Mathematical Modelling on Alcohol Consumption Control and its Effect on Poor Population

K. Chinnadurai , *Member, IAENG,* S. Athithan,* *Member, IAENG,* and M. G. Fajlul Kareem.

***Abstract*—A mathematical model of alcohol consumption by categorizing the alcohol consumption people into four compartments names are susceptible to alcohol drinking, low alcohol drinkers, serious alcohol drinkers, and recovered alcohol drinkers. The variety of motivational research works on the consequences of drinking alcohol were discussed by including the habits like the desire to boost one's sociability, mightiness, capacity to handle issues, ability to enjoy participation in a ritual, and a desire to become intoxicated. The use of alcohol and other drugs may have significant unnecessary impacts on society as increased rates of sexual violence within and outside of the family due to abnormal conduct. Some studies stated that consuming alcohol in moderation rates may have certain health advantages, for example, decreasing the rate of getting heart disease and the possibility of lowering the rate of getting diabetes. Since the worst effects of alcohol drinking are enormous, it considers and assumed as one of the diseases in humans that the drinking/basic reproduction number R_0 computed and sensitivity analysis for showing the effectiveness of R_0 is carried out. The sensitivity indices may help suggest which are beneficial parameters or not for evading drinking habits. The basic reproduction number is a tool to assess the stability of the model equations. When R_0 is less than 1, the system of our model is locally and asymptotically stable (LAS) at disease-free equilibrium (DFE) E^0 and, when R_0 is more than 1, the endemic equilibrium (EE) E^* is stable. The importance of our analytical conclusions is shown by through numerical simulation. The epidemiological impacts of our results are important because the quiver made from our study through our model is the consequences of habitats becoming free from serious alcohol issues.**

***Index Terms*—Basic reproduction number, Sensitivity analysis, Stability analysis, Alcohol consumption.**

I. Introduction

THE problem of alcoholism has escalated to epidemic levels, characterized by the persistent pattern of excessive drinking despite the detrimental effects it has an individuals' employment, physical well-being, educational pursuits, and social engagements are analyzed by S. Sharma and G. P. Samanta [1]. F. Schilbach [2] is studied about the low-income communities, particularly in India and other developing countries, excessive alcohol consumption is prevalent among men. This behavior not only adversely affects individuals and their families but also presents significant gaps in our understanding of its underlying mechanisms. One long-standing association with excessive drinking is a lack of self-control. However, the potential efficacy of commitment devices in addressing these challenges remains relatively unexplored. By implementing sobriety incentives through a randomized selection process, individuals were motivated to save more when given the opportunity. These observed increases in savings cannot be solely attributed to higher income resulting from reduced alcohol expenditure. Instead, they reflect shifts in individuals' decision-making regarding their savings habits. Moreover, it is noteworthy that individuals may have experienced personal satisfaction from maintaining sobriety during the day, even if it was accompanied by increased physical discomfort, thus impacting their ability to perform certain tasks effectively.

A.I. Balsa et al. [3] provided the drinking impact of alcohol use on academic performance, namely the number of days missed from school besides the quality of work completed, such as difficult for focusing in class and getting along with classmates and instructors. For both genders, we can find evidence that self-reports contribute to prejudice since drinking is tie-up by the higher grades for females and worse grades for men. This conceptual breakthroughs might be just as valuable for future research and educational policy as its empirical findings. D'Souza El-Guindy, Nympha B., et al. [4] examined the chronic alcohol addiction leads to progressive damage in multiple organs of the body. Previous laboratory models have primarily focused on studying the liver damage associated with alcohol consumption, which is a major contributor to morbidity and mortality among individuals with alcohol use disorder. It is important to note that the detrimental effects of alcohol extend beyond the liver, affecting various organs such as the lungs, stomach, brain, and muscles. Additionally, alcohol-induced organ damage triggers inflammatory responses that impact virtually every system in the human body, including the immune system, endocrine system, and central nervous system. Thus, alcohol-related organ damage manifests as an inflammatory condition with widespread consequences .

H. Zhang [5] indicates the alcohol differs from other medications in that it works by altering a specific receptor system. It may impact various organs since, it is a medication that spreads broadly throughout the body. It is particularly true of the immune system, where alcohol may have varying effects in various immunological compartments. B. Buonomo and D. Lacitignola [6] are analyzed the health risk, because of the incredible significance of squash health and social effects, alcohol dependence is one of the multiple common healthy hazardous behaviours. Our discoveries highlight the consequence of pinpointing social processes, such as imitation mechanisms, that may oblige or inhibit the stretch of high-risk alcohol-drinking behaviour within an open society

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of people. An adequate understanding of these approaches is needed to fix the effective drinking control regulations, reckon operational therapy techniques, and closely scrutinise all elements that may favour the survival of a drinking culture.

PS Meier [7] narrates the gender dissimilarities in alcohol purchase and consumption habits, besides baseline rates of health risk, exist. The results in disparate reactions to liquor pricing regulations, with alcohol taxation and minimum price policies predicted to result in much greater consumption and harm deduction advantages for men than women. AW Kimball et al. [8] and AW Kimball and LA Friedman [9], to the extent that alcohol use has a linear and accumulative effect on health outcomes. It established the connection between coronary heart disease mortality between the other is not unbent, and this may corresponding be true.

S. Sharma and G. P. Samanta [10] are primarily concerned with reducing alcohol consumption. Because of this, we propose a user model focused on technologies that help people cut down or stop drinking altogether. Alcohol binge drinking has the potential to significantly impact individuals and their communities. The transfer of drinking habits from one individual to another can lead to diverse social repercussions and economic challenges across the nation. Considering excessive alcohol consumption as an epidemic, it resembles an infectious condition wherein individuals exhibit a pattern of excessive drinking despite the negative consequences it imposes on various aspects of their lives, including work, business, social environment, economic stability, and road safety. The author suggests that a sensitivity analysis reveals the preference for prevention over treatment. Consequently, to target high-risk alcohol consumers directly through preventive measures yields more favorable outcomes. This approach provides valuable insights into potential consequences and serves as a valuable guide for consumer therapy.

In this paper, we provide an epidemiologically mathematical model of the dynamics and development of alcohol use. A highly influenced alcohol consumption linked occupational accident, ill payment and the production reduction based on a highly affected in alcohol consumption related occupational accident or sick payment and the production drop. This paper is structured as follows: The basic hypothesis of alcolism model equations presents in section (II) is described the initial assumption of alcolism model equations. This section (III) incorporates the subsections as the invariant region, solution of posivity, and the basic reproduction number R_0 derived. Section (IV) investigates the model's parameter sensitivity. Section (V) equilibria and their stability analysis of $E^0 \& E^*$. Section (VI) as we consult the numerical results in the deterministic model of the numerical simulations to show the results. Finally, we have given the results conclusion of the section (VII).

II. The Mathematical Model and Assumption

We developed a model of alcohol consumption misuse. We considering a alcohol consuming adults population is calculated into four different category of named as susceptible alcohol drinkers $S(t)$, low alcohol drinkers $I_L(t)$, heavy alcohol drinkers in treatment $I_H(t)$, recovered alcohol drinkers class $R(t)$. We form a frame for representing spread of alcohol habits in one class to another class mentioned in our model([11],[12],[13]).

The passage of persons from one class to the next is shown in the flowchart (1) and the parameters are described in the Table (I). The following system of ordinary differential

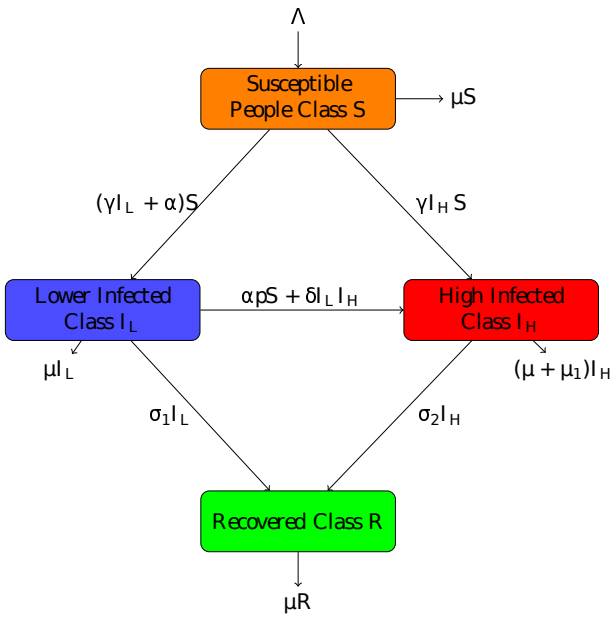

Fig. 1: Diagram of the model with four distinct classes.

equations are used to represent the model:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \alpha S - \mu S - (I_L + I_H)\gamma S, \\
\frac{dI_L}{dt} &= (1-p)\alpha S + \gamma I_L S - \delta I_L I_H - (\sigma_1 + \mu) I_L, \\
\frac{dI_H}{dt} &= \alpha p S + \gamma I_H S + \delta I_L I_H - (\sigma_2 + \mu + \mu_1) I_H, \\
\frac{dR}{dt} &= \sigma_1 I_L + \sigma_2 I_H - \mu R.
\end{aligned} \tag{1}$$

with initial conditions $S(0) > 0, I_L(0) \geq 0, I_H(0) \geq 0, R(0) \geq 0$.

Clearly the model involves the following assumptions:

(i)As a consequence, the total population remains constant. The subgroups are mutually exclusive, meaning each individual in the population belongs to only one of these subgroups. Consequently, we can express the total population size, denoted as N, as the sum of the individuals in each subgroup, represented as $S(t)$, $I_L(t)$, $I_H(t)$, and $R(t)$. Thus, we have $N = S(t) + I_L(t) + I_H(t) + R(t)$.

(ii) As everyone in society interacts similarly, the probability of becoming a binge drinker is uniform for all individuals.

(iii) Low and vulnerable drinkers are exposed to the risky drinking habits of heavy drinkers who are not undergoing treatment.

(iv) Moderate and infrequent drinkers, as well as those in treatment, are at risk of developing alcoholism from heavy drinkers who are not in treatment.

(v) Patients in rehabilitation are not contagious to moderate, heavy, low drinkers, or occasional susceptible drinkers.

(vi) Alcohol abstainers can be categorized into two groups: those who are temporarily sober and those who may relapse into heavy drinking.

TABLE I: Description of parameters

Parameter	Description	Values (E^*)	Source
Λ	Recruitment rate of poor people	10	Estimated
μ	Natural death rate	0.02 yr^{-1}	([14],[15], [20])
μ_1	Death rate due to Alcohol related disease infected and with out immunity	0.03	Estimated
p	Probability of fast progression to drinking habit of the poor people	0.01	[16]
γ	Transmission rate due to contact	0.002	[22]
α	Rate of transmission to self influence	0.003	[21]
δ	Rate of interaction between the lower and higher alcohol consumed peoples	0.0015	Estimated
σ_1	Rate of treatment in low alcohol consumed people are moves into recovered class	0.03	Estimated
σ_2	Rate of treatment in high alcohol consumed people are moves into recovered class	0.02	[22]

(vii) Individuals in the partially recovered class experienced a relapse due to their interactions with heavy and low drinkers.

III. Basic Properties

A. Invariant Region

Theorem III.1. *The feasible invariant region F defined by*

$$F = \{(S(t), I_L(t), I_H(t), R(t)) \in \mathbf{R}^4_+ : 0 < B \leq \frac{\Lambda}{\mu + \alpha}\}$$

with initial conditions $S(0) > 0, I_L(0) \geq 0, I_H(0) \geq 0, R(0) \geq 0$ is positively invariant.

Proof: Adding the system of equation (1) we obtain.

$$\frac{dM}{dt} = \Lambda - \mu M - \mu_1 I_H$$
$$\leq \Lambda - \mu M$$

The solution M(t) of the different a given system
$0 \leq M(t) \leq M(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t})$
Where $M(0)$ represents the sum of the initial values of the variables. As $t \to \infty$, $0 \leq M \leq \frac{\Lambda}{\mu}$. So, if $M(0) \leq \frac{\Lambda}{\mu}$ then $\lim_{t\to\infty} M(t) \leq \frac{\Lambda}{\mu}$. This means that $\frac{\Lambda}{\mu}$ is the upper bounded of M. On the other side, if $M(0) \geq \frac{\Lambda}{\mu}$, then the solution $(S(t), I_L(t), I_H(t), R(t))$ enters $\mathbb{F}$ or approaching if asymtotically. Hence completely positively invarint under the flow induced by the system. This, $\mathbb{F}$, the model(1) is well-posed epidemiologically and mathematically. Hence it is sufficient to studying the dynamic of the model in $\mathbb{F}$. ■

B. Positivity of Solutions

Theorem III.2. *Given the initial condition of the system are $S(0) > 0, I_L(0) \geq 0, I_H \geq 0$ and $R(0) \geq 0$. $S(t), I_L(t), I_H(t)$ and $R(t)$ are positive for all $t \geq \bar{t}$, where $\bar{t} = \inf\{t > 0 : S(t) > 0, I_L(t) > 0, I_H(t) > 0, R(t) > 0\}$*

Proof:

Here, $\bar{t} = \inf\{t > 0 : S(t) > 0, I_L(t) > 0, I_H(t) > 0, R(t) > 0\}$

Thus, $\bar{t} > 0$ and it follow from the 1^{st} equation of the system (1) that

$$\frac{dS}{dt} = \Lambda - [\mu + \alpha + \gamma(I_L + I_H)]S(t)$$

We thus have,

$$S(t) = S(\bar{t}) \exp[-\{\mu(t - \bar{t}) + \int_{\bar{t}}^{t} \alpha + \gamma(I_L + I_H)\}]$$
$$+ \exp[-\{\mu t + \int_0^t \alpha + \gamma(I_L + I_H)ds\}]$$
$$\int_{\bar{t}}^{t} \Lambda \exp\{\mu t + \int_0^t \alpha + \gamma(I_L + I_H)\}dv > 0$$

From the second equation of (1), we have

$$\frac{dI_L}{dt} \geq (\gamma S - \delta I_H - \sigma_1 - \mu)I_L(t)$$
$$= I_L(t) \geq I_L(\bar{t}) \exp[(\gamma S - \delta I_H - \sigma_1 - \mu)(t - \bar{t})] > 0$$

From the third equation of (1), we have

$$\frac{dI_H}{dt} \geq (\gamma S + \delta I_L - \sigma_2 - (\mu + \mu_1))I_H(t)$$
$$= I_L(t) \geq I_L(\bar{t}) \exp[(\gamma S + \delta I_L - \sigma_2 - (\mu + \mu_1))(t - \bar{t})] > 0$$

Similarly, from the last equation of (1)

$$\frac{dR}{dt} \geq -\mu R(t)$$
$$= R(t) \geq R(\bar{t}) \exp[-\mu(t - \bar{t})] > 0$$

Therefore, we can see that
$S(t) > 0, I_L(t) > 0, I_H(t) > 0, R(t) > 0, \forall t \geq \bar{t} > 0$
Hence is proved. ■

C. The Basic Reproduction Number R_0

When a low drinker transitions into the population of heavy alcohol drinkers, the basic reproduction number R_0 is determined to refer [10] S. Sharma and G. P. Samanta. In this model, the reproduction rate can be estimated by dividing the transmission coefficient from light to heavy drinkers by sum of the total number of people who pass away naturally, those who perish due to alcohol-related causes, or those who seek treatment.

We use the next-generation matrix method to find the basic reproduction number. Using this method we obtain two non-negative matrices $\mathbf{F}$ and $\mathbf{V}$ evaluated at E^0 such that,

$$\mathbf{F} = \begin{pmatrix} \gamma S & 0 \\ 0 & 0 \end{pmatrix} \text{ and } \mathbf{V} = \begin{pmatrix} \sigma_1 + \mu & 0 \\ 0 & \sigma_2 + \mu + \mu_1 \end{pmatrix}$$

For definition and evaluation of matrices $\mathbf{F}$ and $\mathbf{V}$ refer to ([10], [20]). Now the spectral radius of $(\mathbf{FV}^{-1})$, which corresponds to basic reproduction number is

$$R_0 = \frac{\gamma\Lambda}{(\alpha+\mu)(\sigma_1+\mu)}$$

IV. Sensitivity Analysis of R_0

R_0's sensitivity to each of its characteristics will be evaluated using this procedure followed in the reference S. Sharma and G. P. Samanta [10], The normalised ahead sensitivity index is computed at each of the parameters.

$$\begin{aligned}
\frac{\partial R_0}{\partial \Lambda}\frac{\Lambda}{R_0} &= 1 > 0,\\
\frac{\partial R_0}{\partial \gamma}\frac{\gamma}{R_0} &= 1 > 0,\\
\frac{\partial R_0}{\partial \alpha}\frac{\alpha}{R_0} &= -\frac{\alpha}{(\alpha+\mu)} < 0,\\
\frac{\partial R_0}{\partial \sigma_1}\frac{\sigma_1}{R_0} &= -\frac{\sigma_1}{\sigma_1+\mu} < 0,\\
\frac{\partial R_0}{\partial \mu}\frac{\mu}{R_0} &= -\frac{\mu(\sigma_1+\alpha+2\mu)}{(\alpha+\mu)(\sigma_1+\mu)} < 0.
\end{aligned}$$

Our analysis concludes that changes in the parameter γ have the most significant impact on the fundamental reproduction number (R_0). Increasing or decreasing γ will have an equal effect on R_0. Conversely, an increase in any of the three variables will lead to a decrease in R_0, but the magnitude of the decrease will be proportionally smaller. It is important to note that μ represents the population's regular mortality rate, while μ_1 represents the death rate from alcohol-related causes among untreated heavy drinkers. It is morally unacceptable and unadvisable to intentionally raise any of these rates.

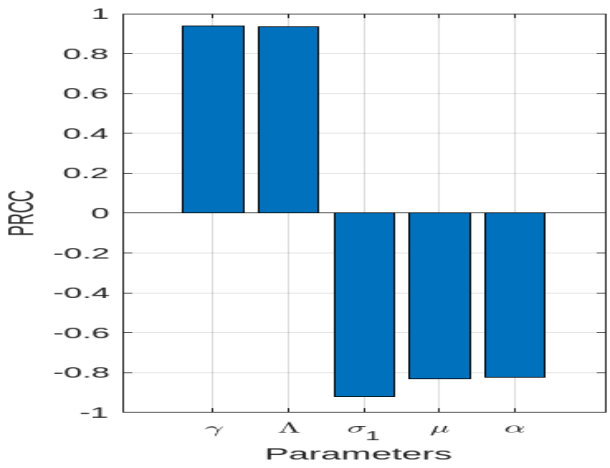

Fig. 2: PRCC findings illustrating model parameter sensitivity with respect to R_0.

Considering the above, we choose to focus on one of two parameters: either the percentage of drinkers undergoing treatment, δ, or the rate at which moderate and infrequent drinkers transition to heavy drinking, γ. While reducing γ may appear as the optimal strategy, our sensitivity analysis indicates that prevention outweighs treatment. Preventative interventions are more effective in controlling the spread of habitual drinking compared to solely increasing the number of individuals seeking treatment. In essence, the results of this sensitivity analysis consistently highlight the superiority of prevention over treatment.

See the fig(2), the Partial Ranked Correlation Coefficient (PRCC) is a sample-based method used to evaluate the correlation between a model's output variable and its parameters. It relies on sample points generated through Latin Hypercube Sampling (LHS). In order to assess the correlation between parameter values and the R_0's output, specifically the time-dependent nuclear γ-catenin concentration, PRCC values are computed for each time point. This ranked correlation coefficient, similar to PRCC, serves as a robust sensitivity metric, particularly effective for identifying non-linear yet monotonic relationships are used in the reference [17] Srivastav, A. & Ghosh, M. PRCC values adhere to the same scale as standard correlation coefficients, ranging from -1 (indicating a perfect negative correlation) to +1 (indicating a perfect positive correlation).

A. Data Analysis

Over the span of a decade, from 2011 to 2021, Finland experienced a consistent reduction in alcohol-related fatalities. The peak occurred in 2012, with a total of 1,960 recorded deaths. However, by 2021, this number had dropped to 1,646, reflecting a notable decline of 70 deaths compared to the preceding year, its referred in Statista [18]. See the fig.(3), these data collected from source is statista and shown the effect of alcohol-related death rate in yearwise parameter μ_1.

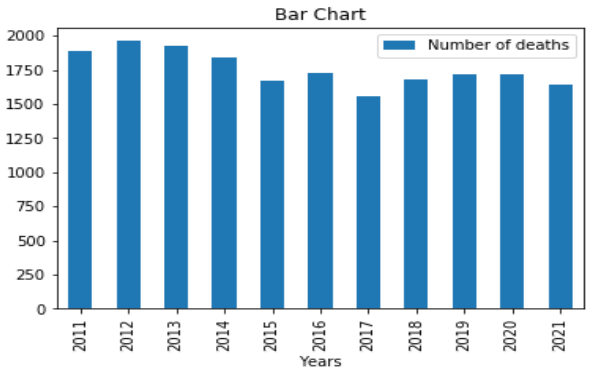

Fig. 3: Alcohol-Related Mortalities in Finland from 2011 to 2021

NDSAP shared the relevant real-world scenario for implementing a mathematical model is readily observable in society. Presently, our efforts are focused on refining and validating our results by integrating them with empirical data. Within this framework, we have incorporated with data pertaining to confirmed alcohol-related cases in India spanning the period from 2006 to 2016, as referenced in [19]. It's noteworthy that despite the presence of previous studies on the count of individuals affected in accidents caused by drivers under the influence of alcohol or drugs, our work contributes further insights. Acquire data pertaining to road accidents categorized by State/Union Territory (UT), with a specific emphasis on incidents associated with alcohol/drug consumption and drivers exceeding lawful speed limits. This dataset encompasses critical information, including the total

number of road accidents, the fatalities count, and the number of individuals injured.

We present a comprehensive comparison between our model (designated as Model (1)) and actual data spanning the time frame from 2006 to 2016. Figure 4 visually depicts this comparison, offering a graphical representation that demonstrates the alignment between real-world data of susceptiple population due to alcohol and our model's compartment namely S predictions for instances of substantial alcohol consumption over the same period. Notably, the simulated results closely track with the observed data for alcohol-related cases, as highlighted in Figure 4.

Furthermore, it provides an insightful display of total cases for different states in India. These figure presents an overview of the annual confirmed cases of alcohol-related incidents involving drivers in India throughout the study period. This depiction sheds light on the annual prevalence of heavy alcohol consumption cases across various states within India.

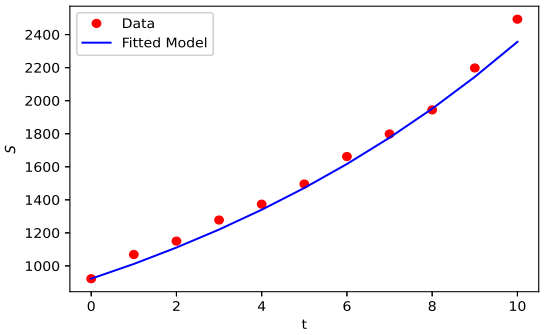

Fig. 4: Plot of Susceptible population S vs. alcohol/drug-related accident data by State/UT, 2006-2016.

V. Equilibria and Stability Analysis

A. Existence of Equilibria

The system of equation (1) has the following two equilibriums are:

(i) Disease Free Equilibrium (DFE) $E^0(S^0, 0, 0, 0) = (\frac{\Lambda}{\mu+\alpha}, 0, 0, 0)$

(ii) Endemic Equilibrium (EE) $E^*\ (S^*, I_L^*, I_H^*, R^*)$ where,

$$
\begin{aligned}
S^* &= \frac{\Lambda}{\mu + \alpha + \gamma(I_L + I_H)},\\
I_L^* &= \Lambda\alpha(1-p) + \gamma\Lambda I_L - (\sigma_1+\mu)(\mu+\alpha)I_L\\
&\quad - \gamma I_L(\sigma_1+\mu)(I_L+I_H) - (\mu+\alpha)\delta I_L I_H\\
&\quad - \delta\gamma I_L I_H(I_L+I_H),\\
I_H^* &= \Lambda\alpha p + \gamma\Lambda I_H - [\sigma_2 + (\mu+\mu_1)](\mu+\alpha)I_H\\
&\quad - \gamma I_H[\sigma_2 + (\mu+\mu_1)](I_L+I_H) + \delta I_L I_H(\mu+\alpha)\\
&\quad - \delta\gamma I_L I_H(I_L+I_H),\\
R^* &= \frac{\sigma_1 I_L + \sigma_2 I_H}{\mu}.
\end{aligned}
$$

B. Stability analysis of disease (drinking) free equilibrium

Theorem V.1. *Given the disease free equilibrium of the equation 1 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof:

The $\mathbf{J^0}$ matrix of the system (1)

$$
\mathbf{J^0} = \begin{pmatrix} i_{11} & i_{12} & i_{13} & 0\\ i_{21} & i_{22} & 0 & 0\\ i_{31} & 0 & i_{33} & 0\\ 0 & \sigma_1 & \sigma_2 & -\mu \end{pmatrix}
$$

where

$$
\begin{aligned}
i_{11} &= -(\mu+\alpha),\\
i_{12} &= -\gamma S,\\
i_{13} &= -\gamma S,\\
i_{21} &= \alpha(1-p),\\
i_{22} &= \gamma S - (\sigma_1+\mu),\\
i_{31} &= \alpha p,\\
i_{33} &= \gamma S - (\mu+\mu_1+\sigma_2).
\end{aligned}
$$

One of the eigenvalue of this $\mathbf{J^0}$ matrix is -μ and the other three eigenvalues are the roots of the following cubic equation

$$\lambda^3 + j_1\lambda^2 + j_2\lambda + j_3 = 0$$

where

$$
\begin{aligned}
j_1 &= -(1_{33} + i_{11} + 1_{22}),\\
j_2 &= -(1_{12}b1 + 1_{13}i_{31} - i_{11}i_{22} - i_{11}i_{33} - i_{22}i_{33}),\\
j_3 &= -(i_{11}i_{22}i_{33} - i_{12}i_{21}i_{33} - i_{13}i_{31}i_{22}).
\end{aligned}
$$

We can show that all eigenvalues of $\mathbf{J^0}$ are negative by Routh Hurwitz criteria, roots of the cubic equation will have negative real parts if $j_1 > 0, j_3 > 0, j_1 j_2 > j_3$. Hence the equilibrium point E^0 is locally asymptotically stable provided $R_0 < 1$. The disease free equilibrium is locally asymptotically stable if $R_0 < 1$. ■

C. Stability analysis of endemic equilibrium

Theorem V.2. *Endemic equilibrium E^*, whenever it exists, is locally asymptotically stable when $R_0 > 1$.*

Proof: The $\mathbf{J}^*$ matrix of the system (1)

$$
\mathbf{J^1} = \begin{pmatrix} a_{11} & a_{12} & a_{13} & 0\\ b_{11} & b_{12} & b_{13} & 0\\ c_{11} & c_{12} & c_{13} & 0\\ 0 & \sigma_1 & \sigma_2 & -\mu \end{pmatrix}
$$

where

$$
\begin{aligned}
a_{11} &= -(\mu+\alpha),\\
a_{12} &= -\gamma S,\\
a_{13} &= -\gamma S,\\
b_{11} &= \alpha(1-p),\\
b_{12} &= \gamma S - (\sigma_1+\mu+\delta I_H),\\
b_{13} &= -\delta I_L,\\
c_{11} &= \alpha p,\\
c_{12} &= \delta I_H,\\
c_{13} &= \delta I_L + \gamma S - (\mu+\mu_1+\sigma_2).
\end{aligned}
$$

The eigenvalue of this $\mathbf{J^1}$ matrix is λ_1 = -μ and the remaining three values are the roots of the following cubic equation

$$\lambda^3 + k_1\lambda^2 + k_2\lambda + k_3 = 0$$

where the coefficients of λ are as follows,

$$k_1 = -(c_{13} + a_{11} + b_{12}),$$
$$k_2 = -(c_{12}b_{13} + a_{12}b_{11} + a_{13}c_{11} - a_{11}b_{12} - a_{11}c_{13} - b_{12}c_{13}),$$
$$k_3 = -(a_{11}b_{12}c_{13} - a_{11}c_{12}b_{13} - a_{12}b_{11}c_{13} - a3c_{11}b_{12}).$$

By Routh Hurwitz criteria, roots of the cubic equation will have negative real parts if $k_1 > 0, k_3 > 0, k_1k_2 > k_3$. Hence the equilibrium point E^* is locally asymptotically stable provided $R_0 > 1$ [23]. ■

VI. Result Discussion

In this part, we will present several numerical results of model (1) for a variety of different parameter values.

In order to demonstrate the dynamical behaviour of our findings, we make use of numerical simulations, in which we assume that the whole population is always one hundred percent. and choose S = 500, I_L = 10, I_H = 25 and R = 10. Table (1) is shows the additional parameters that would be utilised in this values to the endemic equlibrium point E^* is locally asymtotically stable. Its shown in numerical result in fig.(6) and the fig.(5) is shown in the disease free equilibrium point is locally asymtotically stable S_1. When the values of parameter set $S_1 = \{\Lambda = 10, \alpha = 0.001, \gamma = 0.001, \delta = 0.87, p = 0.92, \sigma_1 = 0.03, \sigma_2 = 0.05, \mu = 0.014, \mu_1 = 0.02\}$

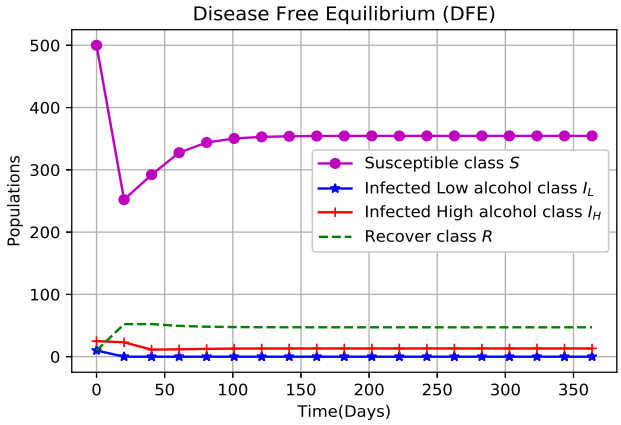

Fig. 5: The Stability of DFE (E^0) with S, I_L, I_H, R over time.

In fig.(10, 11, 12, 13) are shown in the effect of parameter the level of point-wise variation and how it worked in the particular compartment. Each value shows how the populations increase/decrease during the period.

In fig.(10), The transition between alcohol consumption categories exhibits a notable pattern. Initially, changes occur within the low alcohol drinker class, where the values show incremental shifts. These changes are linked to susceptible individuals gradually transitioning to either the low or heavy drinker class. Consequently, heavy drinkers may develop an addiction to alcohol, leading to significant physical and mental repercussions.

The bodies of heavy drinkers are particularly susceptible to alcohol-related health issues, such as memory loss and increased vulnerability to viruses like HIV, N1H1, and others.

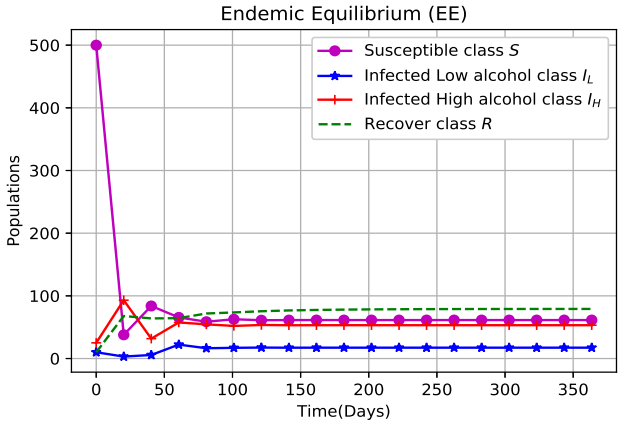

Fig. 6: The variation of S, I_L, I_H, R over time demonstrating the stability of EE (E^*).

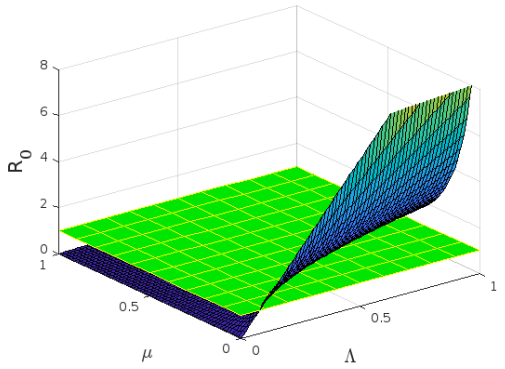

Fig. 7: R_0 value in terms of μ and Λ.

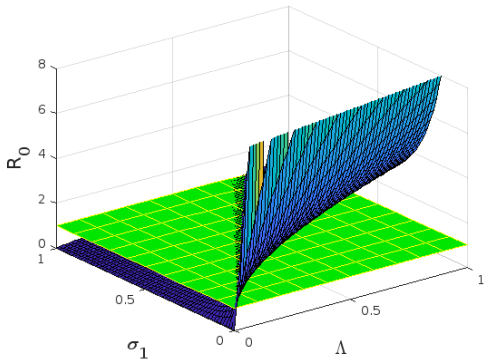

Fig. 8: R_0 value in terms of σ_1 and Λ.

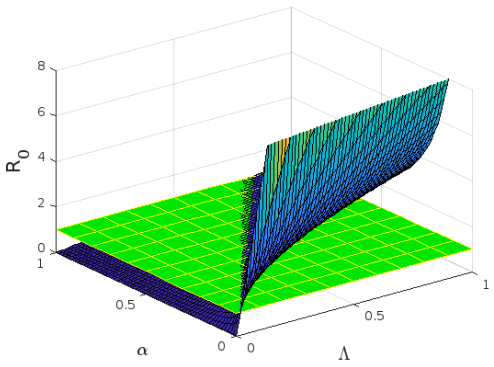

Fig. 9: R_0 value in terms of α and Λ.

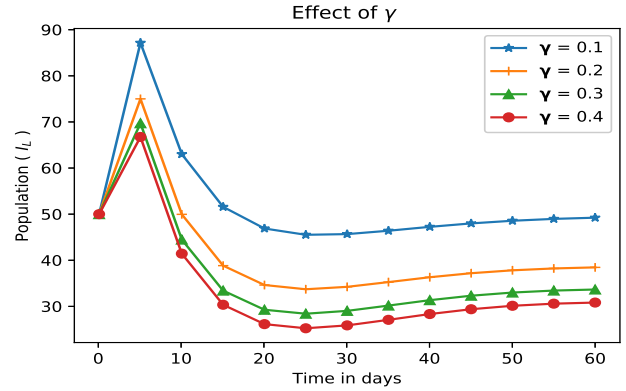

Fig. 10: Effect of γ on the low drinking population

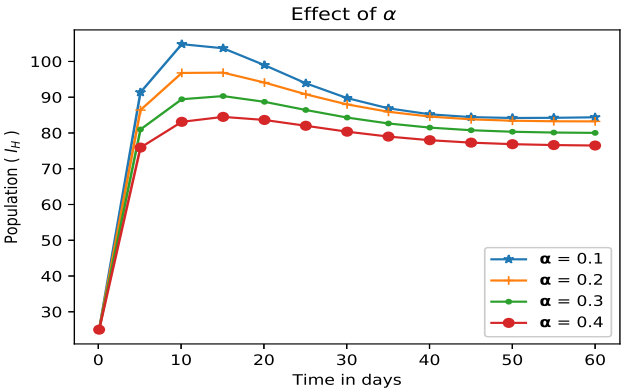

Fig. 11: Effect of α on the heavy drinking population.

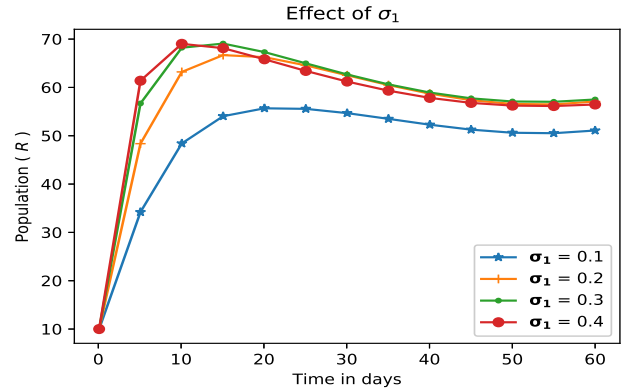

Fig. 12: Effect of σ_1 on the recovered population.

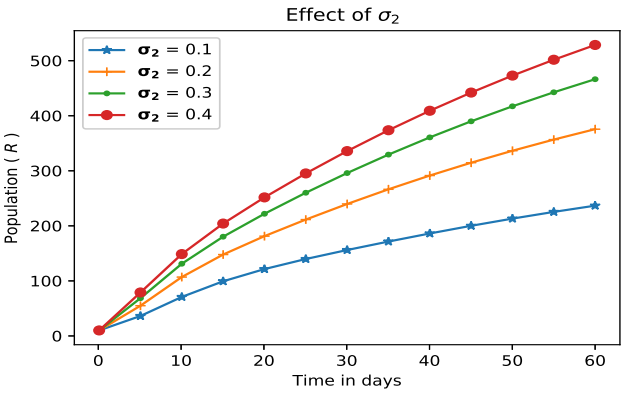

Fig. 13: Effect of σ_2 on the recovered population.

Additionally, vital organs, including the liver, lungs, heart, brain, stomach, and kidneys, may not function properly due to the adverse effects of excessive alcohol consumption. Fig. (11) Once individuals initiate alcohol consumption, they often persist with this behavior in their daily lives, leading to a detrimental change in their lifestyle.

We have try to improve the low and heavy alcohol consumer to recovered in this behaviour. It shown in fig.(12, 13) detailed. As the values of σ_1 and σ_2 increase, we observe a corresponding increase in the number of recovered individuals. However, for alcohol binge drinkers, their ability to function effectively in their day-to-day lives is compromised, leading to numerous challenges in completing routine tasks. As a result, our aim is to provide guidance and support to help them withdraw from alcohol consumption or educate them on the potential health risks if they continue drinking.

Alcohol addiction can severely impact various organs, raising concerns about potential diseases that may arise. By addressing these health concerns, individuals are encouraged to seek treatment through hospitalization, rehabilitation programs, or voluntarily dropping their drinking behavior to achieve recovery from alcohol addiction. Our goal is to offer comprehensive assistance in facilitating a healthier and alcohol-free lifestyle for those struggling with alcohol dependence.

Furthermore to intoxication or drunkenness, excessive alcohol consumption, whether in a short period or over an extended period, can lead to various adverse conditions. These conditions may include memory loss, seizures, headaches, memory blackouts, coordination issues, and dehydration.

Fig.(7, 8, 9) are shown in the R_0 effects to influence the different parameter in different figure. The parameter namely α, σ_1, μ, Λ.

WHO [24] says that the harmful use of alcohol contributes to over 200 diseases and injuries. In every year, 3 million people kills in the worldwide through the hazardous alcohol usage. This accounts for 5.3% of all fatalities.The responsible of alcohol is 5.1% of the worldwide the illness of burden and injury, as assessed in DALYs(Disability-Adjusted Life Years). Aside from the health repercussions, irresponsible alcohol consumption effects considerable socially and economically damage to individuals and public. Early in life, alcohol intake causes mortality and disability. Alcohol accounts for around 13.5 per cent of all fatalities in adults aged 20 to 39. There is a link between problematic alcohol consumption and various mental and behavioural problems, noncommunicable diseases, and accidents. There is a link between problematic drinking and the occurrence or outcome of infectious illnesses such as TB and HIV/AIDS. An expecting mother's use of alcohol may result in foetal alcohol syndrome (FAS) and pre-term delivery problems.

Centers for Disease Control and Prevention [25] is controlled the excessive alcohol consumption stands as a significant preventable cause of mortality in the United States, leading to an average reduction in lifespan by 26 years for those who succumb to its effects. Binge drinking is characterized by the consumption of more than four drinks in a sitting for women, or more than five drinks for men. On the other hand, massive drinking is defined as the intake of more than eight drinks per week for women and fifteen or more drinks per week for men. It is essential for the alcohol

consumption is particularly risky for pregnant women and individuals younger than 21 years old.

VII. Conclusions

We have identified R_0 as (the basic reproduction number) the system (1), it enables to comprehend the dynamic behavior of the system. To describe the mathematical alcohol drinking model and explore the individual and group behavior of world wide drinking dynamics, we employed stability analysis theory of nonlinear systems.

For the drinking-free equilibrium, denoted as E^0, it can be locally asymptotically stable if the threshold quantity $R_0 \leq 1$. Conversely, if $R_0 > 1$, the alcohol-present equilibrium, labeled as E^*, exhibits local asymptotic stability. These stability assessments provide crucial insights into the long-term behavior of the system, shedding light on the potential implications for controlling alcohol-related dynamics.

We conducted a sensitivity analysis of the model parameters to identify the key factors that have a significant impact on the reproduction number, denoted as R_0. Determining these influential parameters is crucial for effectively targeting interventions aimed at reducing R_0. Our sensitivity analysis results emphasize the importance of prevention over treatment, indicating that efforts to enhance prevention strategies are more effective in controlling the spread of excessive drinking habits compared to focusing solely on increasing the number of individuals receiving treatment. This highlights the significance of proactive measures in mitigating the prevalence of habitual drinking.

It focuses on assessing the influence of key risk factors on different risk categories and employs numerical simulations to validate the findings of the sensitivity analysis. In order to effectively reduce the health risks associated with alcoholism, it is imperative to enhance public awareness. Notably, public education campaigns have a higher likelihood of success when they involve influential individuals, as social influence plays a critical role in recruiting others to participate in such initiatives. An intriguing aspect of this study involves combining alcohol-related hazards with the risk of contracting sexually-transmitted infections within a single model, which warrants further investigation.

B. Buonomo and D. Lacitignola [6] provided a comprehensive of these processes is essential to establish efficacious regulations for controlling drinking behavior, evaluate operational therapy techniques, and meticulously monitor all factors that contribute to the persistence of alcohol consumption patterns. To reduce the intercourse rate between non-drinkers and heavy drinkers is insufficient to effectively address the alcoholism epidemic. In addition to this measure, it is imperative to increase the number of individuals seeking treatment for alcoholism and to educate people about the potential dangers associated with excessive drinking. By promoting both treatment-seeking behavior and awareness of the risks involved, we can contribute to a more comprehensive approach towards combating alcoholism.

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