^*)$ denotes the constant solution of system (3.1.1). This equilibrium satisfies

$$\begin{cases} A-(\mu+\beta_1 O_2^*+\beta_2 C_2^*)S_2^* & = 0, \\ (\beta_1 S_2^*-(\mu+\delta_1+\lambda_2 C_2^*))O_2^* & = 0, \\ (\beta_2 S_2^*+\lambda_2 O_2^*-(\mu+\delta_2+\alpha_2))C_2^* & = 0, \\ \alpha_2 C_2^*-(\mu+\delta_4)T_2^* & = 0. \end{cases} \tag{3.3.19}$$

We have $O_2^* \neq 0$ and $C_2^* \neq 0$. The first equation of system (3.3.19) implies

$$S_2^* = \frac{A}{\mu+\beta_1 O_2^* \beta_2 C_2^*}. \tag{3.3.20}$$

The second equation of system (3.3.19) leads to

$$C_2^* = \frac{\beta_1 S_2^* - (\mu+\delta_1)}{\lambda_2}. \tag{3.3.21}$$

From the third equation of system (3.3.19), we get

$$O_2^* = \frac{(\mu+\delta_2+\alpha_2) - \beta_2 S_2^*}{\lambda_2}. \tag{3.3.22}$$

From the fourth equation of system (3.3.19), we find

$$T_2^* = \frac{\alpha_2 C_2^*}{\mu+\delta_4}. \tag{3.3.23}$$

Substituting (3.3.21) and (3.3.22) into (3.3.20), we find

$$S_2^* = \frac{\lambda_2 A}{\lambda_2\mu + \beta_1(\mu+\delta_2+\alpha_2) - \beta_2(\mu+\delta_1)}. \tag{3.3.24}$$

By replacing (3.3.22) in (3.3.21) and (3.3.22), we get

$$C_2^* = \frac{\beta_1\lambda_2 A - (\mu+\delta_1)\Big(\lambda_2\mu + \beta_1(\mu+\delta_2+\alpha_2) - \beta_2(\mu+\delta_1)\Big)}{\lambda_2\Big(\lambda_2\mu + \beta_1(\mu+\delta_2+\alpha_2) - \beta_2(\mu+\delta_1)\Big)}, \tag{3.3.25}$$

and

$$O_2^* = \frac{(\mu+\delta_2+\alpha_2)\Big(\lambda_2\mu + \beta_1(\mu+\delta_2+\alpha_2) - \beta_2(\mu+\delta_1)\Big) - \beta_2\lambda_2 A}{\lambda_2(\lambda_2\mu + \beta_1(\mu+\delta_2+\alpha_2) - \beta_2(\mu+\delta_1))}. \tag{3.3.26}$$

We substitute (3.3.25) in (3.3.23), we obtain

$$T_2^* = \frac{\alpha_2[\beta_1\lambda_2 A - (\mu+\delta_1)(\lambda_2\mu + \beta_1(\mu+\delta_2+\alpha_2) - \beta_2(\mu+\delta_1))]}{\lambda_2(\mu+\delta_4)[\lambda_2\mu + \beta_1(\mu+\delta_2+\alpha_2) - \beta_2(\mu+\delta_1)]}. \tag{3.3.27}$$

Then, the diabetes free equilibrium $E_2^* = (S_2^*, O_2^*, C_2^*, 0, T_2^*)$ is given by

$$\begin{cases} S_2^* = \frac{\lambda_2 A}{\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)}, & O_2^* = \frac{(\mu+\delta_2+\alpha_2)\Big(\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)\Big)-\beta_2\lambda_2 A}{\lambda_2(\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1))}, \\ C_2^* = \frac{\beta_1\lambda_2 A-(\mu+\delta_1)\Big(\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)\Big)}{\lambda_2\Big(\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)\Big)}, & T_2^* = \frac{\alpha_2\Big(\beta_1\lambda_2 A-(\mu+\delta_1)(\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1))\Big)}{\lambda_2(\mu+\delta_4)\Big(\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)\Big)}, \quad D_2^* = 0. \end{cases} \tag{3.3.28}$$

E_2^* is positive if the following conditions are satisfied

$$\beta_1 > \beta_2, \quad \delta_2 > \delta_1, \quad \frac{(\mu+\delta_2+\alpha_2)}{\lambda_2} > \frac{\beta_2 A}{\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)}, \quad \frac{\beta_1 A}{\lambda_2\mu+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)} > \frac{(\mu+\delta_1)}{\lambda_2}.$$

■

Proposition 3.3.0 *System (3.1.1) admits a Cardiovascular, Diabetes, and Hypertension-free equilibrium point* $E_3^* = (S_3^*, O_3^*, 0, 0, 0)$ *with the following condition of existence*

$$\frac{\beta_1 A}{\mu(\mu+\delta_1)} > 1. \tag{3.3.29}$$

Proof. The Cardiovascular, Diabetes, and Hypertension free Equilibrium $E_3^* = (S_3^*, O_3^*, 0, 0, 0)$ verifies the following system

$$\begin{cases} A - \mu S_3^* - \beta_1 O_3^* S_3^* & = 0, \\ (\beta_1 S_3^* - (\mu+\delta_1)) O_3^* & = 0, \end{cases} \tag{3.3.30}$$

with $O_3^* \neq 0$. The second equation of system (3.3.30) gives

$$S_3^* = \frac{\mu+\delta_1}{\beta_1}. \tag{3.3.31}$$

From the first equation of system (3.3.30), we get

$$O_3^* = \frac{A}{\mu+\delta_1} - \frac{\mu}{\beta_1}. \tag{3.3.32}$$

Therefore, the cardiovascular, diabetes, hypertension free equilibrium $E_3^* = (S_3^*, O_3^*, 0, 0, 0)$ exists for $\frac{A}{\mu+\delta_1} - \frac{\mu}{\beta_1} > 0$, with

$$S_3^* = \frac{\mu+\delta_1}{\beta_1} \quad O_3^* = \frac{A}{\mu+\delta_1} - \frac{\mu}{\beta_1}. \tag{3.3.33}$$

E_3^* exists if and only if

$$\frac{\beta_1 A}{\mu(\mu+\delta_1)} > 1.$$

■

Proposition 3.3.0 *System (3.1.1) admits a obesity-cardiovascular free equilibrium point* $E_4^* = (S_4^*, 0, 0, D_4^*, T_4^*)$ *that exists if and only if*

$$\frac{\beta_3 A}{\mu(\mu+\delta_3+\alpha_1)} > 1. \tag{3.3.34}$$

Proof. The obesity-cardiovascular-free equilibrium $E_4^* = (S_4^*, 0, 0, D_4^*, T_4^*)$, which satisfies

$$\begin{cases} A - \mu S_4^* - \beta_3 D_4^* S_4^* & = 0, \\ (\beta_3 S_4^* - (\mu + \delta_3 + \alpha_1)) D_4^* & = 0, \\ \alpha_1 D_4^* - (\mu + \delta_4) T_4^* & = 0, \end{cases} \tag{3.3.35}$$

The fourth equation of system (3.3.35)implies

$$S_4^* = \frac{\mu + \delta_3 + \alpha_1}{\beta_3}. \tag{3.3.36}$$

The first equation of system (3.3.36) gives

$$D_4^* = \frac{A}{\mu + \delta_3 + \alpha_1} - \frac{\mu}{\beta_3}. \tag{3.3.37}$$

From the fifth equation of system (3.3.36), we find that

$$T_4^* = \frac{\alpha_1}{\mu + \delta_4} \left(\frac{A}{\mu + \delta_3 + \alpha_1} - \frac{\mu}{\beta_3} \right). \tag{3.3.38}$$

Therefore, we write the obesity-cardiovascular-free Equilibrium $E_4^* = (S_4^*, 0, 0, D_4^*, T_4^*)$ as follows

$$S_4^* = \tfrac{\mu+\delta_3+\alpha_1}{\beta_3} \quad O_4^* = 0 \quad C_4^* = 0 \quad D_4^* = \tfrac{A}{\mu+\delta_3+\alpha_1} - \tfrac{\mu}{\beta_3} \quad T_4^* = \tfrac{\alpha_1}{\mu+\delta_4}\left(\tfrac{A}{\mu+\delta_3+\alpha_1} - \tfrac{\mu}{\beta_3}\right), \tag{3.3.39}$$

which exists if and only if

$$\frac{\beta_3 A}{\mu(\mu + \delta_3 + \alpha_1)} > 1.$$

■

Proposition 3.3.0 *System (3.1.1) admits a obesity-diabetes-free equilibrium point* $E_5^* = (S_5^*, 0, C_5^*, 0, T_5^*)$ *if*

$$\frac{\beta_2 A}{\mu(\mu + \delta_2 + \alpha_2)} > 1. \tag{3.3.40}$$

Proof. The obesity-diabetes-free equilibrium $E_5^* = (S_5^*, 0, C_5^*, 0, T_5^*)$. This equilibrium solves

$$\begin{cases} A - \mu S_5^* - \beta_2 C_5^* S_5^* & = 0, \\ (\beta_2 S_5^* - (\mu + \delta_2 + \alpha_2)) C_5^* & = 0, \\ \alpha_2 C_5^* - (\mu + \delta_4) T_5^* & = 0. \end{cases} \tag{3.3.41}$$

From the second equation of (3.3.41), we get

$$S_5^* = \frac{\mu+\delta_2+\alpha_2}{\beta_2}. \tag{3.3.42}$$

From the first equation of (3.3.41), we obtain

$$C_5^* = \frac{A}{\mu+\delta_2+\alpha_2} - \frac{\mu}{\beta_2}. \tag{3.3.43}$$

According to the third equation of (3.3.41), we have

$$T_5^* = \frac{\alpha_2}{\mu+\delta_4}\left(\frac{A}{\mu+\delta_2+\alpha_2} - \frac{\mu}{\beta_2}\right). \tag{3.3.44}$$

Hence, the obesity-diabetes-free equilibrium $E_5^* = (S_5^*, 0, C_5^*, 0, T_5^*)$ is given by

$$S_5^* = \tfrac{\mu+\delta_2+\alpha_2}{\beta_2}, \quad O_5^* = 0, \quad C_5^* = \tfrac{A}{\mu+\delta_2+\alpha_2} - \tfrac{\mu}{\beta_2}, \quad D_5^* = 0, \quad T_5^* = \tfrac{\alpha_2}{\mu+\delta_4}\left(\tfrac{A}{\mu+\delta_2+\alpha_2} - \tfrac{\mu}{\beta_2}\right).$$

E_5^* is positive if the following inequality holds

$$\frac{\beta_2 A}{\mu(\mu+\delta_2+\alpha_2)} > 1.$$

■

Now, we determine the endemic equilibrium point, which represents the persistence of all classes. The obtained results are as follows

Proposition 3.3.0 *System (3.1.1) admits a endemic equilibrium that is* $E_6^* = (S_6^*, O_6^*, C_6^*, D_6^*, T_6^*)$ *if*

$$\begin{cases} \lambda_2 > \lambda_1, \quad \beta_1 > \beta_3, \quad \beta_1 > \beta_2, \quad \beta_3 > \beta_2, \\ \frac{\mu\lambda_1+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)}{\lambda_2\beta_3-\lambda_1\beta_2} > \frac{\lambda_1 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\alpha_2+\delta_2)}, \\ \frac{\lambda_2 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)} > \frac{\mu\lambda_2+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)}{\lambda_2\beta_3-\lambda_1\beta_2}. \end{cases} \tag{3.3.45}$$

Proof. The endemic equilibrium $E_6^* = (S_6^*, O_6^*, C_6^*, D_6^*, T_6^*)$, with $S_6^* \neq 0, O_6^* \neq 0, C_6^* \neq 0, D_6^* \neq 0,$ *and* $T_6^* \neq 0$. This equilibrium is the solution of the following system

$$\begin{cases} A-(\mu+\beta_1 O_6^*+\beta_2 C_6^*+\beta_3 D_6^*)S_6^* &= 0, \\ (\beta_1 S_6^*-(\mu+\delta_1+\lambda_1 D_6^*+\lambda_2 C_6^*)O_6^* &= 0, \\ (\beta_2 S_6^*+\lambda_2 O_6^*-(\mu+\delta_2+\alpha_2))C_6^* &= 0, \\ (\beta_3 S_6^*+\lambda_1 O_6^*-(\mu+\delta_3+\alpha_1))D_6^* &= 0, \\ \alpha_1 D_6^*+\alpha_2 C_6^*-(\mu+\delta_4)T_6^* &= 0. \end{cases} \tag{3.3.46}$$

We multiply third equation of system(3.3.46) by λ_1 and the fourth equation of system(3.3.46) by $-\lambda_2$. By addition these equations, we find

$$S_6^* = \frac{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)}{\lambda_2\beta_3-\lambda_1\beta_2}. \tag{3.3.47}$$

From the third equation of system (3.3.46) and (3.3.47), we get

$$O_6^* = \frac{\beta_3(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_3+\alpha_1)}{\lambda_2\beta_3-\beta_2\lambda_1}. \tag{3.3.48}$$

By multiplying the first equation of system 3.3.46) by λ_1 and the second equation of system(3.3.46) by $-\beta_2$. By addition these equations, we obtain

$$C_6^* = \frac{\mu\lambda_1+\beta_1(\mu+\delta_3+\alpha_1)-(\mu+\delta_1)\beta_3}{\lambda_2\beta_3-\lambda_1\beta_2} - \frac{A\lambda_1}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)}. \tag{3.3.49}$$

By multiplying the first equation of system 3.3.46) by λ_2 and the second equation of system(3.3.46) by $-\beta_2$. By summing these equations, we obtain

$$D_6^* = \frac{\lambda_2 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)} - \frac{\mu\lambda_2+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)}{\lambda_2\beta_3-\lambda_1\beta_2}. \tag{3.3.50}$$

By replacing (3.3.49) and (3.3.50) in fifth equation of system 3.3.46), we get

$$\begin{aligned} T_6^* &= \frac{\alpha_2}{\mu+\delta_4}\left(\frac{\mu\lambda_1+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)}{\lambda_2\beta_3-\lambda_1\beta_2} - \frac{\lambda_1 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)}\right) \\ &+ \frac{\alpha_1}{\mu+\delta_4}\left(\frac{\lambda_2 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)} + \frac{\beta_2(\mu+\delta_1)-\mu\lambda_2-\beta_1(\mu+\delta_2+\alpha_2)}{\lambda_2\beta_3-\lambda_1\beta_2}\right). \end{aligned} \tag{3.3.51}$$

So, The endemic equilibrium $E_6^* = (S_6^*, O_6^*, C_6^*, D_6^*)$ is

$$\begin{cases} S_6^* = & \frac{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)}{\lambda_2\beta_3-\lambda_1\beta_2}, \\ O_6^* = & \frac{\beta_3(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_3+\alpha_1)}{\lambda_2\beta_3-\beta_2\lambda_1}, \\ C_6^* = & \frac{\mu\lambda_1+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)}{\lambda_2\beta_3-\lambda_1\beta_2} - \frac{\lambda_1 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\alpha_2+\delta_2)}, \\ D_6^* = & \frac{\lambda_2 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)} - \frac{\mu\lambda_2+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)}{\lambda_2\beta_3-\lambda_1\beta_2}, \\ T_6^* = & \frac{\alpha_2}{\mu+\delta_4}\left(\frac{\mu\lambda_1+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)}{\lambda_2\beta_3-\lambda_1\beta_2} - \frac{\lambda_1 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)}\right) \\ & + \frac{\alpha_1}{\mu+\delta_4}\left[\frac{\lambda_2 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)} + \frac{\beta_2(\mu+\delta_1)-\mu\lambda_2-\beta_1(\mu+\delta_2+\alpha_2)}{\lambda_2\beta_3-\lambda_1\beta_2}\right]. \end{cases} \tag{3.3.52}$$

E_6^* is positive if the following conditions are satisfied

$$\begin{cases} \lambda_2 > \lambda_1, & \beta_1 > \beta_3, & \beta_1 > \beta_2, & \beta_3 > \beta_2, \\ \frac{\mu\lambda_1+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)}{\lambda_2\beta_3-\lambda_1\beta_2} & > \frac{\lambda_1 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\alpha_2+\delta_2)}, & \frac{\lambda_2 A}{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)} & > \frac{\mu\lambda_2+\beta_1(\mu+\delta_2+\alpha_2)-\beta_2(\mu+\delta_1)}{\lambda_2\beta_3-\lambda_1\beta_2}. \end{cases} \tag{3.3.53}$$

■

4 The local stability of the model

In this section, we consider the local stability of all equilibria given in the last section. The Jacobian matrix of system (3.1.1) at an arbitrary equilibrium (S, O, C, D, T) is as follows

$$J = \begin{bmatrix} -(\mu+\beta_1 O+\beta_2 C+\beta_3 D) & -\beta_1 S & -\beta_2 S & -\beta_3 S & 0 \\ \beta_1 O & \beta_1 S-(a+\lambda_1 D+\lambda_2 C) & -\lambda_2 O & -\lambda_1 O & 0 \\ \beta_2 C & \lambda_2 C & \beta_2 S+\lambda_2 O-b & 0 & 0 \\ \beta_3 D & \lambda_1 D & 0 & \beta_3 S+\lambda_1 O-c & 0 \\ 0 & 0 & \alpha_2 & \alpha_1 & -d \end{bmatrix},$$

with $a=\mu+\delta_1$, $b=\mu+\delta_2+\alpha_2$, $c=\mu+\delta_3+\alpha_1$, $d=\mu+\delta_4$. We start with the local stability of E_0. The Jacobian of the system (3.1.1) at E_0 is written as follows

$$J(E_0)=\begin{bmatrix} -\mu & -\beta_1\frac{A}{\mu} & -\beta_2\frac{A}{\mu} & -\beta_3 S & 0 \\ 0 & \beta_1\frac{A}{\mu}-(\mu+\delta_1) & 0 & 0 & 0 \\ 0 & 0 & -(\mu+\delta_2+\alpha_2)+\beta_2\frac{A}{\mu} & 0 & 0 \\ 0 & 0 & 0 & -(\mu+\delta_3+\alpha_1)+\beta_3\frac{A}{\mu} & 0 \\ 0 & 0 & \alpha_2 & \alpha_1 & -(\mu+\delta_4) \end{bmatrix}.$$

We notice that $J(E_0)$ has three eigenvalues $\lambda_1=-\mu$, $\lambda_2=\beta_1\frac{A}{\mu}-(\mu+\delta_1)$, $\lambda_3=-(\mu+\delta_4)$, $\lambda_4=\beta_2\frac{A}{\mu}-(\mu+\delta_2+\alpha_2)$, and $\lambda_5=\beta_3\frac{A}{\mu}-(\mu+\delta_3+\alpha_1)$. This means that $\lambda_1=-\mu<0$, $\lambda_2=-(\mu+\delta_4)<0$. Thus, E_0 is locally asymptotically stable if

$$\begin{cases} \lambda_2=\beta_1\frac{A}{\mu}-(\mu+\delta_1)<0, \\ \lambda_4=\beta_2\frac{A}{\mu}-(\mu+\delta_2+\alpha_2)<0, \\ \lambda_5=-(\mu+\delta_3+\alpha_1)+\beta_3\frac{A}{\mu}<0. \end{cases}$$

Next, we prove the local stability of E_1^*. The Jacobian matrix at E_1^* is given by

$$J(E_1^*)=\begin{bmatrix} -A_1 & -A_2 & -A_3 & -A_4 & 0 \\ A_5 & A_6 & -A_7 & -A_8 & 0 \\ 0 & 0 & A_9 & 0 & 0 \\ A_{10} & A_{11} & 0 & A_{12} & 0 \\ 0 & 0 & g & f & -A_{13} \end{bmatrix},$$

with $A_1=\mu+\beta_1 O_1^*+\beta_3 D_1^*$, $A_2=\beta_1 S_1^*$, $A_3=\beta_2 S_1^*$, $A_4=\beta_3 S_1^*$, $A_5=\beta_1 O_1^*$, $A_6=\beta_1 S_1^*-(a+\lambda_1 D_1^*)$, $A_7=\lambda_2 O_1^*$, $A_8=\lambda_1 O_1^*$, $A_9=\beta_2 S_1^*+\lambda_2 O_1^*-b$, $A_{10}=\beta_3 D_1^*$, $A_{11}=\lambda_1 D_1^*$, $A_{12}=\beta_3 S_1^*+\lambda_1 O_1^*-c$, $A_{13}=d$, $f=\alpha_1$, $g=\alpha_2$.

Notice that $\lambda_{1,1}=-d=-(\mu+\delta_4)$, $\lambda_{1,2}=A_9=\beta_2 S_1^*+\lambda_2 O_1^*-b$ are eigenvalues of the matrix $J(E_1^*)$, such that $\lambda_{1,1}$ is a negative eigenvalue and $\lambda_{1,2}$ will be a negative eigenvalue under the condition

$$\lambda_1\beta_2 S_1^*+\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_2\beta_3 S_1^*-\lambda_1(\mu+\delta_2+\alpha_2)<0$$

is equivalent to

$$\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)<(\lambda_2\beta_3-\lambda_1\beta_2)S_1^*.$$

This leads to

$$S_1^*>\frac{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)}{\lambda_2\beta_3-\lambda_1\beta_2}$$

Then, it remains to determine the nature of the eigenvalues of the following reduced matrix of the previous one

$$J_1(E_1^*)=\begin{bmatrix}-A_1 & -A_2 & -A_4\\ A_5 & A_6 & -A_8\\ A_{10} & A_{11} & A_{12}\end{bmatrix}$$

Therefore, the characteristic polynomial of this matrix is

$$P(\lambda)=\lambda^3+C_{1,1}\lambda^2+C_{1,2}\lambda+C_{1,3}.$$

where,

$$\begin{aligned}C_{1,1} &= (A_1-A_6-A_{12}),\\ C_{1,2} &= A_2A_5-A_1A_6+A_4A_{10}+A_8A_{11}-A_{12}(A_1-A_6),\\ C_{1,3} &= (A_1-A_6)(A_4A_{10}+A_8A_{11})-A_{10}(A_1A_4+A_2A_8)+A_{12}(A_1A_6-A_2A_5)+A_{11}(A_4A_5+A_6A_8).\end{aligned}$$

Using Lienard-Chipart criteria, the system is locally asymptotically stable if the coefficients $C_{1,1}$, $C_{1,3}$ are positive and the Hurwitz determinant

$$H_{1,2}=\begin{Vmatrix} C_{1,1} & C_{1,3}\\ 1 & C_{1,2}\end{Vmatrix}$$

is positive.

To complete the demonstration, we must show that the previous conditions are satisfied. We start by proving that the coefficients $C_{1,1}$, and $C_{1,3}$ are positive, we get

$$\begin{aligned}
C_{1,1} &= A_1 - A_6 - A_{12}, \\
&= \Big(\mu+\beta_1\Big(\frac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)+\beta_3\Big(\frac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big)-\Big(\beta_1 S_1^*-\Big((\mu+\delta_1)+\lambda_1\Big(\frac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big)\Big) \\
&\quad -\Big(\beta_3 S_1^*+\lambda_1\Big(\frac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)-(\mu+\delta_3+\alpha_1)\Big), \\
&= \frac{1}{\lambda_1}\big(\mu\beta_1+\mu\lambda_1-\mu\beta_3+\alpha_1\beta_1+\beta_1\delta_3-\beta_3\delta_1\big).
\end{aligned}$$

Now, we show that $C_{1,3}$ is positive. So, we have

$$\begin{aligned}
C_{1,3} = &\Big(\Big(\mu+\beta_1\Big(\tfrac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)+\beta_3\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big)-\Big(\beta_1 S_1^*-\Big((\mu+\delta_1)+\lambda_1\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big)\Big)\Big) \\
&\Big(\beta_3 S_1^*\Big(\beta_3\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big)+\Big(\lambda_1\Big(\tfrac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)\Big)\Big(\lambda_1\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big)\Big)-\Big(\beta_3\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big) \\
&\Big(\Big(\mu+\beta_1\Big(\tfrac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)+\beta_3\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big)\beta_3 S_1^*+\beta_1 S_1^*\Big(\lambda_1\Big(\tfrac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)\Big)\Big) \\
&+\Big(\beta_3 S_1^*+\lambda_1\Big(\tfrac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)-(\mu+\delta_3+\alpha_1)\Big)\Big(\Big(\mu+\beta_1\Big(\tfrac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)+\beta_3\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big) \\
&\Big(\beta_1 S_1^*-\Big((\mu+\delta_1)+\lambda_1\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big)\Big)-\beta_1 S_1^*\Big(\beta_1\Big(\tfrac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)\Big)\Big)+\Big(\lambda_1\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big) \\
&\Big(\beta_3 S_1^*\Big(\beta_1\Big(\tfrac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)\Big)+\Big(\beta_1 S_1^*-\Big((\mu+\delta_1)+\lambda_1\Big(\tfrac{\beta_1 S_1^*-(\mu+\delta_1)}{\lambda_1}\Big)\Big)\Big)\Big(\lambda_1\Big(\tfrac{(\mu+\delta_3+\alpha_1)-\beta_3 S_1^*}{\lambda_1}\Big)\Big)\Big), \\
= &\ -\tfrac{1}{\lambda_1}\big(\mu+\delta_1-\beta_1 S_1^*\big)\big(\mu+\alpha_1+\delta_3-\beta_3 S_1^*\big)\big(\mu\beta_1+\mu\lambda_1-\mu\beta_3+\alpha_1\beta_1+\beta_1\delta_3-\beta_3\delta_1\big).
\end{aligned}$$

We evaluate the Hurwitz determinant, we find

$$\begin{aligned}
H_{1,2} &= C_{1,1}C_{1,2}-C_{1,3}, \\
&= \Big(\tfrac{1}{\lambda_1}\big(\mu\beta_1+\mu\lambda_1-\mu\beta_3+\alpha_1\beta_1+\beta_1\delta_3-\beta_3\delta_1\big)\Big)\Big(-\tfrac{1}{\lambda_1}\Big[\mu^2\lambda_1-\beta_1\beta_3^2(S_1^*)^2+\beta_1^2\beta_3(S_1^*)^2-\mu\beta_1^2 S_1^* \\
&\quad +\mu\beta_3^2 S_1^*-\alpha_1\beta_1^2 S_1^*-\beta_1^2\delta_3 S_1^*+\beta_3^2\delta_1 S_1^*+\mu\alpha_1\lambda_1+\mu\lambda_1\delta_1+\mu\lambda_1\delta_3+\alpha_1\lambda_1\delta_1+\lambda_1\delta_1\delta_3-\mu\beta_1\lambda_1 S_1^* \\
&\quad -\mu\lambda_1\beta_3 S_1^*-\alpha_1\beta_1\lambda_1 S_1^*-\beta_1\lambda_1\delta_3 S_1^*-\lambda_1\beta_3\delta_1 S_1^*+\beta_1\lambda_1\beta_3(S_1^*)^2\Big]\Big) \\
&\quad -\Big(-\tfrac{1}{\lambda_1}\big(\mu+\delta_1-\beta_1 S_1^*\big)\big(\mu+\alpha_1+\delta_3-\beta_3 S_1^*\big)\big(\mu\beta_1+\mu\lambda_1-\mu\beta_3+\alpha_1\beta_1+\beta_1\delta_3-\beta_3\delta_1\big)\Big), \\
&= \tfrac{1}{\lambda_1^2}S_1^*\Big(\mu\beta_1+\mu\lambda_1-\mu\beta_3+\alpha_1\beta_1+\beta_1\delta_3-\beta_3\delta_1\Big)\Big(\mu\beta_1^2-\mu\beta_3^2+\alpha_1\beta_1^2+\beta_1^2\delta_3-\beta_3^2\delta_1+\beta_1\beta_3^2 S_1^* \\
&\quad -\beta_1^2\beta_3 S_1^*\Big).
\end{aligned}$$

4. The local stability of the model

Next, we demonstrate the local stability of E_2^*. The Jacobian matrix at E_2^* is given by

$$J(E_2^*)=\begin{bmatrix} -B_1 & -B_2 & -B_3 & -B_4 & 0 \\ B_5 & B_6 & -B_7 & -B_8 & 0 \\ B_9 & B_{10} & B_{11} & 0 & 0 \\ 0 & 0 & 0 & B_{12} & 0 \\ 0 & 0 & g & f & -B_{13} \end{bmatrix},$$

with $B_1=(\mu+\beta_1 O_2^*+\beta_2 C_2^*)$, $B_2=\beta_1 S_2^*$, $B_3=\beta_2 S_2^*$, $B_4=\beta_3 S_2^*$, $B_5=\beta_1 O_2^*$, $B_6=\beta_1 S_2^*-(a+\lambda_2 C_2^*)$, $B_7=\lambda_2 O_2^*$, $B_8=\lambda_1 O_2^*$, $B_9=\beta_2 C_2^*$, $B_{10}=\lambda_2 C_2^*$, $B_{11}=\beta_2 S_2^*+\lambda_2 O_2^*-b$, $B_{12}=\beta_3 S_2^*+\lambda_1 O_2^*-c$, $B_{13}=d$, $f=\alpha_2$, $g=\alpha_1$.

It is clear that $\lambda_{2,1}=-d=-(\mu+\delta_4)$ and $\lambda_{2,2}=B_{12}=\beta_3 S_2^*+\lambda_1 O_2^*-(\mu+\delta_3+\alpha_1)$ are eigenvalues of the matrix $J(E_2^*)$, such that $\lambda_{2,1}$ is a negative eigenvalue and from which $\lambda_{2,1}$ is a negative eigenvalue if

$$\beta_3 S_2^*+\lambda_2\Big(\frac{(\mu+\delta_3+\alpha_1-\beta_3 S_2^*}{\lambda_1}-(\mu+\delta_3+\alpha_1)\Big)<0,$$

which is equivalent to

$$\Big(\lambda_2-\lambda_1)(\mu+\delta_3+\alpha_1)<(\lambda_2-\lambda_1)\beta_3 S_2^*.$$

This leads to

$$S_2^*>\frac{\mu+\delta_3+\alpha_1}{\beta_3}.$$

Now, we need to study the local stability of matrix $J_1(E_2^*)$ which is

$$J_1(E_2^*)=\begin{bmatrix} -B_1 & -B_2 & -B_3 \\ B_5 & B_6 & -B_7 \\ B_9 & B_{10} & B_{11} \end{bmatrix}$$

Therefore, the characteristic polynomial of the Jacobian matrix is given by

$$P(\lambda)=\lambda^3+C_{2,1}\lambda^2+C_{2,2}\lambda+C_{2,3}.$$

where, $C_{2,1}=B_1-B_6-B_{11}$,

$C_{2,2}=B_2B_5-B_1B_6+B_3B_9+B_7B_{10}-B_{11}(B_1-B_6)$,

$C_{2,3}=(B_1-B_6)(B_3B_9+B_7B_{10})-B_9(B_1B_3+B_2B_7)+B_{11}(B_1B_6-B_2B_5)+B_{10}(B_3B_5+B_6B_7)$.

Using Lienard-Chipart criteria, the system is locally asymptotically stable if the coefficients $C_{2,1}$, and $C_{2,3}$ are positive and the Hurwitz determinant

$$H_{2,2} = \begin{Vmatrix} C_{2,1} & C_{2,3} \\ 1 & C_{2,2} \end{Vmatrix}$$

is positive. Now, we check the positivity of the previously mentioned coefficients. We begin by $C_{2,1}$, then

$$\begin{aligned} C_{2,1} &= B_1 - B_6 - B_{11}, \\ &= \Big(\mu+\beta_1\Big(\frac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)+\beta_2\Big(\frac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big)-\Big(\beta_1 S_2^*-\Big((\mu+\delta_1)+\lambda_2\Big(\frac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big)\Big) \\ &\quad -\Big(\beta_2 S_2^*+\lambda_2\Big(\frac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)-(\mu+\delta_2+\alpha_2)\Big), \\ &= \frac{1}{\lambda_2}\big(\mu\beta_1-\mu\beta_2+\mu\lambda_2+\alpha_2\beta_1+\beta_1\delta_2-\beta_2\delta_1\big).. \end{aligned}$$

We can write $C_{2,3}$ as follows

$$\begin{aligned} C_{2,3} &= \Big(\Big(\mu+\beta_1\Big(\tfrac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)+\beta_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big)-\Big(\beta_1 S_2^*-\Big((\mu+\delta_1)+\lambda_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big)\Big)\Big) \\ &\quad \times\Big(\beta_2 S_2^*\Big(\beta_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big)+\Big(\lambda_2\Big(\tfrac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)\Big)\Big(\lambda_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big)\Big)-\beta_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big) \\ &\quad \times\Big(\Big(\mu+\beta_1\Big(\tfrac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)+\beta_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big)\beta_2 S_2^*+\beta_1 S_2^*\Big(\lambda_2\Big(\tfrac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)\Big)\Big) \\ &\quad +\Big(\beta_2 S_2^*+\lambda_2\Big(\tfrac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)-(\mu+\delta_2+\alpha_2)\Big)\Big(\Big(\mu+\beta_1\Big(\tfrac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)+\beta_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big) \\ &\quad \times\Big(\beta_1 S_2^*-\Big((\mu+\delta_1)+\lambda_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big)\Big)-\beta_1 S_2^*\Big(\beta_1\Big(\tfrac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)\Big)\Big)+\Big(\lambda_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big) \\ &\quad \times\Big(\beta_2 S_2^*\Big(\beta_1\Big(\tfrac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)\Big)+\Big(\beta_1 S_2^*-\Big((\mu+\delta_1)+\lambda_2\Big(\tfrac{\beta_1 S_2^*-(\mu+\delta_1)}{\lambda_2}\Big)\Big)\Big)\Big(\lambda_2\Big(\tfrac{(\mu+\delta_2+\alpha_2)-\beta_2 S_2^*}{\lambda_2}\Big)\Big)\Big), \\ &= -\tfrac{1}{\lambda_2}\big(\mu+\delta_1-\beta_1 S_2^*\big)\big(\mu+\alpha_2+\delta_2-\beta_2 S_2^*\big)\big(\mu\beta_1-\mu\beta_2+\mu\lambda_2+\alpha_2\beta_1+\beta_1\delta_2-\beta_2\delta_1\big). \end{aligned}$$

Next, $H_{2,2}$ can be written

$$\begin{aligned} H_{2,2} &= C_{2,1}C_{2,2}-C_{2,3} \\ &= \Big(\tfrac{1}{\lambda_2}\big(\mu\beta_1-\mu\beta_2+\mu\lambda_2+\alpha_2\beta_1+\beta_1\delta_2-\beta_2\delta_1\big)\Big)\Big(-\tfrac{1}{\lambda_2}\Big(\mu^2\lambda_2-\beta_1\beta_2^2(S_2^*)^2+\beta_1^2\beta_2(S_2^*)^2-\mu\beta_1^2 S_2^* \\ &\quad +\mu\beta_2^2 S_2^*-\alpha_2\beta_1^2 S_2^*-\beta_1^2\delta_2 S_2^*+\beta_2^2\delta_1 S_2^*+\mu\alpha_2\lambda_2+\mu\lambda_2\delta_1+\mu\lambda_2\delta_2+\alpha_2\lambda_2\delta_1+\lambda_2\delta_1\delta_2 \\ &\quad -\mu\beta_1\lambda_2 S_2^*-\mu\beta_2\lambda_2 S_2^*-\alpha_2\beta_1\lambda_2 S_2^*-\beta_1\lambda_2\delta_2 S_2^*-\beta_2\lambda_2\delta_1 S_2^*+\beta_1\beta_2\lambda_2(S_2^*)^2\Big)\Big) \\ &\quad -\Big(-\tfrac{1}{\lambda_2}\big(\mu+\delta_1-\beta_1 S_2^*\big)\big(\mu+\alpha_2+\delta_2-\beta_2 S_2^*\big)\big(\mu\beta_1-\mu\beta_2+\mu\lambda_2+\alpha_2\beta_1+\beta_1\delta_2-\beta_2\delta_1\big)\Big), \\ &= \tfrac{1}{\lambda_2^2}S_2^*\big(\mu\beta_1-\mu\beta_2+\mu\lambda_2+\alpha_2\beta_1+\beta_1\delta_2-\beta_2\delta_1\big)\Big(\mu\beta_1^2-\mu\beta_2^2+\alpha_2\beta_1^2+\beta_1^2\delta_2-\beta_2^2\delta_1+\beta_1\beta_2^2 S_2^* \\ &\quad -\beta_1^2\beta_2 S_2^*\Big)\Big). \end{aligned}$$

To prove the local stability of E_3^*, we evaluate Jacobian matrix at E_3^*, that is

$$J(E_3^*)=\begin{bmatrix} -D_1 & -D_2 & -D_3 & -D_4 & 0 \\ D_5 & D_6 & -D_7 & -D_8 & 0 \\ 0 & 0 & D_9 & 0 & 0 \\ 0 & 0 & 0 & D_{10} & 0 \\ 0 & 0 & g & f & -D_{11} \end{bmatrix},$$

with $D_1=\mu+\beta_1 O_3^*$, $D_2=\beta_1 S_3^*$, $D_3=\beta_2 S_3^*$, $D_4=\beta_3 S_3^*$, $D_5=\beta_1 O_3^*$, $D_6=\beta_1 S_3^*-a$, $D_7=\lambda_2 O_3^*$, $D_8=\lambda_1 O_3^*$, $D_9=\beta_2 S_3^*+\lambda_2 O_3^*-b$, $D_{10}=\beta_3 S_3^*+\lambda_1 O_3^*-c$, $D_{11}=d$, $f=\alpha_2$, $g=\alpha_1$.

Noting that $\lambda_{3,1}=-d=-(\mu+\delta_4)$ and $\lambda_{3,2}=\beta_1 S_3^*-a$ are eigenvalues of the matrix $J(E_3^*)$. Knowing that $\lambda_{3,1}=-(\mu+\delta_4)$ is a negative eigenvalue. To check local stability of $J(E_3^*)$ the eigenvalue $\lambda_{3,2}=\beta_1 S_3^*-a$ must be negative, i.e under the condition $\beta_1 S_3^*<a$. Now, we need to verify that the matrix $J_1(E_3^*)$ is locally stable, where

$$J_1(E_3^*)=\begin{bmatrix} -D_1 & -D_3 & -D_4 \\ 0 & D_9 & 0 \\ 0 & 0 & D_{10} \end{bmatrix}.$$

Noting that $\lambda_{3,3}=-D_1=-(\mu+\beta_1 O_3^*)=-\frac{\beta_1 A}{\mu+\delta_1}$ is a negative eigenvalue of the matrix $J_1(E_3^*)$. This gives the matrix

$$J_2(E_3^*)=\begin{bmatrix} D_9 & 0 \\ 0 & D_{10} \end{bmatrix},$$

which has $\lambda_{3,4}=D_9=\beta_2 S_3^*+\lambda_2 O_3^*-b$ and $\lambda_{3,5}=D_{10}=\beta_3 S_3^*+\lambda_1 O_3^*-c$ eigenvalues, which must be negative to achieve local stability of E_3^* i.e when the following conditions are satisfied

$$\begin{cases} \frac{\beta_2(\mu+\delta_1)}{\beta_1}+\frac{\lambda_2 A}{\mu+\delta_1}<\frac{\lambda_2\mu}{\beta_1}+(\mu+\delta_2+\alpha_2), \\ \frac{\beta_3(\mu+\delta_1)}{\beta_1}+\frac{\lambda_1 A}{\mu+\delta_1}<\frac{\lambda_1\mu}{\beta_1}+(\mu+\delta_3+\alpha_1). \end{cases} \tag{3.4.54}$$

After that, we prove the local stability of E_4^*. The Jacobian matrix at E_4^* is given by

$$J(E_4^*)=\begin{bmatrix} -E_1 & -E_2 & -E_3 & -E_4 & 0 \\ 0 & E_5 & 0 & 0 & 0 \\ 0 & 0 & E_6 & 0 & 0 \\ E_7 & E_8 & 0 & E_9 & 0 \\ 0 & 0 & g & f & -E_{10} \end{bmatrix}$$

where $E_1=\mu+\beta_3 D_4^*$, $E_2=\beta_1 S_4^*$, $E_3=\beta_2 S_4^*$, $E_4=\beta_3 S_4^*$, $E_5=\beta_1 S_4^*-(a+\lambda_1 D_4^*)$, $E_6=\beta_2 S_4^*-b$, $E_7=\beta_3 D_4^*$, $E_8=\lambda_1 D_4^*$, $E_9=\beta_3 S_4^*-c$, $E_{10}=d$, $f=\alpha_2$, $g=\alpha_1$.

We notice that $\lambda_{4,1}=-d=-(\mu+\delta_4)$, $\lambda_{4,2}=E_5=\beta_1 S_4^*-(a+\lambda_1 D_4^*)$, $\lambda_{4,3}=E_6=\beta_2 S_4^*-b$ are eigenvalues of the matrix $J(E_4^*)$, with $\lambda_{4,1}$ is a negative eigenvalue. In addition, $\lambda_{4,2}$ will be a negative eigenvalue if

$$\beta_1\Big(\frac{\mu+\delta_3+\alpha_1}{\beta_3}\Big)-\Big((\mu+\delta_1)+\lambda_1\Big(\frac{A}{\mu+\delta_3+\alpha_1}-\frac{\mu}{\beta_3}\Big)\Big)<0$$

which gives

$$\beta_1\Big(\mu+\delta_3+\alpha_1\Big)^2+\lambda_1\mu\Big(\mu+\delta_3+\alpha_1\Big)<\lambda_1 A\beta_3+\beta_3\Big(\mu+\delta_1\Big)\Big(\mu+\delta_3+\alpha_1\Big),$$

and $\lambda_{4,3}$ which is a negative eigenvalue under the following condition

$$\beta_2\Big(\mu+\delta_3+\alpha_1\Big)<\beta_3\Big(\mu+\delta_2+\alpha_2\Big)$$

Then, it remains to verify the local stability of the matrix $J_1(E_4^*)$, where

$$J_1(E_4^*)=\begin{bmatrix}-E_1 & -E_4\\ E_7 & E_9\end{bmatrix}.$$

Therefore, we have the trace and the determinant of the matrix $J_1(E_4^*)$ are given by

$$\begin{aligned} tr(J_1(E_4^*)) &= -E_1+E_9\\ &= -\Big(\mu+\beta_3\Big(\frac{A}{\mu+\delta_3+\alpha_1}-\frac{\mu}{\beta_3}\Big)\Big)+\Big(\beta_3\Big(\frac{\mu+\delta_3+\alpha_1}{\beta_3}\Big)-\Big(\mu+\delta_3+\alpha_1\Big)\Big)\\ &= -A\frac{\beta_3}{\mu+\alpha_1+\delta_3}, \end{aligned}$$

and

$$\begin{aligned} \det(J_1(E_4^*)) &= -E_1E_9+E_7E_4,\\ &= -\Big(\mu+\beta_3\Big(\frac{A}{\mu+\delta_3+\alpha_1}-\frac{\mu}{\beta_3}\Big)\Big)\Big(\beta_3\Big(\frac{\mu+\delta_3+\alpha_1}{\beta_3}\Big)-(\mu+\delta_3+\alpha_1)\Big)+\Big(\beta_3\Big(\frac{A}{\mu+\delta_3+\alpha_1}\frac{\mu}{\beta_3}\Big)\Big)\\ &\quad\times\Big(\beta_3\Big(\frac{\mu+\delta_3+\alpha_1}{\beta_3}\Big)\Big),\\ &= A\beta_3-\mu^2-\mu\alpha_1-\mu\delta_3. \end{aligned}$$

Next, we study the local stability of E_5^*. The Jacobian matrix at E_5^* is

$$J(E_5^*)=\begin{bmatrix} -F_1 & -F_2 & -F_3 & -F_4 & 0 \\ 0 & F_5 & 0 & 0 & 0 \\ F_6 & F_7 & F_8 & 0 & 0 \\ 0 & 0 & 0 & F_9 & 0 \\ 0 & 0 & g & f & -F_{10} \end{bmatrix},$$

with $F_1=\mu+\beta_2 C_5^*$, $F_2=\beta_1 S_5^*$, $F_3=\beta_2 S_5^*$, $F_4=\beta_3 S_5^*$, $F_5=\beta_1 S_5^*-(a+\lambda_2 C_5^*)$, $F_6=\beta_2 C_5^*$, $E_7=\lambda_2 C_5^*$, $E_8=\beta_2 S_5^*-b$, $F_9=\beta_3 S_5^*-c$, $F_{10}=d$, $f=\alpha_2$, $g=\alpha_1$.

It readily seen that $\lambda_{4,1}=-d=-(\mu+\delta_4)$, $\lambda_{4,2}=F_5=\beta_1 S_5^*-(a+\lambda_2 C_5^*)$ and $\lambda_{4,3}=F_9=\beta_3 S_5^*-c$ are eigenvalues of the matrix $J(E_5^*)$, where $\lambda_{4,1}$ is a negative eigenvalue. We have $\lambda_{4,2}$ is a negative eigenvalue if

$$\beta_1(\mu+\delta_2+\alpha_2)^2+\lambda_2\beta_2 A<(\beta_2(\mu+\delta_1)+\lambda_2\mu)(\mu+\delta_2+\alpha_2).$$

And from which $\lambda_{4,3}$ is a negative eigenvalue under the condition

$$\beta_3(\mu+\delta_2+\alpha_2)<\beta_2(\mu+\delta_3+\alpha_1)$$

Then we study the local stability of the matrix $J_1(E_5^*)$, where

$$J_1(E_5^*)=\begin{bmatrix} -F_1 & -F_3 \\ F_6 & F_80 \end{bmatrix}.$$

Now, we evaluate the determinant and the trace of the matrix $J_1(E_5^*)$ as follows

$$\begin{aligned} tr(J_1(E_5^*)) &= -\Big(\mu+\beta_2\Big(\frac{A}{\mu+\delta_2+\alpha_2}-\frac{\mu}{\beta_2}\Big)\Big)+\Big(\beta_2\Big(\frac{\mu+\delta_2+\alpha_2}{\beta_2}\Big)-(\mu+\delta_2+\alpha_2)\Big), \\ &= -A\frac{\beta_2}{\mu+\alpha_2+\delta_2} \\ det(J_1(E_5^*)) &= -\Big(\mu+\beta_2\Big(\frac{A}{\mu+\delta_2+\alpha_2}-\frac{\mu}{\beta_2}\Big)\Big)\Big(\beta_2\Big(\frac{\mu+\delta_2+\alpha_2}{\beta_2}\Big)-(\mu+\delta_2+\alpha_2)\Big)+\Big(\beta_2\Big(\frac{A}{\mu+\delta_2+\alpha_2}-\frac{\mu}{\beta_2}\Big)\Big) \\ &\quad \times\Big(\beta_2\Big(\frac{\mu+\delta_2+\alpha_2}{\beta_2}\Big)\Big), \\ &= A\beta_2-\mu^2-\mu\alpha_2-\mu\delta_2. \end{aligned}$$

Therefore, the system is locally asymptotically stable if $tr(J_1(E_5^*))$ is negative and $det(J_1(E_5^*))$ is positive.

Finally, we investigate the local stability of E_6^*. The Jacobian matrix at E_6^* is given by

$$J(E_6^*)=\begin{bmatrix} -G_1 & -G_2 & -G_3 & -G_4 & 0 \\ G_5 & G_6 & -G_7 & -G_8 & 0 \\ G_9 & G_{10} & G_{11} & 0 & 0 \\ G_{12} & G_{13} & 0 & G_{14} & 0 \\ 0 & 0 & g & f & -G_{15} \end{bmatrix},$$

where $G_1 = \mu+\beta_1 O_6^*+\beta_2 C_6^*+\beta_3 D_6^*$, $G_2=\beta_1 S_6^*$, $G_3=\beta_2 S_6^*$, $G_4=\beta_3 S_6^*$, $G_5=\beta_1 O_6^*$, $G_6=\beta_1 S_6^*-(a+\lambda_1 D_6^*+\lambda_2 C_6^*$, $G_7=\lambda_2 O_6^*$, $G_8=\lambda_1 O_6^*$, $G_9=\beta_2 C_6^*$, $G_{10}=\lambda_2 C_6^*$, $G_{11}=\beta_2 S_6^*+\lambda_2 O^*-b$, $G_{12}=\beta_3 D_6^*$, $G_{13}=\lambda_1 D_6^*$, $G_{14}=\beta_3 S_6^*+\lambda_1 O_6^*-c$, $G_{15}=d$, $f=\alpha_2$, $g=\alpha_1$.

Clearly, $\lambda_{4,1}=-d=-(\mu+\delta_4)$ is an eigenvalue of the matrix $J(E_5^*)$ which is negative. Then we need to verify the local stability of the matrix $J_1(E_6^*)$, where

$$J_1(E_6^*)=\begin{bmatrix} -G_1 & -G_2 & -G_3 & -G_4 \\ G_5 & G_6 & -G_7 & -G_8 \\ G_9 & G_{10} & G_{11} & 0 \\ G_{12} & G_{13} & 0 & G_{14} \end{bmatrix}.$$

Therefore, the characteristic polynomial of the Jacobian matrix is given by

$$P(\lambda)=\lambda^4+C_{6,1}\lambda^3+C_{6,2}\lambda^2+C_{6,3}\lambda+C_{6,4}.$$

where, $C_{6,1}=G_1-G_6-G_{11}-G_{14}$,

$C_{6,2}=G_{14}(G_6-G_1+G_{11})-G_1G_6+G_2G_5+G_3G_9+G_4G_{12}+G_7G_{10}+G_8G_{13}-G_{11}(G_1-G_6)$,

$C_{6,3}=(G_1-G_6)(G_3G_9+G_7G_{10})-G_{14}(G_2G_5-G_1G_6+G_3G_9+G_7G_{10}-G_{11}(G_1-G_6))-(G_4G_{12}+G_8G_{13})(G_6-G_1+G_{11})-G_9(G_1G_3+G_2G_7)+G_{11}(G_1G_6-G_2G_5)-G_{12}(G_1G_4+G_2G_8+G_{10}(G_3G_5+G_6G_7)+G_{13}(G_4G_5+G_6G_8)$,

$C_{6,4}=(G_4G_{12}+G_8G_{13})(G_2G_5-G_1G_6+G_3G_9+G_7G_{10}-G_{11}(G_1-G_6))-G_{14}((G_1-G_6)(G_3G_9+G_7G_{10})-G_9(G_1G_3+G_2G_7)+G_{11}(G_1G_6-G_1G_5)+G_{10}(G_3G_5+G_6G_7))-G_{12}(G_2(G_4G_5+G_6G_1)-G_1(G_1G_4+G_2G_8)+G_3(G_4G_9+G_8G_{10}))-G_{13}(G_5(G_1G_4+G_2G_8)-G_6(G_4G_5+G_6G_8)+G_7(G_4G_9+G_8G_{10}))+(G_{12}(G_1G_4+$

$G_2G_8)-G_{13}(G_4G_5+G_6G_8))(G_6-G_1+G_{11})).$

Using Lienard-Chipart criteria, the system is locally asymptotically stable if the coefficients $C_{6,1}$, $C_{6,3}$, $C_{6,4}$ are positive and the Hurwitz determinant

$$H_{6,3}=\begin{Vmatrix} C_{6,1} & C_{6,3} & 0 \\ 1 & C_{6,2} & C_{6,4} \\ 0 & C_{6,1} & C_{6,3} \end{Vmatrix}.$$

is positive. Based on the above mentioned discussion we have the following result.

Theorem 3.4.1 *The following assertions are valid.*

1. *The disease free equilibrium E_0 is locally asymptotically stable if the following conditions are satisfied*

$$\begin{cases} \beta_1 A < \mu(\mu+\delta_1), \\ \beta_2 A < \mu(\mu+\delta_2+\alpha_2), \\ \beta_3 A < \mu(\mu+\delta_3+\alpha_1). \end{cases} \tag{3.4.55}$$

2. *The disease free equilibrium E_1 is locally asymptotically stable if*

$$S_1^* > \frac{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)}{\lambda_2\beta_3-\lambda_1\beta_2}. \tag{3.4.56}$$

Moreover, the coefficients $C_{1,1}$, $C_{1,3}$ and the Hurwitz determinant $H_{1,2}$ are positive.

3. *The disease free equilibrium E_2 is locally asymptotically stable if*

$$S_2^* > \frac{\mu+\delta_3+\alpha_1}{\beta_3}. \tag{3.4.57}$$

Furthermore, the coefficients $C_{2,1}$, $C_{2,3}$ and the Hurwitz determinant $H_{2,2}$ are positive.

4. *If the following conditions are satisfied*

$$\begin{cases} \beta_1 S_3^* & < & a \\ \frac{\beta_2(\mu+\delta_1)}{\beta_1}+\frac{\lambda_2 A}{\mu+\delta_1} & < & \frac{\lambda_2\mu}{\beta_1}+(\mu+\delta_2+\alpha_2), \\ \frac{\beta_3(\mu+\delta_1)}{\beta_1}+\frac{\lambda_1 A}{\mu+\delta_1} & < & \frac{\lambda_1\mu}{\beta_1}+(\mu+\delta_3+\alpha_1). \end{cases} \tag{3.4.58}$$

Then, the disease free equilibrium E_3 is locally asymptotically stable.

5. *From which the disease free equilibrium E_4 is locally asymptotically stable, if*

$$\begin{cases} \beta_1\big(\mu+\delta_3+\alpha_1\big)^2+\lambda_1\mu\big(\mu+\delta_3+\alpha_1\big) & < & \lambda_1 A\beta_3+\beta_3\big(\mu+\delta_1\big)\big(\mu+\delta_3+\alpha_1\big), \\ \beta_2\big(\mu+\delta_3+\alpha_1\big) & < & \beta_3\big(\mu+\delta_2+\alpha_2\big), \\ -A\frac{\beta_3}{\mu+\alpha_1+\delta_3} & < & 0, \\ A\beta_3-\mu^2-\mu\alpha_1-\mu\delta_3 & > & 0. \end{cases} \tag{3.4.59}$$

6. *The disease-free equilibrium E_5 is locally asymptotically stable under the following conditions*

$$\begin{cases} \beta_1(\mu+\delta_2+\alpha_2)^2+\lambda_2\beta_2 A & < & (\beta_2(\mu+\delta_1)+\lambda_2\mu)(\mu+\delta_2+\alpha_2), \\ \beta_3(\mu+\delta_2+\alpha_2) & < & \beta_2(\mu+\delta_3+\alpha_1), \\ -A\frac{\beta_2}{\mu+\alpha_2+\delta_2} & < & 0, \\ A\beta_2-\mu^2-\mu\alpha_2-\mu\delta_2 & < & 0. \end{cases} \tag{3.4.60}$$

7. *If the coefficients of Lienard-Chipart criteria $C_{6,1}$, $C_{6,3}$ and the Hurwitz determinant $H_{6,3}$ are positive. Then the endemic equilibrium E_6 is locally asymptotically stable.*

5 Global stability

In this section, we study the global stability of the disease free equilibrium E_0.

Theorem 3.5.1 *The disease-free equilibrium E_0 of model (3.1.1) is globally asymptotically stable.*

Proof. A Lyapunov function for the DFE $E_0=(\frac{A}{\mu},0,0,0,0)$ is defined as

$$V(t)=S_0h(\frac{S(t)}{S_0})+O(t)+C(t)+D(t), \quad \text{for all } t\geq 0, \tag{3.5.61}$$

where the function h is given by

$$X\longmapsto X-1-ln(X).$$

We have V is a positive definite function, $V \in C^1$, and $V(E_0)=0$. Now, we verify that if $V'(t) \leq 0$, for all $t \geq 0$. Thus, differentiating (3.5.61) gives

$$\begin{aligned}
V'(t) &= \Big(1-\frac{S_0}{S}\Big)\frac{dS(t)}{dt}+\frac{d}{dt}\Big(O(t)+C(t)+D(t)\Big),\\
&= \Big(1-\frac{S_0}{S}\Big)\Big(A-\mu S-\beta_1 OS-\beta_2 CS-\beta_3 DS\Big)+\beta_1 SO-(\mu+\delta_1)O-\lambda_1 DO-\lambda_2 CO+\beta_2 SC+\lambda_2 OC\\
&\quad -(\mu+\delta_2+\alpha_2)C+c_1\beta_3 SD+c_1\lambda_1 OD-(\mu+\delta_3+\alpha_1)D,\\
&= \mu S_0\Big(1-\frac{S_0}{S}\Big)\Big(1-\frac{S}{S_0}\Big)+\Big(1-\frac{S_0}{S}\Big)\Big(-\beta_1 OS-\beta_2 CS-\beta_3 DS\Big)+\beta_1 SO\\
&\quad -(\mu+\delta_1)O-\lambda_1 DO-\lambda_2 CO+\beta_2 SC+\lambda_2 OC-(\mu+\delta_2+\alpha_2)C+\beta_3 SD+\lambda_1 OD-(\mu+\delta_3+\alpha_1)D,\\
&= \mu S_0\Big(1-\frac{S_0}{S}\Big)\Big(1-\frac{S}{S_0}\Big)+\beta_1 OS_0+\beta_2 CS_0+\beta_3 DS_0-\beta_1 OS-\beta_2 CS-\beta_3 DS+\beta_1 SO-(\mu+\delta_1)O-\lambda_1 DO\\
&\quad +\beta_2 SC+\lambda_2 OC-(\mu+\delta_2+\alpha_2)C+\beta_3 SD+\lambda_1 OD-(\mu+\delta_3+\alpha_1)D,\\
&= \mu S_0\Big(1-\frac{S_0}{S}\Big)\Big(1-\frac{S}{S_0}\Big)+\beta_1 OS_0+\beta_2 CS_0+\beta_3 DS_0-(\mu+\delta_1)O-(\mu+\delta_2+\alpha_2)C-(\mu+\delta_3+\alpha_1)D,\\
&= \mu S_0\Big(1-\frac{S_0}{S}\Big)\Big(1-\frac{S}{S_0}\Big)+\Big(\beta_1 S_0-(\mu+\delta_1)\Big)O+\Big(\beta_2 S_0-(\mu+\delta_2+\alpha_2)\Big)C+\Big(\beta_3 S_0-(\mu+\delta_3+\alpha_1)\Big)D.
\end{aligned}$$

Noting that

$$\Big(1-\frac{S_0}{S}\Big)\Big(1-\frac{S}{S_0}\Big)<0,$$

due to

$$\begin{cases} \text{if } S_0>S, \text{ then } \frac{S_0}{S}>1 \text{ and } \frac{S}{S_0}<1,\\ \text{if } S_0<S, \text{ then } \frac{S_0}{S}<1 \text{ and } \frac{S}{S_0}>1. \end{cases}$$

Since E_0 is locally asymptotically stable if

$$\begin{cases} \beta_1 A<\mu(\mu+\delta_1),\\ \beta_2 A<\mu(\mu+\delta_2+\alpha_2),\\ \beta_3 A<\mu(\mu+\delta_3+\alpha_1) \end{cases}$$

Hence, $V'(t)<0$ if

$$\begin{cases} K_1=\frac{\beta_1 A}{\mu(\mu+\delta_1)}<1,\\ K_2=\frac{\beta_2 A}{\mu(\mu+\delta_2+\alpha_2)}<1,\\ K_3=\frac{\beta_3 A}{\mu(\mu+\delta_3+\alpha_1)}<1. \end{cases}$$

We have

$V'(S,O,C,D)=0$ if and only if $O=C=D=0$, and $S=S_0=\frac{A}{\mu}$.

Therefore, $V'(S,O,C,D)=0$ is equivalent to $(S,O,C,D)=(\frac{A}{\mu},0,0,0)$.

Applying the Lyapunov Theorem, we deduce that E_0 is globally asymptotically stable if $K_1 < 1$, $K_2 < 1$, and $K_3 < 1$. ■

6 Graphical representations

In this section, we perform a numerical simulation of the results obtained using Matlab.

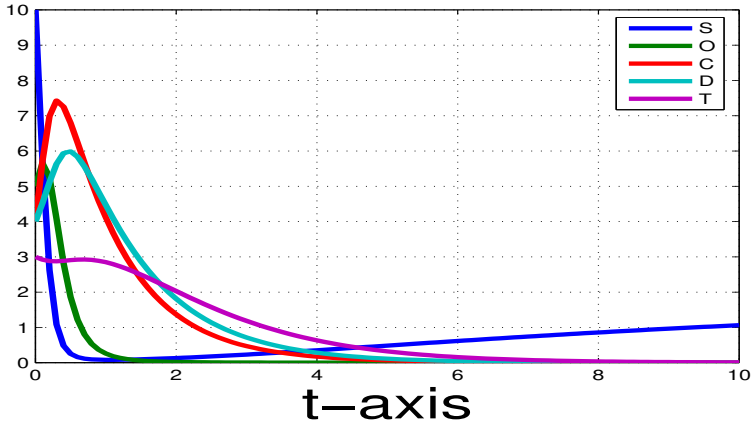

Figure 29: The local asymptotic stability of E_0.

Figure 29 shows that the disease free equilibrium E_0 is locally asymptotically stable for the parameters $A = 0.2$, $\mu = 0.1$, $\alpha_1 = 0.3$, $\alpha_2 = 0.1$, $\beta_1 = 0.63$, $\beta_2 = 0.7$, $\beta_3 = 0.05$, $\delta_1 = 0.2$, $\delta_2 = 0.6$, $\delta_3 = 0.5$, $\delta_4 = 0.7$, $\lambda_1 = 0.34$, $\lambda_2 = 0.08$, with initial conditions $S_0 = 10$, $O_0 = 5$, $C_0 = 4$, $D_0 = 4$, $T_0 = 3$.

The conditions of local stability of E_0 is satisfied

$$\begin{cases} \beta_1 \frac{A}{\mu} = 0,26 < \mu + \delta_1 = 0.3, \\ \beta_2 \frac{A}{\mu} = 0,8 < \mu + \delta_2 + \alpha_2 = 1, \\ \beta_3 \frac{A}{\mu} = 0,1 < \mu + \delta_3 + \alpha_1 = 0.9. \end{cases}$$

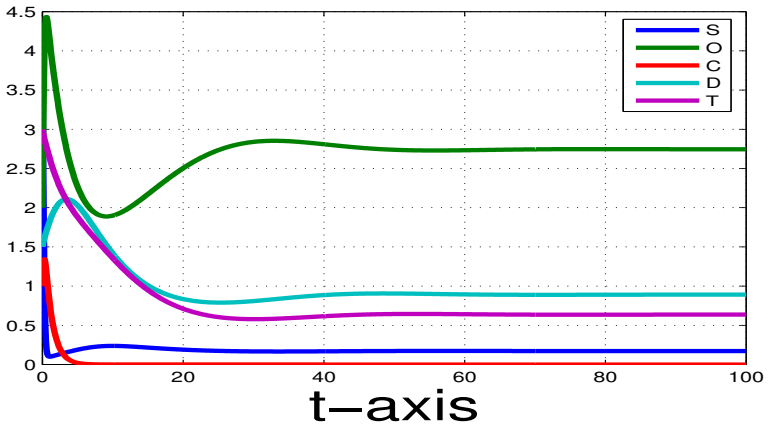

Figure 30: The local asymptotic stability of E_1.

Figure 30 illustrates that system (3.1.1) accepts the disease-free equilibrium E_1 which is locally asymptotically stable for the parameters $A = 0.5$, $\mu = 0.08$, $\alpha_1 = 0.2$, $\alpha_2 = 0.09$, $\beta_1 = 1$, $\beta_2 = 0.7$, $\beta_3 = 0.09$, $\delta_1 = 0.003$, $\delta_2 = 0.9$, $\delta_3 = 0.01$, $\delta_4 = 0.2$, $\lambda_1 = 0.1$, $\lambda_2 = 0.03$ with initial conditions $S_0 = 4$, $O_0 = 2$, $C_0 = 1$, $D_0 = 1.5$, $T_0 = 3$.

The conditions of existence of E_1 are satisfied

$$\begin{cases} \beta_1 = 1 > \beta_3 = 0.09, \quad \delta_3 = 0.01 > \delta_1 = 0.003, \\ \frac{(\mu+\delta_3+\alpha_1)}{\lambda_1} = 2,9 > \frac{\beta_3 A}{\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)} = 0.155, \\ \frac{\beta_1 A}{\lambda_1\mu+\beta_1(\mu+\delta_3+\alpha_1)-\beta_3(\mu+\delta_1)} = 1;72 > \frac{(\mu+\delta_1)}{\lambda_1} = 0.83. \end{cases}$$

The conditions of local stability of E_1 are verified

$$S_1^* = 0,172099 > \frac{\lambda_2(\mu+\delta_3+\alpha_1)-\lambda_1(\mu+\delta_2+\alpha_2)}{\lambda_2\beta_3-\lambda_1\beta_2} = -0.0673.$$
$$C_{1,1} = 2.9053 > 0, \quad C_{1,3} = 0.0710 > 0, \quad H_{1,2} = 1.3761 > 0.$$

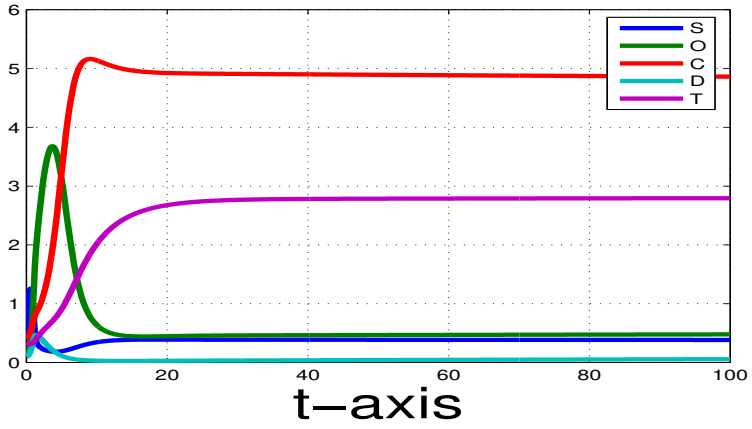

Figure 31: The local asymptotic stability of E_2.

The parameters in Figure 31 indicates that the disease free equilibrium E_2 is locally asymptotically stable when $A = 0.5$, $\mu = 0.08$, $\alpha_1 = 0.2$, $\alpha_2 = 0.09$, $\beta_1 = 1.6$, $\beta_2 = 0.9$, $\beta_3 = 0.07$, $\delta_1 = 0.00003$, $\delta_2 = 0.05$, $\delta_3 = 0.1$, $\delta_4 = 0.2$, $\lambda_1 = 0.08$, $\lambda_2 = 0.06$

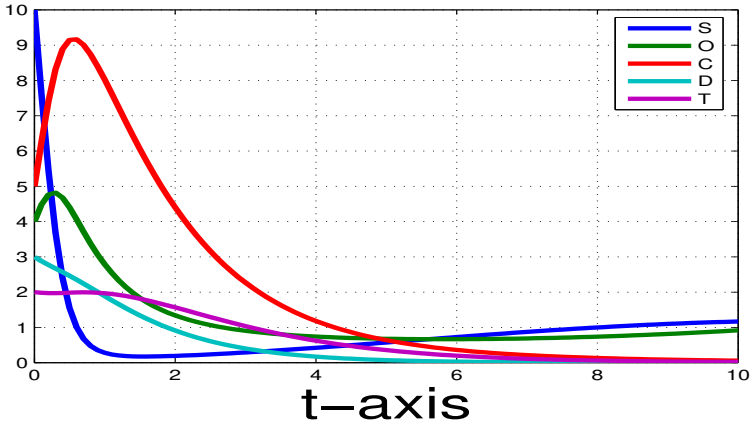

Figure 32: The local asymptotic stability of E_3.

Through figure 32, we can see that the disease free equilibrium E_3 of system (3.1.1) is locally asymptotically stable for the parameters $A = 0.4$, $\mu = 0.1$, $\alpha_1 = 0.3$, $\alpha_2 = 0.1$, $\beta_1 = 0.2$, $\beta_2 = 0.3$, $\beta_3 = 0.01$, $\delta_1 = 0.001$, $\delta_2 = 0.6$, $\delta_3 = 0.5$, $\delta_4 = 0.7$, $\lambda_1 = 0.1$, $\lambda_2 = 0.08$, at initial condi-

tions $S_0 = 10$, $O_0 = 4$, $C_0 = 5$, $D_0 = 3$, $T_0 = 2$.

The condition of existence of E_3 is satisfied

$$\frac{\beta_1 A}{\mu(\mu+\delta_1)} = 7.9207 > 1$$

The conditions of local stability of E_3 are verified

$$\begin{cases} \beta_1 S_3^* = 0.00505 & < & 0.101 \\ \frac{\beta_2(\mu+\delta_1)}{\beta_1} + \frac{\lambda_2 A}{\mu+\delta_1} = 0,4683 & < & \frac{\lambda_2\mu}{\beta_1} + (\mu+\delta_2+\alpha_2) = 0.84. \\ \frac{\beta_3(\mu+\delta_1)}{\beta_1} + \frac{\lambda_1 A}{\mu+\delta_1} = 0,4010 & < & \frac{\lambda_1\mu}{\beta_1} + (\mu+\delta_3+\alpha_1) = 0.95. \end{cases}$$

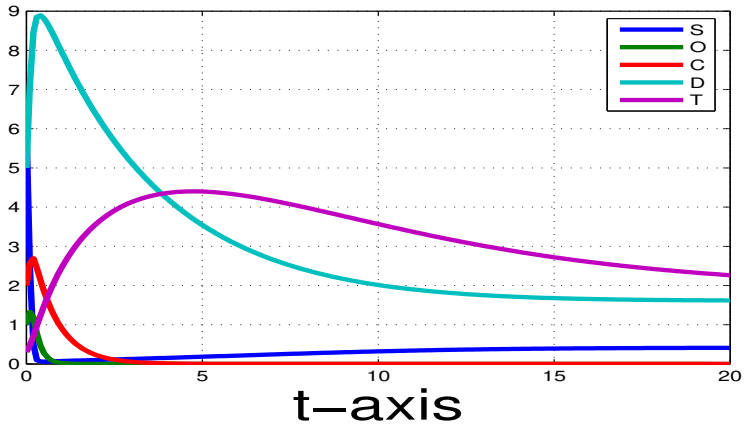

Figure 33: The local asymptotic stability of E_4.

When $A = 0.5$, $\mu = 0.08$, $\alpha_1 = 0.3$, $\alpha_2 = 0.1$, $\beta_1 = 1$, $\beta_2 = 0.7$, $\beta_3 = 0.09$, $\delta_1 = 0.0007$, $\delta_2 = 0.9$, $\delta_3 = 0.01$, $\delta_4 = 0.2$, $\lambda_1 = 0.34$, $\lambda_2 = 0.08$, and for the initial conditions $S_0 = 6$, $O_0 = 1$, $C_0 = 2$, $D_0 = 5$, $T_0 = 0,3$, the disease-free equilibrium E_4 of system (3.1.1) is locally asymptotically stable as shown in figure 33.

The conditions of existence of E_4 is satisfied

$$\frac{\beta_3 A}{\mu(\mu+\delta_3+\alpha_1)} = 15.0862 > 1.$$

The conditions of local stability of E_4 are verified

$$\begin{cases} \beta_1\left(\frac{\mu+\delta_3+\alpha_1}{\beta_3}\right)-\left((\mu+\delta_1)+\lambda_1\left(\frac{A}{\mu+\delta_3+\alpha_1}-\frac{\mu}{\beta_3}\right)\right) & = & -1.0130<0, \\ \beta_2\left(\mu+\delta_3+\alpha_1\right)=0.203 & < & \beta_3\left(\mu+\delta_2+\alpha_2\right)=0.966, \\ -A\frac{\beta_3}{\mu+\alpha_1+\delta_3} & < & 0, \\ A\beta_3-\mu^2-\mu\alpha_1-\mu\delta_3=0.3268 & > & 0. \end{cases}$$

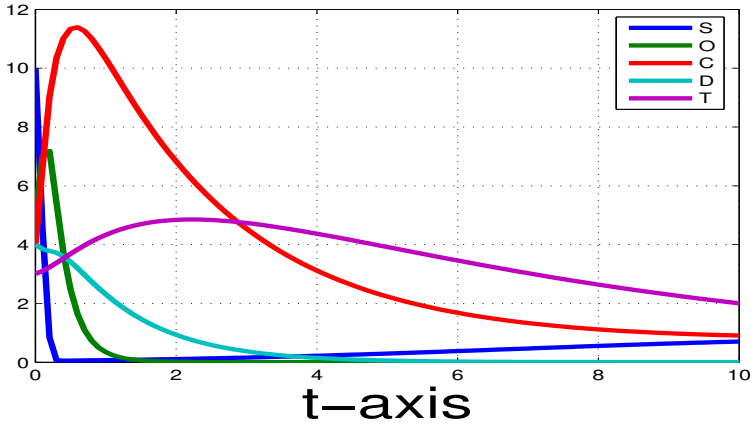

Figure 34: The local asymptotic stability of E_5.

Figure 34 demonstrates that the disease free equilibrium E_5 exhibits local asymptotic stability for the parameters $A=0.5$, $\mu=0.08$, $\alpha_1=0.3$, $\alpha_2=0.1$, $\beta_1=0.6$, $\beta_2=0.6$, $\beta_3=0.001$, $\delta_1=0.7$, $\delta_2=0.3$, $\delta_3=0.5$, $\delta_4=0.1$, $\lambda_1=0.1$, $\lambda_2=0.2$, with the initial conditions $S_0=10$, $O_0=5$, $C_0=4$, $D_0=4$, $T_0=3$.

The conditions of existence of E_5 is satisfied

$$\frac{\beta_2 A}{\mu(\mu+\delta_2+\alpha_2)}=7.8125>1.$$

The conditions of local stability of E_5 are verified

$$\begin{cases} \beta_1(\mu+\delta_2+\alpha_2)^2+\lambda_2\beta_2 A=0,19824 & < & (\beta_2(\mu+\delta_1)+\lambda_2\mu)(\mu+\delta_2+\alpha_2)=0.23132, \\ \beta_3(\mu+\delta_2+\alpha_2)=0,00048 & < & \beta_2(\mu+\delta_3+\alpha_1)=0.528, \\ -A\frac{\beta_2}{\mu+\alpha_2+\delta_2} & < & 0, \\ A\beta_2-\mu^2-\mu\alpha_2-\mu\delta_2=0.2616 & > & 0. \end{cases}$$

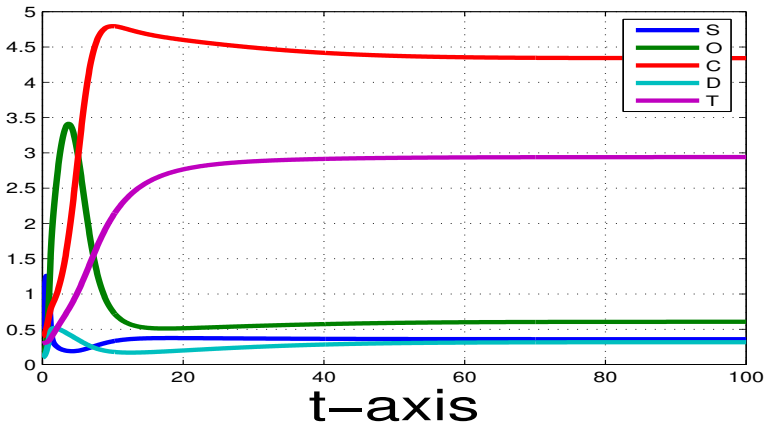

Figure 35: The local asymptotic stability of E_6.

For the parameters $A = 1$, $\mu = 0.1$, $\alpha_1 = 0.01$, $\alpha_2 = 0.1$, $\beta_1 = 0.7$, $\beta_2 = 0.4$, $\beta_3 = 0.1$, $\delta_1 = 0.00002$, $\delta_2 = 0.01$, $\delta_3 = 0.0001$, $\delta_4 = 0.001$, $\lambda_1 = 0.05$, $\lambda_2 = 0.01$, and with $S_0 = 10$, $O_0 = 5$, $C_0 = 4$, $D_0 = 4$, $T_0 = 3$, the endemic equilibrium E_6 of system (3.1.1) is locally asymptotically stable, as in figure 35.

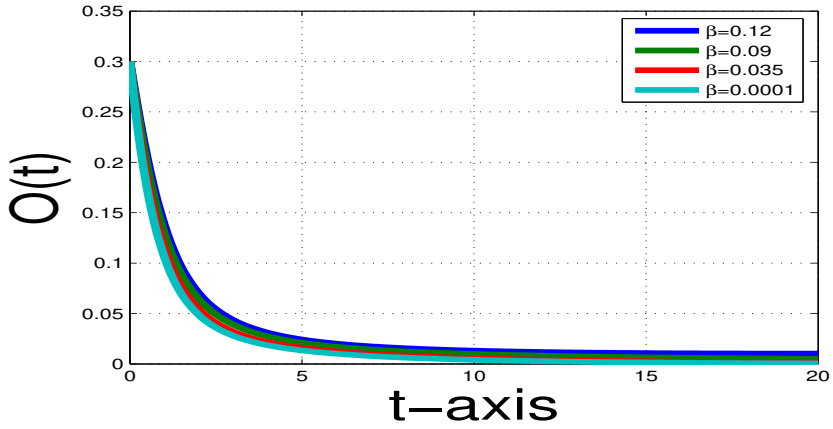

Figure 36: The effect of β_1 on obesity population.

Figure 36 illustrates how decreasing peer pressure β_1 in a population of obese patients leads to a reduction in obesity population. Ultimately eliminating it altogether. This suggests

that manipulating β_1 could be a way to manage and monitor the disease, potentially, leading to its minimization.

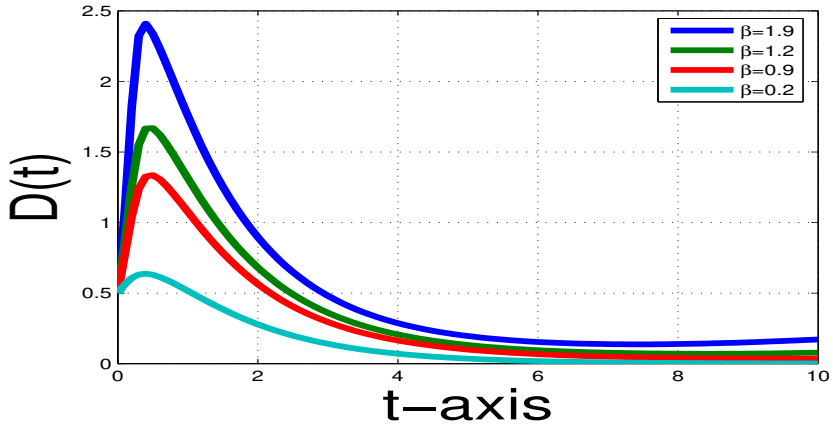

Figure 37: The effect of β_3 on diabetes population.

As shown in figure 37, a decrease in peer pressure among diabetes patients β_3 is associated with a corresponding decrease in diabetes population, eventually resulting in its complete absence. This finding implies that by influencing β_3, we might be able to control and monitor the development of diabetes, potentially minimizing its prevalence.

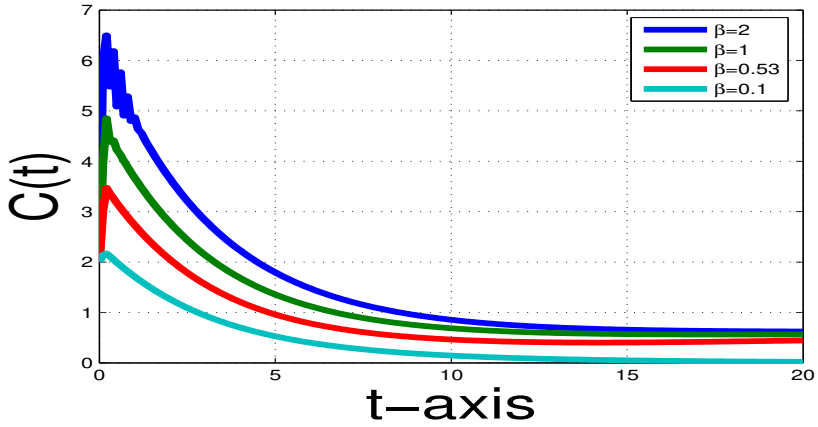

Figure 38: The effect of β_2 on cardiovascular population.

Figure 38 presents that as peer pressure within a population of cardiovascular β_2 diminishes, their cardiovascular obesity also decreases, ultimately disappearing entirely. This strongly suggests that β_2 could be a powerful tool for managing and monitoring obesity, potentially leading to significant reductions in its impact.

Final remarks

Conclusion

In this thesis, we presented a mathematical model describing obesity and its complications in order to reduce the obesity population and to decrease the prevalence of type 2 diabetes, cardiovascular disease, and high blood pressure. We identified social connections as a significant factor contributing to the prevalence and severity of obesity with its complications in the population. These diseases are transmitted indirectly in the population as a result of the acquisition of unhealthy lungial habits. We showed this by studying the effect of these factors on the evolution of the number of people in the obesity population, diabetes population, and cardiovascular population. This is confirmed by the numerical simulation of theoretical results. We conclude that the prevalence of obesity and its complications can be controlled and minimized by reducing the impact of social factors.

Prespectives

In the future work, we will investigate the spatial effect on the obisity outbrerack, which is more challenging and more relevant that the temporal analysis.
The study of this model allows us to better understand the mechanisms of the spread of obesity and to evaluate the impact of different interventions. We can develop new mathematical models to analyze obesity taking into account other parameters such as body mass index (BMI), genetic factors of obesity and gender of individuals... .

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