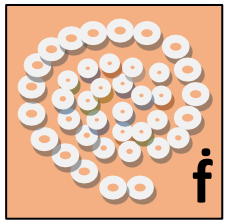

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Mathematical modeling of alcoholism as a social contagion: dynamics, bifurcation, and control

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Abstract

Alcoholism remains a critical social and public health problem worldwide, characterized by behavioral transmission through peer influence and social reinforcement. In this study, we develop and analyze a deterministic compartmental model describing alcoholism dynamics as a social contagion process. We derive the basic reproduction number, examine local and global stability, and investigate bifurcation behavior. Sensitivity analysis identifies key parameters driving the persistence of alcohol abuse. Finally, we introduce optimal control interventions to guide public health policies. Numerical simulations reveal that combined interventions—awareness, rehabilitation, and relapse prevention—are the most effective in reducing alcohol dependence. The study provides qualitative insights into alcoholism dynamics rather than quantitative prediction.

Keywords and phrases: Alcoholism modeling; Social contagion; Basic reproduction number; Bifurcation analysis; Optimal control

1 Introduction

Alcoholism remains one of the most persistent public health challenges worldwide, contributing significantly to morbidity, mortality, and long-term socioeconomic burden [1]. Reports from global health authorities indicate that harmful alcohol consumption is responsible for millions of deaths annually and is associated with a wide range of non-communicable diseases, mental health disorders, and accidental injuries [2]. Beyond its physiological effects, alcoholism imposes profound social and economic consequences, including reduced productivity, family instability, and increased healthcare costs. These multidimensional impacts have driven extensive research aimed at understanding the mechanisms underlying alcohol dependence and its persistence in human populations [3].

While alcoholism has traditionally been studied from biomedical and psychological perspectives, growing evidence suggests that it also exhibits characteristics analogous to transmissible processes. Social exposure—through peer pressure, family influence, cultural practices, and media reinforcement—plays a crucial role in shaping drinking behavior [4]. In many communities, particularly among young individuals, alcohol initiation and escalation occur within social networks where drinking is normalized. This interaction-driven spread of behavioral traits aligns with the concept of social contagion, whereby behaviors and norms propagate through interpersonal interactions in a manner similar to infectious diseases [1, 4]. Alcoholism is modeled as a socially transmitted behavior rather than a biological infectious disease. Here, 'infection' represents behavioral adoption through peer influence. This perspective provides a strong theoretical foundation for modeling alcoholism using compartmental frameworks and dynamical systems.

The burden of alcoholism is especially pronounced in developing regions, including many African countries, where cultural acceptance, socioeconomic challenges, and limited access to mental health services increase vulnerability [11]. Alcohol consumption is often embedded in social traditions and daily

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life, making abstinence difficult. Additional factors such as unemployment, economic stress, urbanization, and the availability of unregulated alcoholic products further contribute to rising prevalence. Adolescents and young adults are particularly at risk due to heightened susceptibility to peer influence [7]. These realities highlight the need for analytical tools capable of capturing the complex interplay between social behavior and addiction dynamics.

Mathematical modeling provides a powerful framework for analyzing these dynamics and evaluating intervention strategies. By adapting concepts from infectious disease modeling, such as compartmental structures, threshold parameters, and stability analysis, it becomes possible to describe transitions between behavioral states and assess the impact of interventions, including awareness campaigns, rehabilitation programs, and preventive policies [11]. Several studies have contributed to this area, including deterministic and fractional-order models that incorporate treatment effects, relapse dynamics, and social influences [6, 8, 10, 12]. Despite these advances, many existing models remain limited in their ability to provide comprehensive predictive insights for long-term dynamics and policy evaluation.

Motivated by these gaps, this study develops a deterministic compartmental model that captures the essential mechanisms of alcoholism dynamics, including social influence, progression to addiction, recovery, and relapse. The model is analyzed to determine equilibrium states, derive the basic reproduction number R_0, and investigate stability and bifurcation behavior. In addition, sensitivity and optimal control analyses are performed to evaluate the effectiveness of intervention strategies and identify key parameters influencing the persistence of alcohol abuse. The results provide a quantitative foundation for evidence-based policy design, particularly in resource-constrained settings where efficient allocation of interventions is critical. Unlike many existing alcoholism models that focus primarily on transmission and treatment dynamics, the present study simultaneously incorporates awareness-induced protection, relapse mechanisms, bifurcation analysis, sensitivity analysis, and optimal control within a unified deterministic framework.

The remainder of this paper is organized as follows: section 2 presents the model formulation, section 3 provides the mathematical analysis, section 4 discusses numerical simulations, section 5 introduces the optimal control framework, and section 6 concludes with a discussion of the results and policy implications.

2 Model Formulation

The total population is divided into five compartments: susceptible (non-drinkers but at risk) denoted by $S(t)$, the light-drinkers (occasional consumers) represented by $L(t)$, the addicted individuals (problem drinkers) represented by $A(t)$, the recovered individuals (rehabilitated) represented by $R(t)$ and the protected individuals (educated and resistant) represented by $P(t)$. The protected compartment is introduced to explicitly track individuals who develop long-term behavioral resistance through awareness and education programs. The transition occurs from S to L due to peer influence at rate β, where the term βSL represents behavioral transmission through peer influence and social interaction rather than biological infection, from L to A due to addiction progression at rate α, from A to R due to rehabilitation at the rate γ, relapse from R to A at the rate ρ and from S to P at rate θ due to awareness/education (protection). The flow diagram is shown in Figure 1.

2.1 Flow Dynamics

From the above flowchart we derive the system of equations below:

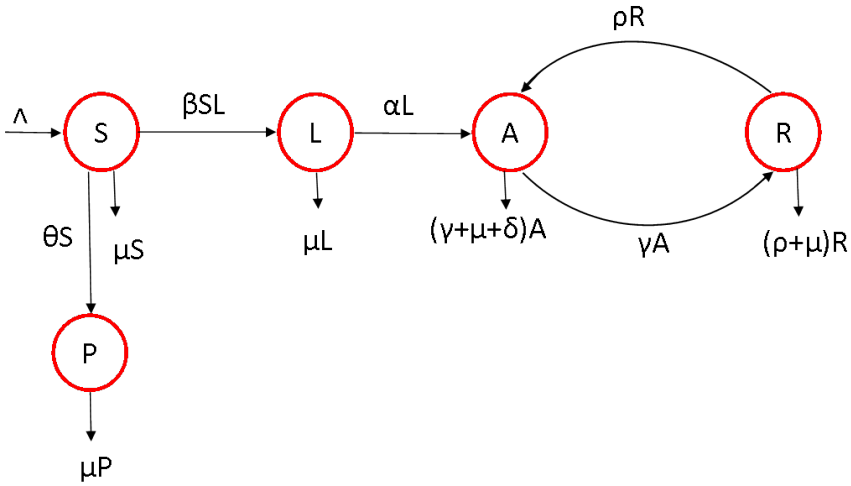


Figure 1: Flow diagram of the alcoholism transmission model

2.2 System of Equations

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SL - \mu S - \theta S, \\
\frac{dL}{dt} &= \beta SL - (\alpha + \mu)L, \\
\frac{dA}{dt} &= \alpha L + \rho R - (\gamma + \mu + \delta)A, \\
\frac{dR}{dt} &= \gamma A - (\rho + \mu)R, \\
\frac{dP}{dt} &= \theta S - \mu P.
\end{aligned} \tag{1}$$

Where Λ is recruitment, μ is natural death, θ is awareness rate, β is peer initiation, α is addiction rate, γ is rehabilitation rate, ρ is relapse rate, and δ is alcoholism-induced death. The density-dependent incidence term βSL is adopted under the assumption that the intensity of peer influence increases with the absolute number of interacting drinkers in the population. This assumption is appropriate in environments such as social gatherings, campuses, bars, and urban communities where alcohol exposure rises with increasing population interaction. Unlike biological infection models where contact rates often saturate with population size, social exposure to alcohol consumption may intensify in densely interactive environments, making density-dependent transmission appropriate for qualitative behavioral analysis.

2.3 Parameter Description

A detailed table can be included to define each model parameter with its biological interpretation and baseline value.

Table 1: Description of state variables of the model

Variable	Description
$S(t)$	Number of susceptible individuals at time t
$L(t)$	Number of light-drinkers
$A(t)$	Number of addicted drinkers
$R(t)$	Number of recovered individuals at time t
$P(t)$	Number of protected/aware individuals

Table 2: Description of parameters used in the model

Parameter	Description
Λ	Recruitment (birth/immigration) rate into the population
β	Social influence rate between susceptible and light-drinkers individuals
μ	Natural death rate
θ	protection/awareness rate of susceptible individuals
α	Progression rate from light drinking to addiction
γ	Recovery rate from active alcoholism class
δ	alcoholism-induced death rate among addicted individuals
ρ	Relapse rate from recovered to addicted class

3 Model Assumptions

The model is based on the following biologically and socially motivated assumptions:

- The population is homogeneously mixed.
- Recruitment into the population occurs at a constant rate.
- The population has no explicit age or spatial structure.
- The total population is closed except for recruitment and natural death.
- Alcohol consumption is transmitted through social interaction.
- Transition rates between compartments are time-continuous and deterministic.

4 Basic Properties of the Model

In this section, we establish the fundamental qualitative properties of the model, including positivity and boundedness of solutions, to ensure that the model is mathematically and behaviorally well posed.

4.1 Existence and Uniqueness of Solutions

Since the right-hand side of system (1) consists of continuously differentiable functions, the system satisfies the local Lipschitz condition. Therefore, by the Picard–Lindelöf theorem, there exists a unique solution corresponding to any nonnegative initial condition.

4.2 Positivity of Solutions

We want to show that if the initial conditions of system (1) are non-negative then all the solutions of the system remain non-negative for future time. That is if

$$S(0) \geq 0, \quad L(0) \geq 0, \quad A(0) \geq 0, \quad R(0) \geq 0, \quad P(0) \geq 0, \tag{2}$$

then the solution remains non-negative for all $t \geq 0$.

$$\frac{dS}{dt} = \Lambda - (\mu + \theta)S - \beta SL \geq -(\mu + \theta)S. \tag{3}$$

By Gronwall's inequality, $S(t) \geq S(0)e^{-(\mu+\theta)t} \geq 0$. Similarly,

$$\frac{dL}{dt} = L(\beta S - (\alpha + \mu)) \geq -(\alpha + \mu)L. \tag{4}$$

Since $L(0) \geq 0$, $L(t) \geq 0$.

$$\frac{dA}{dt} = \alpha L + \rho R - (\gamma + \mu + \delta)A \geq -(\gamma + \mu + \delta)A. \tag{5}$$

With $L, R \geq 0$ and $A(0) \geq 0$, we have $A(t) \geq 0$.

$$\frac{dR}{dt} = \gamma A - (\rho + \mu)R \geq -(\rho + \mu)R. \tag{6}$$

Thus $R(t) \geq 0$ for $R(0) \geq 0$.

$$\frac{dP}{dt} = \theta S - \mu P \geq -\mu P. \tag{7}$$

Hence $P(t) \geq 0$ for $P(0) \geq 0$. All solutions of the system remain non-negative for all $t \geq 0$ provided the initial conditions are non-negative:

$$S(t) \geq 0, \quad L(t) \geq 0, \quad A(t) \geq 0, \quad R(t) \geq 0, \quad P(t) \geq 0, \quad \forall t \geq 0. \tag{8}$$

4.3 Boundedness of Solutions

The total population is

$$N(t) = S(t) + L(t) + A(t) + R(t) + P(t). \tag{9}$$

Adding all the equations, we get

$$\begin{aligned}
\frac{dN}{dt} &= \frac{dS}{dt} + \frac{dL}{dt} + \frac{dA}{dt} + \frac{dR}{dt} + \frac{dP}{dt} \\
&= \Lambda - (\mu + \theta)S - (\alpha + \mu)L - (\gamma + \mu + \delta)A - (\rho + \mu)R - \mu P + \beta SL + \alpha L + \rho R + \gamma A + \theta S \\
&= \Lambda - \mu(S + L + A + R + P) - \delta A \\
&= \Lambda - \mu N - \delta A \leq \Lambda - \mu N.
\end{aligned} \tag{10}$$

$$\frac{dN}{dt} \leq \Lambda - \mu N. \tag{11}$$

The solution of the corresponding equality is

$$N(t) = \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu}\right) e^{-\mu t}. \tag{12}$$

Thus, as $t \to \infty$,

$$N(t) \leq \frac{\Lambda}{\mu}. \tag{13}$$

Since all compartments are non-negative (from positivity analysis) and the total population is bounded above, we have

$$0 \leq S(t), L(t), A(t), R(t), P(t) \leq N(t) \leq \frac{\Lambda}{\mu}, \quad \forall t \geq 0. \tag{14}$$

Hence, the solutions of the system are positive and bounded for all time. Therefore, the feasible region of the model is given by

$$\Omega = \left\{ (S, L, A, R, P) \in \mathbb{R}^5_+ : N(t) \leq \frac{\Lambda}{\mu} \right\}. \tag{15}$$

The region Ω is positively invariant and attracts all solutions of system (1). Hence, the solutions of the system are positive and bounded for all time.

5 Equilibrium Analysis

Two equilibria exist in our model. These are alcohol-free equilibrium (AFE), that is no alcohol-consuming individuals and endemic alcoholism equilibrium (EAE), that is persistent drinking.

5.1 Alcohol-Free Equilibrium

The alcohol-free equilibrium (AFE) occurs when there are no drinkers, i.e.,

$$L^* = 0, \quad A^* = 0. \tag{16}$$

From the system of equations (1):

$$\begin{aligned} 0 &= \Lambda - \beta S^* L^* - (\mu+\theta)S^* \implies S^* = \frac{\Lambda}{\mu+\theta}, \\ 0 &= \gamma A^* - (\rho+\mu)R^* \implies R^* = 0, \\ 0 &= \theta S^* - \mu P^* \implies P^* = \frac{\theta S^*}{\mu} = \frac{\theta\Lambda}{\mu(\mu+\theta)}. \end{aligned} \tag{17}$$

Thus, the alcohol-free equilibrium (AFE) point is

$$(S^*, L^*, A^*, R^*, P^*) = \left(\frac{\Lambda}{\mu+\theta},\ 0,\ 0,\ 0,\ \frac{\theta\Lambda}{\mu(\mu+\theta)}\right) \tag{18}$$

5.2 Endemic Alcoholism Equilibrium (EAE)

The endemic alcoholism equilibrium point occurs when the alcohol-use behavior persists in the population. By setting the right-hand side of system (1) equal to zero and solve for the variables we get

$$\begin{aligned} S^* &= \frac{\alpha+\mu}{\beta}, \\ L^* &= \frac{\Lambda\beta - (\mu+\theta)(\alpha+\mu)}{\beta(\alpha+\mu)}, \\ A^* &= \frac{\alpha(\rho+\mu)}{(\gamma+\mu+\delta)(\rho+\mu)-\rho\gamma}L^*, \\ R^* &= \frac{\gamma}{\rho+\mu}A^*, \\ P^* &= \frac{\theta(\alpha+\mu)}{\mu\beta}. \end{aligned} \tag{19}$$

The endemic alcoholism equilibrium exists only if $L^* > 0 \implies \mathcal{R}_0 > 1$.

5.3 Basic Reproduction Number $\mathcal{R}_0$

Using the next-generation matrix approach, $\mathcal{R}_0$ is computed as the expected number of new drinkers produced by one drinker in a fully susceptible population. At the alcohol-free equilibrium (AFE), $S^* = \frac{\Lambda}{\mu+\theta}$, $L^* = A^* = R^* = 0$, $P^* = \frac{\theta\Lambda}{\mu(\mu+\theta)}$. Although relapse contributes to persistence of alcoholism, the recovered class does not generate new drinkers directly. Hence the infected subsystem is taken as (L, A). The new behavioral transition matrix F and behavioral transition matrix V are:

$$F = \begin{bmatrix} \beta S^* & 0 \\ 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} \alpha+\mu & 0 \\ -\alpha & \gamma+\mu+\delta \end{bmatrix}. \tag{20}$$

The determinant of V is

$$|V| = (\alpha+\mu)(\gamma+\mu+\delta). \tag{21}$$

Hence, the inverse of V is

$$V^{-1} = \frac{1}{(\alpha+\mu)(\gamma+\mu+\delta)} \begin{bmatrix} \gamma+\mu+\delta & 0 \\ \alpha & \alpha+\mu \end{bmatrix}. \tag{22}$$

Therefore,

$$V^{-1} = \begin{bmatrix} \frac{1}{\alpha+\mu} & 0 \\ \frac{\alpha}{(\alpha+\mu)(\gamma+\mu+\delta)} & \frac{1}{\gamma+\mu+\delta} \end{bmatrix} \tag{23}$$

The next generation matrix is

$$K = FV^{-1} = \begin{bmatrix} \beta S^* & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\alpha+\mu} & 0 \\ \frac{\alpha}{(\alpha+\mu)(\gamma+\mu+\delta)} & \frac{1}{\gamma+\mu+\delta} \end{bmatrix}. \tag{24}$$

Hence, the basic reproduction number is

$$\mathcal{R}_0 = \frac{\beta S^*}{\alpha+\mu} = \frac{\beta\Lambda}{(\mu+\theta)(\alpha+\mu)}. \tag{25}$$

The relapse parameter ρ does not explicitly appear in $\mathcal{R}_0$ because relapse does not generate new drinkers directly, but instead contributes indirectly to persistence through the addicted and recovered classes.

The basic reproduction number $\mathcal{R}_0$ represents the average number of new light-drinkers generated by a single light drinker introduced into a completely alcohol-free population. It serves as a threshold parameter governing the transmission dynamics of alcoholism as a socially driven behavior. When $\mathcal{R}_0 < 1$, each individual engaged in alcohol use produces less than one secondary case on average, indicating that peer influence is insufficient to sustain alcohol consumption and that locally asymptotically stable. Conversely, when $\mathcal{R}_0 > 1$, each individual generates more than one secondary case on average, implying that social interaction and peer pressure are strong enough to maintain and propagate alcohol-use behavior, leading to persistence of alcoholism in the population. Thus, reducing R_0 below unity through interventions such as reducing peer influence, increasing awareness, enhancing rehabilitation, and limiting progression to addiction is essential for effective control and eventual elimination of alcoholism.

6 Stability Analysis

This section examines the local and global stability of the equilibrium points to determine the long-term behavior of the alcoholism dynamics in the population.

6.1 Local Stability Analysis of the Alcohol-Free Equilibrium (AFE)

Consider the system of equations (1) Let $X = (S, L, A, R, P)^T$. The Jacobian J of the system is:

$$J = \begin{bmatrix} -(\mu+\theta) & -\beta S & 0 & 0 & 0 \\ 0 & \beta S - (\alpha+\mu) & 0 & 0 & 0 \\ 0 & \alpha & -(\gamma+\mu+\delta) & \rho & 0 \\ 0 & 0 & \gamma & -(\rho+\mu) & 0 \\ \theta & 0 & 0 & 0 & -\mu \end{bmatrix}. \tag{26}$$

At the alcohol-free equilibrium (AFE):

$$(S^*, L^*, A^*, R^*, P^*) = \left(\frac{\Lambda}{\mu+\theta},\ 0,\ 0,\ 0,\ \frac{\theta\Lambda}{\mu(\mu+\theta)} \right), \tag{27}$$

the Jacobian becomes

$$J_{AFE} = \begin{bmatrix} -(\mu+\theta) & -\frac{\beta\Lambda}{(\mu+\theta)} & 0 & 0 & 0 \\ 0 & \frac{\beta\Lambda}{(\mu+\theta)} - (\alpha+\mu) & 0 & 0 & 0 \\ 0 & \alpha & -(\gamma+\mu+\delta) & \rho & 0 \\ 0 & 0 & \gamma & -(\rho+\mu) & 0 \\ \theta & 0 & 0 & 0 & -\mu \end{bmatrix}. \tag{28}$$

The Jacobian is block lower-triangular. The eigenvalues are:

$$\begin{aligned} \lambda_1 = -(\mu+\theta), \quad \lambda_2 = -\mu, \quad \lambda_3 = \frac{\beta\Lambda}{(\mu+\theta)} - (\alpha+\mu), \\ \lambda_4, \lambda_5 = \text{eigenvalues of } \begin{bmatrix} -(\gamma+\mu+\delta) & \rho \\ \gamma & -(\rho+\mu) \end{bmatrix}. \end{aligned} \tag{29}$$

The first two eigenvalues are negative. The 2×2 block corresponding to (A, R) has eigenvalues with negative real parts. The eigenvalue corresponding to the light drinking compartment L is:

$$\lambda_3 = \frac{\beta\Lambda}{(\mu+\theta)} - (\alpha+\mu) = (\alpha+\mu)(\mathcal{R}_0 - 1), \tag{30}$$

where the basic reproduction number is

$$\mathcal{R}_0 = \frac{\beta S^*}{\alpha+\mu} = \frac{\beta\Lambda}{(\mu+\theta)(\alpha+\mu)}. \tag{31}$$

$$\begin{cases} \mathcal{R}_0 < 1 \implies \text{all eigenvalues have negative real parts} \implies \text{AFE is locally asymptotically stable,} \\ \mathcal{R}_0 > 1 \implies \lambda_3 > 0 \implies \text{AFE is unstable.} \end{cases} \tag{32}$$

6.2 Global Stability of the Alcohol-Free Equilibrium (AFE)

In this section, the global asymptotic stability of the alcohol-free equilibrium is established using the approach of Castillo-Chavez and Song.

Consider system (1) written in the form

$$\begin{aligned} \frac{dX}{dt} &= F(X, Z), \\ \frac{dZ}{dt} &= G(X, Z), \end{aligned} \tag{33}$$

where

$$X = (S, P)$$

represents the uninfected compartments and

$$Z = (L, A, R)$$

represents the alcoholism-related compartments.

The alcohol-free equilibrium is

$$E_0 = \left(\frac{\Lambda}{\mu+\theta}, 0, 0, 0, \frac{\theta\Lambda}{\mu(\mu+\theta)} \right). \tag{34}$$

The subsystem corresponding to the uninfected classes is

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - (\mu+\theta)S, \\ \frac{dP}{dt} &= \theta S - \mu P. \end{aligned} \tag{35}$$

Solving system (35) gives

$$S(t) \to \frac{\Lambda}{\mu+\theta}, \qquad P(t) \to \frac{\theta\Lambda}{\mu(\mu+\theta)}$$

as $t \to \infty$.

Hence, the alcohol-free equilibrium of subsystem (35) is globally asymptotically stable.

Now consider the alcoholism subsystem:

$$\begin{aligned} \frac{dL}{dt} &= \beta SL - (\alpha + \mu)L, \\ \frac{dA}{dt} &= \alpha L + \rho R - (\gamma + \mu + \delta)A, \\ \frac{dR}{dt} &= \gamma A - (\rho + \mu)R. \end{aligned} \tag{36}$$

This system can be written as

$$G(X, Z) = AZ - \widehat{G}(X, Z), \tag{37}$$

where

$$A = D_Z G(E_0, 0)$$

is the Jacobian matrix evaluated at the alcohol-free equilibrium:

$$A = \begin{bmatrix} \beta S^* - (\alpha + \mu) & 0 & 0 \\ \alpha & -(\gamma + \mu + \delta) & \rho \\ 0 & \gamma & -(\rho + \mu) \end{bmatrix}, \tag{38}$$

with

$$S^* = \frac{\Lambda}{\mu + \theta}.$$

When $R_0 < 1$,

$$\beta S^* - (\alpha + \mu) < 0,$$

and all eigenvalues of matrix A have negative real parts. Hence, A is a Metzler stable matrix.

Furthermore,

$$\widehat{G}(X, Z) = \begin{bmatrix} \beta(S^* - S)L \\ 0 \\ 0 \end{bmatrix} \geq 0$$

for all $(X, Z) \in \Omega$, since

$$S \leq S^*.$$

Therefore, the conditions of the Castillo-Chavez theorem are satisfied:

1. the alcohol-free equilibrium of the uninfected subsystem is globally asymptotically stable;
2. $G(X, Z)$ satisfies
$$G(X, Z) = AZ - \widehat{G}(X, Z), \qquad \widehat{G}(X, Z) \geq 0.$$

Hence, by the theorem of Castillo-Chavez and Song, the alcohol-free equilibrium E_0 of system (1) is globally asymptotically stable whenever

$$R_0 \leq 1.$$

■

6.3 Local Stability of the Endemic Alcoholism Equilibrium (EAE)

The Jacobian matrix evaluated at EAE is:

$$J_{EAE} = \begin{bmatrix} -(\mu+\theta)-\beta L^* & -\beta S^* & 0 & 0 & 0 \\ \beta L^* & \beta S^* - (\alpha+\mu) & 0 & 0 & 0 \\ 0 & \alpha & -(\gamma+\mu+\delta) & \rho & 0 \\ 0 & 0 & \gamma & -(\rho+\mu) & 0 \\ \theta & 0 & 0 & 0 & -\mu \end{bmatrix}. \tag{39}$$

$$\begin{aligned} \det(M-\lambda I) = -(\lambda+\mu)\Big(\lambda^2 + \lambda(\delta+\gamma+2\mu+\rho) + (\delta\mu+\delta\rho+\gamma\mu+\mu^2+\mu\rho)\Big) \\ \times\Big(\lambda^2 + \lambda\big(\beta L^* - \beta S^* + \alpha + 2\mu + \theta\big) \\ +\big(\beta L^*\alpha + \beta L^*\mu - \beta S^*\mu - \beta S^*\theta + \alpha\mu + \alpha\theta + \mu^2 + \mu\theta\big)\Big). \end{aligned} \tag{40}$$

Thus, the characteristic equation is

$$\begin{aligned} (\lambda+\mu)\Big[\lambda^2 + \lambda(\delta+\gamma+2\mu+\rho) + (\delta\mu+\delta\rho+\gamma\mu+\mu^2+\mu\rho)\Big] \\ \times\Big[\lambda^2 + \lambda(\beta L^* - \beta S^* + \alpha + 2\mu + \theta) \\ +\big(\beta L^*\alpha + \beta L^*\mu - \beta S^*\mu - \beta S^*\theta + \alpha\mu + \alpha\theta + \mu^2 + \mu\theta\big)\Big] = 0. \end{aligned} \tag{41}$$

Let

$$\begin{aligned} a_1 &= \delta+\gamma+2\mu+\rho, \\ b_1 &= \delta\mu+\delta\rho+\gamma\mu+\mu^2+\mu\rho, \\ a_2 &= \beta L^* - \beta S^* + \alpha + 2\mu + \theta, \\ b_2 &= \beta L^*\alpha + \beta L^*\mu - \beta S^*\mu - \beta S^*\theta + \alpha\mu + \alpha\theta + \mu^2 + \mu\theta. \end{aligned} \tag{42}$$

The eigenvalues of the matrix are:

$$\begin{aligned} \lambda_1 &= -\mu, \\ \lambda_{2,3} &= \frac{-a_1 \pm \sqrt{a_1^2 - 4b_1}}{2}, \\ \lambda_{4,5} &= \frac{-a_2 \pm \sqrt{a_2^2 - 4b_2}}{2}. \end{aligned} \tag{43}$$

The eigenvalue corresponding to P is $-\mu < 0$. The remaining 4×4 block (S, L, A, R) determines the stability of the endemic alcoholism equilibrium. Since all coefficients of the characteristic polynomials are positive for $\mathcal{R}_0 > 1$, and the corresponding Routh–Hurwitz determinants are positive, all eigenvalues have negative real parts. Hence, the endemic alcoholism equilibrium is locally asymptotically stable whenever $\mathcal{R}_0 > 1$.

$$\mathcal{R}_0 > 1 \implies \text{EAE exists and is locally asymptotically stable.}$$

7 Bifurcation Analysis

In this section, the occurrence of bifurcation in system (1) is investigated using the center manifold theory of Castillo-Chavez and Song. Consider the system

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SL - \mu S - \theta S = f_1,\\
\frac{dL}{dt} &= \beta SL - (\alpha+\mu)L = f_2,\\
\frac{dA}{dt} &= \alpha L + \rho R - (\gamma+\mu+\delta)A = f_3,\\
\frac{dR}{dt} &= \gamma A - (\rho+\mu)R = f_4,\\
\frac{dP}{dt} &= \theta S - \mu P = f_5.
\end{aligned} \tag{44}$$

Let the state variables be ordered as

$$X = (S, L, A, R, P)^T. \tag{45}$$

The alcohol-free equilibrium (AFE) of system (44) is

$$E_0 = \left(\frac{\Lambda}{\mu+\theta}, 0, 0, 0, \frac{\theta\Lambda}{\mu(\mu+\theta)}\right). \tag{46}$$

The basic reproduction number is

$$\mathcal{R}_0 = \frac{\beta\Lambda}{(\mu+\theta)(\alpha+\mu)}. \tag{47}$$

Setting $R_0 = 1$, the critical bifurcation value is obtained as

$$\beta^* = \frac{(\mu+\theta)(\alpha+\mu)}{\Lambda}. \tag{48}$$

7.1 Jacobian Matrix at the AFE

The Jacobian matrix evaluated at E_0 is

$$J(E_0) = \begin{bmatrix}
-(\mu+\theta) & -\beta\dfrac{\Lambda}{\mu+\theta} & 0 & 0 & 0\\
0 & \beta\dfrac{\Lambda}{\mu+\theta} - (\alpha+\mu) & 0 & 0 & 0\\
0 & \alpha & -(\gamma+\mu+\delta) & \rho & 0\\
0 & 0 & \gamma & -(\rho+\mu) & 0\\
\theta & 0 & 0 & 0 & -\mu
\end{bmatrix}. \tag{49}$$

At $\beta = \beta^*$, the Jacobian becomes

$$J(E_0, \beta^*) = \begin{bmatrix}
-(\mu+\theta) & -(\alpha+\mu) & 0 & 0 & 0\\
0 & 0 & 0 & 0 & 0\\
0 & \alpha & -(\gamma+\mu+\delta) & \rho & 0\\
0 & 0 & \gamma & -(\rho+\mu) & 0\\
\theta & 0 & 0 & 0 & -\mu
\end{bmatrix}. \tag{50}$$

The matrix $J(E_0, \beta^*)$ possesses a simple zero eigenvalue.

7.2 Right Eigenvector

Let

$$W = (w_1, w_2, w_3, w_4, w_5)^T \tag{51}$$

be the right eigenvector associated with the zero eigenvalue such that

$$J(E_0, \beta^*)W = 0. \tag{52}$$

Solving the resulting system yields

$$w_1 = -\frac{\alpha + \mu}{\mu + \theta} w_2, \tag{53}$$

$$w_3 = \frac{\alpha(\rho + \mu)}{D} w_2, \tag{54}$$

$$w_4 = \frac{\alpha\gamma}{D} w_2, \tag{55}$$

$$w_5 = -\frac{\theta(\alpha + \mu)}{\mu(\mu + \theta)} w_2, \tag{56}$$

where

$$D = (\gamma + \mu + \delta)(\rho + \mu) - \rho\gamma. \tag{57}$$

Choosing $w_2 = 1$, the right eigenvector becomes

$$W = \begin{bmatrix} -\frac{\alpha + \mu}{\mu + \theta} \\ 1 \\ \frac{\alpha(\rho + \mu)}{D} \\ \frac{\alpha\gamma}{D} \\ -\frac{\theta(\alpha + \mu)}{\mu(\mu + \theta)} \end{bmatrix}. \tag{58}$$

7.3 Left Eigenvector

Let

$$V = (v_1, v_2, v_3, v_4, v_5) \tag{59}$$

be the left eigenvector satisfying

$$VJ(E_0, \beta^*) = 0. \tag{60}$$

Solving gives

$$v_1 = v_3 = v_4 = v_5 = 0. \tag{61}$$

Choosing $v_2 = 1$, the left eigenvector is

$$V = (0, 1, 0, 0, 0). \tag{62}$$

7.4 Computation of the Bifurcation Coefficients

The only nonlinear term in system (44) is

$$f_2 = \beta SL - (\alpha + \mu)L. \tag{63}$$

Hence, the only nonzero second-order partial derivatives are

$$\frac{\partial^2 f_2}{\partial S \partial L} = \frac{\partial^2 f_2}{\partial L \partial S} = \beta, \tag{64}$$

and

$$\frac{\partial^2 f_2}{\partial L \partial \beta} = S. \tag{65}$$

At the AFE,

$$S^0 = \frac{\Lambda}{\mu + \theta}. \tag{66}$$

Using the Castillo-Chavez and Song formulation,

$$a = \sum_{k,i,j} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0, \beta^*), \tag{67}$$

and

$$b = \sum_{k,i} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(E_0, \beta^*). \tag{68}$$

Since only $v_2 \neq 0$, we obtain

$$a = 2\beta^* w_1 w_2. \tag{69}$$

Substituting $w_2 = 1$ and

$$w_1 = -\frac{\alpha + \mu}{\mu + \theta}, \tag{70}$$

gives

$$a = 2\beta^* \left(-\frac{\alpha + \mu}{\mu + \theta} \right). \tag{71}$$

Using

$$\beta^* = \frac{(\mu + \theta)(\alpha + \mu)}{\Lambda}, \tag{72}$$

we obtain

$$a = -\frac{2(\alpha + \mu)^2}{\Lambda}. \tag{73}$$

Similarly,

$$b = w_2 S^0. \tag{74}$$

Since $w_2 = 1$,

$$b = \frac{\Lambda}{\mu + \theta}. \tag{75}$$

Because

$$a < 0 \qquad \text{and} \qquad b > 0, \tag{76}$$

system (1) undergoes a forward (transcritical) bifurcation at $R_0 = 1$. Therefore, the endemic alcoholism equilibrium exists and is locally asymptotically stable whenever $R_0 > 1$, while the alcohol-free equilibrium is locally asymptotically stable for $R_0 < 1$.

7.5 Bifurcation Direction

To determine the endemic alcoholism equilibrium of system (1), we set

$$\frac{dS}{dt} = \frac{dL}{dt} = \frac{dA}{dt} = \frac{dR}{dt} = \frac{dP}{dt} = 0. \tag{77}$$

Thus,

$$\begin{aligned} 0 &= \Lambda - \beta SL - (\mu + \theta)S, \\ 0 &= \beta SL - (\alpha + \mu)L, \\ 0 &= \alpha L + \rho R - (\gamma + \mu + \delta)A, \\ 0 &= \gamma A - (\rho + \mu)R, \\ 0 &= \theta S - \mu P. \end{aligned} \tag{78}$$

Since the P-equation is decoupled from the remaining equations, it does not influence the bifurcation structure. From the fourth equation of (78),

$$R = \frac{\gamma}{\rho + \mu} A. \tag{79}$$

Substituting (79) into the third equation of (78) gives

$$\alpha L + \rho \left(\frac{\gamma}{\rho + \mu} A \right) = (\gamma + \mu + \delta)A. \tag{80}$$

Hence,

$$L = \frac{(\gamma + \mu + \delta)(\rho + \mu) - \rho\gamma}{\alpha(\rho + \mu)} A \equiv k_1 A. \tag{81}$$

From the second equation of (78), assuming $L \neq 0$,

$$\beta S = \alpha + \mu,$$

so that

$$S = \frac{\alpha + \mu}{\beta}. \tag{82}$$

Substituting (81) and (82) into the first equation of (78) yields

$$0 = \Lambda - (\alpha + \mu)k_1 A - \frac{(\mu + \theta)(\alpha + \mu)}{\beta}.$$

Thus,

$$A = \frac{\Lambda\beta - (\mu + \theta)(\alpha + \mu)}{\beta(\alpha + \mu)k_1}. \tag{83}$$

Since

$$R_0 = \frac{\beta\Lambda}{(\mu + \theta)(\alpha + \mu)}, \tag{84}$$

equation (83) can be written as

$$A = \frac{(\mu + \theta)(\alpha + \mu)(R_0 - 1)}{\beta(\alpha + \mu)k_1}. \tag{85}$$

Hence,

$$A > 0 \qquad \text{if and only if} \qquad R_0 > 1. \tag{86}$$

Therefore, the endemic alcoholism equilibrium exists uniquely whenever $R_0 > 1$, confirming the absence of backward bifurcation.

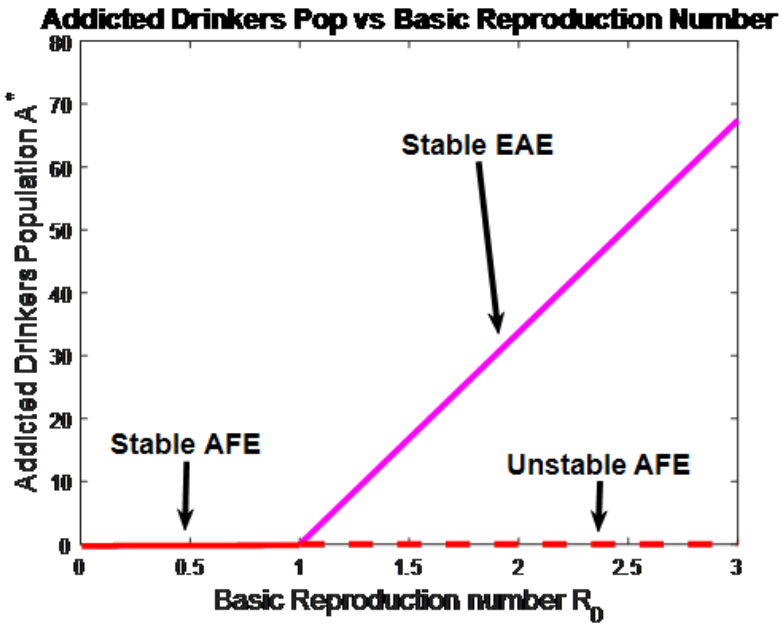


Figure 2: Forward bifurcation diagram of system (1)

8 Sensitivity Analysis

We compute normalized sensitivity indices of $\mathcal{R}_0$ to identify parameters with the greatest influence on alcoholism transmission dynamics (e.g., β, θ, and α).

$$S_\beta = 1, \qquad S_\Lambda = 1. \tag{87}$$

$$S_\theta = \frac{\partial \ln \mathcal{R}_0}{\partial \ln \theta} = -\frac{\theta}{\mu+\theta}, \qquad S_\alpha = -\frac{\alpha}{\alpha+\mu}. \tag{88}$$

$$S_\mu = \frac{\partial \ln \mathcal{R}_0}{\partial \ln \mu} = -\frac{\mu}{\mu+\theta} - \frac{\mu}{\alpha+\mu}. \tag{89}$$

8.1 Interpretation of Sensitivity Indices

The normalized forward sensitivity indices measure the relative change in the basic reproduction number $\mathcal{R}_0$ due to a relative change in a model parameter. A positive sensitivity index indicates that increasing the corresponding parameter increases alcoholism transmission, whereas a negative sensitivity index implies that increasing the parameter reduces the spread and persistence of alcoholism.

Using the parameter values in Table 4,

$$\Lambda = 10, \quad \beta = 0.0005, \quad \mu = 0.02, \quad \theta = 0.03, \quad \alpha = 0.05, \tag{90}$$

the numerical sensitivity indices are computed as follows:

$$S_\beta = 1, \qquad S_\Lambda = 1, \tag{91}$$

$$S_\theta = -\frac{\theta}{\mu+\theta} = -\frac{0.03}{0.05} = -0.6, \tag{92}$$

$$S_\alpha = -\frac{\alpha}{\alpha+\mu} = -\frac{0.05}{0.07} = -0.7143, \tag{93}$$

$$S_\mu = -\frac{\mu}{\mu+\theta} - \frac{\mu}{\alpha+\mu} = -\frac{0.02}{0.05} - \frac{0.02}{0.07} = -0.6857. \tag{94}$$

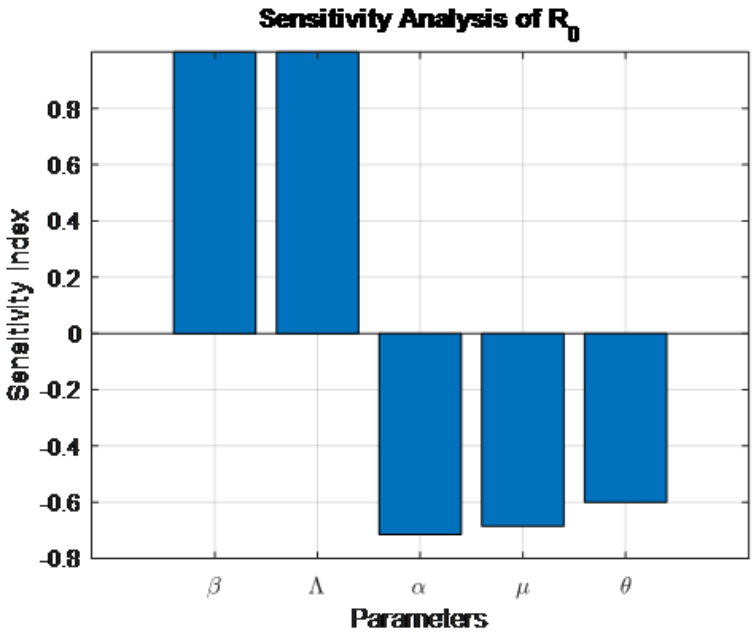


Figure 3: Figure showing the normalized sensitivity analysis of R_0 with respect to model parameters

The positive indices of β and Λ indicate that increased peer influence and recruitment into the susceptible population strongly promote alcoholism persistence. In contrast, the negative indices of θ, α, and μ indicate that increasing awareness, progression out of the light-drinking stage, and natural removal reduce alcoholism transmission.

The ranking of the sensitivity indices in decreasing order of influence is given by

$$|S_\beta| = |S_\Lambda| > |S_\alpha| > |S_\mu| > |S_\theta|. \tag{95}$$

This ranking shows that peer influence and population recruitment are the most influential parameters driving alcoholism spread, while awareness programs also contribute significantly toward reducing addiction prevalence.

Table 3: Normalized sensitivity indices of R_0

Parameter	Sensitivity Index	Interpretation
β	1.0000	Strong positive influence on alcoholism spread
Λ	1.0000	Increased recruitment increases alcoholism persistence
α	-0.7143	Reduces duration of light drinking stage
μ	-0.6857	Natural removal reduces persistence
θ	-0.6000	Awareness reduces susceptibility to alcoholism

9 Optimal Control Analysis

To investigate effective intervention strategies for alcoholism control, we incorporate three time-dependent control functions into model (1). The controls are defined as follows:

- $u_1(t)$: prevention and education effort aimed at reducing susceptibility to alcoholism,
- $u_2(t)$: rehabilitation and treatment effort for addicted individuals,
- $u_3(t)$: relapse and transmission reduction effort through social regulation, awareness campaigns, and restricted alcohol access.

The controls satisfy

$$0 \leq u_i(t) \leq 1, \qquad i = 1, 2, 3, \quad t \in [0, T]. \tag{96}$$

9.1 Controlled Model

Incorporating the control measures into system (1) yields the following controlled system:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta(1-u_3)SL - \mu S - \theta u_1 S, \\
\frac{dL}{dt} &= \beta(1-u_3)SL - (\alpha+\mu)L, \\
\frac{dA}{dt} &= \alpha L + \rho R - (\gamma+\mu+\delta)A - u_2 A, \\
\frac{dR}{dt} &= \gamma A + u_2 A - (\rho+\mu)R, \\
\frac{dP}{dt} &= \theta u_1 S - \mu P,
\end{aligned} \tag{97}$$

subject to the initial conditions

$$S(0) = S_0, \quad L(0) = L_0, \quad A(0) = A_0, \quad R(0) = R_0, \quad P(0) = P_0 \geq 0. \tag{98}$$

9.2 Objective Functional

The objective is to minimize the number of light-drinkers and addicted drinkers while simultaneously minimizing the implementation costs of the controls over the time interval $[0, T]$. Thus, the objective functional is defined by

$$J(u_1, u_2, u_3) = \int_0^T \left[w_1 L(t) + w_2 A(t) + \frac{1}{2} c_1 u_1^2(t) + \frac{1}{2} c_2 u_2^2(t) + \frac{1}{2} c_3 u_3^2(t) \right] dt, \tag{99}$$

where w_1, w_2 denote the relative societal costs associated with light-drinkers and addicted drinkers, respectively, while $c_1, c_2, c_3 > 0$ represent the costs associated with implementing the controls. The admissible control set is given by

$$\mathcal{U} = \{(u_1, u_2, u_3) : u_i(\cdot) \text{ are measurable}, \ 0 \leq u_i(t) \leq 1, \ i = 1, 2, 3\}. \tag{100}$$

The optimal control problem consists of finding an optimal control triple

$$(u_1^*, u_2^*, u_3^*) \in \mathcal{U} \tag{101}$$

such that

$$J(u_1^*, u_2^*, u_3^*) = \min_{(u_1, u_2, u_3) \in \mathcal{U}} J(u_1, u_2, u_3), \tag{102}$$

.

9.3 Pontryagin's Maximum Principle

subject to the controlled system (97) To derive the necessary conditions for optimality, we apply Pontryagin's Maximum Principle. Let

$$\lambda_i(t), \qquad i = 1, \ldots, 5, \tag{103}$$

be the adjoint variables associated with the state variables S, L, A, R, and P, respectively. The Hamiltonian is defined as

$$\begin{aligned}\mathcal{H} = {} & w_1 L + w_2 A + \frac{1}{2}c_1 u_1^2 + \frac{1}{2}c_2 u_2^2 + \frac{1}{2}c_3 u_3^2 \\ & + \lambda_1\Big(\Lambda - \beta(1-u_3)SL - \mu S - \theta u_1 S\Big) \\ & + \lambda_2\Big(\beta(1-u_3)SL - (\alpha+\mu)L\Big) \\ & + \lambda_3\Big(\alpha L + \rho R - (\gamma+\mu+\delta)A - u_2 A\Big) \\ & + \lambda_4\Big(\gamma A + u_2 A - (\rho+\mu)R\Big) \\ & + \lambda_5\Big(\theta u_1 S - \mu P\Big).\end{aligned} \tag{104}$$

9.4 Adjoint Equations

According to Pontryagin's Maximum Principle, the adjoint equations satisfy

$$\dot{\lambda}_i(t) = -\frac{\partial \mathcal{H}}{\partial x_i}, \qquad i = 1, \ldots, 5, \tag{105}$$

with transversality conditions

$$\lambda_i(T) = 0, \qquad i = 1, \ldots, 5. \tag{106}$$

Hence, the adjoint system is given by

$$\begin{aligned}\frac{d\lambda_1}{dt} &= \mu\lambda_1 + \beta(1-u_3)L(\lambda_1 - \lambda_2) + \theta u_1(\lambda_1 - \lambda_5), \\ \frac{d\lambda_2}{dt} &= -w_1 + \beta(1-u_3)S(\lambda_1 - \lambda_2) + (\alpha+\mu)\lambda_2 - \alpha\lambda_3, \\ \frac{d\lambda_3}{dt} &= -w_2 + (\gamma+\mu+\delta+u_2)\lambda_3 - (\gamma+u_2)\lambda_4, \\ \frac{d\lambda_4}{dt} &= -\rho\lambda_3 + (\rho+\mu)\lambda_4, \\ \frac{d\lambda_5}{dt} &= \mu\lambda_5.\end{aligned} \tag{107}$$

9.5 Characterization of the Optimal Controls

The optimal controls are obtained by minimizing the Hamiltonian with respect to u_1, u_2, and u_3. Setting

$$\frac{\partial \mathcal{H}}{\partial u_i} = 0, \qquad i = 1, 2, 3, \tag{108}$$

gives the following characterizations.

For u_1,

$$\frac{\partial \mathcal{H}}{\partial u_1} = c_1 u_1 + \theta S(\lambda_5 - \lambda_1), \tag{109}$$

which yields

$$u_1^*(t) = \min\left\{1, \max\left(0, \frac{\theta S(t)\big(\lambda_1(t) - \lambda_5(t)\big)}{c_1}\right)\right\}. \tag{110}$$

For u_2,

$$\frac{\partial \mathcal{H}}{\partial u_2} = c_2 u_2 + A(\lambda_4 - \lambda_3), \tag{111}$$

thus

$$u_2^*(t) = \min\left\{1, \max\left(0, \frac{A(t)\big(\lambda_3(t) - \lambda_4(t)\big)}{c_2}\right)\right\}. \tag{112}$$

Similarly, for u_3,

$$\frac{\partial \mathcal{H}}{\partial u_3} = c_3 u_3 + \beta S L(\lambda_1 - \lambda_2), \tag{113}$$

which gives

$$u_3^*(t) = \min\left\{1, \max\left(0, \frac{\beta S(t) L(t)\big(\lambda_2(t) - \lambda_1(t)\big)}{c_3}\right)\right\}. \tag{114}$$

The state system (97), adjoint system (107), transversality conditions, and control characterizations (110)–(114) together constitute the optimality system.

9.6 Existence of an Optimal Control

The existence of an optimal control follows from standard results in optimal control theory [14, 13]. In particular, the admissible control set

$$\mathcal{U} = \{(u_1, u_2, u_3) : u_i(\cdot) \text{ are measurable}, \ 0 \le u_i(t) \le 1, \ i = 1, 2, 3\} \tag{115}$$

is nonempty, convex, and closed.

Furthermore, the right-hand side of the controlled system (97) is continuously differentiable with respect to the state variables and depends linearly on the control variables u_1, u_2, and u_3. Hence, for bounded controls and initial conditions, the state system admits bounded solutions and satisfies the Lipschitz condition.

In addition, the integrand of the objective functional

$$w_1 L(t) + w_2 A(t) + \frac{1}{2} c_1 u_1^2(t) + \frac{1}{2} c_2 u_2^2(t) + \frac{1}{2} c_3 u_3^2(t) \tag{116}$$

is convex in the control variables since $c_1, c_2, c_3 > 0$.

Therefore, by standard existence results of optimal control theory, there exists an optimal control triple

$$(u_1^*, u_2^*, u_3^*) \in \mathcal{U} \tag{117}$$

that minimizes the objective functional $J(u_1, u_2, u_3)$.

9.7 Numerical Scheme

The optimality system can be solved numerically using the forward–backward sweep method as follows:

1. Initialize the controls $u_1^{(0)}(t)$, $u_2^{(0)}(t)$, and $u_3^{(0)}(t)$.
2. Solve the state system (97) forward in time using the initial conditions.
3. Solve the adjoint system (107) backward in time subject to the terminal conditions

$$\lambda_i(T) = 0, \qquad i = 1, \ldots, 5. \tag{118}$$

4. Update the controls using equations (110), (112), and (114).
5. Repeat steps 2–4 until convergence is achieved.

10 Numerical Simulations

To illustrate the analytical results, numerical simulations of the controlled and uncontrolled systems were performed using MATLAB ode45 on the interval $0 \leq t \leq 100$. The forward-backward sweep algorithm was iterated until the relative error between successive control profiles was less than 10^{-6}. The parameter values used in the simulations are adopted from existing literature, as summarized in Table 1. The parameter values are chosen within biologically plausible ranges from related literature to illustrate qualitative dynamics rather than quantitative prediction. These values are used for qualitative illustration of the model dynamics due to the absence of region-specific epidemiological data. The following biologically feasible initial conditions were used:

$$S(0) = 80, \quad L(0) = 20, \quad A(0) = 10, \quad R(0) = 5, \quad P(0) = 0. \tag{119}$$

The computed basic reproduction number for the system is

$$\mathcal{R}_0 = 1.4286. \tag{120}$$

Since $\mathcal{R}_0 > 1$, the numerical result confirms that alcoholism persists in the population in the absence of control measures. However, under optimal control intervention, the effective reproduction dynamics are significantly reduced, leading to a decline in addiction prevalence over time. The control functions $u_1(t)$, $u_2(t)$, and $u_3(t)$ obtained from the optimality system are implemented numerically using the forward-backward sweep method. For comparison purposes, two scenarios are considered:

- **Uncontrolled case:** $u_1(t) = u_2(t) = u_3(t) = 0$
- **Controlled case:** optimal controls (u_1^*, u_2^*, u_3^*) from Pontryagin's Maximum Principle

The system is solved using MATLAB ode45, combined with an iterative forward-backward sweep algorithm. The state system is solved forward in time, while the adjoint system is solved backward in time. Controls are updated at each iteration until convergence is achieved.

Table 4: Parameter values used in the Simulation

Parameter	Values	Source
Λ	10	Estimated for qualitative illustration
β	0.0005	[2]
μ	0.02	[2]
θ	0.03	[3]
α	0.05	[7]
γ	0.04	[10]
δ	0.01	[10]
ρ	0.03	[6]

Subfigure 5(a) shows the dynamics of the susceptible population, where implementing control strategies leads to a faster decline as individuals transition into protected or other compartments. Subfigure 5(b) illustrates the behavior of light drinkers, indicating a significant reduction under control due to decreased initiation and progression. Subfigure 5(c) presents the addicted population, demonstrating that combined interventions effectively suppress addiction levels over time compared to the uncontrolled case. Subfigure 5(d) depicts the recovered individuals, where control measures enhance recovery rates and sustain higher recovery levels. Finally, subfigure 5(e) represents the protected class, showing a steady increase under control strategies due to awareness and preventive measures. Overall, the comparison highlights that the application of combined controls substantially mitigates alcoholism prevalence and improves population outcomes.

The results demonstrate that combining prevention, treatment, and awareness strategies significantly reduces the burden of alcoholism compared to the uncontrolled scenario. This supports the analytical result that optimal intervention strategies can effectively drive the system toward reduced addiction levels.

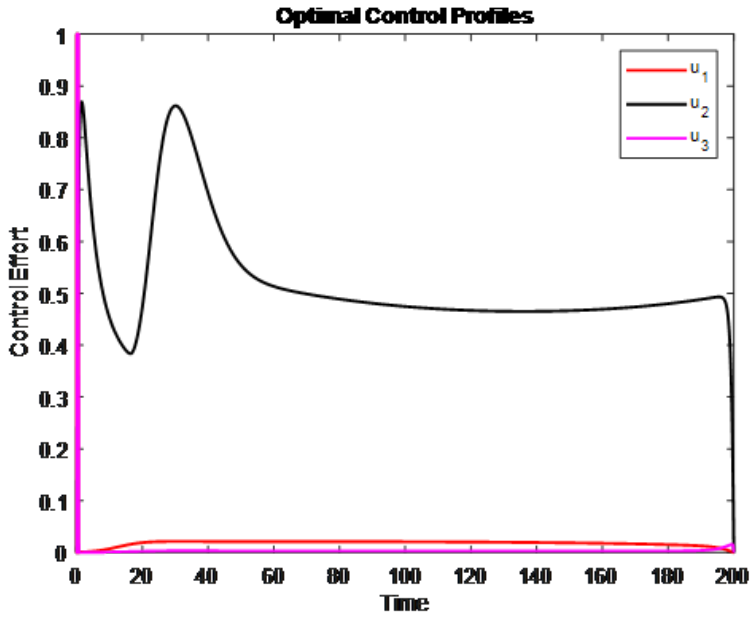


Figure 4: Time evolution of the alcoholism dynamics under optimal control intervention

11 Discussion

The mathematical analysis of the model establishes the basic reproduction number R_0 as a key threshold parameter governing the dynamics of alcoholism in the population. Biologically and behaviorally, R_0 determines whether alcohol abuse dies out or persists. while the alcohol-free equilibrium is globally asymptotically stable. This result highlights the fundamental role of peer influence and social interaction in sustaining alcohol consumption within a population.

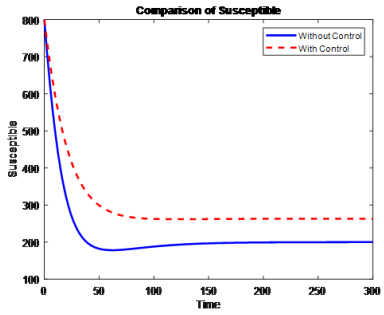
From a mathematical perspective, the stability results confirm that the system undergoes a forward (transcritical) bifurcation at $\mathcal{R}_0 = 1$, ensuring a smooth transition between alcohol-free and endemic states. This behavior is consistent with classical results in epidemic and social contagion models, where threshold dynamics determine long-term outcomes. The absence of backward bifurcation in the model further implies that reducing $\mathcal{R}_0$ below unity is sufficient for complete eradication of alcoholism without the need for more complex control strategies.

In comparison with previous studies on alcoholism and behavioral contagion dynamics [1, 11, 12], the present model agrees that peer influence (captured by β) is the dominant driver of alcohol initiation. However, the inclusion of awareness and protection compartments in this study extends earlier frameworks by explicitly quantifying the mitigating effect of education and behavioral resistance on alcohol uptake.

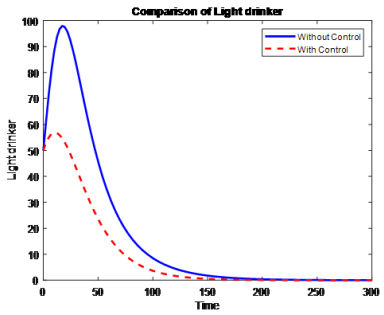
From a policy perspective, the results provide clear guidance for intervention strategies. Reducing peer influence through social regulation, limiting exposure to drinking environments, and strengthening rehabilitation programs are essential for reducing alcoholism prevalence. In particular, increasing awareness rates (θ) reduces susceptibility and shifts individuals into a protected class, thereby lowering the effective reproduction number and weakening transmission pathways.

The relapse parameter ρ plays a crucial role in sustaining alcoholism dynamics. It represents the tendency of recovered individuals to return to alcohol consumption, often due to psychological dependence, social pressure, or inadequate rehabilitation support. Mathematically, relapse acts as a feedback mechanism that replenishes the addicted class, thereby stabilizing the endemic equilibrium even when recovery efforts are present.

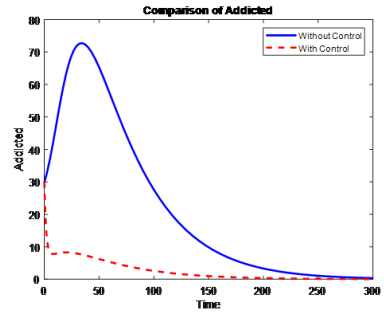
The model also highlights the importance of awareness programs in controlling alcoholism. Awareness not only reduces susceptibility but also strengthens long-term behavioral resistance by moving individuals into a protected class. This suggests that continuous education campaigns, community outreach



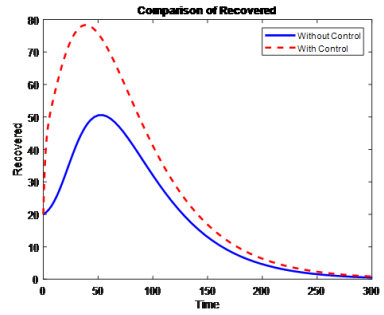
(a) Dynamics of the susceptible population with and without control interventions



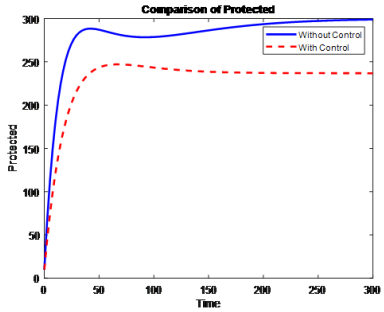
(b) plot of light-drinkers with control and without control



(c) Dynamics of the addicted individuals with control and without control



(d) Dynamics of the recovered with control and without control



(e) Dynamics of the protected with control and without control

Figure 5: Dynamics of the variables with and without control

programs, and school-based interventions are essential components of effective alcoholism prevention strategies.

Despite these contributions, the model has some limitations. The parameters used in the numerical simulations are hypothetical and not fitted to real-world epidemiological or behavioral data. Additionally, the model assumes homogeneous mixing within the population, ignoring heterogeneity in social networks, cultural influences, and individual behavioral differences. Furthermore, the deterministic framework does not capture random effects that may influence drinking behavior at the individual level.

Future work may address these limitations by incorporating age structure, spatial heterogeneity, stochastic effects, delayed relapse mechanisms, and data-driven parameter estimation. Such extensions would improve the realism and predictive power of the model, making it more suitable for direct policy application.

12 Conclusion

In this study, a deterministic compartmental model for alcoholism dynamics was formulated by incorporating peer influence, awareness, rehabilitation, relapse, and protection mechanisms. The model treats alcoholism as a socially transmitted behavior and provides a mathematical framework for understanding its spread within a population.

The analysis established that the model is mathematically well posed, with solutions remaining positive and bounded in a feasible region. Two equilibria were identified: the alcohol-free equilibrium (AFE) and the endemic alcoholism equilibrium (EAE). The basic reproduction number $\mathcal{R}_0$, derived using the next-generation matrix approach, was shown to govern the long-term behavior of the system.

The alcohol-free equilibrium is locally asymptotically stable when $\mathcal{R}_0 < 1$, while a forward transcritical bifurcation occurs at $\mathcal{R}_0 = 1$, indicating that reducing $\mathcal{R}_0$ below unity is sufficient for alcoholism elimination. Sensitivity analysis identified peer influence β and recruitment Λ as key drivers of alcoholism persistence, whereas awareness θ and natural removal μ reduce transmission.

Optimal control and numerical simulations showed that combined prevention, rehabilitation, and relapse-reduction strategies significantly lower addiction prevalence compared to the uncontrolled case. Overall, the results emphasize the importance of awareness programs, rehabilitation support, and policies that reduce peer-driven alcohol initiation. Future work may incorporate stochastic effects, age structure, spatial heterogeneity, and data-driven parameter estimation to improve model realism and policy relevance.

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