
Association Between Alcohol Consumption and Hypertension Risk in Adults with Type 2 Diabetes: Insights from a Population-Based Study

Ikram Imken*, Nadia Idrissi Fatmi

LIPIM Laboratory ENSA Khouribga, Sultan Moulay Slimane University, Beni Mellal, Morocco

Email address:

ikramimken@gmail.com (Ikram Imken), nadidriss200133@gmail.com (Nadia Idrissi Fatmi)

*Corresponding author

To cite this article:

Ikram Imken, Nadia Idrissi Fatmi. (2024). Association Between Alcohol Consumption and Hypertension Risk in Adults with Type 2 Diabetes: Insights from a Population-Based Study. *American Journal of Applied Mathematics*, 12(6), 266-285.

<https://doi.org/10.11648/j.ajam.20241206.16>

Received: 17 November 2024; **Accepted:** 5 December 2024; **Published:** 24 December 2024

Abstract: Hypertension, or high blood pressure, is a chronic condition where blood pressure in the arteries remains elevated, leading to severe complications such as heart disease, stroke, and kidney failure. Managing hypertension is particularly challenging when comorbid conditions like type 2 diabetes are present. This paper investigates the combined impact of type 2 diabetes and alcohol consumption on hypertension progression using a deterministic mathematical model. We analyze the model's dynamics, calculate the basic reproduction number (R_0), and perform a sensitivity analysis to identify key parameters influencing the progression of hypertension. Stability analysis shows that the system is stable at both the drinking-free equilibrium and the equilibrium with alcohol consumption. Our findings indicate that reducing alcohol intake significantly lowers the risk of hypertension in diabetic patients. The paper also explores strategies to manage hypertension and diabetes through family support, patient education, and lifestyle modifications such as diet and physical activity. Additionally, Pontryagin's maximum principle is used to optimize these intervention strategies. The results, solved numerically using Matlab, validate the effectiveness of these optimized approaches in controlling hypertension and improving the overall health of patients. Future research may explore the impact of psychological stress on disease progression and incorporate stochastic elements into the model to better reflect real-world variability.

Keywords: Mathematical Model, Hypertension, Alcohol, Diabetes 2, Stability Local and Global, Sensitivity Index, Optimal Control

1. Introduction

Hypertension, also known as high or raised blood pressure, is a condition characterized by persistently elevated pressure in the blood vessels. Blood is transported from the heart to various parts of the body through these vessels. Each time the heart beats, it pumps blood into the vessels, creating blood pressure as the blood pushes against the walls of the arteries. The higher the pressure, the harder the heart must work. It is estimated that 1.28 billion adults aged 30-79 years worldwide have hypertension, with two-thirds of them living in low- and middle-income countries. One of the global targets for noncommunicable diseases is to reduce the prevalence of

hypertension by 33% between 2010 and 2030 [1, 2].

Hypertension is the leading cause of premature cardiovascular disease and death, responsible for millions of disability-adjusted life-years lost each year due to its effects on ischemic heart disease, strokes (both hemorrhagic and ischemic), and chronic kidney disease [1]. In 2010, the global prevalence of hypertension was estimated at 1.4 billion, affecting approximately 31.1% of adults, with projections indicating a significant rise, especially in low- and middle-income countries. However, the prevalence had been declining in high-income countries until the American College of Cardiology/American Heart Association (ACC/AHA) revised their hypertension guidelines in 2017, lowering the threshold

for diagnosis. This change led to a significant increase in the number of people meeting the new criteria for hypertension. As hypertension is a major risk factor for cardiovascular and kidney diseases, it continues to pose a serious global public health challenge [3–7].

Heavy alcohol consumption has been associated with high blood pressure since 1915 [3]. However, light to moderate alcohol intake does not show a strong correlation with hypertension. In fact, it has been linked to improved cardiovascular outcomes when compared to non-drinkers, often represented by a U-shaped curve in various epidemiological studies [4–7]. Due to the cardiovascular benefits associated with reduced atherosclerosis, blood clotting, and platelet aggregation, which in turn lower the risk of coronary artery disease, light to moderate alcohol consumption is often recommended as beneficial and cardioprotective in preventing cardiovascular diseases [8, 9].

Two recent studies, which did not specifically select patients with diabetes mellitus, have highlighted through meta-analyses a linear relationship between the amount of alcohol consumed and the degree of hypertension [10, 11]. New research published in the *Journal of the American Heart Association*, an open-access journal of the American Heart Association, suggests that consuming eight or more alcoholic drinks per week may increase the risk of high blood pressure in adults with Type 2 diabetes [12].

Researchers investigated the relationship between alcohol consumption and blood pressure in over 10,000 adults with Type 2 diabetes (average age 63, 61% male), all of whom participated in the Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial. This large-scale, long-term trial, conducted from 2001 to 2005 across 77 centers in the U.S. and Canada, aimed to compare different treatment strategies for reducing heart disease risk in adults with Type 2 diabetes. Alcohol consumption was categorized as none, light (1-7 drinks per week), moderate (8-14 drinks per week), and heavy (15 or more drinks per week). One alcoholic drink was defined as a 12-ounce beer, a 5-ounce glass of wine, or 1.5 ounces of hard liquor. Participants self-reported their weekly alcohol intake via a questionnaire at study enrollment. Blood pressure was classified according to the 2017 American College of Cardiology/American Heart Association guidelines as normal (below 120/80 mm Hg), elevated (120-129/80 mm Hg), Stage 1 high blood pressure (130-139/80-89 mm Hg), or Stage 2 high blood pressure (140/90 mm Hg or higher). Since most participants were already taking one or more blood pressure medications, the blood pressure readings were adjusted to account for the effects of these medications and to estimate the underlying level of hypertension.

1. Light drinking was not linked to elevated blood pressure or any stage of high blood pressure.
2. Moderate drinking was associated with a 79% higher likelihood of elevated blood pressure, a 66% higher likelihood of Stage 1 high blood pressure, and a 62% higher likelihood of Stage 2 high blood pressure.
3. Heavy drinking was associated with a 91% higher likelihood of elevated blood pressure, a 149% higher

likelihood of Stage 1 high blood pressure (a 2.49-fold increase), and a 204% higher likelihood of Stage 2 high blood pressure (a 3.04-fold increase).

Excessive alcohol consumption is identified as a risk factor for elevated blood pressure, particularly among individuals with Type 2 diabetes, who already face a heightened susceptibility to hypertension. The American Heart Association advises moderate alcohol intake, if any, emphasizing the importance for individuals to consider its potential health implications.

Several mathematicians have worked to understand the dynamics of alcohol consumption and its impact on both individuals and society, aiming to reduce harm and minimize the number of addicted drinkers. For instance, S. H. Ma et al. [13] modeled alcoholism as a contagious disease and used optimal control to study their mathematical model, incorporating awareness programs and time delays. S. Sharma et al. [14] developed a model of alcohol abuse, examining the existence and stability of drinking-free and endemic equilibria, as well as performing sensitivity analysis of R_0 . B. Benedict [15] employed an SIR-type model to analyze alcoholism, calculating the reproductive number and discussing the stability of two equilibrium states. H. F. Huo et al. [16] proposed a new social epidemic model to depict alcoholism with media coverage, demonstrating its effectiveness in encouraging people to quit drinking. I. K. Adu et al. [17] utilized a non-linear SHTR mathematical model to study the dynamics of drinking epidemics. IMKEN [18] studied a unique mathematical model to describe the dynamics of COVID-19 transmission in diabetic populations, introducing optimal controls in 2023. Additionally, IMKEN [19] developed a novel model to investigate alcohol consumption among diabetics, particularly in the context of anti-diabetic treatments, highlighting that moderate consumption is beneficial for diabetics without complications.

This is the first mathematical study based on that explores the link between alcohol consumption and hypertension in individuals with Type 2 diabetes [12], aiming to clarify the risks associated with alcohol in a population that had been living with Type 2 diabetes for an average of 10 years before participating in the study. In addition to their diabetes, these individuals were at heightened risk for cardiovascular events due to pre-existing cardiovascular disease or signs of potential cardiovascular issues. Specifically, the key contributions of this paper are summarized as follows:

1. Proposing a novel compartmental model based on a comprehensive study. This is the first large-scale study to specifically examine the relationship between alcohol consumption and hypertension in adults with Type 2 diabetes. While previous research has indicated a link between heavy alcohol consumption and high blood pressure, the association between moderate alcohol consumption and hypertension remained unclear [12].
2. Modeling individuals with Type 2 diabetes for 10 years, who are at risk of cardiovascular disease, alongside moderate drinkers, heavy drinkers, and

hypertensive patients (Stage 1 and Stage 2). The four compartments are represented through a system of differential equations to analyze the temporal dynamics.

3. Using the Routh-Hurwitz criteria and constructing Lyapunov functions to determine the local and global stability of both alcohol-free and alcohol consumption equilibria.
4. Performing a sensitivity analysis of model parameters to identify those that have the most significant impact on the reproduction number R_0 .

The structure of the paper is as follows. In section 2, we derive the formulation of the model along with its basic properties. section 3 presents the equilibrium of the proposed model, discusses its stability, and provides some numerical simulations. The issue of parameter sensitivity is explored in section 4. Finally, in section 5, we introduce the optimal controls problem for the proposed model, present results related to the optimal controls, and use Pontryagin’s maximum principle to characterize these controls, concluding with numerical simulations in MATLAB at the end of the

section.

2. Mathematical Model Formulation

2.1. Model Representation

We introduce a continuous non-linear model, denoted as $D_2M_{D_2}H_{D_2}H_{yp}$, designed to explore the dynamics between various categories of drinkers who are living with Type 2 diabetes and psychological disorders, such as stress and depression [20]. The model divides the population into four distinct compartments: individuals with Type 2 diabetes who are also experiencing stress and are at risk of cardiovascular diseases, denoted as $D_2(t)$; moderate alcohol consumers, represented by $M_{D_2}(t)$; heavy alcohol consumers, denoted by $H_{D_2}(t)$; and those who develop hypertension as a result of the interaction between alcohol consumption and Type 2 diabetes, denoted as $H_{yp}(t)$.

The graphical representation of the proposed model is illustrated in Figure 1.

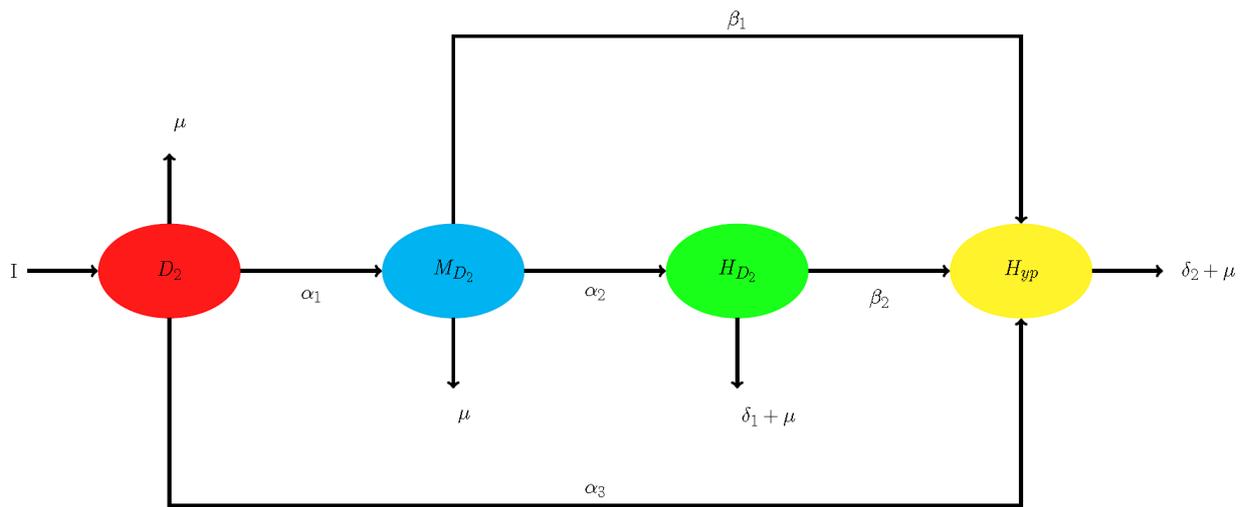


Figure 1. Compartments Model.

We examine the following system of four non-linear differential equations

$$\begin{cases} \frac{dD_2}{dt} = I - (\alpha_3 + \mu)D_2(t) - \alpha_1 \frac{D_2(t)M_{D_2}(t)}{N} \\ \frac{dM_{D_2}}{dt} = \alpha_1 \frac{D_2(t)M_{D_2}(t)}{N} - (\alpha_2 + \beta_1 + \mu)M_{D_2}(t) \\ \frac{dH_{D_2}}{dt} = \alpha_2 M_{D_2}(t) - (\beta_2 + \delta_1 + \mu)H_{D_2}(t) \\ \frac{dH_{yp}}{dt} = \beta_2 H_{D_2}(t) + \beta_1 M_{D_2}(t) + \alpha_3 D_2(t) - (\mu + \delta_2)H_{yp}(t) \end{cases}$$

Where $D_2(0) \geq 0, M_{D_2}(0) \geq 0, H_{D_2}(0) \geq 0, H_{yp}(0) \geq 0$ are the given initial states.

Personnes souffrant de diabète, de troubles psychologiques et de risque cardiovasculaire $D_2(t)$

The compartment D_2 encapsulates individuals in adolescence and adulthood who, due to lifestyle factors such as

stress and anxiety, are predisposed to psychological disorders and are at heightened risk of developing cardiovascular diseases [12]. This population may also engage in alcohol consumption influenced by these stressors. The size of this

compartment grows through recruitment at a rate denoted by I and diminishes due to effective interactions with individuals who consume alcohol in controlled amounts at a rate α_1 , as well as through natural mortality at a rate μ . Additionally, it increases at a rate α_3 when individuals with Type 2 diabetes transition into a state of hypertension driven by psychological distress.

$$\frac{dD_2(t)}{dt} = I - \alpha_1 \frac{D_2(t)M_{D_2}}{N} - (\alpha_3 + \mu)D_2(t) \quad (1)$$

Moderate Drinkers M_{D_2} :

$$\frac{dD_2(t)}{dt} = \alpha_1 \frac{D_2(t)M_{D_2}}{N} - (\alpha_2 + \beta_1 + \mu)M_{D_2}(t) \quad (2)$$

This compartment M_{D_2} consists of diabetic individuals who consume alcohol in a controlled or occasional manner, typically during specific events or situations, often unnoticed within their social environment. Controlled alcohol consumption is categorized as drinking between 8 – 14 units per week [12]. This Studies show that such consumption is linked to an increased risk of progressing to stage 1 hypertension (β_1), transitioning to excessive alcohol use (β_2), and natural mortality (μ).

Heavy Drinkers H_{D_2} :

$$\frac{dH_{D_2}(t)}{dt} = \alpha_2 M_{D_2}(t) - (\alpha_3 + \beta_2 + \delta_1 + \mu)H_{D_2}(t) \quad (3)$$

The compartment H_{D_2} comprises individuals classified as excessive alcohol consumers, defined as those consuming 14 to 26 units per week, and struggling with alcohol dependency. This compartment decreases at a rate β_2 , representing the transition of some individuals to a state of high blood pressure [12]. It also diminishes due to mortality caused by psychological disorders linked to alcohol addiction (δ_1) and natural mortality (μ). Additionally, it increases at a rate α_2 .

Person with High Blood Pressure H_{yp} :

$$\frac{dH_{D_{yp}}(t)}{dt} = \beta_2 H_{D_2}(t) + \beta_1 M_{D_2}(t) + \alpha_3 D_2(t) - (\mu + \delta_2)H_{yp}(t) \quad (4)$$

$$\frac{dD_2(t)}{dt} \times \exp\left(\int_0^t A(s)ds\right) + A(t)D_2(t) \times \int_0^t (A(s)ds) \geq 0$$

Implies that

$$\frac{d}{dt} \left(D_2(t) \exp\left(\int_0^t A(s)ds\right) \right) \geq 0 \quad (8)$$

Integrating (8) between 0 and t gives

$$D_2(t) \geq D_2(0) \exp\int_0^t (-A(s))ds \quad (9)$$

So, the solution $D_2(t)$ is positive.

Similarly, from the second equation system (1), we have:

$$M_{D_2}(t) \geq M_{D_2}(0) \exp\int_0^t (-B(s))ds \quad (10)$$

The compartment H_{yp} represents the number of individuals with high blood pressure. This group decreases due to natural mortality (μ) and deaths caused by cardiovascular diseases [21, 22], which are a consequence of hypertension. The total population size at any given time is represented by $N(t)$.

$$N(t) = D_2(t) + M_{D_2}(t) + H_{D_2}(t) + H_{yp}(t)$$

2.2. Primary Properties

2.2.1. Positivity of Model Solutions

To ensure the biological relevance of the model, it is essential to demonstrate that all solutions of the system (1) remain positive for all, provided the initial conditions are positive. This guarantees that the model variables, representing population sizes or concentrations, do not take non-physical negative values over time.

Theorem 2.1. If $D_2(0) \geq 0$ and $H_{yp}(0) \geq 0$, then the solution of system equations (1) $D_3(t), M_{D_2}(t), H_{D_2}(t), H_{yp}(t)(t)$ are positive for all $t > 0$. *Proof*

$$\begin{aligned} \frac{dD_2(t)}{dt} &= I - (\alpha_3 + \mu)D_2(t) - \alpha_1 \frac{D_2(t)M_{D_2}(t)}{N} \\ &= I - \left[(\alpha_3 + \mu) + \alpha_1 \frac{M_{D_2}(t)}{N} \right] D_2(t) \end{aligned} \quad (5)$$

We consider

$$A(t) = (\alpha_3 + \mu) + \alpha_1 \frac{M_{D_2}(t)}{N} \quad (6)$$

It follows from the equation (5) that

$$\frac{dD_D(t)}{dt} + A(t)D_2(t) \geq 0 \quad (7)$$

We multiply inequality (7) by

$$\exp\left(\int_0^t A(s)ds\right)$$

We find:

Where:

$$B(t) = \alpha_1 \frac{D_2(t)}{N} - (\alpha_2 + \beta_2 + \mu) \tag{11}$$

From, the other equations of system (1), we have.

$$H_{D_2}(t) \geq H_{D_2}(0) \exp\left(-(\beta_2 + \delta_1 + \mu)t\right) \geq 0 \tag{12}$$

$$H_{yp}(t) = H_{yp}(0) \exp\left(-(\mu + \delta_2)t\right) \geq 0 \tag{13}$$

Therefore, we can see that $D_2(t) \geq 0, M_{D_2}(t) \geq 0, H_{D_2}(t), H_{yp}(t) \geq 0 \forall t \geq 0$ this completes the proof

2.2.2. Invariant Region

Lemma 2.1. All feasible Solution $D_2(t), M_{D_2}(t), H_{D_2}(t)$ and $H_{yp}(t)$ of system equation (1) are bounded by the region.

$$\Omega = \left\{ (D_2, M_{D_2}, H_{D_2}, H_{yp}) \in \mathbb{R}_+^4 : D_2 + M_{D_2} + H_{D_2} + H_{yp} \leq \frac{I}{\mu} \right\} \tag{14}$$

Proof From the system (1)

$$\begin{aligned} \frac{dN(t)}{dt} &= \frac{dD_2(t)}{dt} + \frac{dM_{D_2}(t)}{dt} + \frac{dH_{D_2}(t)}{dt} + \frac{dH_{yp}(t)}{dt} \\ &= I - \mu N(t) - \delta_1 H_{D_2}(t) - \delta_2 H_{yp}(t) \end{aligned} \tag{15}$$

Implies that

$$\frac{dN(t)}{dt} \leq I - \mu N(t).$$

It follows that

$$N(t) \leq \frac{I}{\mu} + N(0)e^{-\mu t} \tag{16}$$

Where $N(0)$ is the initial value of total number of people, this

$$\lim_{t \rightarrow \infty} \sup N(t) \leq \frac{I}{\mu}$$

Then

$$D_2(t) + M_{D_2} + H_{D_2} + H_{yp}(t) \leq \frac{I}{\mu}$$

Hence, for the analysis of model (1). We get the region which is given by the set

$$\Omega = \left\{ (D_2, M_{D_2}, H_{D_2}, H_{yp}) \in \mathbb{R}_+^4 : D_2 + M_{D_2} + H_{D_2} + H_{yp} \leq \frac{I}{\mu} \right\}$$

A positively invariant set for (1) must be identified, so we need to examine the dynamics of the system within the non-negative solution set Ω .

3. Equilibrium Points and Stability Evaluation

The first three equations in the system (1) are independent of the variable H_{yp} . Therefore, the dynamics of the full system (1) are equivalent to the dynamics of the reduced system given by:

$$\begin{cases} \frac{dD_2}{dt} = I - (\alpha_3 + \mu)D_2(t) - \alpha_1 \frac{D_2(t)M_{D_2}(t)}{N} \\ \frac{dM_{D_2}}{dt} = \alpha_1 \frac{D_2(t)M_{D_2}(t)}{N} - (\alpha_2 + \beta_1 + \mu)M_{D_2}(t) \\ \frac{dH_{D_2}}{dt} = \alpha_2 M_{D_2}(t) - (\beta_2 + \delta_1 + \mu)H_{D_2} \end{cases} \tag{17}$$

3.1. Equilibrium Point

The conventional approach is employed to analyze the model (17). This model yields two equilibrium points: one corresponding to a drinking-free state and the other representing a drinking-present state. These equilibrium points are obtained by setting the right-hand sides of equations (1) - (3) equal to zero.

The drinking-free equilibrium, $P_0 \left(\frac{I}{\alpha_3 + \mu}, 0, 0 \right)$, occurs in the absence of alcohol consumption, i.e., when $M_{D_2} = H_{D_2} = 0$. The equilibrium with alcohol consumption, $P_1 (D_2^*, M_{D_2}^*, H_{D_2}^*)$, is reached when individuals are drinking, i.e., when $M_{D_2} \neq 0$ and $H_{D_2} \neq 0$, where

$$D_2^* = \frac{I}{(\alpha_3 + \mu)R_0}$$

$$M_{D_2}^* = \frac{I(\alpha_3 + \mu)(R_0 - 1)}{\alpha_1\mu}$$

$$H_{D_2}^* = \frac{\alpha_2(\alpha_3 + \mu)(R_0 - 1)I}{\alpha_1(\beta_2 + \delta_1 + \mu)\mu}$$

$$J(P) = \begin{pmatrix} -(\alpha_3 + \mu) - \alpha_1 \frac{M_{D_2}}{N} & -\alpha_1 \frac{D_2}{N} & 0 \\ \alpha_1 \frac{M_{D_2}}{N} & -(\alpha_2 + \beta_1 + \mu) + \alpha_1 \frac{D_2}{N} & 0 \\ 0 & \alpha_2 & -(\beta_2 + \delta_1 + \mu) \end{pmatrix}$$

The Jacobian matrix corresponding to the equilibrium in the absence of drinking is given by

$$J(P_0) = \begin{pmatrix} -(\alpha_3 + \mu) & \frac{-\alpha_1 I}{N(\mu + \alpha_3)} & 0 \\ 0 & -(\alpha_2 + \beta_1 + \mu) + \frac{\alpha_1 I}{N(\alpha_3 + \mu)} & 0 \\ 0 & \alpha_2 & -(\beta_2 + \delta_1 + \mu) \end{pmatrix}$$

The characteristic equation of this matrix is expressed as $\det(J(P_0) - \lambda I_3) = 0$, where I_3 is a 3×3 identity matrix. Consequently, the eigenvalues of $J(P_0)$ are given by

$$\begin{cases} X_1 = -(\beta_2 + \delta_1 + \mu) \\ X_2 = -(\alpha_3 + \mu) \\ X_3 = -(\alpha_2 + \beta_1 + \mu)(1 - R_0) \end{cases}$$

Therefore, all the eigenvalues of the characteristic equation are real and negative if $R_0 < 1$. Thus, we conclude that the equilibrium in the absence of drinking is locally asymptotically stable when $R_0 < 1$, and it becomes unstable if $R_0 > 1$.

3.2.2. Drinking Present Equilibrium

Theorem 3.2. The equilibrium where drinking is present, P_1^* , is locally asymptotically stable if $R_0 > 1$ and unstable otherwise.

Proof We denote $P_1 (D_2^*, M_{D_2}^*, H_{D_2}^*)$ as the equilibrium corresponding to the presence of drinking in system (16), where $D_2^* \neq 0$, $M_{D_2}^* \neq 0$, and $H_{D_2}^* \neq 0$. The Jacobian matrix is given by...

$$J(P_1^*) = \begin{pmatrix} -(\alpha_3 + \mu) - \alpha_1 \frac{M_{D_2}^*}{N} & -\alpha_1 \frac{D_2^*}{N} & 0 \\ \alpha_1 \frac{M_{D_2}^*}{N} & -(\alpha_2 + \beta_1 + \mu) + \alpha_1 \frac{D_2^*}{N} & 0 \\ 0 & \alpha_2 & -(\beta_2 + \delta_1 + \mu) \end{pmatrix}$$

R_0 is the basic reproduction number that measures the average number of new drinkers generated by single drinker in a population of potential drinkers. The value of R_0 will indicate the epidemic could occur or not, the reproduction basic number can be determined by using the next generation matrix method formulated in (Drissche et al (2022))

$$R_0 = \frac{\alpha_1\mu}{(\alpha_3 + \mu)(\alpha_2 + \beta_1 + \mu)N}$$

3.2. Local Stability Analysis

The stability properties of the equilibria P_0 and P_1 are now investigated.

3.2.1. The Drinking-Free Equilibrium

This section focuses on assessing the local stability of the drinking-free equilibrium.

Theorem 3.1. The equilibrium in the absence of drinking, $P_0 \left(\frac{I}{\alpha_3 + \mu}, 0, 0 \right)$, of system (17) is asymptotically stable if $R_0 \leq 1$ and unstable if $R_0 > 1$.

Proof The jacobain matrix at P is given by

Where:

$$\begin{cases} D_1^* = \frac{I}{(\alpha_3 + \mu)R_0} \\ M_{D_2}^* = \frac{I(\alpha_3 + \mu)(R_0 - 1)}{\alpha_1\mu} \\ H_{D_2}^* = \frac{\alpha_2(\alpha_3 + \mu)(R_0 - 1)I}{\alpha_1(\beta_2 + \delta_1 + \mu)\mu} \end{cases}$$

We observe that the characteristic equation $P(X)$ of $J(P_1^*)$ has an eigenvalue $X_1 = -(\beta_2 + \delta_1 + \mu)$, whose real part is negative. Therefore, to determine the stability of the drinking-present equilibrium of model (17), we analyze the roots of the following equation $P(Q)$:

$$P(Q) = Q^2 + a_1Q + b_1$$

where

$$\begin{cases} a_1 = \left((\alpha_3 + \mu) + (\alpha_2 + \beta_2 + \mu) + \frac{\alpha_1}{N}(M_D^* - D_2^*) \right) \\ b_1 = N(\alpha_2 + \beta_1 + \mu)(\alpha_3 + \mu)(R_0 - 1) \end{cases}$$

by Routh-Hurwitz Criterrin, the system (17) is locally asymptotically stable if $a_1 > 0$ and $b_1 > 0$.

Therefore, the equilibrium P_1^* of system (17), where drinking is present, is locally asymptotically stable.

3.3. Global Stability

3.3.1. Global Stability of the Drinking-free Equilibrium

To demonstrate that system (17) is globally asymptotically stable, we apply Lyapunov function theory to both the non-drinking equilibrium and the alcohol-consuming equilibrium. Initially, we establish the global stability of the non-drinking equilibrium P_0 when $R_0 \leq 1$.

Theorem 3.3. the drinking-free equilibrium P_0 is globally asymptotically stable if $R_0 < 1$ and unstable otherwise.

Proof Consider the following Lyapunov function.

$$v(D_2, M_{D_2}, H_{D_2}, H_{yp}) = M_{D_2}$$

The derivate of $v(D_2, M_{D_2}, H_{D_2}, H_{yp})$ with respect to t gives

$$\begin{aligned} \frac{dv}{dt} &= \frac{dM}{dt} \\ &= \alpha_1 \frac{D_2 M_{D_2}}{N} - 2ig(\alpha_2 + \beta_1 + \mu)M_{D_2} \\ &= M_{D_2} \left(\alpha_2 \frac{D_2}{N} - (\alpha_2 + \beta_1 + \mu) \right) \\ &= M_{D_2} \left(\alpha_1 \frac{I}{N(\alpha_3 + \mu)} - (\alpha_2 + \beta_1 + \mu) \right) \\ &= M_{D_2} \left(\alpha_1 \frac{I}{N(\alpha_3 + \mu)(\alpha_2 + \beta_1 + \mu)} - 1 \right) \\ &= M_{D_2}(R_0 - 1) \end{aligned}$$

So, $\frac{dv}{dt} \leq 0$ if $R_0 \leq 1$

Furthermore, $\frac{dv}{dt} = 0$ if and only if $M_{D_2} = 0$. Hence, by LaSalle’s invariance principle [23], P_0 is globally asymptotically stable.

3.3.2. Global Stability of the Alcohol-Consuming Equilibrium

The final result of the global stability of P_1^* in this section is as follows:

Theorem 3.4. The alcohol-consuming equilibrium point P_1^* is globally asymptotically stable if $R_0 > 1$.

Proof. let as construct the Lyapunov function as follows:

$V: \Gamma \rightarrow \mathbb{R}$ where $\Gamma = \{(D_2, M_{D_2}), D_2 > 0, M_{D_2} > 0\}$

$$V(D_2, M_{D_2}) = D_2 + D_2^* \ln\left(\frac{D_2^*}{D_2}\right) + M_{D_2} + M_{D_2}^* \ln\left(\frac{M_{D_2}^*}{M_{D_2}}\right)$$

Differentiating V

$$\begin{aligned} \implies \dot{V}(D_2, M_{D_2}) &= \frac{\partial V(D_2, M_{D_2})}{\partial D_2} dD_2 + \frac{\partial V(D_2, M_{D_2})}{\partial M_{D_2}} dM_{D_2} \\ &= \left(1 - \frac{D_2^*}{D_2}\right) dD_2 + \left(1 - \frac{M_{D_2}^*}{M_{D_2}}\right) dM_{D_2} \\ &= \left(\frac{D_2 - D_2^*}{D_2}\right) \left(I - (\alpha_3 + \mu)D_2 - \alpha_1 \frac{D_2 M_{D_2}}{N} \frac{M_{D_2} - M_{D_2}^*}{M_{D_2}} \left(\frac{\alpha D_2 M_{D_2}}{N} - (\alpha_2 + \beta_1 + \mu)M_{D_2}\right)\right) \end{aligned}$$

So

$$\begin{aligned} \dot{V}(D_2, M_{D_2}) &= I - \frac{ID_2^*}{D_2} - D_2(\alpha_3 + \mu) + D_2^*(\alpha_3 + \mu) - \frac{\alpha_1 D_2 M_{D_2}}{N} + \frac{\alpha_1 D_2^* M_{D_2}}{N} + \frac{\alpha_1 M_{D_2} D_2}{N} \\ &\quad - \frac{\alpha_1 M_{D_2}^* D_2}{N} - M_{D_2}(\alpha_2 + \beta_1 + \mu) + M_{D_2}^*(\alpha_2 + \beta_1 + \mu) \end{aligned} \tag{18}$$

Let K be the positive term and L the negative term of (18) that is

$$\begin{aligned} K &= I + D_2^*(\alpha_3 + \mu) + \frac{\alpha_1 D_2^* M_{D_2}}{N} + \frac{\alpha_1 D_2 M_{D_2}}{N} + M_{D_2}^*(\alpha_2 + \beta_1 + \mu) \\ L &= \frac{D_2^*}{D_2} + D_2(\alpha_3 + \mu) + \frac{\alpha_1 D_2 M_{D_2}}{N} + \frac{\alpha_1 M_{D_2}^* D_2}{N} + M_{D_2}(\alpha_2 + \beta_1 + \mu) \end{aligned}$$

Then,

$$\frac{dV}{dt} = K - L$$

If $K < L$, then

$$\frac{dV}{dt} < 0$$

and

$$\frac{dV}{dt} = 0$$

if and only if $D_2^* = D_2$ and $M_{D_2}^* = M_{D_2}$.

The largest compact invariant set in $(D_2, M_{D_2}) \in \mathbb{R}_+^2$,

$$\frac{dV}{dt} = 0$$

is the single ton of P_1^* It implies that P_1^* globally asymptotically stable in Γ if $K < L$ by Lasalle’s invariant principle [24].

3.4. Numerical Simulation

Table 1. Parameters and values for the cases $R_0 > 1$ et $R_0 < 1$.

Parameter	Description	$R_0 > 1$	$R_0 < 1$
μ	Natural Mortality	0.05	0.045
β_1	Rate of hypertensive people due to moderate consumption	0.1	0.2
β_2	Rate of moderate drinkers becoming heavy drinkers	0.1	0.2
α_1	Contact rate with moderate drinkers	0.66	0.44
α_2	Rate of heavy drinkers	0.02	0.1
α_3	Rate of hypertensive persons due to psychological disorder	0.01	0.1
δ_1	Cardiovascular disease rate	0.02	0.02
δ_2	Mortality due to psychological disorder	0.03	0.03
I	Incidence	510	510

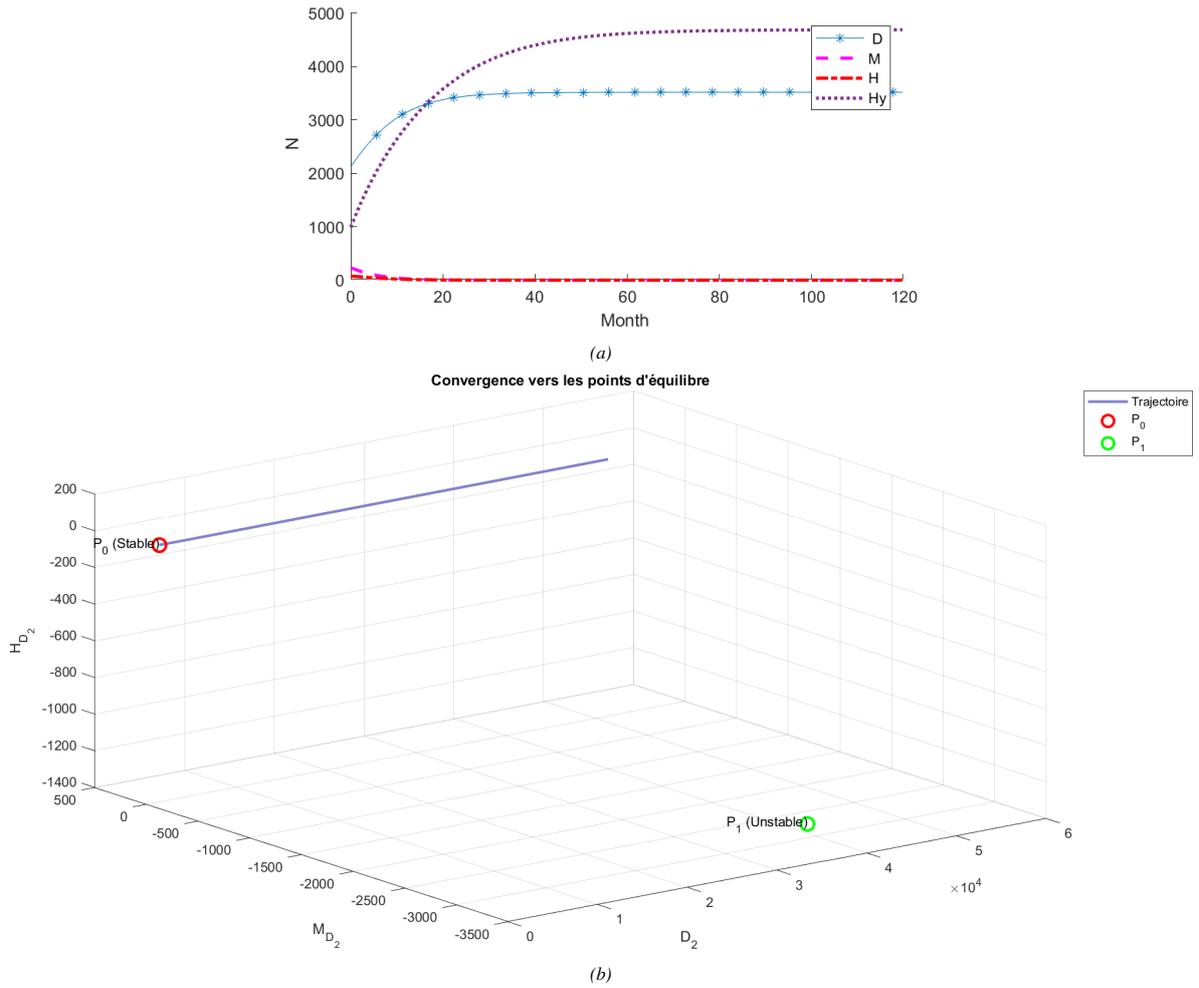


Figure 2. When $R_0 < 1$ the Drinking-absent equilibrium P_0 is locally asymptotically stable.

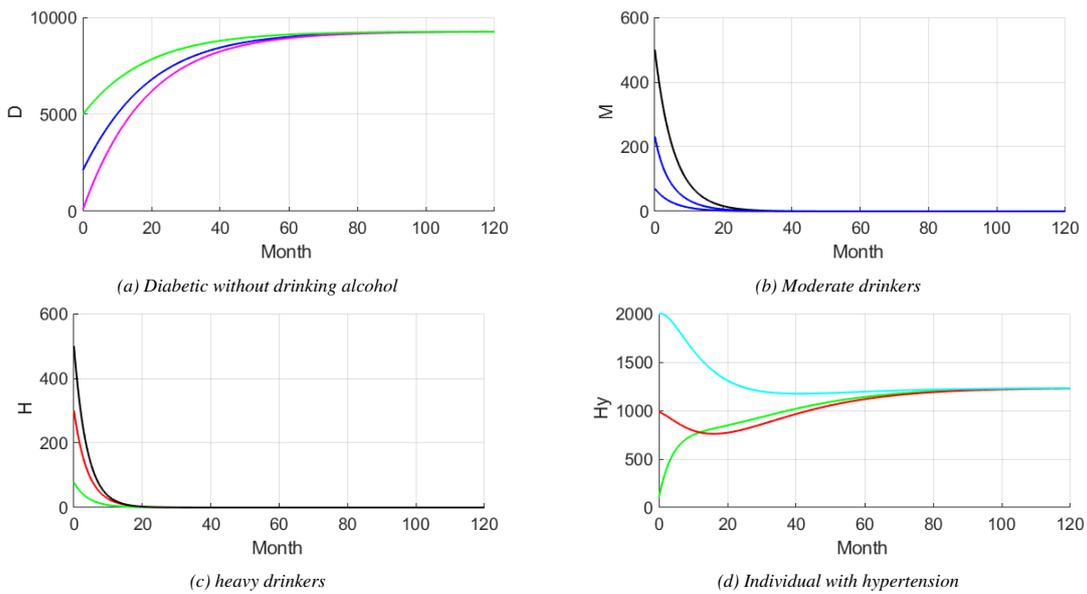


Figure 3. When $R_0 < 1$ the Drinking-absent equilibrium P_0 is locally asymptotically stable.

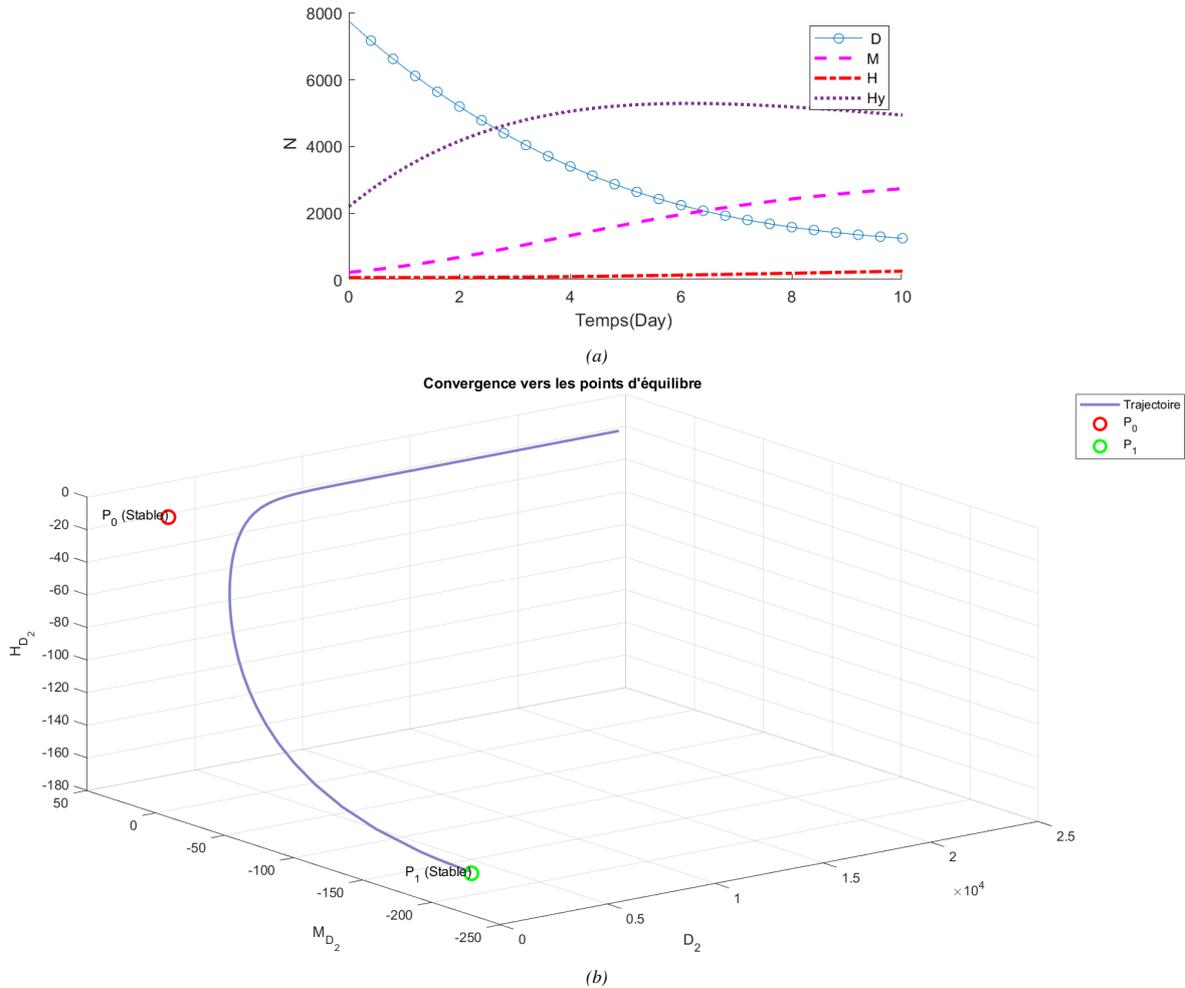


Figure 4. When $R_0 > 1$ the Drinking-Present equilibrium P_1 is locally asymptotically stable.

Table 2. Initial Values of Populations by Category and Health Status.

Population	Description	Value	source
$D_2(0)$	Diabetic without alcohol	2124	[12]
$M_{D_2}(0)$	Moderate drinkers	232	[12]
$H_{D_2}(0)$	Heavy drinkers	77	[12]
$H_{yp}(0)$	Hypertensive	987	[12]

In this section, we present numerical simulations of the model (1) for varying parameter values. The system (1) is solved using the Gauss-Seidel-like implicit finite-difference method, known as the GSS1 method, developed by Gumel et al. (2001) and outlined in Karrakchou et al. (2006). The initial conditions are chosen such that $D_2 + M_{D_2} + H_{D_2} + H_{yp} = 3420$. Numerical simulations are conducted to illustrate the theoretical findings, and we use the parameters and population values provided in table 1 and table 2.

We begin by graphically representing the no-alcohol equilibrium, denoted as P_0 . According to Theorem 3.1, the no-alcohol equilibrium P_0 of system (1) is locally asymptotically stable within the domain Ω . For the given parameters, the equilibrium point is $P_0 = (3517, 24; 0; 0; 4688.77)$. As shown

in Figure 2, the system clearly converges to the no-alcohol equilibrium P_0 . Furthermore, as discussed in Theorem 3.3, we conclude that the no-alcohol equilibrium P_0 is globally asymptotically stable in the domain Ω , a result confirmed by the simulation shown in Figure 3.

At the second stage, we present a graphic representation of the present alcohol equilibrium P_1 . We have $P_1 = (2069.56; 2269.47; 266.99; 3428.71)$. According to Theorem 3.4, the present alcohol equilibrium P_1 of the system (1) is locally asymptotically stable on Ω (see Figure 4). As a consequence, from Theorem 3.4, the present alcohol equilibrium is globally asymptotically stable, as displayed in Figure 5.

In summary, the combination of theoretical results

and graphical representations provides a comprehensive understanding of the stability properties of the equilibrium point. The local asymptotic stability, as shown in Figures 2 and 4, confirms that small perturbations will not lead to a departure from equilibrium, while the global asymptotic

stability, as demonstrated in Figures 3 and 5, guarantees that the equilibrium point is the ultimate state for any initial condition within the domain Ω . These findings together underscore the robustness and reliability of the equilibrium in the context of System (1)

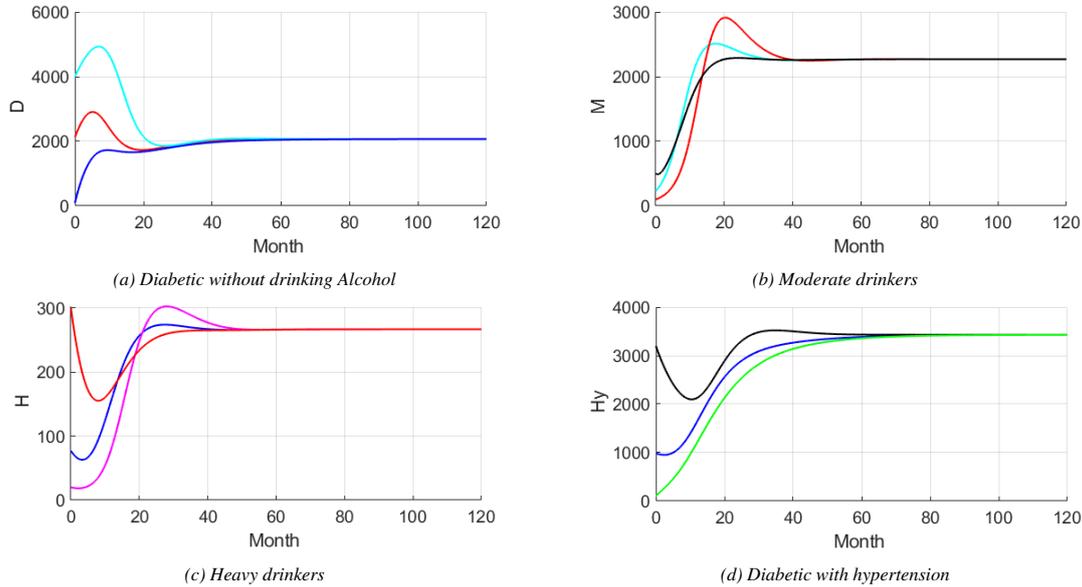


Figure 5. When $R_0 > 1$ the Drinking-present equilibrium P_1 is globally asymptotically stable.

4. Sensitivity Analysis of R_0

In this section, we calculate the sensitivity index for each model parameter related to the basic reproduction number R_0 . This index measures the relative importance of each parameter within the model that describes alcohol consumption in the diabetic population. The sensitivity index is employed to identify the parameter that has the greatest impact on R_0 , which can then be targeted for intervention strategies.

4.1. Sensitivity Index Calculation

In this section, we calculate the sensitivity index for each model parameter related to the basic reproduction number, R_0 . This index helps assess the relative importance of each parameter in the model that describes alcohol consumption within a diabetic population. By determining the sensitivity index, we can identify the parameters that have the most significant impact on R_0 , which can then be targeted for intervention purposes.

Definition 4.1. The normalized sensitivity index is calculated by differentiating R_0 with respect to the parameter m as follows:

$$\Gamma_m^{R_0} = \frac{m}{R_0} \frac{\partial R_0}{\partial m}. \tag{*}$$

This sensitivity index will be used to evaluate the influence of each parameter on the epidemic dynamics of the disease, guiding the implementation of control measures. The

sensitivity index for each parameter is derived from the expression for R_0 and calculated using the parameter values presented in table 1 and the formula shown in equation (*). For instance, the sensitivity index of R_0 with respect to the parameter m is expressed as:

$$R_0 = \frac{\alpha_1 I}{(\alpha_3 + \beta_1 + \mu)N}$$

$$\Gamma_{\alpha_1}^{R_0} = 1 \geq 0$$

$$\Gamma_{\mu}^{R_0} = \frac{-(\alpha_3 + \beta_1 + \alpha_2 + 2\mu)}{(\alpha_3 + \mu)(\alpha_2 + \beta_1 + \mu)\alpha_1 I} \leq 0$$

$$\Gamma_{\alpha_3}^{R_0} = \frac{-\alpha_3}{(\alpha_3 + \mu)\alpha_1 I} \leq 0$$

$$\Gamma_{\beta_1}^{R_0} = \frac{-\beta_1}{\alpha_1(\alpha_2 + \beta_1 + \mu)I} \leq 0$$

$$\Gamma_{\alpha_2}^{R_0} = \frac{-\alpha_2}{I\alpha_1(\alpha_2 + \beta_1 + \mu)} \leq 0$$

Table 3. Sensitivity indices of the effective reproduction R_0 using parameter value in table 1.

Parameter	Sensitivity Index
α_1	+1
α_2	-0.25
α_3	-2.08
μ	-0.14
β_1	-0.28

4.2. Interpretation of the Sensitivity

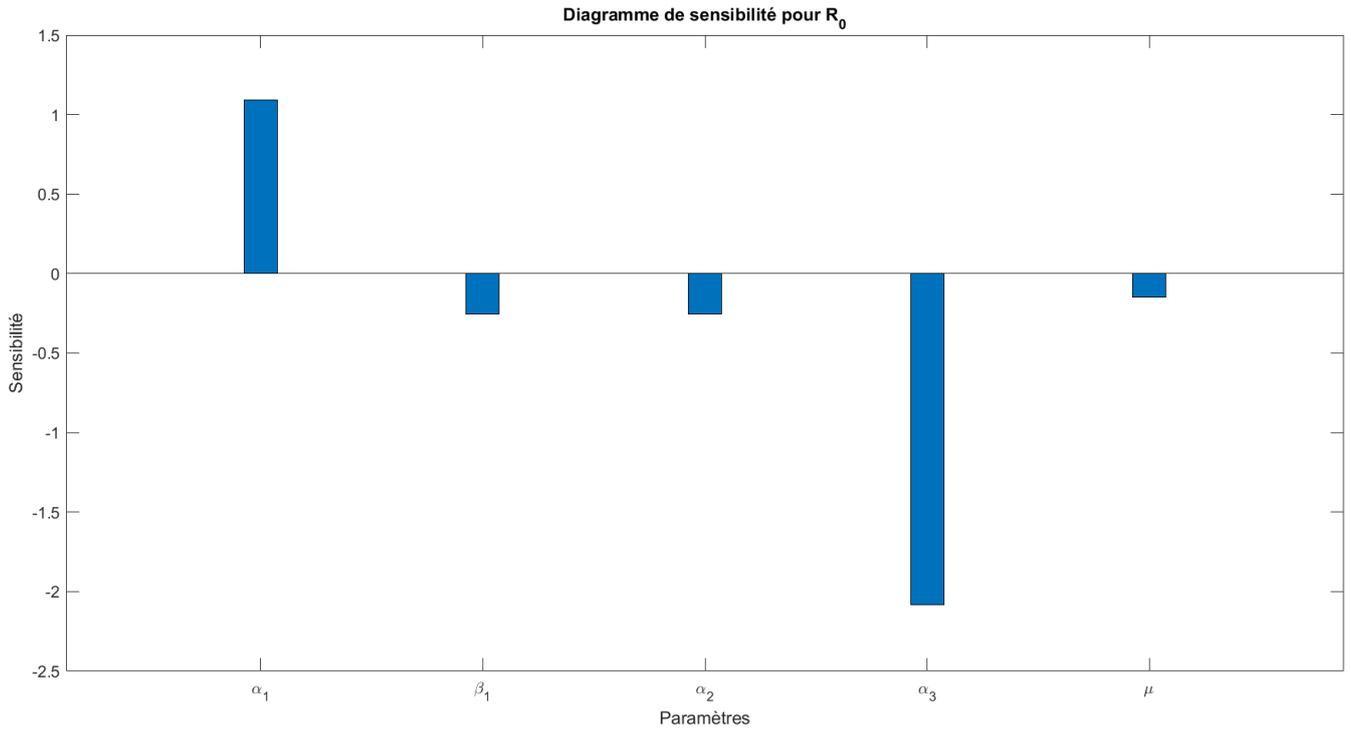


Figure 6. Graph of sensitivity indices of R_0 with respect to the model parameters.

Figure 6 presents the sensitivity profiles of the model with respect to the parameters influencing the basic reproduction number R_0 . The analysis indicates that the parameter α_1 exhibits a positive sensitivity index, whereas the parameters μ , β_1 , α_2 , and α_3 are characterized by negative sensitivity indices. Notably, α_1 emerges as the most influential positive parameter, implying that variations in its value lead to proportional increases or decreases in alcohol consumption within the diabetic population. Conversely, α_3 is identified as the most impactful negative parameter, suggesting that an increase in α_3 is associated with a reduction in the transmission dynamics of rabies within this cohort.

strategies for familial support, psychological treatment, and glycemic control. The objective is to maximize the diabetic population who abstain from alcohol consumption while minimizing both the population of heavy drinkers and those suffering from hypertension.

Simultaneously, the optimal control strategy minimizes the cost associated with implementing psychological and medical treatment interventions. To achieve these goals, we define and analyze the following optimal control problem:

$$J(u^*, v^*) = \min\{j(u, v); (u, v) \in U\} \tag{19}$$

where:

$$J(u, v) = \int_0^{t_f} [H_{yp}(t) - D_2(t) + \frac{A_1}{2}u^2(t) + \frac{A_2}{2}v^2(t)] dt$$

Subject to the equation

5. Statement of the Optimal Control Problem

In this section, we aim to determine the optimal control functions, denoted by $(u(t), v(t))$, which represent the best

$$\begin{cases} \frac{dD_2(t)}{dt} = I - \alpha_3(1 - v(t))D_2(t) - \alpha_1(1 - u(t))\frac{D_2(t)M_{D_2}(t)}{N} \\ \frac{dM_{D_2}(t)}{dt} = \alpha_1(1 - u(t))\frac{D_2(t)M_{D_2}(t)}{N} - (\alpha_2 + \beta_1 + \mu)M_{D_2}(t) \\ \frac{dH_{D_2}(t)}{dt} = \alpha_2M_{D_2}(t) - (\beta_2 + \delta_1 + \mu)H_{D_2}(t) \\ \frac{dH_{yp}(t)}{dt} = \beta_2H_{D_2}(t) + \beta_1M_{D_2}(t) + \alpha_3(1 - v(t))D_2(t) - (\mu + \delta_2)H_{yp}(t). \end{cases} \tag{20}$$

The two functions $u(t)$ and $v(t)$ represent:

$v(t)$: psychological treatment.

$u(t)$: Medical treatment + familial support.

These controls function are assumed to be elements of U .

$$U = \left\{ (u, v) : 0 \leq u, v \leq 1, t \in [0, t_f]; t_f \in \mathbb{R}; u, v \text{ are lebesgue measurable} \right\}$$

The constants $A_1 \geq 0, A_2 \geq 0$ are weighted cost with the use of the controls u and v respectively.

Theorem 5.1. (Existence of Optimal Control) Consider the optimal control problem (19) subject to (20). then there exists on optimal control (u^*, v^*) in u and a corresponding solution.

$$f^0(X(t), u(t), v(t)) = H_{yp}(t) - D_2(t) + \frac{A_2}{2}u^2(t) + \frac{A_2}{2}v(t)$$

is convex. i.e

The hessian matrix of f^0

$$H = \begin{pmatrix} A_1 & 0 \\ 0 & A_2 \end{pmatrix}$$

we have $\text{spec}(H) = \{A_1, A_2\} \subset \mathbb{R}_+$, the f^0 at strictly convex

It exists constants $K_1, K_2 > 0$ et $P > 1$ such as the integrand f^0 of objective function verify $f^0(x(t), u(t), v(t)) \geq K_1|(u, v)|^P - K_2$, we have:

$$\begin{aligned} f^0(x(t), u(t), v(t)) &= H_{yp}(t) - D_2(t) + \frac{A_2}{2}u^2(t) + \frac{A_2}{2}v^2(t) \\ &\geq \frac{1}{2} \min(A_1, A_2)(u^2(t) + v^2(t)) + H_{yp}(t) - D_2(t) \\ &\geq \frac{1}{2} \min(A_1, A_2)\|(u, v)\|_2^2 - D_2(t) \end{aligned}$$

$$N(t) = D_2(t) + M_{D_2}(t) + H_{D_2}(t) + H_{yp}(t)$$

is borned, then $D_2(t)$ is borned too. thus it exists, $m_1, m_2 \in \mathbb{R}^+$, such as $m_1 < D_2(t) < m_2 \forall t \in \mathbb{R}_+$. Let $K_1 = \frac{1}{2} \min(A_1, A_2)$ and $K_2 = m_2$. wo get:

$$f^0(x(t), u(t), v(t)) \geq K_1\|(u, v)\|_2^2 - K_2$$

Proposition 5.1. (Hamiltonian Characterization of the Minimization Problem)

The minimization problem (19) leads to a Hamiltonian problem H , defined as:

$$H(x(t), P(t), P^0, u(t), v(t)) = H_{yp}(t) - D_2(t) + \frac{A_2}{2}u^2(t) + \frac{A_2}{2}v^2(t) + \sum_{i=1}^4 \lambda_i f_i \tag{21}$$

Where f_i represents the right-hand side of the differential equation for the i^{th} state variable, $P(\cdot)$ is an absolutely continuous function defined on $[0, t_f]$ as $P : [0, t_f] \rightarrow \mathbb{R}^n \setminus \{0\}$, and P_0 is a non-negative real number and

$$X(t) = (D_2, M_{D_2}, H_{D_2}, H_{yp})$$

Proof. Let

$$P_0 = 1$$

$$P(t) = (\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t))$$

$$f^0(X(t), u(t), v(t)) = H_{yp}(t) - D_2(t) + \frac{A_2}{2}u^2(t) + \frac{A_2}{2}v^2(t)$$

$$f(X(t), u(t), v(t)) = \left(f_1(X(t), u(t), v(t)), f_2(X(t), u(t), v(t)), \dots, f_4(X(t), u(t), v(t)) \right)$$

where $X(t) = (D_2, M_{D_2}, H_{D_2}, H_{yp})$.
and:

$$\begin{aligned} f_1(x(t), u(t), v(t)) &= I - \alpha_3(1 - v(t))D_2(t) - \alpha_1(1 - u(t))\frac{D_2(t)M_{D_2}(t)}{N} \\ f_2(x(t), u(t), v(t)) &= \alpha_1(1 - u(t))\frac{D_2(t)M_{D_2}(t)}{N} - (\alpha_2 + \beta_1 + \mu)M_{D_2}(t) \\ f_3(x(t), u(t), v(t)) &= \alpha_2M_{D_2}(t) - (\beta_2 + \delta_1 + \mu)H_{D_2}(t) \\ f_4(x(t), u(t), v(t)) &= \beta_2H_{D_2}(t) + \beta_1M_{D_2}(t) + \alpha_3(1 - v(t))D_2(t) - (\mu + \delta_2)H_{yp}(t). \end{aligned}$$

Then, the Hamiltonian of optimal problem is defined by

$$\begin{aligned} H(t, x, p, p^0, u, v) &= \langle p, f(x(t), u(t), v(t)) \rangle + p^0 f^0(x(t), u(t), v(t)) \\ &= \langle (\lambda_1, \lambda_2, \lambda_3, \lambda_4) (f_1, f_2, f_3, f_4) \rangle + p^0 f^0 \\ &= \sum_{i=1}^4 \lambda_i f_i + H_{yp}(t) - D_2(t) + \frac{A_1}{2}u^2(t) + \frac{A_2}{2}v^2(t). \\ H(t, x, p, p^0, u, v) &= H_{yp}(t) - D_2(t) + \frac{A_1}{2}u^2(t) + \frac{A_2}{2}v^2(t) + \sum_{i=1}^4 \lambda_i f_i \end{aligned}$$

Proposition 5.2. (Existence Of Adjoint Vector $p(\cdot)$) The application $p(\cdot)$

$$p(\cdot) : [0, t_f] \longrightarrow \mathbb{R}^4$$

$t \mapsto (\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t))$ and verify

$$\begin{aligned} \lambda'_1 &= \lambda_1(\alpha_3(1 - v(t)) + \alpha_1\frac{M_{D_2}}{N}(1 - u(t)) - \alpha_1(1 - u(t))\lambda_2\frac{M_{D_2}}{N} - \alpha_3\lambda_4(1 - v(t))) \\ \lambda'_2 &= \alpha_1(1 - u(t))\frac{D_2(t)}{N}\lambda_1 + \lambda_2\left[(\alpha_2 + \beta_1 + \mu) - \alpha_1(1 - u(t))\frac{D_2}{N}\right] \\ \lambda'_3 &= \lambda_3(\beta_2 + \delta_1 + \mu) - \beta_2\lambda_4 \\ \lambda'_4 &= (\mu + \delta_2)\lambda_4. \\ \lambda_i(t_f) &= 0 \quad \forall i \in \{1, 2, 3, 4\} \end{aligned}$$

Proof. According to Theorem 5.1, the control pair (u^*, v^*) , associated with the solution X^* , minimizes $J(u, v)$ in U . By Pontryagin’s Maximum Principle, there exists an absolutely continuous function $P(\cdot)$ such that:

$$p(\cdot) : [0, t_f] \longrightarrow \mathbb{R}^4$$

$$t \mapsto (\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t))$$

Such as for almost all $t \in [0, t_f]$

$$p^0(t) = \frac{\partial H}{\partial X} \text{ and } p(t_f) = 0$$

then

$$p^0 = \frac{-\partial H}{\partial X} \implies \begin{cases} \lambda'_1 = \frac{-\partial H}{\partial D_2} \\ \lambda'_2 = \frac{-\partial H}{\partial M_{D_2}} \\ \lambda'_3 = \frac{-\partial H}{\partial H_{D_2}} \\ \lambda'_4 = \frac{-\partial H}{\partial H_{yp}} \end{cases}$$

then, we have

$$\begin{aligned} \lambda_1' &= \frac{-\partial H}{\partial D_2}(t, x, p, p^0, u, v) \\ &= \frac{\partial H}{\partial D_2} \left(H_{yp}(t) - D_2(t) + \frac{A^2}{2}u^2(t) + \frac{A^2}{2}v^2(t) + \sum_{i=1}^4 \lambda_i f_i(x, u, v) \right) \\ &= \sum_{i=1}^4 \lambda_i \frac{\partial f_i}{\partial D_2}(x, u, v) \\ &= \lambda_1(\alpha_3(1 - v(t)) + \alpha_1 \frac{M_{D_2}}{N}(1 - u(t)) - \alpha_1(1 - u(t))\lambda_2 \frac{M_{D_2}}{N} - \alpha_3\lambda_4(1 - v(t))) \end{aligned}$$

By the same method, we have:

$$\begin{aligned} \lambda_2' &= \alpha_1(1 - u(t)) \frac{D_2(t)}{N} \lambda_1 + \lambda_2 \left[(\alpha_2 + \beta_1 + \mu) - \alpha_1(1 - u(t)) \frac{D_2}{N} \right] \\ \lambda_3' &= \lambda_3(\beta_2 + \delta_1 + \mu) - \beta_2\lambda_4 \\ \lambda_4' &= (\mu + \delta_2)\lambda_4. \end{aligned}$$

The condition of transversality at final time t_f is $p(t_f) = 0$, then,

$$p(t_f) = 0 \implies \begin{cases} \lambda_1(t_f) = 0 \\ \lambda_2(t_f) = 0 \\ \lambda_3(t_f) = 0 \\ \lambda_4(t_f) = 0 \end{cases}$$

Finally, the characteristics of the vector $p(\cdot): t \mapsto (\lambda_1(t), \lambda_2(t), \lambda_3(t), \lambda_4(t))$ are

$$\begin{cases} \lambda_1' = \lambda_1(\alpha_3(1 - v(t)) + \alpha_1 \frac{M_{D_2}}{N}(1 - u(t)) - \alpha_1(1 - u(t))\lambda_2 \frac{M_{D_2}}{N} - \alpha_3\lambda_4(1 - v(t))) \\ \lambda_2' = \alpha_1(1 - u(t)) \frac{D_2(t)}{N} \lambda_1 + \lambda_2 \left[(\alpha_2 + \beta_1 + \mu) - \alpha_1(1 - u(t)) \frac{D_2}{N} \right] \\ \lambda_3' = \lambda_3(\beta_2 + \delta_1 + \mu) - \beta_2\lambda_4 \\ \lambda_4' = (\mu + \delta_2)\lambda_4. \\ \lambda_i(t_f) = 0 \quad \forall i \in \{1, 2, 3, 4\} \end{cases}$$

Theorem 5.2. (Characterization of Optimal Control) the optimal control (u^*, v^*) is defined by:

$$\begin{aligned} u^* &= \min \left(1, \max \left(0, \frac{(\lambda_2 - \lambda_1)\alpha_1 \frac{D_2 M_{D_2}}{N}}{A_1} \right) \right) \\ v^* &= \min \left(1, \max \left(0, \frac{(\lambda_4 - \lambda_1)D_2\alpha_3}{A_2} \right) \right) \end{aligned}$$

Proof. To prove the characterization of optimal control, we define the Lagrangien associated to the problem. It corresponds to Hamiltonian increased by coefficient of Penalty

$$L(t, x, u, v, p) = H(t, x, p, p^0, u, v) + w_{11}u + w_{12}(1 - u) + w_{21}v + w_{22}(1 - v)$$

where $w_{ij}(t) \geq 0$ are penalization coefficients that verify

$$w_{11}u(t) = w_{12}(1 - u(t)) = 0 \text{ for the control } u^*$$

$$w_{21}v(t) = w_{22}(1 - v(t)) = 0 \text{ for the control } v^*$$

The optimal control (u^*, v^*) obtained is the resultant of application of equation of contrainte

$$\begin{cases} \frac{\partial L}{\partial u} = 0 & \text{in } u^* \\ \frac{\partial L}{\partial v} = 0 & \text{in } v^* \end{cases}$$

that imply,

$$\begin{cases} \frac{\partial H}{\partial u} - w_{11} + w_{12} = 0 & \text{in } u^* \\ \frac{\partial H}{\partial v} - w_{21} + w_{22} = 0 & \text{in } v^* \end{cases}$$

The partial derivative of H in relation to u is given by

$$\begin{aligned} \frac{\partial H}{\partial u} &= \frac{\partial}{\partial u} \left(H_{yp}(t) - D_2(t) + \frac{A_1}{2}u^2(t) + \frac{A_2}{2}v^2(t) + \sum_{i=1}^4 \lambda_i f_i \right) \\ &= A_1 u(t) + \lambda_1 \frac{\partial f_1}{\partial u} + \lambda_2 \frac{\partial f_2}{\partial u} + \lambda_3 \frac{\partial f_3}{\partial u}. \\ &= A_1 u(t) + \lambda_1 \left(\alpha_1 \frac{D_2(t)M_{D_2}(t)}{N} \right) + \lambda_2 \alpha_1 \frac{D_2(t)M_{D_2}(t)}{N} \\ \frac{\partial H}{\partial v} &= A_2 v(t) + \lambda_1 \frac{\partial f_1}{\partial v} + \lambda_4 \frac{\partial f_4}{\partial v} \end{aligned}$$

we obtained

$$\begin{cases} A_1 u(t) + (\lambda_2 - \lambda_1) \alpha_1 \frac{D_2(t)M_{D_2}(t)}{N} - w_{11} + w_{12} = 0 \text{ for } u = u^* \\ A_2 v(t) + (\lambda_4 + \lambda_1) \alpha_3 D_2(t) - w_{21} + w_{22} = 0 \text{ for } v = v^* \end{cases}$$

At u^* and v^* , we have:

$$\begin{aligned} A_1 u^* + (\lambda_2 - \lambda_1) \alpha_1 \frac{D_2^*(t)M_{D_2}^*(t)}{N} - w_{11} + w_{12} &= 0 \\ u^* &= \frac{1}{A_1} \left((\lambda_2 - \lambda_1) \frac{D_2^*(t)M_{D_2}^*(t)}{N} \alpha_1 - w_{11} + w_{12} \right) \\ v^* &= \frac{1}{A_2} \left[(\lambda_4 - \lambda_1) \alpha_3 D_2^*(t) - w_{21} + w_{22} \right]. \end{aligned}$$

Let be the set $\{t : 0 < u^* < 1\}$ we have $w_{11}u^* = w_{12}(1 - u^*) \implies w_{11} = w_{12} = 0$

$$u^* = \frac{(\lambda_2 - \lambda_1) D_2^*(t) M_{D_2}^*(t) \alpha_1}{A_1 N}$$

let be the set $\{t : u^* = 0\}$ we have $w_{12}(1 - u^*) = 0 \implies w_{21} = 0$, therefore

$$0 = u^* = \frac{(\lambda_2 - \lambda_1) D_2^*(t) M_{D_2}^*(t) \alpha_1 + w_{11}}{A_1 N}$$

since $w_{11} \geq 0$, then

$$\frac{(\lambda_2 - \lambda_1) D_2^*(t) M_{D_2}^* \alpha_1 + \varepsilon H_D^*(t) (\lambda_3 - \lambda_1)}{A_1 N} \leq u^* = 0$$

thus, on the set $\{t : 0 \leq u < 1\}$, u^* is defined like the following: $\max(0, B)$, where,

$$B = \frac{(\lambda_2 - \lambda_1) D_2^*(t) M_{D_2}^* \alpha_1}{A_1 N}$$

let be the set: $\{t : u^* = 1\}$

we have $w_{11} \times 1 = w_{12} \times 0 = 0 \implies w_{11} = 0$

then $1 = u^* = B - w_{12}$

since $w_{21} \geq 0$ the: $B \leq u^* = 1$

on the set $\{t, 0 \leq u^* \leq 1\}$ u^* is defined by:

$$u^* = \min \left(1, \max(0, B) \right).$$

By the same method, we get the expression of v^* :

$$v^* = \min \left(1, \max \left(0, \alpha_3 \frac{(\lambda_4 - \lambda_1) D_2^*}{A_2} \right) \right)$$

Finally on the set U the optimal control (u^*, v^*) is given by:

$$u^* = \min\left(1, \max\left(0, \frac{(\lambda_2 - \lambda_1)D_2^*M_{D_2}^*\alpha_1}{A_1N}\right)\right)$$

and

$$v^* = \min\left(1, \max\left(0, \frac{(\lambda_4 - \lambda_1)\alpha_3D_2^*(t)}{A_2}\right)\right)$$

5.1. Numerical Simulations

The optimal control problem (7), along with the adjoint equation (15) and the optimal control characterization (14), has been solved numerically, and the results are presented graphically. In the simulations of the optimal control problem, the time unit is considered in months. The parameter values are taken from the system (1), and the parameters of the model used in the optimal control simulations are as follows:

$\alpha_1 = 0.45; \alpha_2 = 0.06; \alpha_3 = 0.02; \beta_1 = 0.09, \beta_2 = 0.03, \delta_1=0.02, \delta_2=0.004, \mu=0.045$ The numerical value of the weight constants are $A_1 = 1000, A_2 = 100000$, initial values of the variables in simulations are considered to be $D_2(0) = 2124, M_{D_2}(0) = 232, H_{D_2}(0) = 77, H_{yp}(0) = 987$.

We present a numerical method to solve the optimality system (20), with initial conditions given for the state variables at time $t = 0$, and terminal conditions specified for the adjoint variables at $t = t_f$. The numerical simulations are carried out using the forward Euler method for the state system and the backward difference approximation for the adjoint system, as

inspired by [19].

We consider the following two strategies for the numerical simulation of the model:

1. *Strategy A:* $u = 0$ et $v \neq 0$
2. *Strategy B:* $u \neq 0$ et $v \neq 0$

5.2. Strategy A

In this strategy, by setting the control u to zero, we use control v to optimize the objective function J . In Figure 7b, Figure 7c, and Figure 7d, we observe that the populations of moderate and heavy drinkers, as well as hypertensive individuals, are significantly reduced when control is applied, compared to the situation with no control. However, Figure 7a shows that the number of diabetic individuals not consuming alcohol increases during the first two years with family support and awareness. Hence, this strategy proves to be an effective approach to combat alcohol consumption among people with diabetes.

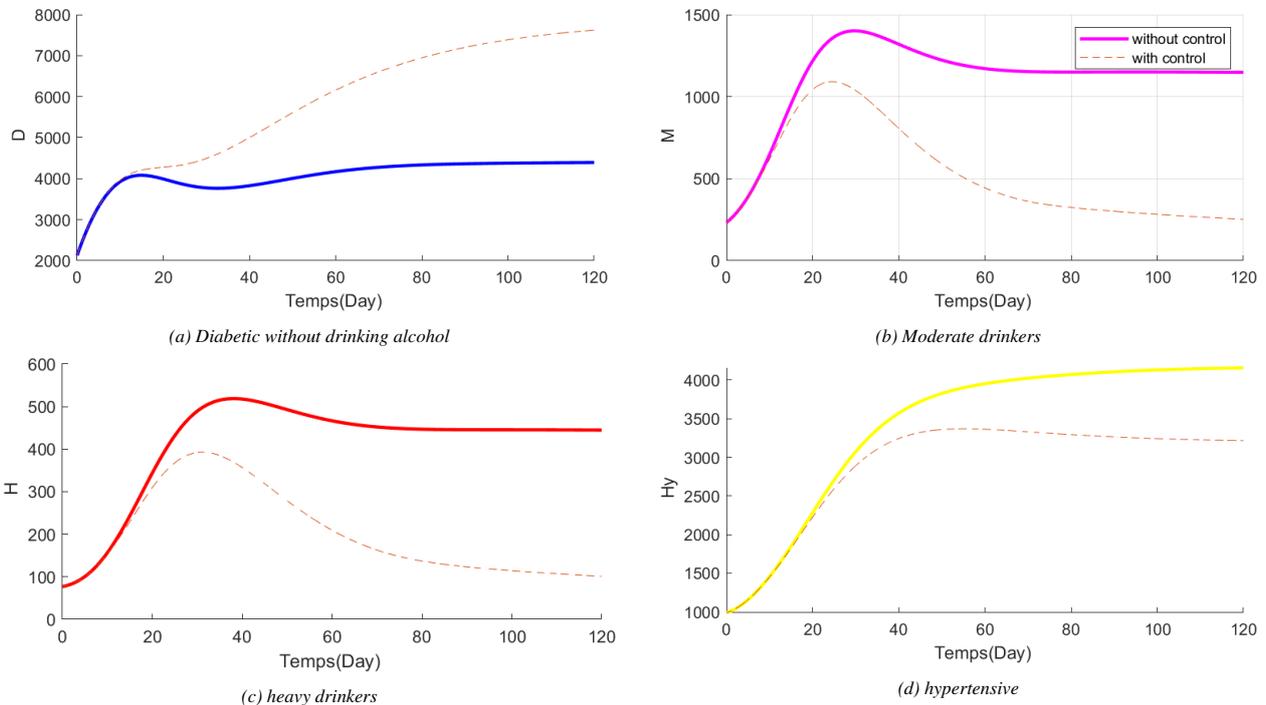


Figure 7. Numbers of D_2, M_{D_2}, H_{D_2} and H_{yp} with control v .

Table 4. Evolution of the number of diabetics with controls v after 10 years.

Groups after 10 years	without control	with control	Pourcentage
Diabetic without drinking alcohol	4393,81	7620,45	+73,43 %
Moderate drinkers	1148,41	40,77	-96,45 %
Heavy drinkers	444,85	21,07	-95,26 %
Hypertensive	4185,51	3216,17	- 23,15 %

For individuals with diabetes, familial support and medical treatment are crucial to manage their condition and avoid excessive alcohol consumption, which can lead to hypertension.

1. **Familial Support: Education and Awareness:** Families should understand diabetes and its complications, including the impact of alcohol on blood sugar levels and blood pressure.
2. **Encouragement and Motivation:** Families can encourage healthy habits and participate in activities like walking or cooking healthy meals together
3. **Monitoring and Emotional Support:** Helping to monitor blood sugar levels, attending medical appointments, and providing emotional support to reduce stress.
4. **Regular Medical Consultations:** Regular check-ups to monitor blood sugar and blood pressure, and adjust treatments as needed.
5. **Medications:** Using antihypertensives if necessary, and diabetes medications to maintain normal blood sugar levels. **Alcohol Management Plan:** Advising on moderate alcohol consumption or abstinence and

suggesting healthy alternatives for social occasions.

By combining strong familial support with appropriate medical treatment, individuals with diabetes can better manage their condition and prevent complications like hypertension.

5.3. Strategy B

In the second case, we perform a simulation of the control system incorporating both strategies u and v . It is evident from Figure 8b, Figure 8c, Figure 8d that the numbers of moderate drinkers (M_{D_2}), heavy drinkers (H_{D_2}), as well as hypertensive individuals (H_{yp}) are significantly reduced when these strategies are applied, compared to the scenario with no control. This indicates that the combined application of strategies u and v is effective in reducing alcohol consumption. Additionally, it maximizes the number of individuals who do not consume alcohol, demonstrating the overall success of the intervention in promoting healthier behaviors across the population Figure 8a. The simulation results clearly show that using both strategies in tandem produces a more substantial impact on reducing the prevalence of alcohol-related health issues.

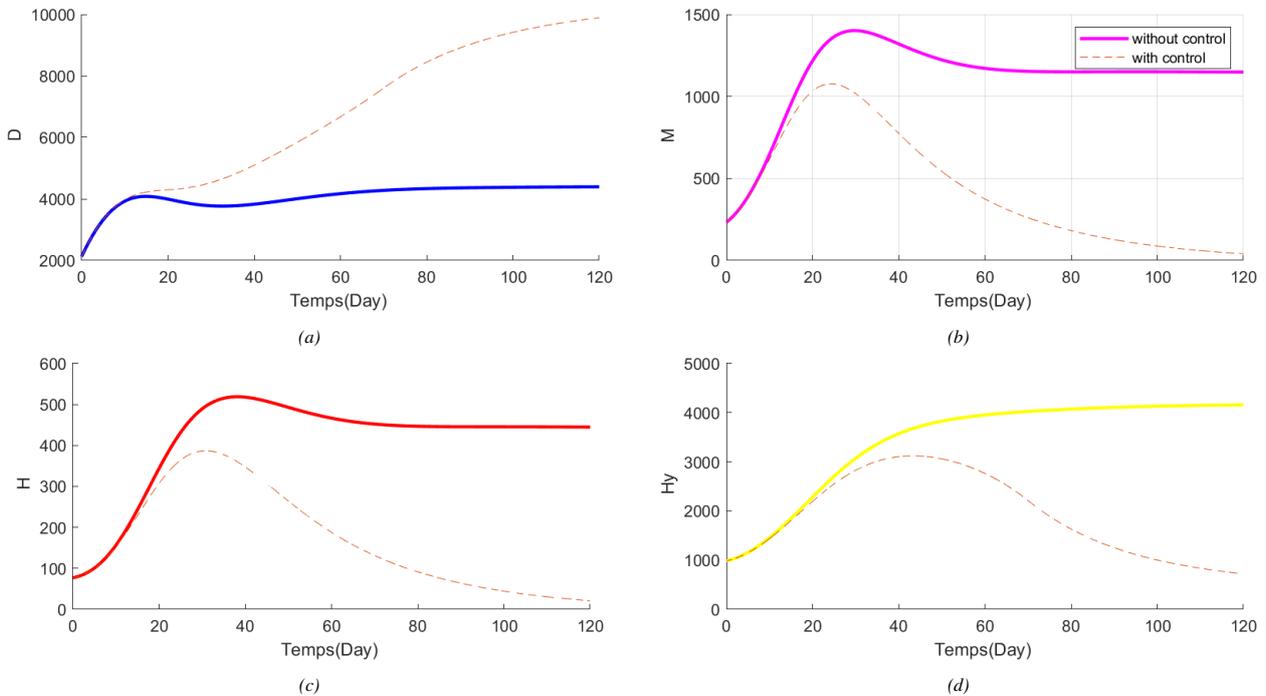


Figure 8. Numbers of D_2 , M_{D_2} , H_{D_2} and H_{yp} with control v and u .

Table 5. Evolution of the number of diabetics with controls *u* and *v* after 10 years.

Groups after 10 years	without control	with control	Pourcentage
Diabetic without drinking alcohol	4393,81	9875,59	+124,79 %
Moderate drinkers	1148,41	254,30	-77,85 %
Heavy drinkers	444,85	101,49	-77,19 %
Hyperetensive	4185,51	725,59	-82,67 %

[25] Psychological treatment for diabetic patients is essential in preventing hypertension. Cognitive Behavioral Therapy (CBT) helps patients identify and change negative thought patterns that cause stress, which can elevate blood pressure. Mindfulness practices like meditation and deep breathing exercises reduce stress effectively. Counseling and support groups provide emotional support, helping patients cope with diabetes-related challenges. Behavioral interventions such as motivational interviewing encourage healthy habits, while lifestyle coaching guides patients in maintaining these changes. Regular psychological assessments monitor mental health and allow for treatment adjustments.

These strategies help manage stress and emotions, crucial for preventing hypertension and improving overall health.

ORCID

0009-0000-3600-2831 (Ikram Imken)

0000-0001-7423-3008 (Nadia Idrissi Fatmi)

Conflicts of Interest

The authors declare no conflicts of interest.

References

- [1] World Health Organization, *Hypertension*, 2024, https://www.who.int/health-topics/hypertension#tab=tab_1, Accessed: 2024-06-09.
- [2] World Health Organization, *Hypertension Fact Sheet*, 2023, <https://www.who.int/news-room/fact-sheets/detail/hypertension>, Accessed: 2024-06-09.
- [3] C.Lian, *L'alcoholisme cause d'hypertension arterielle*, Bull Acad Med, (1915), 74, 8.
- [4] A. L.Klatsky, M. A. Armstrong, G. D. Friedman, *Relations of alcoholic beverage use to subsequent coronary artery disease hospitalization*, American Journal of Cardiology, 58, (1986), 710–714, [https://doi.org/10.1016/0002-9149\(86\)90342-5](https://doi.org/10.1016/0002-9149(86)90342-5)
- [5] Klatsky, A. L., Armstrong, M. A., and Friedman, G. D., *Risk of cardiovascular mortality in alcohol drinkers, ex-drinkers and nondrinkers*, American Journal of Cardiology, (1990), 66, 1237–1242, [https://doi.org/10.1016/0002-9149\(90\)91107-h](https://doi.org/10.1016/0002-9149(90)91107-h)
- [6] C. S. Fuchs, M. J. Stampfer, G. A. Colditz, E. L. Giovannucci, J. E. Manson, I. Kawachi, D. J. Hunter, S. E. Hankinson, C. H. Hennekens, B. Rosner, *Alcohol consumption and mortality among women*, New England Journal of Medicine, (1995), 332, 1245–1250, <https://doi.org/10.1056/NEJM199505113321901>
- [7] E. B. Rimm, E. L. Giovannucci, W. C. Willett, G. A. Colditz, A. Ascherio, B. Rosner, M. J. Stampfer, *Prospective study of alcohol consumption and risk of coronary disease in men*, Lancet, (1991), 338, 464–468, [https://doi.org/10.1016/0140-6736\(91\)90542-w](https://doi.org/10.1016/0140-6736(91)90542-w)
- [8] E. B. Rimm, A. Klatsky, D. Grobbee, M. J. Stampfer, *Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine, or spirits*, BMJ, (1996), 312, 731–736, <https://doi.org/10.1136/bmj.312.7033.731>
- [9] B. Xi, S. P. Veeranki, M. Zhao, C. Ma, Y. Yan, J. Mi, *Relationship of alcohol consumption to all-cause, cardiovascular, and cancer-related mortality in U.S. adults*, Journal of the American College of Cardiology, (2017), 70, 913–922, <https://doi.org/10.1016/j.jacc.2017.06.054>
- [10] B. Taylor, H. M. Irving, D. Baliunas, M. Roerecke, J. Patra, S. Mohapatra, J. Rehm, *Alcohol and hypertension: gender differences in dose? response relationships determined through systematic review and meta-analysis*, Addiction, (2009), 104, 1981–1990, <https://doi.org/10.1111/j.1360-0443.2009.02694.x>
- [11] M. Roerecke, J. Kaczorowski, S. W. Tobe, G. Gmel, O. Hasan, J. Rehm, *The effect of a reduction in alcohol consumption on blood pressure: a systematic review and meta-analysis*, The Lancet Public Health, (2017), 2, e108–e120, [https://doi.org/10.1016/S2468-2667\(17\)30003-8](https://doi.org/10.1016/S2468-2667(17)30003-8)
- [12] J. J. Mayl, *Association of Alcohol Intake With Hypertension in Type 2 Diabetes Mellitus: The ACCORD Trial*, Journal of the American Heart Association, (2020), <https://doi.org/10.1161/JAHA.120.017334>
- [13] S. Ma, H. Huo, X. Meng, *Modelling alcoholism as a contagious disease: a mathematical model with awareness programs and time delay*, Discrete Dynamics in Nature and Society, 2015, (2015), 260195, <https://doi.org/10.1155/2015/260195>

- [14] S. Sharma, G. P. Samanta, *Drinking as an epidemic: a mathematical model with dynamic behaviour*, Journal of Applied Mathematics and Informatics, (2013), 31, 1–25, <https://doi.org/10.14317/JAMI.2013.001>
- [15] B. Benedict, *Modeling alcoholism as a contagious disease: how “infected” drinking buddies spread problem drinking*, SIAM News, (2007), 40, 1–3.
- [16] H. F. Huo, S. R. Huang, X. Y. Wang, et al, *Optimal control of a social epidemic model with media coverage*, Journal of Biological Dynamics, (2017), 11, 226–243, <https://doi.org/10.1080/17513758.2017.1321792>
- [17] I. Adu, M. Osman, C. Yang, *Mathematical model of drinking epidemic*, British Journal of Mathematics & Computer Science, (2017), 22, <https://doi.org/10.9734/bjmcs/2017/33659>
- [18] I. Imken, N. I. Fatmi, S. Elamari, *A new model of the spread of COVID-19 among diabetes population: a mathematical analysis and optimal control approach for intervention strategies*, Communications in Mathematical Biology and Neuroscience, 2023, (2023), Article ID 123, <https://doi.org/10.28919/cmbn/8264>
- [19] I. Imken, N. I. Fatmi, *A new mathematical model of drinking alcohol among diabetes population taking anti-diabetic drugs: an optimal control approach*, Communications in Mathematical Biology and Neuroscience, 2024, (2024), Article ID 5, <https://doi.org/10.28919/cmbn/8359>
- [20] S. V. Bădescu, C. Tătaru, L. Kobylinska, E. L. Georgescu, D. M. Zăhău, A. M. Zăgrean, L. Zăgrean, *The association between diabetes mellitus and depression*, Journal of Medicine and Life, (2016), 9(2), 120–125, PMID 27453739.
- [21] S. P. Veeranki, M. Zhao, C. Ma, Y. Yan, J. Mi, *Relationship of alcohol consumption to all-cause, cardiovascular, and cancer-related mortality in U.S. adults*, Journal of the American College of Cardiology, (2017), 70, 913–922, <https://doi.org/10.1016/j.jacc.2017.06.054>
- [22] J. Rehm, M. Roerecke, *Cardiovascular effects of alcohol consumption*, Trends in Cardiovascular Medicine, (2017), 27, 534–538, <https://doi.org/10.1016/j.tcm.2017.06.002>
- [23] J. P. LaSalle, *The Stability of Dynamical Systems*, SIAM, (1976), Regional Conference Series in Applied Mathematics, Volume 25.
- [24] S. Nababan, *A Filippov-type lemma for functions involving delays and its application to time-delayed optimal control problems*, Journal of Optimization Theory and Applications, (1979), 27, 357–376, <https://doi.org/10.1007/BF00933030>
- [25] D. Mikhailov, A. Pikovsky, *Introduction to Nonlinear Dynamics and Chaos*, Springer, (1999).