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Mathematical Modeling and Analysis of Alcoholism Epidemics: A Case Study in Ethiopia

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Abstract In this paper, we developed deterministic mathematical model of alcoholism epidemics. First, the existence and uniqueness of the formulated model will be studied to show the well-possedness of the model. Second, the major qualitative analysis like alcoholic free equilibrium point (E_0), endemic equilibrium point (E^*), basic reproduction number (R_0), were computed. From the stability analysis, we found that an alcoholic free equilibrium point is locally asymptotically stable if $R_0 < 1$. The global asymptotic stability of alcoholic free equilibrium point is established using LaSalle's invariance principle of Lyapunov functions. The sensitivity of model parameters is done using normalized forward sensitivity index. At the end, numerical simulations on the study were conducted using the ODE 45 to confirm our analytic results. It is pointed out that, minimizing the contact rate between the non-drinkers and heavy drinkers, maximizing the number of drinkers that go into treatment can be useful in combating the alcoholism epidemic.

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Introduction

Alcoholism is, broadly, any drinking of alcohol that ends up in significant mental or physical health problems and is answerable for about 6% of mortality and 5% of disability- adjusted life year's lost worldwide [12], [6]. The World Health Organization (WHO) has estimated that, globally, about 53% aged 15 years and above have ever used alcohol and 39% used it the last year [19]. The study that conducted in South Africa, indicates that 9% of the population aged 15 years or above in risk or harmful drinking. When compared their percentage, more men had hazardous drinking than women, 17% and 2.9% respectively [20].

In our country, Ethiopia, different studies were conducted to point the prevalence of alcohol consumption. The studies shown a significant increment, and usually hazardous drinking and alcohol dependence were more in men than women [28, 8]. Excessive alcohol use can damage all organ systems, but it particularly affects the brain, heart, liver, pancreas and immune system. Alcoholism can result in mental illness, delirium tremens, irregular heartbeat, an impaired immune response, liver cirrhosis and increased cancer risk [7, 21]. Women are generally more sensitive than men to the harmful effects of alcohol, orimerily because to

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their smaller body weight, lower capacity to metabolize alcohol, and higher proportion of body fat [18].

Environment and genetics are the two factors within the risk of development of alcoholism with about half the chance attributed to every [7]. Stress and associated disorders, including anxiety, are some factors in the development of alcoholism as alcohol consumption can temporarily reduce dysphoria [14]. Someone with a parent or sibling with an alcohol use disorder is three to four times more likely to develop an alcohol use disorder them- selves, but only a minority of them do. Environment factors includes social, cultural and behavioral influences. People may still to drink partly to stop or improve symptoms of withdrawal [7]. Medically, alcoholism is taken into account both a physical and mental illness [13].
Prevention of alcoholism could also be somewhat difficult but attempted by reducing the experience of stress and anxiety in individuals, limiting the dimensions of alcohol, taxing alcohol to increase its cost, and providing equation (awareness) and treatment [7, 14]. Treatment of alcoholism may take several forms. Because of medical problems which will occur during withdrawal, alcohol cessation should be controlled carefully. Some common method involves the use of benzodiazepine medications, like diazepam, acamprosate, disulfiram or naltrexone may use to prevent further drinking [29, 2].

It is known that alcoholism incorporates a great social and economic impacts on the country. For this case, it is vital to grasp the dynamics of alcoholism spread among the populations. Many studies and research in social, medical, and political has focused on this study areas [19, 4, 5]. Since alcoholism could be a problem of public health importance, not much has been analyzed in terms of using mathematical modeling to inspire its transmission dynamics at population level.
Mathematical models are used to analyze the spread of infectious disease. It is used as a tool to study the transmission dynamics of diseases and to grasp the effect of different intervention strategies to aware public health policy makers on the implementation to combat infections. So far, researchers are studied different mathematical models to investigate the dynamics of transmission and optimal control strategies; see for instance, [1, 27, 26, 9, 30, 10] . As respect to alcoholism, different mathematical models are formulated and studied to cut back the amount of drinkers. There is a really interesting model on alcoholic abuse proposed by Sanchez et al.[22]. During this model author considered a three-compartment drinking epidemic model. The population is split into three classes, namely, moderate and occasional drinkers (susceptible to drinking epidemic), heavy drinkers and recovered population. The model could be a simple model which does not incorporate the relapse from recovered class to susceptible class, which is extremely unrealistic. The author failed to introduce any treatment or intervention policy during this model. There must be an additional death rate for the heavy drinkers. But the authors failed to introduce additional drinking related death rate during this model, which is not realistic.

A similar quite of work was done by Mulone and Straughan [16]. But during this paper also no treatment is introduced. As drinking is may be a chronic relapsing disease, the relapse of drinkers from treatment should be considered to create the work more realistic, which is missing in [16]. Other models done by Mubayi et al.[15] and Santonja et al.
[23] , do not contain a treatment class. They were split the population into three classes depending on the quantity of alcohol a person consumes. Mubayi et al. [15], also focused on the drinking habits of students. They were interested on checking how a change from low to high risk drinking environments affected the transition from susceptible to heavy
drinker. Santonja et al. [23] do not consider a treatment class, they were despite a private who are spending

50 years within the system as their focus so as to see the health and economic costs of risky alcohol consumption.

We have seen different limitations within the above models. To reduce those limitations and by considering current situations of alcoholism in Ethiopia, we have developed a mathematical modeling of alcoholism epidemics by introducing treatment class. We have also considered drinking related additional death rate, since alcohol might be quite deadly. According to Ethiopia, throughout the year alcohol related deaths occur. To beat this, we have got added additional death rate so as to aware the community to stop themselves from excessive drinking. Here our main contribution is that, by introducing treatment class, which is effective way to reduce the proportion of the population within the alcohol problems. We will identify the parameters of great importance which can help the policy- makers in targeting prevention and treatment resources for maximum effectiveness in Ethiopia.

The rest of the paper is organized as follows. In Section 2, mathematical model is formulated and some basic properties are derived by examining equilibrium point of the model. Section 3 is devoted for the stability analysis of the model. The problem of sensitivity's parameter is discussed in Section 4. Section 5 is devoted for numerical simulations of the model by estimating the parameters. The conclusion is presented in Section 6.

Model Formulation and Description

In this study, we formulate a mathematical model for the dynamics of alcoholism and divide the population into four compartments: Non-drinkers S(t), Heavy drinkers H(t), Drinkers in treatment T (t), and Recovered drinkers R(t). Thus, the governing non-linear system of differential equation represents the model is given by

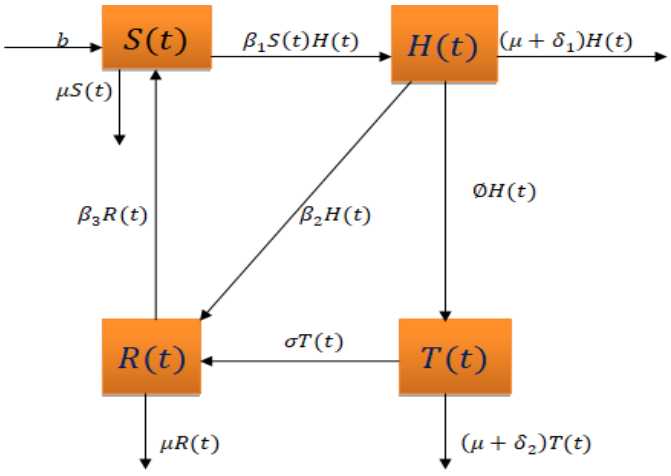

Figure 1: *Compartmental diagram for four drinking classes in the model*

$$\begin{aligned}
\frac{dS(t)}{dt} &= b - \beta_1 S(t)H(t) + \beta_3 R(t) - \mu S(t),\\
\frac{dH(t)}{dt} &= \beta_1 S(t)H(t) - (\beta_2 + \mu + \delta_1 + \phi)H(t),\\
\frac{dT(t)}{dt} &= \phi H(t) - (\delta_2 + \mu + \sigma)T(t),\\
\frac{dR(t)}{dt} &= \sigma T(t) + \beta_2 H(t) - (\beta_3 + \mu)R(t).
\end{aligned} \tag{1}$$

In the model system (1), individuals in the non-drinkers compartment are subjected to a heavy drinker with a contact rate of β_1. From the heavy drinker compartment a portion φH of individuals enter the treatment phase T. The possibility for the individual to die from treatment compartment T infected at the rate of δ_2 due to the drinking in treatment.

The model variables and their descriptions are tabulated in Table 1 and the model parameters and their description are tabulated in Table 2.

Table 1: *Model variables and their description.*

Variables	Description of the variables
$S(t)$	Non-drinker population at time t
$H(t)$	Heavy drinker population at time t
$T(t)$	Drinkers in Treatment population at time t
$R(t)$	Recovered drinker population at time t

Table 2: *Model parameters and their description.*

Parameters	Description of the parameters
B	Birth rate
β_1	The rate of recruitment to alcoholism due to encounters between no drinkers and alcoholics
β_2	The rate of a light problem alcoholic will become a recovered individual due to treatment
β_3	The rate at which recovered drinkers' relapse to no drinkers
μ	The natural death rate which is refer to any death rate not caused by alcohol
Φ	Proportion of drinkers entering treatment compartment
Σ	Recovered rate of Treatment
δ_1	Drinking induced death rate of heavy drinker
δ_2	Drinking induced death rate of drinking in treatment

1.1 Positivity Solution of the Model

In order to show that the model system (1) are to be epidemiologically meaningful and well posed, it is needed to prove that all the state variables are non-negative. This fact has been stated as a theorem and proved in what follows.

Theorem 1. *Let the initial conditions of model system (1) are* $S(0) \geq 0$, $H(0) \geq 0$, $T(0) \geq 0$, $R(0) \geq 0$, *then the solutions* $S(t)$, $H(t)$, $T(t)$, $R(t)$ *of the model system (1) are positively invariant for all* $t \geq 0$.

Proof. Assume that all the state variables are continuous. Then, from the model system (1) of the first equation we have:

$$\frac{dS(t)}{dt} = b - \beta_1 S(t)H(t) + \beta_3 R(t) - \mu S(t).$$

$$\frac{dS(t)}{dt} \geq -\beta_1 S(t)H(t) - \mu S(t),$$

$$\frac{dS(t)}{dt} \geq -\mu S(t).$$

Using separation of variables rule the above inequality becomes:

$$\frac{dS(t)}{S(t)} \geq -\mu dt.$$

Integrating both sides

$$\int \frac{dS(t)}{S(t)} \geq \int -\mu dt,$$

$$\ln S(t) \geq -\mu t + c,$$

$$S(t) \geq e^{-\mu t} e^{c},$$

$$S(t) \geq S(0)e^{-\mu t} \geq 0. \qquad (2)$$

Also, by taking the second equation of the model system (1) we have:

$$\frac{dH(t)}{dt} = \beta_1 S(t)H(t) - (\beta_2 + \mu + \delta_1 + \phi)H(t).$$

$$\frac{dH(t)}{dt} \geq -(\beta_2 + \mu + \delta_1 + \phi)H(t).$$

Now we use separation of variable and taking integration on both sides we have

$$\int \frac{dH(t)}{H(t)} \geq \int -(\beta_2 + \mu + \delta_1 + \phi)dt,$$

$$\ln H(t) \geq -(\beta_2 + \mu + \delta_1 + \phi)t + c,$$

$$H(t) \geq e^{-(\beta_2+\mu+\delta_1+\phi)t}e^{c},$$

$$H(t) \geq H(0)e^{-(\beta_2+\mu+\delta_1+\phi)t} \geq 0. \tag{3}$$

Similarly,

$$T(t) \geq T(0)e^{-(\sigma+\mu+\delta_2)t} \geq 0. \tag{4}$$

$$R(t) \geq R(0)e^{-(\beta_3+\mu)t} \geq 0. \tag{5}$$

Hence, the solution $S(t)$, $H(t)$, $T(t)$, $R(t)$ of the model system (1) are positive for all $t \geq 0$.

□

1.2 Invariant Region

The model under consideration monitors population as such, we assume that all the variables and parameters of the model are positive for all $t \geq 0$. In order to show that the solution of model system (1) is bounded it is needed to prove that the total population size $N(t)$ is bounded.

Theorem 2. All the solutions S(t), H(t), T (t), R(t) of the model system (1) are bounded.

Proof. In order to show that the population sizes of each compartment are bounded we prefer to show that the total population size of the whole system $N(t)$ is bounded. The total population at any time t is given by:

$$N(t) = S(t) + H(t) + T(t) + R(t). \tag{6}$$

Differentiating (6) both sides with respect to time t gives

$$\frac{dN}{dt} = \frac{dS(t)}{dt} + \frac{dH(t)}{dt} + \frac{dT(t)}{dt} + \frac{dR(t)}{dt},$$

$$\frac{dN}{dt} = b - \mu N - \delta_1 H - \delta_2 T. \tag{7}$$

In the absence of mortality due to alcohol (7) becomes:

$$\frac{dN}{dt} \leq b - \mu N. \tag{8}$$

By the separation of variable rule (8) become:

$$\frac{dN}{b - \mu N} \leq dt. \tag{9}$$

Taking integration on both sides, (9) becomes:

$$\int \frac{dN}{b - \mu N} \leq \int dt,$$

$$-\frac{1}{\mu} \ln(b - \mu N) \leq t + c,$$

$$\ln(b - \mu N) \geq -\mu t + c,$$

$$b - \mu N \geq Ae^{-\mu t}, \tag{10}$$

where A is constant. By applying the initial condition $N(0) = N_0$ in (10), we get

$$b - \mu N \geq (b - \mu N_0)e^{-\mu t}, \tag{11}$$

$$N \leq \frac{b}{\mu} - \left(\frac{b - \mu N_0}{\mu}\right) e^{-\mu t}, \tag{12}$$

as $t \to \infty$ in (12) the population size, $N \to \frac{b}{\mu}$ which implies that $0 \leq N \leq \frac{b}{\mu}$. Thus, the feasible solution set of the model system (1) enter and remain in the region Ω for all time t,

$$\Omega = \left\{(S, H, T, R) \in \mathbb{R}_+^4; S, H, T, R \geq 0 : N(t) \leq \frac{b}{\mu}\right\}. \tag{13}$$

Therefore, the model system (1) is well posed epidemiologically and mathematically. Hence, it is sufficient to study the dynamics of the model system in the domain Ω. □

1.3 Alcoholic Free Equilibrium

Let $E_0 = (S^*, H^*, T^*, R^*)$ represents alcoholic free equilibrium point of the model $SHTR$. Alcoholic free equilibrium points are steady state solutions of mathematical model indicating that there is no drinking problem. The compartmental classification of population reveals that the drinker population is distributed only in heavy drinkers and drinkers in treatment compartments. Hence, in the absence of infection we have $H^* = T^* = 0$. To obtain equilibrium point we just set zero in the model system (1), then alcoholic free equilibrium point E_0 will be:

$$b + \beta_3 R^* - \mu S^* = 0, \qquad -(\beta_3 + \mu)R^* = 0.$$

Hence, we have

$$S^* = \frac{b}{\mu}, \quad R^* = 0.$$

Therefore, an alcoholic free equilibrium point, E_0 is

$$(S^*, H^*, T^*, R^*) = \left(\frac{\nu}{\mu}, 0, 0, 0\right). \tag{14}$$

1.4 Determination of Basic Reproduction Number

The basic reproduction number R_0 is a threshold parameter defined as the average number of secondary infections caused by an infectious individual by introducing in to a completely susceptible population. The basic reproductive number provides an invasion criterion for the initial spread of the infection in susceptible population. Now in the present paper, basic reproduction number for the dynamics of alcoholism epidemic model is defined as the number of heavy drinkers produced when a single heavy drinker is introduced into susceptible population. We use next generation matrix approach [31] to define the basic reproduction number R_0. The next generation matrix compromises two matrices F and V, whose elements in matrix are constitute the new infections that will arise and the transfer of infections from one compartment to another respectively. Let us define f_i and v_i as follows:

1. f_i be the rate of appearance of new infection in compartment i.
2. v_i^+ be the rate transfer of individuals into compartment i.
3. v_i^- be the rate transfer of individuals out of compartment i.
4. v_i be transfer of individuals in and out of compartment i. i.e., $v_i = v_i^- - v_i^+$.

Now from the model system (1) the infected compartments to be

$$\begin{aligned} \frac{dH(t)}{dt} &= \beta_1 S(t)H(t) - (\beta_2 + \mu + \delta_1 + \phi)H(t), \\ \frac{dT(t)}{dt} &= \phi H(t) - (\delta_2 + \mu + \sigma)T(t). \end{aligned} \tag{15}$$

From the system (15), we can define f_i and v_i as

$$f_i = \begin{pmatrix} \beta_1 S(t)H(t) \\ 0 \end{pmatrix}, v_i = \begin{pmatrix} (\beta_2 + \mu + \delta_1 + \phi)H(t) \\ (\delta_2 + \mu + \sigma)T(t) - \phi H(t) \end{pmatrix}. \tag{16}$$

Let us take partial differentiation with respect to H and T and evaluate at alcoholic free equilibrium point, E_0

$$F = \frac{\partial f_i}{\partial x_j}(E_0) = \begin{pmatrix} \beta_1 S^* & 0 \\ 0 & 0 \end{pmatrix} = \begin{pmatrix} \frac{b\beta_1}{\mu} & 0 \\ 0 & 0 \end{pmatrix}, V = \frac{\partial v_i}{\partial x_j}(E_0) = \begin{pmatrix} \beta_2 + \mu + \delta_1 + \phi & 0 \\ -\phi & \delta_2 + \mu + \sigma \end{pmatrix}.$$

Therefore, the next generation matrix is given by:

$$FV^{-1} = \begin{pmatrix} \frac{b\beta_1}{\mu(\beta_2+\mu+\delta_1+\phi)} & \frac{b\phi\beta_1}{\mu(\beta_2+\mu+\delta_1+\phi)(\delta_2+\mu+\sigma)} \\ 0 & 0 \end{pmatrix}. \tag{17}$$

Now the eigenvalues of the next generation matrix, FV^{-1} is

$$\lambda_1 = 0, \quad \lambda_2 = \frac{b\beta_1}{\mu(\beta_2 + \mu + \delta_1 + \phi)}. \tag{18}$$

Then the basic reproduction number, $R_0 = \rho(FV^{-1})$, spectral radius (maximum eigen- value) of the matrix of the model is

$$R_0 = \frac{b\beta_1}{\mu(\beta_2 + \mu + \delta_1 + \phi)}. \tag{19}$$

2 Stability Analysis

2.1 Existence of Alcoholic Free Equilibrium

Alcoholic free equilibrium point, E_0 is the state in which the population is free of alcohol. That is $H = 0$, $T = 0$ and $R = 0$. Thus, the model has an alcoholic free equilibrium.

$$E_0 = (S^*, H^*, T^*, R^*) = \left(\frac{b}{\mu}, 0, 0, 0\right). \tag{20}$$

2.2 Local Stability of Alcoholic Free Equilibrium

To determine the stability of the model at an alcoholic free equilibrium point, we will consider the Jacobian matrix of the model system (1) at an alcoholic free equilibrium, E_0:

$$J(E_0) = \begin{pmatrix} -\mu & -\beta_1\frac{b}{\mu} & 0 & \beta_3 \\ 0 & \beta_1\frac{b}{\mu} - (\beta_2+\mu+\delta_1+\phi) & 0 & 0 \\ 0 & \phi & -(\delta_2+\mu+\sigma) & 0 \\ 0 & \beta_2 & \sigma & -(\mu+\beta_3) \end{pmatrix},$$

$$|J(E_0)| = \begin{vmatrix} -\mu & -\beta_1\frac{b}{\mu} & 0 & \beta_3 \\ 0 & \beta_1\frac{b}{\mu} - (\beta_2+\mu+\delta_1+\phi) & 0 & 0 \\ 0 & \phi & -(\delta_2+\mu+\sigma) & 0 \\ 0 & \beta_2 & \sigma & -(\mu+\beta_3) \end{vmatrix}. \qquad (21)$$

Let, $u = (\beta_2 + \mu + \delta_1 + \varphi)$, $v = \delta_2 + \mu + \sigma$ from (21). Then the characteristic equation of (21) becomes:

$$\begin{vmatrix} -\mu-\lambda & -\beta_1\frac{b}{\mu} & 0 & \beta_3 \\ 0 & \beta_1\frac{b}{\mu} - u - \lambda & 0 & 0 \\ 0 & \phi & -v-\lambda & 0 \\ 0 & \beta_2 & \sigma & -(\mu+\beta_3)-\lambda \end{vmatrix} = 0. \qquad (22)$$

Thus, the eigenvalues of the characteristic equation (22) can be

$$(-\mu-\lambda)\begin{vmatrix} \beta_1\frac{b}{\mu} - u - \lambda & 0 & 0 \\ \phi & -v-\lambda & 0 \\ \beta_2 & \sigma & -(\mu+\beta_3)-\lambda \end{vmatrix} = 0,$$

$$= (-\mu-\lambda)(\beta_1\frac{b}{\mu} - u - \lambda)\begin{vmatrix} -v-\lambda & 0 \\ \sigma & -(\mu+\beta_3)-\lambda \end{vmatrix} = 0,$$

$$= (-\mu-\lambda)(\beta_1\frac{b}{\mu} - u - \lambda)(-v-\lambda)(-(\mu+\beta_3)-\lambda) = 0,$$

$$\lambda_1 = -\mu, \quad \lambda_2 = \beta_1\frac{b}{\mu} - u, \quad \lambda_3 = -v, \quad \lambda_4 = -(\mu+\beta_3). \qquad (23)$$

Here, λ_1, λ_3 and λ_4 are clearly real and negative. Now, alcoholic free equilibrium, E_0 is stable if $\lambda_2 < 0$. That is

$$\beta_1\frac{b}{\mu} - u < 0 \Rightarrow \beta_1\frac{b}{\mu u} < 1 \Rightarrow \frac{b\beta_1}{\mu(\beta_2+\mu+\delta_1+\phi)} < 1 \Rightarrow R_0 < 1.$$

Therefore, the model system (1) is locally asymptotically stable at alcoholic free equilibrium, $E_0 = (\frac{b}{\mu}, 0, 0, 0)$ if $R_0 < 1$. So, we arrive to the following result:

Theorem 3. *Alcoholic free equilibrium, E_0 of the model system (1) is locally asymptotically stable if $R_0 < 1$.*

2.3 Global Stability of Alcoholic Free Equilibrium

In this section, we study the global properties of alcoholic free equilibrium. Let us consider the Lyapunov function,

$$L(S, H, T, R) : \mathbb{R}^4_+ \to \mathbb{R}^4_+ \quad \text{defined as:}$$

$$V = H + T. \tag{24}$$

differentiating (24) with respect to t and substituting the respective equations in the model system (1), we have:

$$\frac{dV}{dt} = \frac{dH}{dt} + \frac{dT}{dt} = \beta_1 SH - (\beta_2 + \mu + \delta_1 + \phi)H + \phi H - (\delta_2 + \mu + \sigma)T.$$

Since $S \leq S^*$, and $H = \frac{(\delta_2+\mu+\sigma)T}{\phi}$, we get:

$$\frac{dV}{dt} \leq \beta_1 S^* H - (\beta_2 + \mu + \delta_1 + \phi)H.$$

$$\frac{dV}{dt} = H\left(\frac{\beta_1 b}{\mu} - (\beta_2 + \mu + \delta_1 + \phi)\right),$$

$$\frac{dV}{dt} = H(\beta_2 + \mu + \delta_1 + \phi)\left(\frac{\beta_1 b}{\mu(\beta_2 + \mu + \delta_1 + \phi)} - 1\right),$$

$$\frac{dV}{dt} = H(\beta_2 + \mu + \delta_1 + \phi)(R_0 - 1).$$

So, $\frac{dV}{dt} \leq 0$ if $R_0 < 1$. Furthermore $\frac{dV}{dt} = 0$, if and only if $H = 0$. This implies that the only trajectory of the model system (1) on which $\frac{dV}{dt} \leq 0$ is E_0. Therefore, by LaSalle's invariance principle [11], E_0 is globally asymptotically stable in the region R^4.
So, summarizing the above discussions we come to the following result:

Theorem 4. *An alcoholic free equilibrium, E_0 of the model system (1) is globally asymptotically stable if $R_0 < 1$.*

2.4 Existence of Endemic Equilibrium

If drinking persists with in the population (i.e $S(t) \geq 0$, $H(t) \geq 0$, $T(t) \geq 0$, $R(t) \geq 0$), then the model has an equilibrium called endemic equilibrium denoted by $E^* = (S^*, H^*, T^*, R^*) \neq 0$. It can

be obtained by equating each of the model system (1) to zero. That is

$$\frac{dS}{dt} = \frac{dH}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0.$$

Then, by some simple calculation we obtain

$$\begin{aligned} S^* &= \frac{\beta_2 + \mu + \delta_1 + \phi}{\beta_1}, \\ H^* &= \frac{\mu(\mu + \delta_1 + \phi + \beta_2) - \beta_1 b}{\beta_1 \left[\beta_3 - (\beta_2 + \mu + \delta_1 + \phi)\right]}, \\ T^* &= \frac{\phi \left[\mu(\beta_2 + \mu + \delta_1 + \phi) - \beta_1 b\right]}{\beta_1(\mu + \delta_2 + \sigma)\left[\beta_3 - (\beta_2 + \mu + \delta_1 + \phi)\right]}, \\ R^* &= \frac{\sigma\phi \left[\mu(\beta_2 + \mu + \delta_1 + \phi) - \beta_1 b\right]}{\beta_1(\mu + \beta_3)(\mu + \delta_2 + \sigma)\left[\beta_3 - (\beta_2 + \mu + \delta_1 + \phi)\right]}. \end{aligned} \tag{25}$$

2.5 Local Stability of Endemic Equilibrium

To investigate the stability of the system at endemic equilibrium, we will consider the Jacobian matrix of the model system (1) at endemic equilibrium, E^*.

$$J(E^*) = \begin{pmatrix} -\beta_1 \left[\left(\frac{\mu(\mu+\delta_1+\phi+\beta_2)-\beta_1 b}{\beta_1[\beta_3-(\beta_2+\mu+\delta_1+\phi)]}+\right)+\mu\right] & -(\beta_2+\mu+\delta_1+\phi) & 0 & \beta_3 \\ \beta_1\left(\frac{\mu(\mu+\delta_1+\phi+\beta_2)-\beta_1 b}{\beta_1[\beta_3-(\beta_2+\mu+\delta_1+\phi)]}\right) & 0 & 0 & 0 \\ 0 & \phi & -(\delta_2+\mu+\sigma) & 0 \\ 0 & \beta_2 & \sigma & -(\mu+\beta_3) \end{pmatrix}. \tag{26}$$

Let

$$A_{11} = -\beta_1 \left[\left(\frac{\mu(\mu+\delta_1+\phi+\beta_2)-\beta_1 b}{\beta_1\left[\beta_3-(\beta_2+\mu+\delta_1+\phi)\right]}+\right)+\mu\right], A_{12} = -(\beta_2+\mu+\delta_1+\phi), A_{13} = 0, A_{14} = \beta_3,$$

$$A_{21} = \beta_1\left(\frac{\mu(\mu+\delta_1+\phi+\beta_2)-\beta_1 b}{\beta_1\left[\beta_3-(\beta_2+\mu+\delta_1+\phi)\right]}\right), A_{22} = 0, A_{23} = 0, A_{24} = 0,$$

$$A_{31} = 0, A_{32} = \phi, A_{33} = -(\delta_2+\mu+\sigma), A_{34} = 0,$$

$$A_{41} = 0, A_{42} = \beta_2, A_{43} = \sigma, A_{44} = -(\mu+\beta_3).$$

Substituting A_{ij} into (26), we obtain

$$J(E^*) = \begin{pmatrix} A_{11} & A_{12} & A_{13} & A_{14} \\ A_{21} & A_{22} & A_{23} & A_{24} \\ A_{31} & A_{32} & A_{33} & A_{34} \\ A_{41} & A_{42} & A_{43} & A_{44} \end{pmatrix}. \tag{27}$$

Then the characteristic equation of (27) is

$$\lambda^4+(\text{trace of } J(E^*))\lambda^3+(A_{12}A_{21}-A_{33}A_{44}-A_{11}A_{44}-A_{22}A_{44}-A_{11}A_{33}-A_{22}A_{33}-A_{11}A_{22})\lambda^2+$$

$$(A_{11}A_{33}A_{44}+A_{22}A_{33}A_{44}+A_{11}A_{22}A_{44}+A_{11}A_{22}A_{33}-A_{12}A_{21}A_{33}-A_{12}A_{21}A_{44})\lambda+$$

$$(A_{12}A_{21}A_{33}A_{44}+A_{14}A_{21}A_{32}A_{43}-A_{11}A_{22}A_{33}A_{44})=0.$$

We can write the above equation as:

$$\lambda^4+f_1\lambda^3+f_2\lambda^2+f_3\lambda+f_4=0, \tag{28}$$

where,

$$f_1=A_{11}+A_{22}+A_{33}+A_{44},\ f_2=A_{12}A_{21}-A_{33}A_{44}-A_{11}A_{44}-A_{22}A_{44}-A_{11}A_{33}-A_{22}A_{33}-A_{11}A_{22},$$

$$f_3=A_{11}A_{33}A_{44}+A_{22}A_{33}A_{44}+A_{11}A_{22}A_{44}+A_{11}A_{22}A_{33}-A_{12}A_{21}A_{33}-A_{12}A_{21}A_{44},$$

$$f_4=A_{12}A_{21}A_{33}A_{44}+A_{14}A_{21}A_{32}A_{43}-A_{11}A_{22}A_{33}A_{44}.$$

Using the Routh-Hurwitz criterion [17], it can be seen that all eigenvalues of the characteristic equation (28) has negative real part if and only if

$$f_1>0, f_4>0, f_1f_2-f_3>0 \quad \text{and} \quad (f_1f_2-f_3)f_3-f_1^2f_4>0. \tag{29}$$

Thus, we have the following result:

Theorem 5. *The endemic equilibrium, E^* of the model system (1) is locally asymptotically stable if and only if inequalities (29) are satisfied.*

2.6 Global Stability of Endemic Equilibrium

Let as construct the Lyapunov function as follow:

$$L(S^*, H^*, T^*, R^*) = S - S^* + S^* \ln\left(\frac{S^*}{S}\right) + H - H^* + H^* \ln\left(\frac{H^*}{H}\right) + T - T^* + T^* \ln\left(\frac{T^*}{T}\right) + R - R^* + R^* \ln\left(\frac{R^*}{R}\right). \tag{30}$$

Differentiating (30) we obtain:

$$\frac{dL}{dt} = \left(\frac{S-S^*}{S}\right)\frac{ds}{dt} + \left(\frac{H-H^*}{H}\right)\frac{dH}{dt} + \left(\frac{T-T^*}{T}\right)\frac{dT}{dt} + \left(\frac{R-R^*}{R}\right)\frac{dR}{dt},$$

$$\frac{dL}{dt} = \left(\frac{S-S^*}{S}\right)[b - \beta_1 S(t)H(t) + \beta_3 R(t) - \mu S(t)] + \left(\frac{H-H^*}{H}\right)[\beta_1 S(t)H(t) - (\beta_2 + \mu + \delta_1 + \phi)H(t)] + \left(\frac{T-T^*}{T}\right)[\phi H(t) - (\delta_2 + \mu + \sigma)T(t)] + \left(\frac{R-R^*}{R}\right)[\sigma T(t) + \beta_2 H(t) - (\beta_3 + \mu)R(t)]. \tag{31}$$

Then by simplifying (31) we get:

$$\frac{dL}{dt} = \frac{Sb + \beta_3 RS - bS^* - \beta_3 RS^*}{S} + \beta_1 S^* H + \mu S^* - \beta_1 SH - \mu S + \beta_1 SH + (\beta_2 + \mu + \phi + \sigma)H^* - (\beta_2 + \mu + \phi + \sigma)H - \beta_1 SH^* + \frac{\phi HT - \phi HT^*}{T} - (\delta_2 + \mu + \sigma)T + (\delta_2 + \mu + \sigma)T^* + \frac{\sigma TR + \beta_2 HR - \sigma TR^* - \beta_2 HR^*}{R} - \beta_3 R + \beta_3 R^* - \mu R + \mu R^*,$$

$$\frac{dL}{dt} = b + \beta_3 R + \beta_1 S^* H + \mu_s S^* - \frac{bS^*}{S} - \frac{\beta_3 RS^*}{S} - \beta_1 SH - \mu_s S + B_1 SH + (\beta_2 + \mu + \phi + \sigma)H^* - (\beta_2 + \mu + \phi + \sigma)H - \beta_1 SH^* + \phi H + (\delta_2 + \mu + \sigma)T^* - \frac{\phi HT^*}{T} - (\delta_2 + \mu + \sigma)T + \sigma T + \beta_2 H + \beta_3 R^* + \mu R^* - \mu R - \beta_3 R - \frac{\sigma TR^*}{T} - \frac{\beta_2 HR^*}{T}. \tag{32}$$

Let M be the positive terms and N be the negative terms of (32). That is

$$M = b + \beta_3 R + \beta_1 S^* H + \mu S^* + \beta_1 SH + (\beta_2 + \mu + \phi + \sigma)H^* + \beta_1 SH^* + \phi H + (\delta_2 + \mu + \sigma)T^* + \sigma T + \beta_2 H + \beta_3 R^* + \mu R^*.$$

$$N = \frac{bS^*}{S} + \frac{\beta_3 RS^*}{S} + \beta_1 SH + \mu S + (\beta_2 + \mu + \phi + \sigma)H + \beta_1 SH^* + \frac{\phi ST^*}{T} + (\delta_2 + \mu + \sigma)T + \mu R + \beta_3 R + \frac{\sigma TR^*}{T} + \frac{\beta_2 HR^*}{T}.$$

Then (32) becomes:

$$\frac{dL}{dt} = M - N.$$

If $M < N$, then $\frac{dL}{dt} < 0$ and $\frac{dL}{dt} = 0$ if and only if $S = S^*$, $H = H^*$, $T = T^*$ and $R = R^*$.

The largest compact invariant set in $(S^*, H^*, T^*, R^*) \in R^4 : \frac{dL}{dt} = 0$ is the singleton of E^*.

It implies that E^* globally asymptotically stable in R^4 if $M < N$ by LaSalle's invariant principle [11].

3 Sensitivity Analysis of R_0

Sensitivity analysis is commonly used to determine the model robustness to parameter values, that is, to help us know the parameters that have a high impact on the reproduction number R_0. Using the approach of [3], we calculate the normalized forward sensitivity indices of R_0. Let

$$\Gamma_m^{R_0} = \frac{m}{R_0}\frac{\partial R_0}{\partial m}, \tag{33}$$

denote the sensitivity index of $R_0 = \frac{b\beta_1}{\mu(\beta_2+\mu+\delta_1+\phi)}$ with respect to the parameter m. We

obtain

$$\begin{aligned}
\Gamma_{\beta_1}^{R_0} &= 1 \geq 0, \\
\Gamma_{\mu}^{R_0} &= \frac{-(\beta_2 + 2\mu + \delta_1 + \phi)}{b\beta_1(\beta_2 + \mu + \delta_1 + \phi)} \leq 0, \\
\Gamma_{\delta_1}^{R_0} &= \frac{-\delta_1}{b\beta_1(\beta_2 + \mu + \delta_1 + \phi)} \leq 0, \\
\Gamma_{\phi}^{R_0} &= \frac{-\phi}{b\beta_1(\beta_2 + \mu + \delta_1 + \phi)} \leq 0, \\
\Gamma_{\beta_2}^{R_0} &= \frac{-\beta_2}{b\beta_1(\beta_2 + \mu + \delta_1 + \phi)} \leq 0.
\end{aligned} \tag{34}$$

And it is similar with respect to the remaining parameters.

From the above discussion, we observe that the basic reproduction number R_0 is most sensitive to changes in β_1. If β_1 increases R_0 will also increase with the same proportion, and if β_1 decreases R_0 will also decrease in the same proportion. μ, δ_1, φ and β_2 have an inversely proportional relationship with R_0, so an increase in any of them will cause a decrease in R_0. The increase in either of the rates μ, the natural death rate of the population, δ_1, the drinking induced related death rate of heavy drinkers and β_2, is rate of a light problem alcoholic will become a recovered individual due to treatment is neither ethical nor practical. Thus, we choose to focus on one of two parameters: either φ, the proportion of drinkers who enter treatment or β_1, the rate of transmission from no drinkers to heavy drinkers. As R_0 is more sensitive to changes in β_1 than φ, 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 drinking than efforts to increase the numbers of individual accessing treatment.

4 Numerical Simulations

To demonstrate the theoretical results obtained in this paper, some numerical simulations will be discussed. In this paper, the hypothetical set of initial values (IV) and parameter values will be given as follows.

First, let us consider for the case when $R_0 < 1$ using the parameter values given in Table 3.

Table 3: *Table of parameter symbols and their value*

Parameter	Parameter values	Sources
B	0.4	[25]
β_1	0.7	[24]
β_2	0.2	Assumed

β_3	0.1	Estimated
μ	0.25	[25]
Φ	0.7	[24]
Σ	0.09	[24]
δ_1	0.35	[25]
δ_2	0.03	Estimated

From the data in Table 3, we get the reproduction number of an alcoholic free equilibrium is estimated to be $R_0 = 0.74666667 < 1$. Now here we have only non-drinkers population is present and the heavy drinkers, drinkers in treatment and recovered drinkers' population reduce to zero. This means that the model system is asymptotically stable at $R_0 < 1$ and this indicates that alcoholic free equilibrium E_0 is asymptotically stable. This has been verified numerically in Figure 2.

4.1 Effects of contact rate on heavy drinkers

Here, the simulation results of the effect of contact rate (β_1) on the heavy drinkers was shown. The simulation is displayed by varying the contact rate and by keeping other parameters as constant. From Figure 3, we observe that when the value of contact rate is large, there is high possibility for the individual to be heavy drinker. Therefore, the community must reduce the contact rate in order to minimize the number of drinkers between the population.

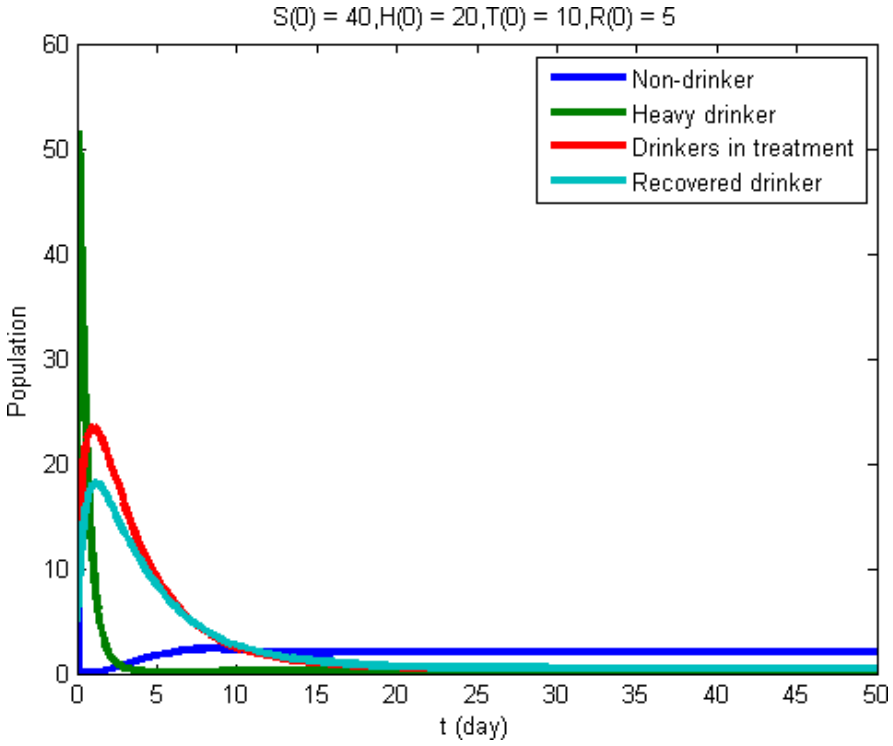

Figure 2: Only non-drinkers exist

4.2 Effects of recovered rate on drinkers in treatment

We investigate the simulation results of impact of recovered rate on drinkers in treatment $T(t)$. Here, as we see it from Figure 4, the heavy drinkers and drinkers in treatment become reduced by increasing the recovered rate of treatment (σ). This indicates that once the individual is at a heavy drinker and drinkers at treatment class, the stake holders must increase the treatment methods to recover the individuals. Therefore, by increasing the recovered rate for drinkers in treatment class, and goes into recovered drinkers we will eliminate alcoholism epidemic from the community. Next, considering the case when $R_0 > 1$, the reproduction number of the endemic equilibrium is to be $R_0 = 1.74545 > 1$ from the data in Table 4. Here the reproduction number greater than one shows that there is coexistence in the population. That is non-drinkers, heavy drinkers, drinkers in treatment

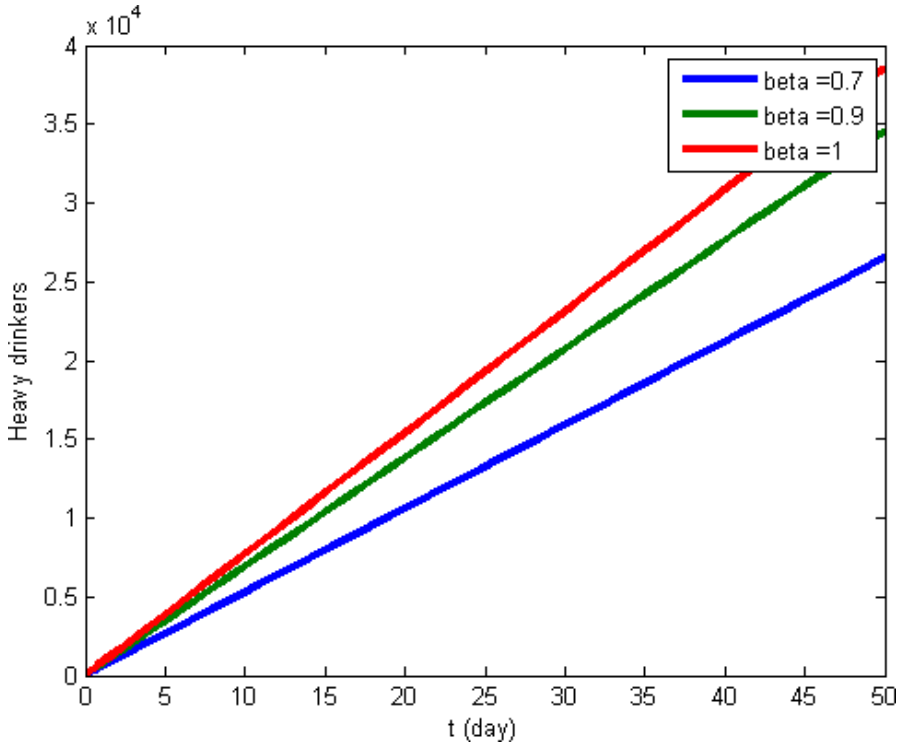

Figure 3: Effect of contact rate on heavy drinkers

Table 4: *Table of parameter symbols and their value*

Parameter	Parameter values	Sources
b	0.9	Assumed
β_1	0.8	[25]
β_2	0.6	Assumed
β_3	0.01	[24]
μ	0.25	[25]
φ	0.5	[24]
σ	0.01	[24]
δ_1	0.3	Assumed
δ_2	0.03	Estimated

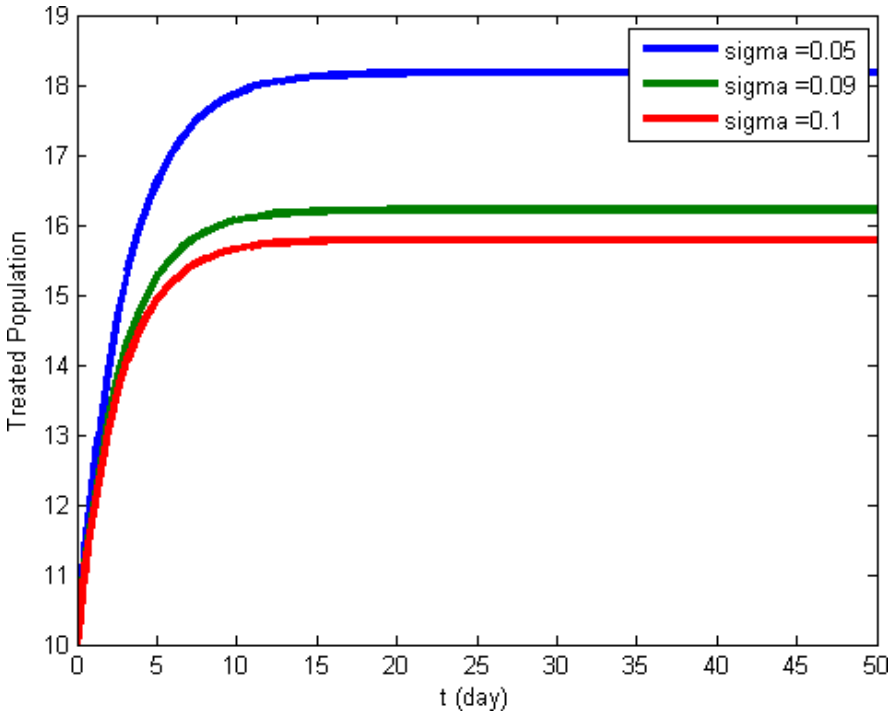

Figure 4: Effect of contact rate on heavy drinkers

and recovered drinkers coexist, which is drinking problem exist in the population or community. Moreover, people with drinking problem continues to transform more non- drinkers into heavy drinkers and drinking free equilibrium is unstable. We verified this numerically in Figure 5.

5 Conclusions

In this work, we presented a deterministic model for an alcoholism epidemic consisting of four compartments, namely, non-drinkers, heavy drinkers, drinkers in treatment and recovered drinkers. Since, alcoholism by nature causes serous health complications, dam- aging nearly every organ and systems in the body. This paper is devoted to implement

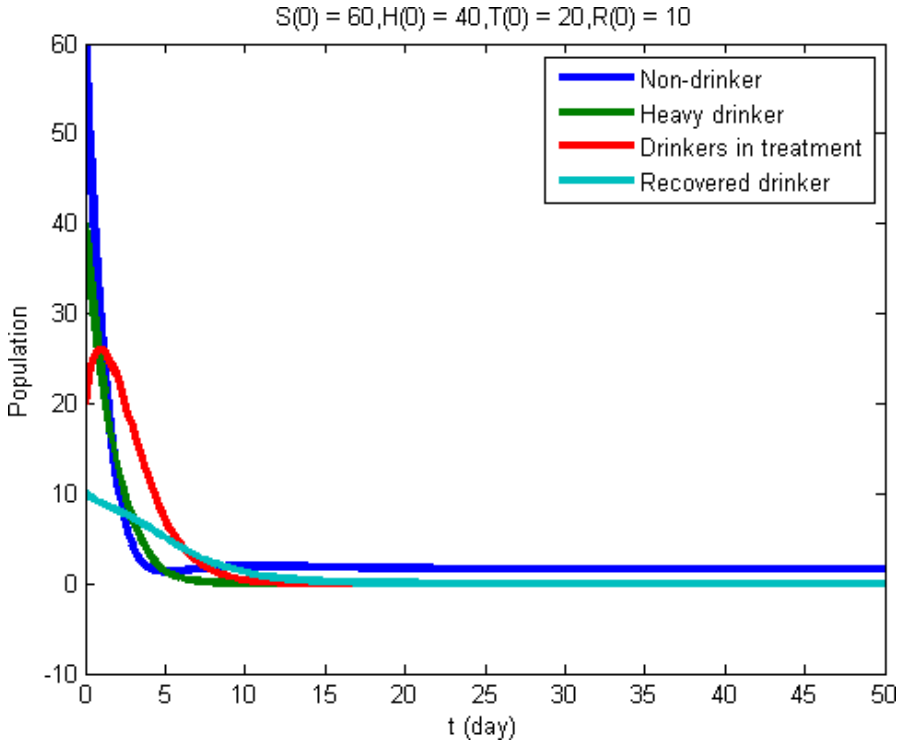

Figure 5: Drinking problem exist in the population

alcoholism mathematical model by introducing treatment class and adding additional death rate. The basic reproduction number of a model system (1) is calculated by using the next generation matrix method. Which is a product of birth rate and the trans- mission coefficient from non-drinkers to heavy drinkers (β_1) divided by the product of natural death rate of population (μ) and recovered individual due to treatment (β_2), natural death rate of population (μ), drinking induced death rate of heavy drinkers (δ_1) and the proportion of individuals who enter treatment (φ).

Then we have discussed the stability analysis of the model system (1) using the basic reproduction number. We analyzed existence and stability of alcohol-free equilibrium point driven. We have found the alcoholic free equilibrium point is locally asymptotically stable when $R_0 < 1$. Also, endemic equilibrium point of the model has been driven and it is globally asymptotically stable when $R_0 < 1$. Sensitivity analysis of R_0 shows that

β_1, the transmission coefficient from non-drinkers to heavy drinkers, as the most useful parameter to the basic reproduction number.

Simulation study and analysis of the model are performed. All our mathematical findings are numerically verified with the help of MATLAB which support our analytical results. From our numerical results we found that alcoholism epidemic can be controlled by minimizing contact rate between the non-drinkers and heavy drinkers and also maximizing the number of drinkers that go into treatment can be useful in combating the epidemic.

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Consent for publication

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References

[1] SO Adewale, GA Adeniran, IA Olopade, SO Ajao, and IT Mohamme. Mathematical and sensitivity analysis of the dynamical spread of cholera. *International Journal of Innovation and Applied Studies*, 19(1):46, 2017.

[2] Richard D Blondell. Ambulatory detoxification of patients with alcohol dependence. *American family physician*, 71(3):495–502, 2005.

[3] Sally M Blower and Hadi Dowlatabadi. Sensitivity and uncertainty analysis of com- plex models of disease transmission: an hiv model, as an example. *International Statistical Review/Revue Internationale de Statistique*, pages 229–243, 1994.

[4] Kate B Carey. Heavy drinking contexts and indices of problem drinking among college students. *Journal of Studies on Alcohol*, 56(3):287–292, 1995.

[5] Vivian M Gonzalez, R Lorraine Collins, and Clara M Bradizza. Solitary and social heavy drinking, suicidal ideation, and drinking motives in underage college drinkers. *Addictive Behaviors*, 34(12):993–999, 2009.

[6] Linda R Gowing, Robert L Ali, Steve Allsop, John Marsden, Elizabeth E Turf, Robert West, and John Witton. Global statistics on addictive behaviors: 2014 status report. *Addiction*, 110(6):904–919, 2015.

[7] Craig Harper et al. Diagnostic and statistical manual of mental disorders. 2014.

[8] D Kebede and A Alem. The epidemiology of alcohol dependence and problem drinking in addisababa, ethiopia. *Acta Psychiatrica Scandinavica*, 100:30–34, 1999.

[9] Ashenafi Kelemu Mengistu and Peter J Witbooi. Modeling the effects of vaccination and treatment on tuberculosis transmission dynamics. *Journal of Applied Mathematcs*, 2019, 2019.

[10] Doyo Kereyu and Seleshi Demie. Transmission dynamics model of tuberculosis with optimal control strategies in haramaya district, ethiopia. *Advances in Difference Equations*, 2021(1):1–22, 2021.

[11] Joseph P La Salle. *The stability of dynamical systems*. SIAM, 1976.

[12] Jill Littrell. *Understanding and treating alcoholism: volume I: an empirically based Clinician's handbook for the treatment of alcoholism: volume ii: biological, psychological, and social aspects of alcohol consumption and Abuse*. Psychology Press, 2014.

[13] David J Mersy. Recognition of alcohol and substance abuse. *American family physician*, 67(7):1529–1532, 2003.

[14] Sachin Moonat and Subhash C Pandey. Stress, epigenetics, and alcoholism. *Alcohol research: current reviews*, 2012.

[15] Anuj Mubayi, Priscilla E Greenwood, Carlos Castillo-Chavez, Paul J Gruenewald, and Dennis M Gorman. The impact of relative residence times on the distribution of heavy drinkers in highly distinct environments. *Socio-Economic planning sciences*, 44(1):45–56, 2010.

[16] Giuseppe Mulone and Brian Straughan. Modeling binge drinking. *International Journal of Biomathematics*, 5(01):1250005, 2012.

[17] Geert Jan Olsder and Jacob Willen van der Woude. *Mathematical systems theory*, volume 4. VSSD Delft, 2005.

[18] World Health Organization. *Global status report on alcohol and health 2018*. World Health Organization, 2019.

[19] World Health Organization et al. Who. global status report on alcohol and health 2014. geneva: World health organization; 2014.[cited 2017 jun 17].

[20] K Peltzer, A Davids, and P Njuho. Alcohol use and problem drinking in south africa: findings from a national population-based survey. *African journal of psychiatry*, 14(1), 2011.

[21] Javier Romeo, Julia Wärnberg, Esther Nova, Ligia E D´ıaz, Sonia Gómez-Martinez, and Ascensión Marcos. Moderate alcohol consumption and the immune system: a review. *British Journal of Nutrition*, 98(S1):S111–S115, 2007.

[22] Fabio Sánchez, Xiaohong Wang, Carlos Castillo-Chávez, Dennis M Gorman, and Paul J Gruenewald. Drinking as an epidemica simple mathematical model with recovery and relapse. In *Therapist's Guide to Evidence-Based Relapse Prevention*, pages 353–368. Elsevier, 2007.

[23] Francisco-José Santonja, Emilio Sánchez, Mar´ıa Rubio, and José-Luis Morera. Alcohol consumption in spain and its economic cost: a mathematical modeling approach. *Mathematical and Computer Modelling*, 52(7-8):999–1003, 2010.

[24] Swarnali Sharma and GP Samanta. Drinking as an epidemic: a mathematical model with dynamic behavior. *Journal of applied mathematics & informatics*, 31(1 2):1– 25, 2013.

[25] Swarnali Sharma and GP Samanta. Analysis of a drinking epidemic model. *International Journal of Dynamics and Control*, 3(3):288–305, 2015.

[26] S Side, AM Utami, MI Pratama, et al. Numerical solution of sir model for trans- mission of tuberculosis by runge-kutta method. In *Journal of Physics: Conference Series*, volume 1040, page 012021. IOP Publishing, 2018.

[27] Cristiana J Silva and Delfim FM Torres. An optimal control approach to malaria prevention via insecticide-treated nets. In *Conference Papers in Mathematics*, volume 2013. Hindawi, 2013.

[28] Solomon Teferra, Girmay Medhin, Medhin Selamu, Arvin Bhana, Charlotte Hanlon, and Abebaw Fekadu. Hazardous alcohol use and associated factors in a rural ethiopian district: a cross-sectional community survey. *BMC Public Health*, 16(1):1– 7, 2016.

[29] G Testino, S Leone, and P Borro. Treatment of alcohol dependence: recent progress and reduction of consumption. *Minerva medica*, 105(6):447–466, 2014.

[30] Getachew Teshome Tilahun, Oluwole Daniel Makinde, and David Malonza. Mod- elling and optimal control of pneumonia disease with cost-effective strategies. *Journal of Biological Dynamics*, 11(sup2):400–426, 2017.

[31] Pauline Van den Driessche and James Watmough. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical biosciences*, 180(1-2):29–48, 2002.