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MODELING MATHEMATICAL AND ANALYSIS OF AN ALCOHOL DRINKING WITH n COMPLICATIONS

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Abstract. In this article, we propose a new continuous mathematical approach to model and analyze the dynamics of the population of heavy drinkers and their health complications. The model has several compartments, including a new one that represents the number of heavy drinkers with different complications associated with excessive alcohol consumption. We study the stability of the model using mathematical theories such as the Routh-Hurwitz criteria for local stability, in order to study the equilibrium without and with excessive alcohol consumption, and the construction of Lyapunov functions makes it possible to study global stability. A sensitivity analysis is performed to determine which parameters have the most significant impact on the number of reproductions R0. The results were validated using numerical simulations carried out under MATLAB. This model may be useful in guiding public health policies aimed at reducing the number of drinkers and the complications associated with alcohol consumption.

Keywords: mathematical model; excessive alcohol consumption; complications; global stability; local stability.

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1. INTRODUCTION

Addiction is a dangerous social scourge that must be combated. But the term ”addiction” is defined as a dependence on a substance or behavior, with serious consequences for health and behavior. An addiction is characterized by an often strong, even compulsive desire to consume or engage in a behavior. Thus, so that the person finds himself unable to abstain from taking a substance or engaging in a behaviour; he loses control over substance use or behavior. This consumption or behavior has negative repercussions on the addict and those around him. Specialists distinguish between physical addiction and behavioral addiction. Physical addiction specifically means addiction to drugs, alcohol, and sedatives, while behavioral addiction refers to activities that obsess a person and waste significant time in the practice of this activity, that makes his behavior unacceptable. On the contrary, it is a source of evil for him and those around him. In this context, we can talk about addiction to the Internet, shopping, work, food, sports, food, sex, phone ...

Addiction to psychotropic substances.

It defines dependence on tobacco, drugs, and alcohol ”if the use of drugs or alcohol continues, it impacts physical or psychological dependence, or both. At the beginningThe drug gives the addict at first a feeling of happiness and calm, then he rushes to gradually increase the dose to enter the cycle of addiction and become a prisoner of drugs and alcohol his only obsession is to get it at any cost. The drug and alcohol addict tries to escape from social events, becomes emotional, and may resort to theft or even murder.

The causes of addiction are multiple: some of these reasons are related to social and cultural factors, the problems and pressures experienced by the individual, also it’s related to the accompaniment of bad friends who push young people, especially to the abuse of drugs or alcohol. Moreover, the causes of addiction may be psychological, with some people suffering from mental illness or chronic depression and anxiety, or neurological diseases, or who have a pathological, unstable or antisocial personality, to drugs or alcohol.

Behavioral addiction.

Behavioral addiction can be defined as the control of an idea over a person to turn into an obsession, which takes a lot of his time and interests, and seeks to reach it by all means, despite

his awareness of its consequences and risks. This type of addiction is very common, but it is not as visible and obvious as in the case of physical addiction.

We are interested in this work on alcohol dependence. In recent years, with the improvement of our standard of living, lifestyles have diversified, and alcohol consumption has become increasingly an important part of people's daily lives. Yet, the current situation of alcoholism in the world is truly worrying. Alcohol addiction has become one of the public health and social problems facing the world. Alcoholism has very serious consequences such as marital harm, child abuse, crime, social violence, and other serious consequences of criminal acts. It also contributes to traffic accidents. In general, the higher the volume of alcohol consumption, the more alcohol causes about 200 different types of diseases and conditions, including injuries and mental and behavioral disorders.

According to the WHO in 2016, the harmful use of alcohol resulted in some 3 million deaths (5.3% of all deaths) worldwide and 132.6 million disability-adjusted life years (DALY), or 5.1% of all DALY in that year. Mortality resulting from alcohol consumption is higher than that caused by diseases such as tuberculosis, HIV/AIDS and diabetes. Among men in 2016, an estimated 2.3 million deaths and 106.5 million DALY were attributable to the consumption of alcohol. Women experienced 0.7 million deaths and 26.1 million DALY attributable to the consumption of alcohol.

Alcohol caused approximately 0.4 million of the 11 million deaths worldwide in 2016 from communicable, maternal, perinatal and nutritional diseases, accounting for 3.5% of these deaths.

Harmful use of alcohol caused some 1.7 million deaths from non-communicable diseases in 2016, including some 1.2 million deaths from digestive and cardiovascular diseases (0.6 million for each condition) and 0.4 million deaths from cancer. Globally, an estimated 0.9 million injury deaths are attributable to alcohol, including around 370,000 deaths from road traffic accidents, 150,000 from self-harm and around 90,000 from interpersonal violence . Among road accidents, 187,000 alcohol-attributable deaths involved people other than drivers.[1,2]

Certain diseases caused by excessive alcohol consumption.

Excessive use of alcohol often leads to many harmful consequences for the drinkers themselves and society. And it causes serious health problems, including an increased risk of several diseases, like certain types of cancer such as cancer of the mouth, throat, liver, esophagus, colon and breast, liver diseases (fatty liver, alcoholic liver and cirrhosis), digestive system diseases (gastritis and inflammation of the liver Pancreatic), heart problems lead to high blood pressure and increase the risk of an enlarged heart, heart failure or stroke, neurological complications, weak immune system, sexual function problems and menstrual problems. Alcohol use also contributes to death and disability through road accidents, injuries, violence, crime and suicide, especially among young people. In its Global Status Report on Alcohol and Health, published in 2018, the World Health Organization reported that in 2016, deaths from alcohol consumption were higher than from diseases such as tuberculosis, HIV/AIDS and diabetes. Of the 3 million deaths caused by the harmful use of alcohol (5.3% of all deaths worldwide), 28% of deaths have been attributed to road traffic accidents, violence and suicide, 21% have been attributed to diseases affecting the digestive system, and 19% were attributed to cardiovascular diseases, and 32% were attributed to infectious diseases, cancers, mental disorders, or other conditions.

Mathematical epidemiological models have become important tools that predict the dynamics of infectious diseases and provide effective measures to analyze and study and control their spread. Many studies use epidemiological mathematical models to study the dynamics of alcohol consumption, analyze consumer behavior and propose solutions to reduce the risks to consumers and society as well as to minimize the number of excessive alcohol consumers. For example, S. H. Ma et al.[15] modeled alcoholism as a contagious disease and used optimal control to study their mathematical model with sensitization and delay programs. Wang et al. [19] proposed and analyzed a nonlinear model of alcoholism and used optimal control to prevent the interaction between susceptible individuals and infected individuals. Sharma et al. [12] developed a mathematical model of alcohol abuse and discussed the existence, local and global stability of the endemic equilibrium without alcohol consumption and sensitivity analysis of a number of basic reproduction R_0. Huo et al. [17] focused on the global ownership of a consumption model with public health education campaigns. They conclude that educational campaigns have a positive effect on controlling consumption dynamics. Giacobbe et al. [17]

considered a mathematical model that describes the dynamics of a population divided into three categories and used an additional variable that represents an external influence. They studied the existence of an endemic equilibrium and analyzed the stability of the equilibrium. Agrawal et al. [22] developed a nonlinear SHTR mathematical model of alcohol abuse with a nonlinear incidence rate. The stability analysis of the model they proposed shows that the system is locally asymptotically stable at equilibrium without alcohol E^0 when $R_0 \leq 1$.

Motivated by the fact that mathematical models have proven to be useful in understanding the dynamics of several social phenomena, in this study we propose a new model concerned with the study of excessive alcohol consumption and its complications on diseases. We examined the local stability of this model using the Routh-Hurwitz criteria and discussed its global stability using the Lyapunov function

So, we will study the dynamics and the analysis of a mathematical model of excessive alcohol consumption and their complications $PMHC_1C_2C_3.....C_nQ$ which contains the following additions:

• A compartment C_i that represents the number of the heavy drinkers with i complications associated with prolonged and excessive alcohol consumption where, $i = \{1, 2, ..., n\}$ n complications.

• The death rate induced by the heavy drinkers δ_0.

• The death rate induced by the heavy drinkers with i complications δ_i.

The drinkers classes of this model are divided into n+4 compartments: Potential drinkers (P), Moderate drinkers (M), Heavy drinkers (H), heavy drinkers with i complications (C_i) and quitters of drinking(Q).

Among the opportunities for reducing the harmful use of alcohol worldwide are inclusion of alcohol-related targets in major global policy and strategic frameworks such as the 2030 Agenda for Sustainable Development, increased health consciousness in populations, decreased youth alcohol consumption as observed in a wide range of countries, recognition of the role of alcohol control policies in reducing health and gender inequalities, and accumulating evidence of effectiveness and cost-effectiveness of a number of alcohol control measures [2].

The paper is organized as follows. In Section 2, we present our $PMHC_1C_2C_3.....C_nQ$ mathematical model that illustrates the dynamics of excessive alcohol consumption and their complications.In Section 3; we discuss basic properties and positivity of solutions. In section 4 ; we analyse the local and global stability and

the problem of parameters sensitivity. Numerical simulations are given in Section 5. Finally, we conclude the paper in Section 6.

2. A Mathematical Model

We propose a continuous model $PMHC_1C_2C_3...C_nQ$ to describe and analyze the dynamics of the population of excessive alcohol consumers and their health complications. We divided the population into several compartments, including potential drinkers $P(t)$, moderate drinkers $M(t)$, heavy drinkers and heavy drinkers with different complications $C_i(t)$ and quitters of drinking Q(t).

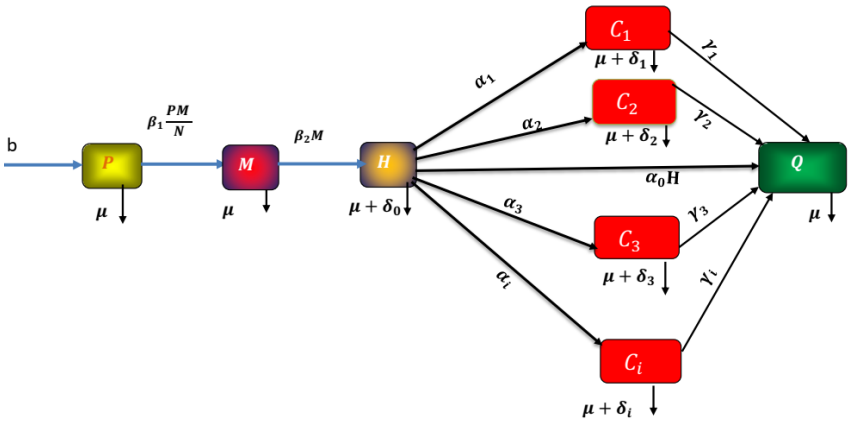

Figure1: Schematic diagram of the fourten drinking classes in the model

We consider the following system of six non-linear differential equations:

$$
(1)\qquad \begin{cases} \frac{dP(t)}{dt} = b - \beta_1 \frac{PM}{N} - \mu P \\ \frac{dM(t)}{dt} = \beta_1 \frac{PM}{N} - (\beta_2 + \mu) M \\ \frac{dH(t)}{dt} = \beta_2 M - (\mu + \alpha_0 + \delta_0 - \sum_{i=1}^{n} \alpha_i) H \\ \frac{dC_i(t)}{dt} = \alpha_i H - (\mu + \gamma_i + \delta_i) C_i(t) \\ \frac{dQ(t)}{dt} = \alpha_0 H + \sum_{i=1}^{n} \gamma_i C_i - \mu Q \end{cases}
$$

Potential drinkers P:

$$\frac{dP(t)}{dt} = b - \beta_1 \frac{PM}{N} - \mu P \tag{2}$$

The potential drinkers $P(t)$ represents individuals who are older than the age of majority, is increased by the recruitment rate denote b and decreased by the rates $\beta_1 \frac{PM}{N}$ and μP , where, μ is the natural death rate, β_1 is the transmission rate from P to M .

Moderate drinkers M:

$$\frac{dM(t)}{dt} = \beta_1 \frac{PM}{N} - \beta_2 M - \mu M \tag{3}$$

The moderate drinkers $M(t)$ is increased by the rates $\beta_1 \frac{PM}{N}$ and decreased by the rates $\beta_2 M$ and μM, where, β_2 is the transmission rate from the M to H .

Heavy drinkers H:

$$\frac{dH(t)}{dt} = \beta_2 M - (\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i) H \tag{4}$$

The number of the heavy drinkers $H(\mathbf{t})$ it comprises the addicted individuals. The compartment become larger as the number of heavy drinkers increases by the rate $\beta_2 M$ and decreases by the rates $\alpha_0 H$, $\sum_{i=1}^{n} \alpha_i H$ and $(\mu + \delta_0)H$. where, δ_0 is the death rate induced by H.

Heavy drinkers with complication C_i**:**

$$\frac{dC_i(t)}{dt} = \alpha_i H - (\mu + \gamma_i + \delta_i) C_i(t) \tag{5}$$

The heavy drinkers with liver complications associated with prolonged and excessive alcohol consumption (alcoholic hepatitis, fibrosis and cirrhosis), is increased by the rate $\alpha_i H$ and decreased by the rates $\gamma_i C_i$, μC_i and $\mu \delta_i$. where, δ_i is the death rate induced by the i complication of heavy drinkers.

four $i = 1$, C_1 represent the number of heavy drinkers with liver disease complications,

four $i = 2$, C_2 represent the number of heavy drinkers with pancreatitis complications,

four $i = 3$, C_3 represent the number of heavy drinkers with cancer complications,

four $i = 4$, C_4 represent the number of heavy drinkers with ulcers and gastrointestinal problems complications,

four $i = 5$, C_5 represent the number of heavy drinkers with immune system dysfunction complications,

four $i = 6$, C_6 represent the number of heavy drinkers with brain damage complications,

four $i = 7$, C_7 represent the number of heavy drinkers with malnourishment and vitamin deficiencies complications,

four $i = 8$, C_8 represent the number of heavy drinkers with osteoporosis complications,

four $i = 9$, C_9 represent the number of heavy drinkers with heart disease complications,

four $i = 10$, C_{10} represent the number of heavy drinkers with accidents and injuries complications,

Quitters of drinking Q :

$$\frac{dQ(t)}{dt} = \alpha_0 H + \sum_{i=1}^{n} \gamma_i C_i - \mu Q \tag{6}$$

$Q(\mathbf{t})$ refers to the individuals who temporarily and permanently quit drinking, is increased by the rates $\alpha_0 H$ and $\sum_{i=1}^{n} \gamma_i C_i$ and decreased by the rate μQ.

The total population size at time t is denoted by $N(t)$ with $N(t)$=$P(t)+M(t)+H(t)+C_1(t)+C_2(t)+.....+C_n(t)+Q(t)$.

We consider system (1) with the following parameter values.

3. Basic Properties

3.1. Invariant Region. It is necessary to prove that all solutions of system (1) with positive initial data will remain positive for all times t > 0. This will be established by the following lemma.

Lemma 1. *All feasible solution $P(t), M(t), H(t), C_1(t), C_2(t).....C_n(t)$ and $Q(t)$ of system equation (1) are bounded by the region*

$$\Omega = \left\{ (P,M,H,C_1,C_2,..,C_n,Q) \in IR_+^6 : P+M+H+C_1+C_2..+C_n+Q \leq \frac{b}{\mu} \right\} \tag{7}$$

Proof. From the system equation(1)

$$\frac{dN(t)}{dt} = \frac{dP(t)}{dt} + \frac{dM(t)}{dt} + \frac{dH(t)}{dt} + \frac{dC_1(t)}{dt} + \frac{dC_2(t)}{dt}..... + \frac{dC_n(t)}{dt} + \frac{dQ(t)}{dt} \tag{8}$$

$$\frac{dN(t)}{dt} = b - \mu N(t) - \delta_0 H - \sum_{i=1}^{n} \delta_i C_i$$

implies that $\frac{dN(t)}{dt} \leq b - \mu N(t)$

It follows that

$$N(t) \leq \frac{b}{\mu} + N(0)e^{-\mu t} \tag{9}$$

Where $N(0)$ is the initial value of total number of people, thus, $\lim_{t\to+\infty} \sup N(t) \leq \frac{b}{\mu}$ Then $P(t)+M(t)+H(t)+C_1(t),.....+C_n(t)+Q(t) \leq \frac{b}{\mu}$

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

$$\Omega = \left\{ (P,M,H,C_1,..,C_n,Q) \in IR^6_+ : P+M+H+C_1+...+C_n+Q \leq \frac{b}{\mu} \right\}$$

Which is a positively invariant set for (1), so we only need to consider dynamics of system on the set Ω non-negative of solutions.

3.2. Positivity of solutions of the model.

Theorem 2. *If* $P(0) \geq 0, M(0) \geq 0, H(0) \geq 0, C_1(0) \geq 0, C_2(0) \geq 0.....,C_n(0) \geq 0$ *and* $Q(0) \geq 0$, *then the solution of system equation*(1) $P(t), M(t), H(t), C_1(t)$ *.....*$,C_n(t)$*and* $Q(t)$ *are positive for all* $t > 0$.

Proof. From the second equation of system (1), we have:

$\frac{dM(t)}{dt} = \beta_1 \frac{PM}{N} - \beta_2 M - \mu M \Rightarrow \frac{dM(t)}{dt} \geq -(\beta_2 + \mu) M$

Using a Gronwall lemma, we have

$$M(t) \geq M(0) \exp[-(\beta_2 + \mu) t] > 0 \tag{10}$$

Similarly, From the third equation of system (1), we have:

$\frac{dH(t)}{dt} = \beta_2 M - (\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i) H \Rightarrow \frac{dH(t)}{dt} \geq -(\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i) H$

implies that

$$H(t) \geq H(0) \exp\left[-(\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i) t \right] > 0 \tag{11}$$

Similarly, From the forth equation of system (1), we have:

$\frac{dC_i(t)}{dt} = \alpha_i H - (\mu + \gamma_i + \delta_i) C_i(t) \Rightarrow \frac{dC_i(t)}{dt} \geq -(\mu + \gamma_i + \delta_i) C_i(t)$

implies that

$$C_i(t) \geq C_i(0)\exp\left[-(\mu+\gamma_i+\delta_i)t\right] > 0 \tag{12}$$

Similarly, From the sixth equation of system (1), we have:
$\frac{dQ(t)}{dt} = \alpha_0 H + \sum_{i=1}^{n} \gamma_i C_i - \mu Q \Rightarrow \frac{dQ(t)}{dt} \geq -\mu Q$
implies that

$$Q(t) \geq Q(0)\exp\left[-\mu t\right] > 0 \tag{13}$$

Therefore, we can see that $P(t) > 0, M(t) > 0, H(t) > 0, C_1(t) > 0, C_1(t) > 0 \ldots\ldots, C_n(t) > 0$ and $Q(t) > 0\ \forall t \geq 0$, this completes the proof.

Since the first tree equations in system (1) are independent of the variables C_i and Q, it is sufficient to consider the following reduced system:

$$\begin{cases} \frac{dP(t)}{dt} = b - \beta_1 \frac{PM}{N} - \mu P \\ \frac{dM(t)}{dt} = \beta_1 \frac{PM}{N} - (\beta_2 + \mu) M \\ \frac{dH(t)}{dt} = \beta_2 M - (\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i) H \end{cases} \tag{14}$$

4. Model Analysis

4.1. Equilibrium states. We first find the equilibrium of the *PMH* model, by setting the right-hand side of the system (14) to zero, we get two equilibrium states, namely the drinking-free state $E^0\left(\frac{b}{\mu}, 0, 0\right)$ and the endemic state $E^*(P^*, M^*, H^*)$.

Where

$$\boxed{P^* = \frac{b}{\mu R_0}} \tag{15}$$

$$\boxed{M^* = \frac{b(R_0-1)}{\beta_1}} \tag{16}$$

$$\boxed{H^* = \frac{b\beta_2(R_0-1)}{\beta_1(\mu+\alpha_0+\delta_0+\sum_{i=1}^{n}\alpha_i)}} \tag{17}$$

$$\boxed{R_0 = \frac{\beta_1}{\mu+\beta_2}} \tag{18}$$

R_0 is the basic reproduction number.

4.2. Local stability analysis.

4.2.1. *The drinking-free equilibrium.* In this section, we analyze the local stability of the drinking-free equilibrium

Theorem 3. *The drinking-free equilibrium* $E^0\left(\frac{b}{\mu},0,0\right)$ *of the system* (16) *is asymptotically stable if* $R_0<1$ *and unstable if* $R_0>1$.

Proof. We now consider the stability local of the drinking-free equilibrium, for the system defined by (14), the matrix Jacobian is given by:

$$J(E)=\begin{pmatrix} -\beta_1\frac{M}{N}-\mu & -\beta_1\frac{P}{N} & 0 \\ \beta_1\frac{M}{N} & \beta_1\frac{P}{N}-\beta_2-\mu & 0 \\ 0 & \beta_2 & -(\mu+\alpha_0+\delta_0+\sum_{i=1}^{n}\alpha_i) \end{pmatrix} \tag{19}$$

The Jacobian matrix for the drinking-free equilibrium is given by:

$$J(E^0)=\begin{pmatrix} -\mu & -\beta_1 & 0 \\ 0 & \beta_1-\beta_2-\mu & 0 \\ 0 & \beta_2 & -(\mu+\alpha_0+\delta_0+\sum_{i=1}^{n}\alpha_i \end{pmatrix} \tag{20}$$

where $P_0=\frac{b}{\mu}=N$.

The characteristic equation of this matrix is given by $\det(J(E^0)-\lambda I_3)=0$ where I_3 is a square identity matrix of order 3.

The following eigenvalues where obtained:

$$\begin{array}{|l|}\hline \lambda_1=-\mu \\ \hline \lambda_2=-(\beta_2+\mu-\beta_1)=-(\mu+\beta_2)\left(1-\frac{\beta_1}{\mu+\beta_2}\right) \\ \hline \lambda_3=-(\mu+\alpha_0+\delta_0+\sum_{i=1}^{n}\alpha_i \\ \hline \end{array} \tag{21}$$

$$R_0=\frac{\beta_1}{\mu+\beta_2} \tag{22}$$

Therefore, all the Eigenvalues of the characteristic equation are negatives if $R_0<1$.

Therefore, we conclude the drinking-free equilibrium is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$.

4.2.2. *Endemic equilibrium.* In this section, we analyze the local stability of the endemic equilibrium.

To find the drinking present equilibrium of the system of equation (16) setting $\frac{dP(t)}{dt} = 0, \frac{dM(t)}{dt} = 0$ and $\frac{dH(t)}{dt} = 0$. provided that at least one of the infected compartments is non zero. We evaluate the equilibrium of system (14) by setting the right-hand side of equation of system (14) to zero and then solve for $P^*, M^*$and H^*.

We obtained system (23) :

$$\begin{cases} \frac{dP(t)}{dt} = b - \beta_1 \frac{PM}{N} - \mu P \\ \frac{dM(t)}{dt} = \beta_1 \frac{PM}{N} - (\beta_2 + \mu) M \\ \frac{dH(t)}{dt} = \beta_2 M - (\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i) H \end{cases} \tag{23}$$

From the fourth equation in the system (23), we have

$$M = \frac{\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i}{\beta_2} H \tag{24}$$

From the second equation in the system (23), we have

$$P^* = N \left[\frac{(\mu + \beta_2)}{\beta_1} \right] \tag{25}$$

$$P^* = \frac{b}{\mu R_0} \tag{26}$$

From the first equation in the system (23), we have

$$M^* = \frac{b(R_0 - 1)}{\beta_1} \tag{27}$$

equation (3) gives

$$H^* = \frac{b\beta_2(R_0 - 1)}{\beta_1(\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i)} \tag{28}$$

Theorem 4. *if $R_0 > 1$, E^* is locally asymptotically stable .*

Proof. We present $E^*(P^*, M^*, H^*)$ as endemic equilibrium of system (23) and $P^* \neq 0, M^* \neq 0$, $H^* \neq 0$

The Jacobian matrix is

$$J(E^*) = \begin{pmatrix} -\beta_1 \frac{M^*}{N} - \mu & -\beta_1 \frac{P^*}{N} & 0 \\ \beta_1 \frac{M^*}{N} & \beta_1 \frac{P^*}{N} - \beta_2 - \mu & 0 \\ 0 & \beta_2 & -(\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i) \end{pmatrix} \tag{29}$$

where

$$\begin{aligned} P^* &= \frac{b}{\mu R_0} \\ M^* &= \frac{b(R_0 - 1)}{\beta_1} \\ H^* &= \frac{b\beta_2(R_0 - 1)}{\beta_1(\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i)} \end{aligned} \tag{30}$$

The eigenvalue of matrix $J(E^*)$ is $\lambda_1 = -(\mu + \alpha_0 + \delta_0 + \sum_{i=1}^{n} \alpha_i)$. The characteristic equation of this matrix is given by $\det(J(E^*) - \lambda I_2) = 0$, where I_2 is a square identity matrix of order 2.

$$P(\lambda) = a_1\lambda^2 + a_2\lambda + a_3 \tag{31}$$

where

$$\begin{aligned} P^* &= \frac{b}{\mu R_0} \\ M^* &= \frac{b(R_0 - 1)}{\beta_1} \end{aligned} \tag{32}$$

$$\begin{aligned} a_1 &= 1 > 0 \\ a_2 &= \beta_1 \frac{M^*}{N} + \mu + \beta_2 + \mu - \beta_1 \frac{P^*}{N} \\ a_2 &= \beta_1 \frac{M^*}{N} + \mu > 0 \ \ if\ R_0 > 1. \\ a_3 &= \left(\beta_1 \frac{M^*}{N} + \mu\right)\left(\beta_2 + \mu - \beta_1 \frac{P^*}{N}\right) + \beta_1^2 \frac{P^*}{N}\frac{M^*}{N} \\ a_3 &= \beta_1^2 \frac{P^*}{N}\frac{M^*}{N} > 0 \ if\ R_0 > 1. \end{aligned}$$

By Routh-Hurwitz criterion [22], the system(2) is locally asymptotically stable if $a_1 > 0$, $a_2 > 0$, $a_3 > 0$ and $a_1 a_2 > a_3$.

The jury criterion [22] implies that he two roots λ_1, λ_2 of the equation $P(\lambda) = 0$ satisfy $|\lambda_i| \leq 0$ for $i = 1, 2$. The linearization theory implies that the positive equilibrium $E^*(P^*, M^*, H^*)$ of system (2) is locally asymptotically stable if $R_0 > 1$, i.e, the endemic equilibrium E^* of system (2) is locally asymptotically stable.

4.3. Global stability.

4.3.1. *Global stability of the drinking-free equilibrium.* We will investigate the global stability of E^0 when $R_0 \leq 1$:

Theorem 5. *If $R_0 \leq 1$, E^0 is globally asymptotically stable .*

Proof. *Consider the following Lyapunov function [...],*

$$V = M \tag{33}$$

$$\begin{aligned} \frac{dV}{dt} &= \frac{dM(t)}{dt} \\ &= \left[\frac{\beta_1 P}{N} - (\mu + \beta_2)\right] M \qquad (34) \\ &= \beta_1 \left[\frac{R_0 P - N}{R_0}\right] M < 0 \qquad (35) \end{aligned}$$

implies that

$$\frac{dV}{dt} < 0 \tag{36}$$

$\frac{dV}{dt} = 0$ *implies that* $M = 0$. *Hence, by Lasalle's invariance principle* [23], E^0 *is globally asymptotically stable.*

4.3.2. *Global stabilty of the endemic equilibrium.* Our final result in this section is for the global stability of E^*.

Theorem 6. *Drinking-present equilibrium point E^* is globally asymptotically stable if $R_0 > 1$.*

Proof. *Consider the Lyapunov function V:*

$$\begin{aligned} V &: \Gamma \to IR \\ V(P,M) &= c_1 \left[P - P^* \ln(\frac{P}{P^*})\right] + c_2 \left[M - M^* \ln(\frac{M}{M^*})\right] \qquad (37) \end{aligned}$$

where c_1 and c_2 are positive constant to be chosen latter and $\Gamma = \{(P,M) \in \Gamma / P > 0, M > 0\}$

Then, the time derivative of the Lyapunov function is given by

$$\frac{dV(P,M)}{dt} = -bc_1\frac{[P-P^*]^2}{PP^*} + \frac{\beta_1}{N}(c_2-c_1)[P-P^*][M-M^*]. \tag{38}$$

Then, the time derivative of the Lyapunov function is given by

$$\frac{dV(P,M)}{dt} = -bc_1\frac{[P-P^*]^2}{PP^*} + \frac{\beta_1}{N}(c_2-c_1)[P-P^*][M-M^*]. \tag{39}$$

For $c_1 = c_2 = 1$, we have

$$\frac{dV(P,M)}{dt} = -b\frac{[P-P^*]^2}{PP^*} \leq 0 \tag{40}$$

Also, we obtain

$$\frac{dV(P,M)}{dt} = 0 \Longrightarrow P = P^* \tag{41}$$

Hence by LaSalle's invariance principle [23] *the free equilibrium point E^* is globally asymptotically stable on* Γ.

4.4. Sensitivity Analysis of R_0. To examine the sensitivity of R_0 to each of its parameters, following normalized forward sensitivity index with respect to each of the parameters is computed [3].

Using the approach in Chitnis et al. [24], we calculate the normalized forward sensitivity indices of R_0. Let

$$A_m^{R_0} = \frac{m}{R_0} * \frac{\partial R_0}{\partial m} \tag{42}$$

denote the sensitivity index of R_0 with respect to the parameter m. We get

$$R_0 = \frac{\beta_1}{\mu+\beta_2} \tag{43}$$

$$A_{\beta_1}^{R_0} = 1 \tag{44}$$

$$A_{\beta_2}^{R_0} = -\frac{\beta_2}{\mu+\beta_2} < 0 \tag{45}$$

$$A_{\mu}^{R_0} = -\frac{\mu}{\mu+\beta_2} < 0 \tag{46}$$

From the above discussion we observe that the basic reproduction number R_0 is most sensitive to changes in β_1. if β_1 will increase R_0 will also increase with same proportion and if β_1 will decrease in same proportion, μ and β_2 have an inversely proportional relationship with R_0.

We conclude that the basic reproduction number (R_0) is most sensitive to changes in β_1. An increase in β_1 will cause an increase in R_0 with same proportion and a decrease in β_1 will cause a decrease in R_0 in same proportion. μ and β_2 have an inversely proportional relationship with R_0, so an increase in any of them will bring about a decrease in R_0. However, the size of the decrease will be proportionally smaller. Recall that μ is the natural death rate of the population. It is clear that increase in either of these rates is neither ethical nor practical. Thus we choose to focus on one parameters: $\beta_1$the transmission rate from potential drinker to moderate drinker. Given R_0's sensitivity to β_1, it seems sensible to focus efforts on the reduction of β_1. In other words, this sensitivity analysis tells us that prevention is better than cure. Efforts to increase prevention are more effective in controlling the spread of habitual drinkers than efforts to increase the numbers of individuals accessing treatment.

Parameter	Description	Sensitivity index
β_1	The effective contact rate	$+1$
μ	The natural death rate	$-\mathbf{0},\mathbf{2}$
β_2	coefficient of transmission the M at H	$-\mathbf{0},\mathbf{1}$

In this section, we illustrate some numerical solutions of model (1) for different values of the parameters to compair it with the qualitative results. We use the following different initial values such that $P+M+H+C_1+C_2.....+C_n+Q=1000$.

5. Numerical Simulations

This section includes the numerical simulation of the model proposition 1 describing the dynamics of excessive alcohol consumption and its complications, the resolution of the system (1) was created using the technique of implicit finite differences of the Gauss-Seidel type developed by Gumel et al [28], presented in [29] and noted GSS1 method. We start with a graphical representation of the equilibrium without consumption E0 = (1000;0;0;0;0), using the estimated values of the parameters shown in Table 1, R_0= 0 and $R_0 \prec 0$ and the state variables initial are chosen as P + M + H + Ci + Q = 1000.

We consider system (1) with the following parameter values.

Table 1: Description of parameters of the model (1).

Parameter	Desciption	value	Source
b	Recruitment rate of potentiel drinkers	100	Assumed
β_1	the transmission rate from P to M	0.02	Assumed
β_2	the transmission rate from M to H	0.160	Assumed
μ	The natural death rate	0.4	Assumed
α_0	The rate of the H they becomes R and Q	0.05	Assumed
α_1	The rate of H reached by with C1	0.04	Assumed
α_2	The rate of H reached by C2	0.03	Assumed
α_3	The rate of H reached by C3	0.03	Assumed
α_4	The rate of H reached by C4	0.04	Assumed
δ_0	The death rate induced by the H	0.02	Assumed
δ_1	The death rate induced by the C1	0.003	Assumed
δ_2	The death rate induced by the C2	0.004	Assumed
δ_3	The death rate induced by the C3	0.025	Assumed
δ_4	The death rate induced by the C4	0.0015	Assumed
γ_1	The cure rate of C1 and R and Q	0.001	Assumed
γ_2	The cure rate of C2 and R and Q	0.002	Assumed
γ_3	The cure rate of C3 and R and Q	0.002	Assumed
γ_4	The cure rate of C3 and R and Q	0.0015	Assumed

Therefore, the solution converges to the equilibrium $E^0(P(0), 0, 0, 0, 0, 0)$. It is clearly globally asymptotically stable as soon as $R0 < 1$, this numerical verification confirms the result stated in model 1 concerning the stability.

Fig: 2. Global asymptotic stability equilibrium without excessive alcohol consumption E0 for different initial values of each variable state,

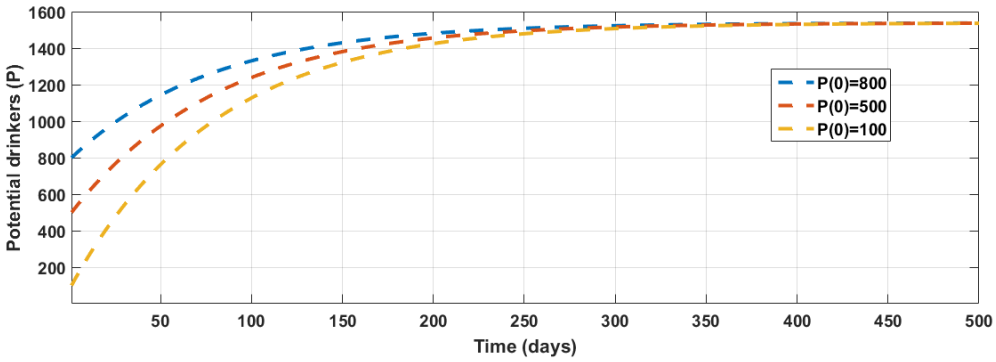

Fig:2(a): the number of potential drinkers increases and approaches the number P(0) =1000

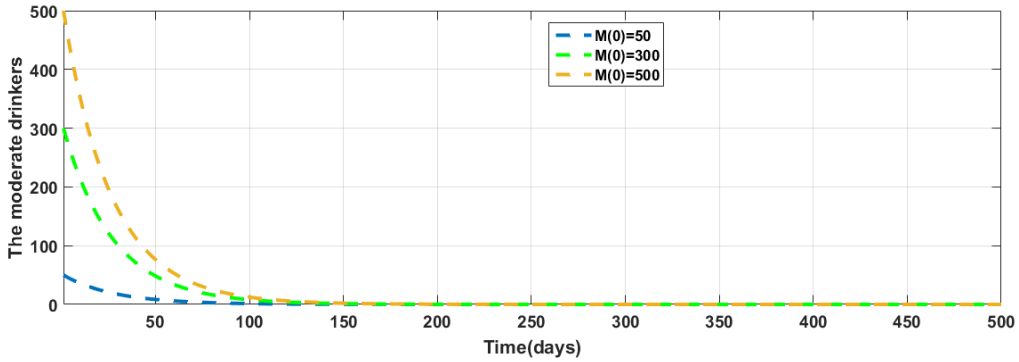

Fig: 2(b)

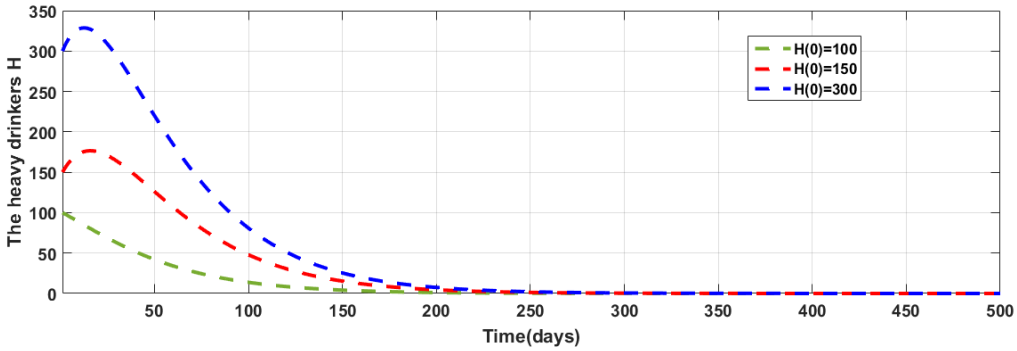

Fig: 2(c)

Fig: 2(b and c) the number of moderate drinkers and the number of haevy drinkers decreases and approaches zero

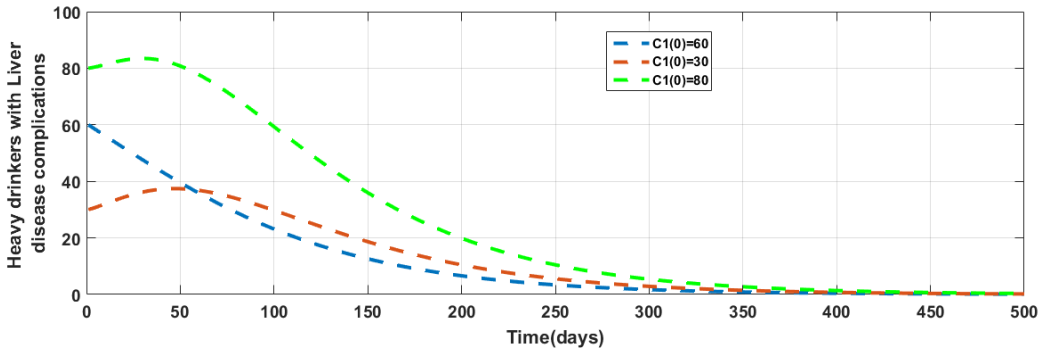

Fig: 2(d)

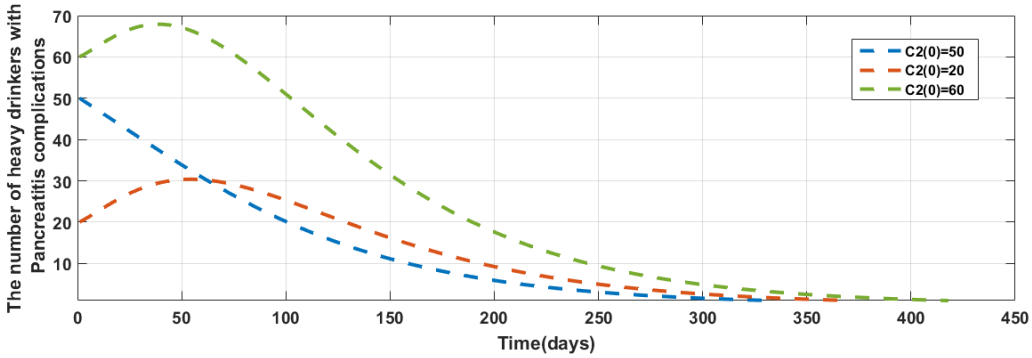

Fig: 2 (e)

Fig: 2(d) the number of heavy drinkers with complications of liver disease is decreasing and approaching zero

Fig: 2(e) the number of heavy drinkers with complications of pancreatitis disease is decreasing and approaching zero

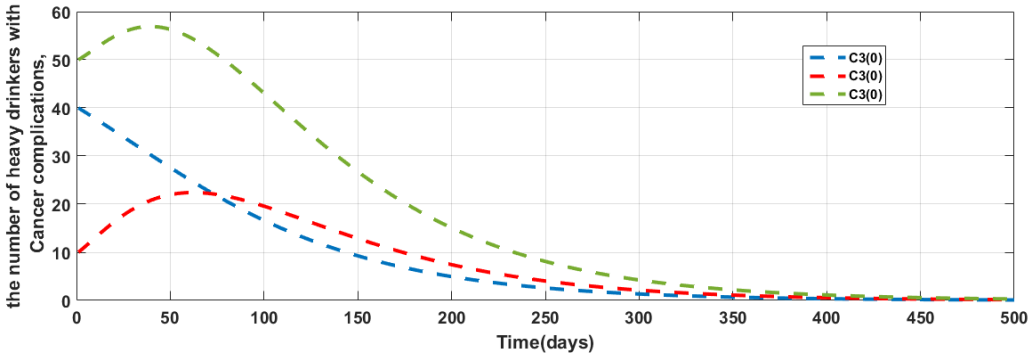

Fig: 2(f)

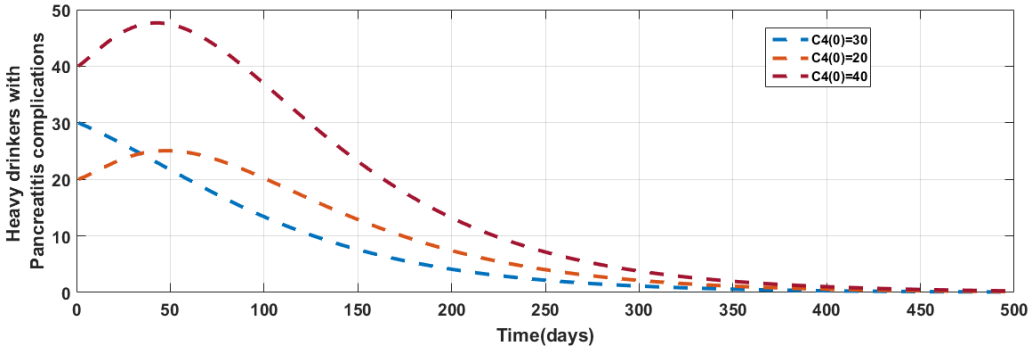

Fig: 2(g)

Fig: 2(f) the number of heavy drinkers with complications of Cancer disease is decreasing and approaching zero

Fig: 2(g) the number of heavy drinkers with disease complications of ulcers and gastrointestinal problems is decreasing and approaching zero

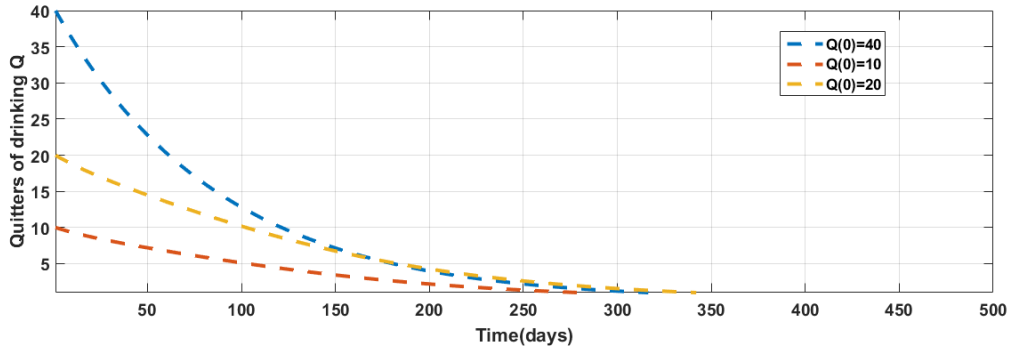

Fig: 2(h) the number of quitters of drinking decreases and approaching zero

We start from a graphic representation of the balance E* of excessive alcohol consumption and its complications and we take the same parameters and different initial values given in table 1, From these ... figures, using the different values of the variables P (0), M(0), H(0), Ci (0), R(0) and Q(0), we obtained the following remarks (Figure 3):

(i) the number of potential drinkers increases and approaches the the value P* = 275(see Figure 3(a))

(ii) the number of moderate drinkers decreases rapidly at first, then increases slightly and approaches the value M* = 400 (see Figure 3(b))

(iii) the number of heavy drinkersdecreases and approaches the value H*=460 (see Figure 2(c)).

(iv) the number of heavy drinkers with complications of liver disease decreases and approaches the value C1*=120 (see Figure 2(d))

(v) the number of heavy drinkers with complications of pancreatitis disease decreases and approaches the value C2*=110 (see Figure 2(e))

(vi) the number of heavy drinkers with complications of Cancer decreases and approaches the value C3*=92.5 (see Figure 2(f))

(vii) the number of heavy drinkers with disease complications of ulcers and gastrointestinal problems decreases and approaches the value C4*=82(see Figure 2(g))

(viii) the number of quitters of drinking decreases and approaches the value Q= 17.5 (see Figure 3(h))

Therefore, the solution curves to the equilibrium E*(P*, M*, H*, C_i*, R*, Q*) when R0>1. Hence, model (1) is globally asymptotically stable.

Fig. 3. **Global asymptotic stability equilibrium with excessive alcohol consumption E0 for different initial values of each variable state,**

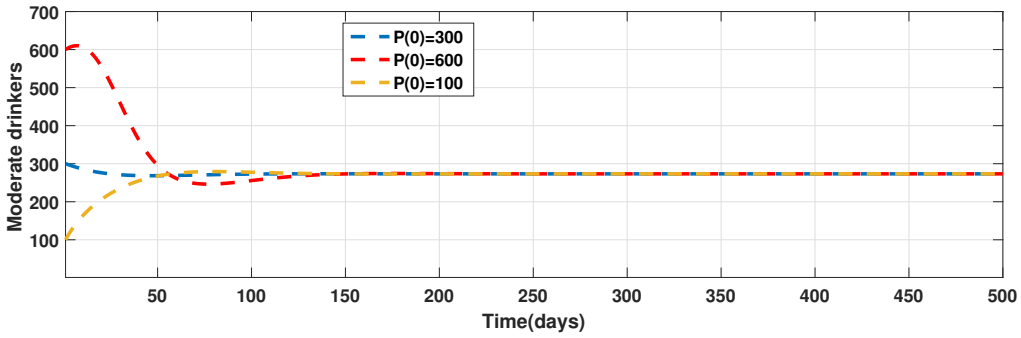

Fig: 3(a)

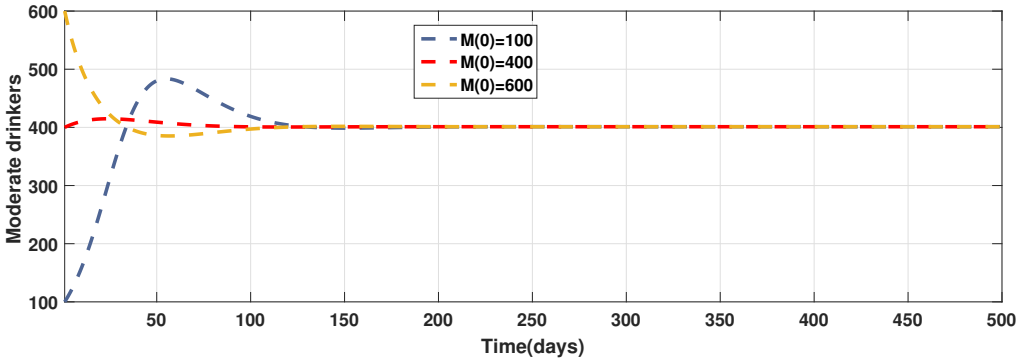

Fig: 3(b)

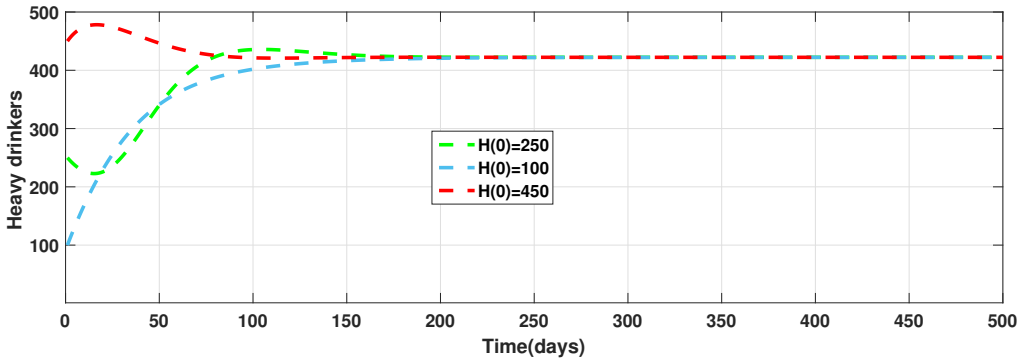

Fig: 3(c)

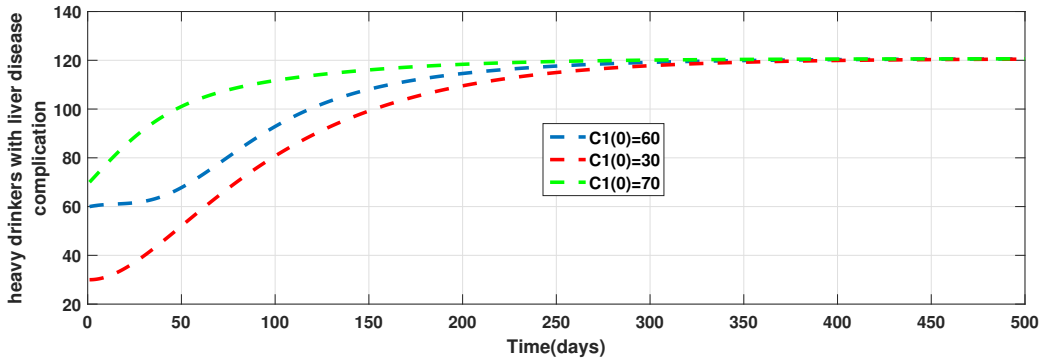

Fig: 3(d)

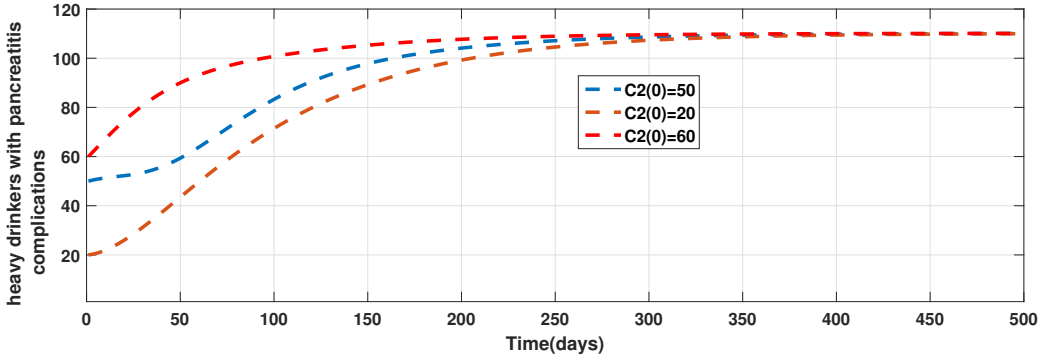

Fig:3(e)

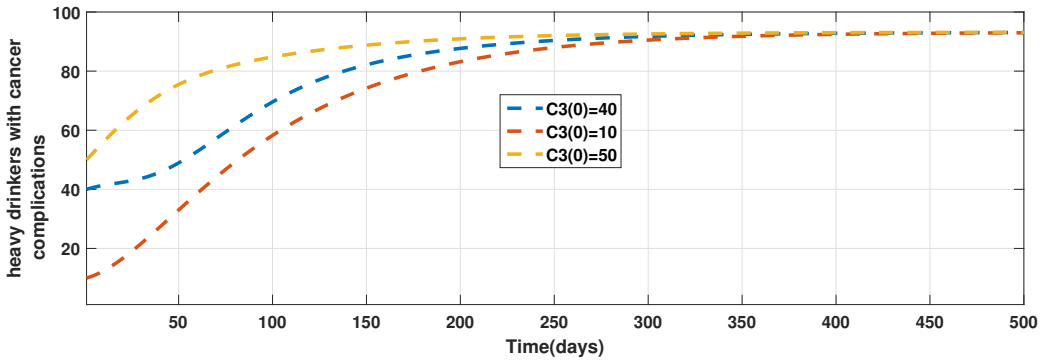

Fig:3(f)

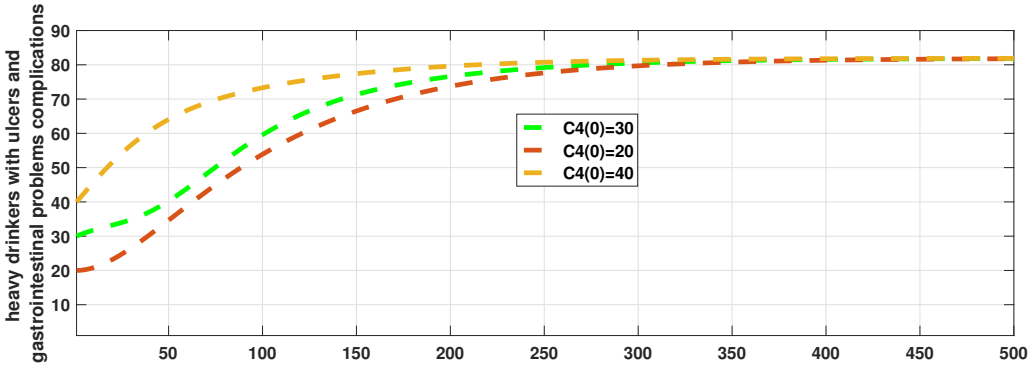

Fig: 3(g)

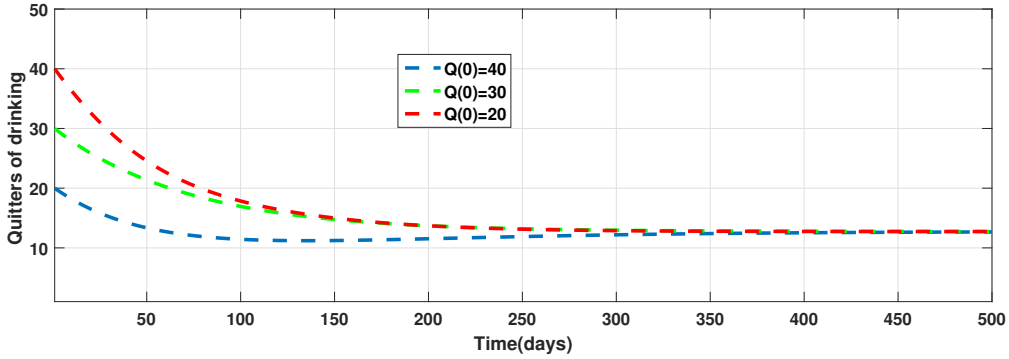

Fig: 3(h)

6. Discussion

In this work, we formulated a continuous mathematical model that describes the population dynamics of heavy drinkers and their complications. We studied the stability of this model using the Routh-Hurwitz criteria to analyze the local stability, we proved that the equilibrium point E0 is local asymptotic stable if $R_0<1$. We also examined the global stability at 1 using the Lyapunov function and we also demonstrated that if R>0 then the equilibrium point with

alcohol consumption is globally asymptotically stable. We calculated the basic reproduction number R_0 and studied the sensitivity analysis of the model parameters to determine which parameters have a high impact on the reproduction number. Numerical simulations are carried out by MATHLAB to illustrate the theoretical results. These findings could be used to inform public policies and public health interventions aimed at reducing excessive alcohol consumption and its health consequences.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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