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Optimal control and stability analysis of an alcoholism model with treatment centers

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ABSTRACT

Alcoholism affects individuals across all demographics and is a major global health challenge, contributing significantly to mortality rates. This study develops and analyzes a mathematical model of alcoholism, focusing on the dynamics of drinking behaviors within a population. The model identifies two equilibrium points: the non-endemic equilibrium and the endemic equilibrium, whose stability depends on the basic reproduction number (R_0). The non-endemic equilibrium is stable when $R_0 < 1$, while the endemic equilibrium becomes stable when $R_0 > 1$. Sensitivity analysis highlights the critical role of the contact rate between at-risk individuals and moderate drinkers, as well as the rate of alcohol cessation among moderate drinkers. The study incorporates control strategies, including educational campaigns and government policy measures, to reduce the spread of alcoholism. Numerical simulations demonstrate the effectiveness of a combined approach in significantly lowering alcoholism prevalence and its social and economic impacts. This study offers practical insights for designing evidence-based policies to address this issue. Some key features of the proposed method include:

- Utilizing the next-generation matrix (NGM) approach to calculate R_0.
- Conducting equilibrium point analysis to examine the stability of the system.
- Applying Pontryagin's maximum principle to determine optimal control policies.

Specifications table

Subject area:	Mathematics and Statistics
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Name of your method:	Next-Generation Matrix (NGM) and Pontryagin's maximum principle
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Background

Alcoholism is a condition where a person cannot control their alcohol consumption, often leading to serious problems for both individuals and society. This condition affects people of all ages, education levels, and social backgrounds [1]. Alcohol is commonly

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used during social activities, especially in environments where drinking is widespread. Many people see alcohol as a way to have fun or to escape from their personal problems [2,3]. However, alcohol abuse contributes to many health and social issues around the world [4]. Each year, about 3 million people die from alcohol-related causes, and it is responsible for 5.1 % of all disease-related deaths. For people aged 15 to 49, alcohol is a major risk factor, causing about 10 % of deaths in this age group [5].

Globally, the amount of alcohol consumed remains significant. For example, the average annual consumption of pure alcohol per person was 5.7 liters in 2000, increased to 6.4 liters in 2016, and slightly decreased to 6.2 liters in 2018 [5]. Despite this slight decline, alcohol consumption remains a major global health concern. Excessive drinking is associated with severe physical health issues, including liver damage, cardiovascular diseases, and weakened immune systems, which make individuals more vulnerable to infections. Additionally, alcohol abuse negatively impacts mental health, leading to unstable behavior, impaired judgment, and increased risks of harm to oneself and others. These factors contribute to alcohol being a leading cause of preventable deaths and diseases worldwide [6].

Mathematical modeling is a valuable tool [7] and can be used for developing strategies to address its impact on individuals and society [8,9]. These models provide a systematic framework to understand how alcohol consumption spreads, identify key factors driving its dynamics, and evaluate the effectiveness of various interventions. By simulating real-world scenarios, mathematical models allow researchers and policymakers to predict outcomes, optimize resource allocation, and design targeted strategies to mitigate the problem. Earlier studies [8,10] have categorized populations based on drinking behavior, such as non-drinkers, moderate drinkers, heavy drinkers, individuals undergoing treatment, and those who have stopped drinking. Such compartmentalization helps capture the transitions between behaviors and the factors influencing them. In addition, some models have incorporated complexities like liver complications [11] and access to treatment centers [12,13] to reflect the broader health and social challenges associated with alcoholism. These models have demonstrated the critical role of education, treatment efforts, and government policies in reducing heavy drinking and supporting recovery, providing insights into how tailored interventions can improve public health outcomes.

In this study, we build on this foundation by developing a new mathematical model of alcohol consumption that integrates treatment centers and introduces additional control measures. Unlike previous models, our approach considers education as a preventive strategy aimed at moderate drinkers and government policies targeting heavy drinking. The model also accounts for behavioral changes, such as heavy drinkers transitioning to moderate drinking due to self-awareness or moderate drinkers ceasing alcohol consumption entirely. Furthermore, the model reflects the critical role of treatment centers in facilitating recovery, highlighting their influence on heavy drinkers who may only stop drinking through structured interventions.

To ensure a comprehensive analysis, we examine the stability of the models equilibrium points, identifying conditions under which alcohol consumption can be minimized or eradicated. We also conduct numerical simulations to evaluate the impact of the introduced control measures. These simulations illustrate the potential effectiveness of combined education, policy, and treatment strategies in reducing the prevalence of heavy drinking, mitigating its harmful effects, and fostering recovery. By using these findings, this study aims to provide actionable insights for designing and implementing interventions that address the root causes of alcoholism and its societal burden.

The structure of this paper reflects the systematic approach taken in this study. Section outlines the methods, including the formulation of the alcoholism model with treatment centers. It provides details about the compartments, parameters, and interactions within the system. This is followed by an examination of the model's basic properties, such as stability analysis and parameter sensitivity, to understand its underlying dynamics. Additionally, the section introduces the optimal control formulation. Section validates the methods by presenting numerical simulations that illustrate the impact of various interventions. Finally, it summarizes the findings, highlights key conclusions, and suggests directions for future research.

Method details

Formulation of an alcoholism model with treatment centers

In this section, a mathematical model of an alcoholism with treatment centers is formulated. The assumptions used for the model construction are as follows:

1. The rate of increase in the human population at risk of consuming alcohol per unit of time is constant.
2. The natural death rate per unit of time for each population is constant.
3. Heavy alcohol drinkers can return to being moderate alcohol drinkers.
4. Moderate alcohol drinkers can become individuals who quit drinking alcohol due to self-awareness emerging within moderate drinkers.
5. Death caused by excessive alcohol consumption occurs only in the population of heavy drinkers.
6. The number of individuals receiving treatment increases due to government policy efforts as a form of control.
7. The number of individuals who quit drinking alcohol increases due to educational efforts as a form of control.

The human population is divided into six compartments: individuals at risk of consuming alcohol (P), moderate alcohol drinkers (M), heavy alcohol drinkers (H), affluent heavy drinkers enrolled in private treatment centers (T^r), disadvantaged heavy drinkers enrolled in public treatment centers (T^p), and individuals who have quit drinking (Q). Based on the assumptions, we can set up the transmission diagram that is shown in Fig. 1. The defining parameters can be seen in Table 1, which are assumed constant and non-negative. From the diagram in Fig. 1, transmission models can be formulated as follows:

$$\frac{dP}{dt} = b - \beta_1 \frac{PM}{b} - \mu P, \tag{1}$$

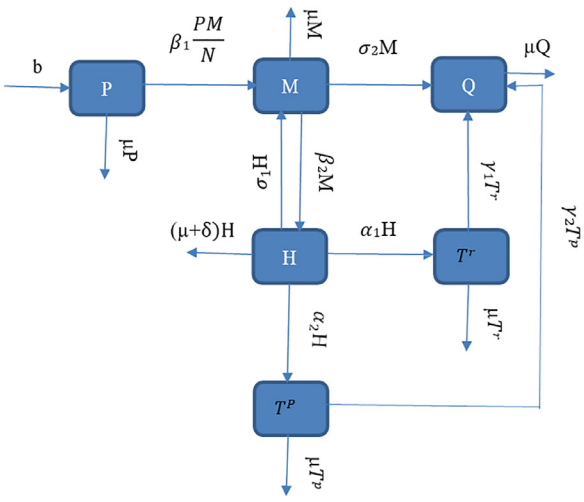

Fig. 1. Transmission diagram: Mathematical model of alcoholism with treatment centers.

Table 1
Parameters. Description of parameters in the mathematical model of an alcoholism with treatment centers.

Par	Description
b	The rate of increase in new individuals at risk of consuming alcohol.
δ	The number of deaths caused by heavy drinkers.
μ	The natural death rate.
β_1	The contact rate between individuals at risk of consuming alcohol and moderate alcohol drinkers.
β_2	The contact rate between moderate alcohol drinkers and heavy alcohol drinkers.
α_1	The rate of transition from heavy drinkers to affluent heavy drinkers joining private treatment centers.
α_2	The rate of transition from heavy drinkers to disadvantaged heavy drinkers joining public treatment centers.
σ_1	The rate at which heavy drinkers return to being moderate drinkers.
σ_2	The rate at which moderate drinkers quit drinking.
γ_1	The rate at which affluent heavy drinkers quit drinking.
γ_2	The rate at which disadvantaged heavy drinkers quit drinking.

$$\frac{dM}{dt} = \beta_1 \frac{PM}{b} + \sigma_1 H - (\beta_2 + \sigma_2 + \mu) M, \tag{2}$$

$$\frac{dH}{dt} = \beta_2 M - (\sigma_1 + \alpha_1 + \alpha_2 + \mu + \delta) H, \tag{3}$$

$$\frac{dT^r}{dt} = \alpha_1 H - (\mu + \gamma_1) T^r, \tag{4}$$

$$\frac{dT^p}{dt} = \alpha_2 H - (\mu + \gamma_2) T^p, \tag{5}$$

$$\frac{dQ}{dt} = \sigma_1 M + \gamma_1 T^r + \gamma_2 T^p - \mu Q. \tag{6}$$

The non-endemic equilibrium point for the mathematical model of an alcoholism with treatment centers occurs if there are no drinkers in a population, either moderate or heavy alcohol drinkers. Suppose the non endemic equilibrium point for the mathematical model of an alcoholism with treatment centers is expressed as E_0. Substituting $M = H = 0$ into 1-6 will get

$$E_0 = (P_0, M_0, H_0, T_0^r, T_0^p, Q_0)) = \left(\frac{b}{\mu}, 0, 0, 0, 0, 0\right).$$

Next, we will determine the basic reproduction number (R_0) which has the important role in the modeling [14,15]. The basic reproduction number (R_0) can be computed using the next generation matrix on the alcoholism model 1-6. Using the approach in [16], the matrices F and V at disease free equilibrium (DFE) are given as follows:

$$F = \begin{pmatrix} \beta_1 \frac{\mu P}{b} & 0 \\ 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \mu + \beta_2 + \sigma_2 & -\sigma_1 \\ -\beta_2 & \sigma_1 + \alpha_1 + \alpha_2 + \mu + \delta \end{pmatrix}.$$

The basic reproduction number, denoted as R_0, is a crucial threshold parameter in modeling the spread of alcoholism, determining whether alcohol consumption can persist and grow within a population. It represents the expected number of new alcohol consumers influenced by a single excessive drinker in a fully susceptible population. In this study, R_0 is derived using the Next-Generation Matrix (NGM) method, a standard approach for compartmental models in behavioral dynamics. This method involves linearizing the system around the alcohol-free equilibrium and analyzing the initiation and transition dynamics of individuals in different drinking states. To apply the NGM method, we first identify the relevant compartments representing alcohol consumers and construct two key matrices: F, the initiation matrix representing the rate at which new individuals start consuming alcohol, and V, the transition matrix representing the rate at which individuals leave the consuming compartments due to recovery or cessation. These matrices are evaluated to capture the early-stage dynamics of alcohol spread. The next-generation matrix is then computed as FV^{-1}, where V^{-1} represents the inverse of the transition matrix. Finally, the basic reproduction number R_0 is obtained as the spectral radius (dominant eigenvalue) of the next-generation matrix, expressed as $R_0 = \rho\left(FV^{-1}\right)$, where $\rho(A)$ denotes the largest absolute eigenvalue of matrix A. The NGM method thus provides a rigorous mathematical framework for analyzing the spread of alcoholism and informing effective intervention strategies, which is given by

$$R_0 = \frac{\beta_1\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta\right)}{\left(\mu+\beta_2+\sigma_2\right)\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta\right)-\beta_2\sigma_1}. \tag{7}$$

Furthermore, the endemic equilibrium point for the mathematical model of an alcoholism with treatment centers is obtained when there are moderate and heavy drinkers in a population. Suppose the endemic equilibrium point for the mathematical model of the spread of alcoholism with treatment centers is expressed as E_1. Substituting $M \neq 0$ and $H \neq 0$ into Eqs. 1–6 will get $E_1 = (P^*, M^*, H^*, T^{r*}, T^{*p}, Q^*)$ where

$$\begin{aligned}
P^* &= \frac{b^2}{\beta_1\mu M^* + \mu b},\\
M^* &= \frac{\left(\mu b^2\right)\left(\left(\mu+\beta_2+\sigma_2\right)\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta\right)-\beta_2\sigma_1\right)\left(1-R_0\right)}{\sigma_1\beta_2 b\beta_1\mu-\left(\mu+\beta_2+\sigma_2\right)\left(b\beta_1\mu\right)\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta\right)},\\
H^* &= c_1 M^*,\\
T^{r*} &= c_2 c_1 M^*,\\
T^{p*} &= c_3 c_1 M^*,\\
Q^* &= \left(\frac{\sigma_2 M^* + \gamma_1 c_2 c_1 + \gamma_2 c_3 c_1}{-\mu}\right) M^*,
\end{aligned}$$

with

$$\begin{aligned}
c_1 &= \frac{\beta_2}{\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta\right)},\\
c_2 &= \frac{\alpha_1}{\left(\mu+\gamma_1\right)},\\
c_3 &= \frac{\alpha_2}{\left(\mu+\gamma_2\right)}.
\end{aligned}$$

Local stability analysis of the equilibrium point is crucial for understanding the system's behavior as it approaches equilibrium. In our study, we modeled the spread of alcoholism, considering treatment centers, using a system of non-linear differential equations. To analyse the stability of the equilibrium points, we linearized the system around these points using the Jacobian matrix. The local stability of an equilibrium point is determined by calculating the eigenvalues of the Jacobian matrix.

We specifically examined the stability of the non-endemic equilibrium point by substituting E_0 into the Jacobian matrix. The non-endemic equilibrium point is asymptotically stable if all the eigenvalues of the Jacobian matrix have negative real parts. The obtained eigenvalues are

$$\lambda_1 = \lambda_2 = -\mu, \quad \lambda_3 = -k_4, \quad \lambda_4 = -k_3$$

where $k_3 = \mu + \gamma_1$ and $k_4 = \mu + \gamma_2$. The remaining eigenvalues are derived from the roots of the characteristic equation:

$$\lambda^2 + a_1\lambda + a_2 = 0, \tag{8}$$

where $a_1 = k_2 - \beta_1 + k_1$ and $a_2 = -\beta_1 k_2 + k_1 k_2 - \sigma_1\beta_2$. For the equilibrium point E_0 to be asymptotically stable, all the roots of the characteristic equation (λ_i) must be negative. Since λ_1, λ_2 and λ_3 are already negative (given that μ, k_1, k_2, γ_1 and γ_2 are positive parameters). According to the Routh-Hurwitz stability criterion, the quadratic Eq. 8 will have negative roots if and only if $a_1, a_2 > 0$. We analyze a_1 and it will positive if

$$\frac{\beta_1^2\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta\right)}{\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta+\beta_2+\sigma_2+\mu\right)\left(\left(\mu+\beta_2+\sigma_2\right)\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta\right)-\beta_2\sigma_1\right)} < R_0, \tag{9}$$

where R_0 is already defined in Eq. 7. Defining:

$$R_1 = \frac{\beta_1^2\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta\right)}{\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta+\beta_2+\sigma_2+\mu\right)\left(\left(\mu+\beta_2+\sigma_2\right)\left(\sigma_1+\alpha_1+\alpha_2+\mu+\delta\right)-\beta_2\sigma_1\right)}. \tag{10}$$

Table 2
Parameter value in a mathematical model of alcoholism with treatment centers.

Parameter	Unit	Value	Source
b	person/time	65	[13]
β_1	1/time	0.56	Assumed
β_2	1/time	0.14	[13]
μ	1/time	0.065	[13]
δ	1/time	0.002	[13]
α_1	1/time	0.001	[13]
α_2	1/time	0.001	[13]
σ_1	1/time	0.09	Assumed
σ_2	1/time	0.08	Assumed
γ_1	1/time	0.001	[13]
γ_2	1/time	0.002	[13]

Table 3
Initial values of the mathematical model of alcoholism with treatment centers.

Variable	Initial value		
	1	2	3
$P(0)$	950	900	850
$M(0)$	700	650	580
$H(0)$	500	410	325
$T^r(0)$	390	285	195
$T^p(0)$	165	110	90
$Q(0)$	95	50	25

From 9 and 10, we obtain $R_1 < R_0$. In addition, we analyze a_2 and it will positive if $1 - R_0 > 1$. Thus the equilibrium poin E_0 is locally asymtotically stable if $R_0 < 1$.

The stability of the endemic equilibrium point of the mathematical model for alcoholism with treatment centers, was analyzed by substituting the endemic equilibrium point E_1 into the Jacobian matrix. We faced challenges due to our model yielding a six-degree polynomial, which makes a detailed analytical analysis of the endemic equilibrium extremely difficult. Due to the complexity of analytically determining the eigenvalues of the Jacobian matrix for this non-linear system, we employed numerical simulations to assess the stability of the endemic equilibrium points. These simulations involved the use of phase planes to visualize the system's trajectories and observe their behavior around the equilibrium point. By setting specific parameter values and initial conditions, we were able to simulate the dynamics of the alcoholism model. The parameter values and initial conditions used in these simulations are detailed in Tables 2 and 3. The initial conditions are chosen arbitrarily to demonstrate, through numerical simulations, that the system's behavior is independent of the initial values. Regardless of the starting point, all trajectories converge to a single equilibrium, illustrating the stability of the system. Although these values are selected for numerical purposes, they remain within a reasonable range that reflects common scenarios.

The simulation was carried out over a time span from $t = 0$ to $t = 50$. Three distinct initial values were used in the simulation to assess the convergence of solutions from each starting point. This approach aimed to determine whether the system consistently converges to the endemic equilibrium point regardless of initial conditions, thereby providing a robust validation of the model's stability. Each set of initial values represents different starting conditions for the compartments in the alcoholism model, allowing us to observe how the system evolves over time from various initial states. The behavior of the trajectories from these initial values helps to illustrate the stability and potential attractor properties of the endemic equilibrium point.

Fig. 2 illustrates the phase trajectory of alcoholism dynamics under different initial conditions, showing how the system evolves over time in the $(M, H)-$plane where M and H represent relevant population compartments associated with alcoholism. Despite varying initial conditions, all trajectories exhibit a similar trend, converging towards a single steady-state point $(M;\ H) = (200.47;\ 176.52)$ which point is the endemic equilibrium point $E_1 = (365.99;\ 200.47;\ 176.52;\ 2.89;\ 2.81;\ 247.46)$. Furthermore, based on the parameter values in Table 2, the value $R_0 = 2,72 > 1$ that conclude alcoholism is expected to persist rather than die out.

Parameter sensitivity analysis

The sensitivity analysis of parameters aims to identify which parameters significantly influence the stability conditions of both the non-endemic and endemic equilibrium points. The magnitude of the parameter's influence is determined based on the sensitivity index (e_m). The sensitivity index (e_m) for parameter m, according to Chitnis et al. [17], is formulated as follows:

$$e_m = \left(\frac{\partial R_0}{\partial m}\right)\frac{m}{R_0}. \tag{11}$$

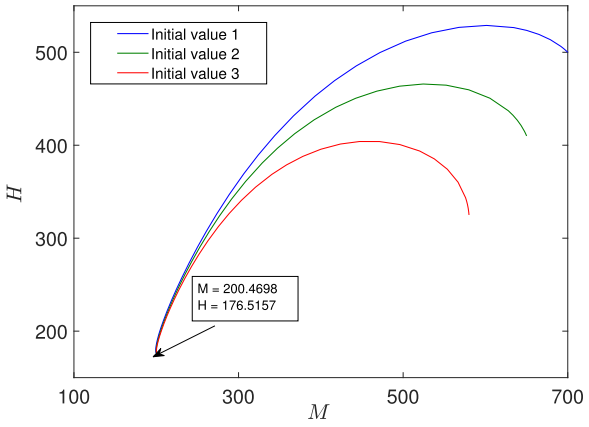

Fig. 2. Phase portrait of alcoholism dynamics with treatment centers.

Table 4
Index of parameter sensitivity.

Parameter (p)	Sensitivity Index	$R_0 = 2.722$			
		$p-10\%$	$p-15\%$	$p+10\%$	$p+15\%$
σ_1	0.167	2.537	2.649	2.766	2.786
σ_2	-0.389	2.832	2.89	2.619	2.572
α_1	-0.002	2.722	2.723	2.721	2.72
α_2	-0.002	2.722	2.723	2.721	2.72
μ	-0.473	2.859	2.935	2.6	2.544
δ	-0.005	2.723	2.724	2.72	2.72
β_1	1	2.449	2.313	2.994	3.13
β_2	-0.295	2.804	2.848	2.644	2.606

The stability of the non-endemic and endemic equilibrium points in the mathematical model of alcoholism with treatment center is determined by the value of R_0. There are eight parameters that affect the value of R_0, namely $\sigma_1, \sigma_2, \alpha_1, \alpha_2, \mu, \delta, \beta_1$ and β_2. The sensitivity indices of these parameters and the relationship between changes in these parameters and the resulting changes in R_0 are presented in the Table 4.

A sensitivity index that has a positive value indicates that an increase in the parameter's value leads to an increase in R_0. Conversely, a sensitivity index with a negative value implies that an increase in the parameter's value results in a decrease in R_0. Understanding these relationships is crucial for identifying which parameters most significantly impact the basic reproduction number and, consequently, the potential for disease spread. For example, the sensitivity index of $\beta_1 = 1$ means that if the value of β decreases (or increases) by 10 %, R_0 will decrease (or increase) by 10 % as well. On the other hand, a negative sensitivity index means that an increase in the parameter value causes R_0 to decrease. For instance, the sensitivity index of $\sigma_2 = -0.389$ indicates that if σ_2 decreases (or increases) by 10 %, R_0 will increase (or decrease) by 3.89 %. This analysis applies to parameters $\sigma_1, \alpha_1, \alpha_2, \mu, \delta$ and β_2.

The Fig. 3 illustrates the sensitivity of the basic reproduction number (R_0) to variations in β_1, which appears to represent the transmission or contact rate. The results show a clear linear relationship between β_1 and R_0, where R_0 consistently increases as β_1 becomes larger. This relationship holds for all values of σ_2, demonstrating that β_1 is a key driver of R_0. However, the increase in R_0 with β_1 depends on the value of σ_2. Smaller values of σ_2 result in steeper increases in (R_0), whereas larger values of σ_2 produce a more gradual rise. This indicates that while β_1 directly influences the reproduction number, its impact is moderated by σ_2, which adjusts the slope of the relationship. Overall, β_1 plays a dominant role in determining (R_0), with σ_2 acting as a modifying factor.

Focusing on the parameters β_1, it is crucial in demonstrating how the spread of alcoholism. Understanding the interplay between these parameters is essential for developing strategies that can reduce R_0, control alcoholism spread, and ultimately lead to the eradication of the social problem, particularly in alcoholism populations. The parameter β_1, representing the contact rate between individuals at risk of consuming alcohol and moderate alcohol drinkers, is directly affect alcoholism increase. As seen in the numerical simulations Fig. 4, an increase in β_1 leads to a higher heavy drinkers, indicating that contact accelerates the progression of alcoholism spreading.

In Fig. 4, the contact rate (β_1) represents the level of interaction between individuals at risk of consuming alcohol and moderate alcohol drinkers. The simulation results show that as β_1 increases (from 0.56 to 56), the number of heavy drinkers rises rapidly at the beginning and then stabilizes at a higher level over time. This indicates that an increase in the contact rate accelerates the spread of alcoholism by facilitating more interactions between at-risk individuals and moderate drinkers, pushing a larger proportion of the population into heavy drinking. These findings highlight the role of social interactions in promoting alcoholism and suggest that strategies to reduce such interactions could help control the progression of heavy drinking.

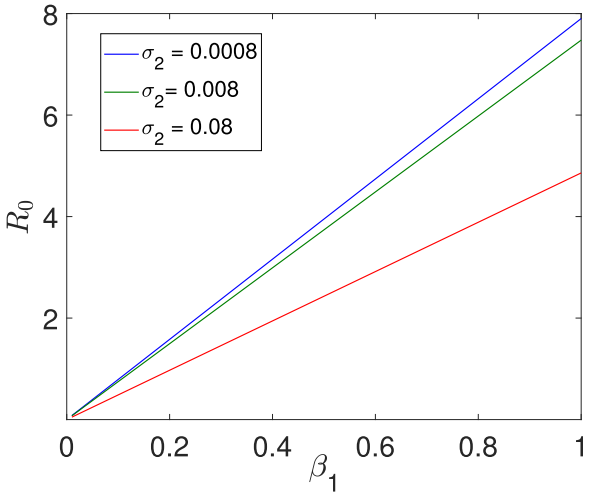

Fig. 3. Sensitivity analysis of parameters β_1 and σ_2.

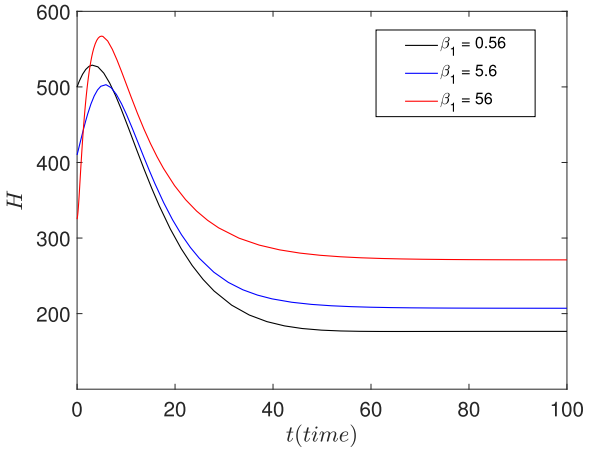

Fig. 4. Effect of contact rate between at-risk individuals and moderate drinkers on the increase in heavy alcohol drinkers.

Application of optimal control

The spread of alcoholism can be effectively controlled through various interventions. In this study, we employed two control measures aimed at reducing the alcoholism population by considering treatment centers. These controls include implementing educational efforts (u_1) to reduce close contact to moderate drinkers, and government policy (u_2) among heavy drinkers populations.

The mathematical model used to simulate the spread of alcoholism by considering treatment centers. By integrating these measures into the model, we can assess their effectiveness in mitigating alcoholism transmission and reducing the overall burden of the social disease within the alcoholism population. The inclusion of control variables allows us to explore the impact of targeted interventions on the dynamics and evaluate their potential for practical implementation in real-world settings.

The mathematical representation of the spread of alcoholism with case detection, accompanied by the control variables, is as follows:

$$\frac{dP}{dt} = b - \beta_1 \frac{PM}{b} - \mu P, \tag{12}$$

$$\frac{dM}{dt} = \beta_1 \frac{PM}{b} + \sigma_1 H - \left(\beta_2 + \sigma_2 + \mu\right) M - u_1 M, \tag{13}$$

$$\frac{dH}{dt} = \beta_2 M - \left(\sigma_1 + \alpha_1 + \alpha_2 + \mu + \delta\right) H - \omega u_2 H, \tag{14}$$

$$\frac{dT^r}{dt} = \alpha_1 H - \left(\mu + \gamma_1\right) T^r + \omega u_2 H, \tag{15}$$

$$\frac{dT^p}{dt} = \alpha_2 H - (\mu + \gamma_2)T^p + \omega u_2 H, \tag{16}$$

$$\frac{dQ}{dt} = \sigma_1 M + \gamma_1 T^r + \gamma_2 T^p - \mu Q + u_1 M. \tag{17}$$

Through numerical simulations and sensitivity analyses, we can evaluate the effectiveness of each control measure in achieving our goal of minimizing alcoholism population and improving public health outcomes among them. This comprehensive approach shows how the importance of different interventions in combating alcoholism and demonstrates the significance of tailored strategies for vulnerable populations. The performance index of the mathematical model of the spread of alcoholism with treatment centers by control variables is defined as follows:

$$MinJ(u_1, u_2) = \int_0^{t_f} A_1 M + A_2 H + \frac{1}{2}\left[B_1 u_1^2 + B_2 u_2^2\right]$$

where $0 \le u_1, u_2 \le 1$ and $A_1, A_2, B_1, B_2 > 0$. The coefficients B_1, B_2 and A_1, A_2 respectively are the weighting constants corresponding to each control and the weighting constants corresponding to the minimized population M and H. Optimal control time out is at an interval $t_0 \le t \le t_f$ that expresses the time of observation made, which is the time when the control is given to the end time of the control. The quadratic function of the control cost is adopted from [18] that represents education as a preventive at moderate drinkers and government policies at heavy drinkers.

Optimal control of the mathematical model of the spread of alcoholism with treatment centers is analyzed using the Pontryagin Maximum Principle (PMP), a powerful mathematical method for determining optimal strategies in dynamic systems. The PMP provides necessary conditions for optimality by transforming the original optimal control problem into a system of differential equations that must be satisfied by the optimal control and state variables. In this approach, we first define an objective functional that represents the desired outcome, typically minimizing the number of alcohol-dependent individuals while considering the costs associated with intervention measures such as treatment and awareness programs. To apply the PMP, we introduce control variables that represent intervention strategies, such as the rate of individuals receiving treatment or prevention efforts. The model is then formulated as a system of differential equations governing the dynamics of alcoholism spread under the influence of these control measures. The Hamiltonian function is then constructed by incorporating the state equations, control variables, and adjoint variables (also known as co-state variables or Lagrange multipliers), which measure the sensitivity of the objective functional to changes in the state variables. The necessary conditions for an optimal control strategy are derived by solving a system of coupled differential equations: the state equations, describing the evolution of the alcoholism model; the adjoint equations, which determine the evolution of the co-state variables; and the optimality condition, which ensures that the chosen control minimizes the Hamiltonian at every point in time. The Hamiltonian is obtained

$$\begin{aligned} H = {} & A_1 M + A_2 H + \frac{1}{2} B_1 u_1^2 + \frac{1}{2} B_2 u_2^2 + \rho_1\left[b - \beta_1 \frac{PM}{b} - \mu P\right] + \rho_2\left[\beta_1 \frac{PM}{b} + \sigma_1 H - (\beta_2 + \sigma_2 + \mu)M - \mu_1 M\right] \\ & + \rho_3\left[\beta_2 M - (\sigma_1 + \alpha_1 + \alpha_2 + \mu + \delta)H - \omega\mu_2 H\right] + \rho_4\left[\alpha_1 H - (\mu + \gamma_1)T^r + \omega u_2 H\right] \\ & + \rho_5\left[\alpha_2 H - (\mu + \gamma_2)T^p + \omega u_2 H\right] + \rho_6\left[\sigma_2 M + \gamma_1 T^r + \gamma_2 T^p - \mu Q + u_1 M\right]. \end{aligned}$$

Optimal conditions are obtained when the Hamiltonian function satisfies the following stationary conditions $\frac{\partial H}{\partial u_1} = 0$, $\frac{\partial H}{\partial u_2} = 0$. So that the optimal controllers u_1 and u_2 are obtained

$$u_1^* = \min\left(1, \max\left(0, \frac{M(\rho_2 - \rho_6)}{B_1}\right)\right),$$

$$u_2^* = \min\left(1, \max\left(0, \frac{\omega H(\rho_3 - \rho_4 - \rho_5)}{B_2}\right)\right).$$

The state variables in u_1^* and u_2^* are obtained by solving the state equation $\dot{x} = \frac{\partial H}{\partial \rho}$, while the Lagrange multiplier for the controls u_1^* and u_2^* is obtained by solving the Lagrange equation $\dot{x} = -\frac{\partial H}{\partial x}$. Then, the state variable and the Lagrange Multiplier are substituted into u^*. The optimal solution from the mathematical model is determined by substituting the control u^* into the state equation.

The controller forms of u_1^*, u_2^* and u_3^* depend on state and co-state variables. The state equations are as follows:

$$\begin{aligned} \frac{dP}{dt} &= \frac{\partial H}{\partial \rho_1} = b - \beta_1 \frac{PM}{b} - \mu P, \\ \frac{dM}{dt} &= \frac{\partial H}{\partial \rho_2} = \beta_1 \frac{PM}{b} + \sigma_1 H - (\beta_2 + \sigma_2 + \mu)M - u_1 M, \\ \frac{dH}{dt} &= \frac{\partial H}{\partial \rho_3} = \beta_2 M - (\sigma_1 + \alpha_1 + \alpha_2 + \mu + \delta)H - \omega u_2 H, \\ \frac{dT^r}{dt} &= \frac{\partial H}{\partial \rho_4} = \alpha_1 H - (\mu + \gamma_1)T^r + \omega u_2 H, \\ \frac{dT^p}{dt} &= \frac{\partial H}{\partial \rho_5} = \alpha_2 H - (\mu + \gamma_2)T^p + \omega u_2 H, \end{aligned}$$

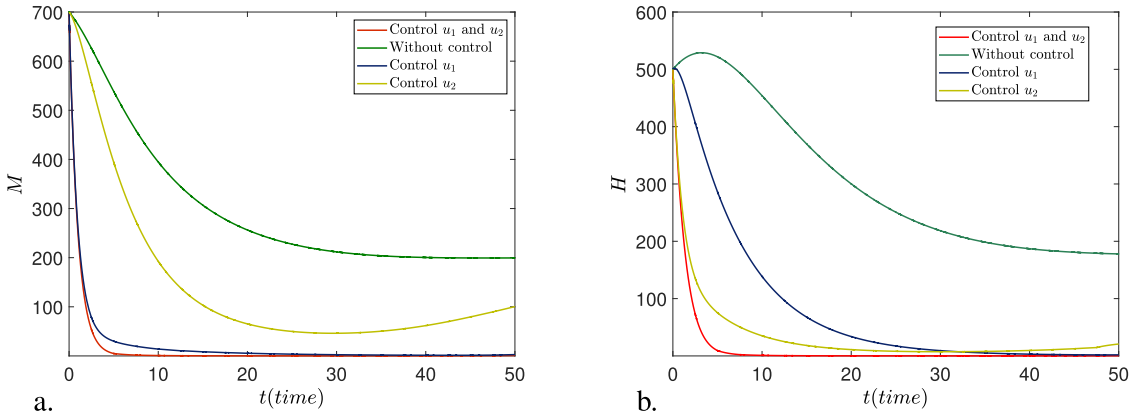

Fig. 5. Comparison of population sizes: (a) moderate and (b) heavy alcohol drinkers, with and without control measures. All parameter values are listed in Table 2.

$$\frac{dQ}{dt} = \frac{\partial H}{\partial \rho_6} = \sigma_1 M + \gamma_1 T^r + \gamma_2 T^p - \mu Q + u_1 M. \tag{18}$$

Meanwhile, the co-state equations are as follows :

$$\begin{aligned}
\dot{\rho_1} &= -\frac{\partial H}{\partial P} = \frac{\beta_1 \mu M}{b}(\rho_1 - \rho_2) + \rho_1 \mu,\\
\dot{\rho_2} &= -\frac{\partial H}{\partial M} = \frac{\beta_1 \mu M}{b}(\rho_1 - \rho_2) + \rho_2(\beta_1 + \sigma_2 + \mu) + \rho_2 u_1 - \rho_3 \beta_2 - \rho_6(\sigma_2 + u_1),\\
\dot{\rho_3} &= -\frac{\partial H}{\partial H} = -\rho_2 \sigma_1 + \rho_3(\sigma_1 + \alpha_1 + \alpha_2 + \mu + \delta - \omega u_2) - \rho_4(\alpha_1 + \omega u_2) - \rho_5(\alpha_2 + \omega u_2),\\
\dot{\rho_4} &= -\frac{\partial H}{\partial T^r} = \rho_4(\mu + \gamma_1) - \rho_6 \gamma_1,\\
\dot{\rho_5} &= -\frac{\partial H}{\partial T^p} = \rho_5(\mu + \gamma_2) - \rho_6 \gamma_2,\\
\dot{\rho_6} &= -\frac{\partial H}{\partial Q} = \rho_6 \mu.
\end{aligned} \tag{19}$$

Based on the description above, to get the values of P, M, H, T^r, T^p and Q from the optimal form u_1^* and u_2^* then it is necessary to solve the non-linear state and co-state equations. The non-linear equation system is hard to be solved analytically, so the solutions to the equation of state are analysed using numerical simulations.

Method validation

Numerical results

The state equation in the numerical simulation program for the mathematical model of the spread of alcoholism with treatment centers is defined as $P = y(1)$, $M = y(2)$, $H = y(3)$, $T^r = y(4)$, $T^p = y(5)$ and $Q = y(6)$. The performance index is defined as a new state equation that is $J = y(7)$ and controls u_1 and u_2 are defined as $u(1)$ dan $u(2)$. The simulation is carried out using initial values, namely $P(t_0) = 950$, $M(t_0) = 700$, $H(t_0) = 500$, $T^r(t_0) = 390$, $T^p(t_0) = 165$, and $Q(t_0) = 95$ with the start and end times respectively $t_0 = 0$ and $t_f = 50$ in years. Defined weighting constants to minimize the population (A) and the costs needed to apply each control respectively are $A_1 = 1$, $A_2 = 1$, $B_1 = 1$ and $B_2 = 1$. They have been standardized to 1 for the purpose of simplifying the calculations. This normalization allows for a more straightforward analysis by eliminating the variability introduced by differing cost magnitudes, thus focusing the analysis on the structural or relative relationships within the model.

The Fig. 5a illustrates the numerical results of moderate alcoholism under four scenarios: without control, using control u_1, using control u_2, and a combination of both controls u_1 and u_2. The first strategy, represented by the green curve, depicts the progression without any control measures. Here, M starts at a high value, around 700, and decreases slowly over time. This slow decline indicates that, without intervention, the effects of moderate alcoholism persist for a significantly longer period. It highlights the need for strategic actions to mitigate the issue. The blue curve represents the scenario where control u_1 is implemented. This control measure results in a more rapid decline in M compared to the uncontrolled case, demonstrating its effectiveness. u_1 represents public awareness campaigns as early intervention programs. Although it accelerates the reduction of alcoholism's impact, its effectiveness is limited when used alone.

The yellow curve corresponds to the implementation of control u_2, which produces a steeper decline in M compared to u_1, u_2 might reflect different measures, such as stricter policies or medical interventions. However, while u_2 performs better than u_1, its impact is still slower compared to the combined control strategy.

Table 5
Comparison of the number of alcoholism sufferers at the end of the observation M, H with and without control.

Condition	Total population (end of observation)		Total alcoholism	Cost value
	M	H		
No Control	200	178	378	-
Control u_1	3	2	5	5445.628
Control u_2	101	21	122	9233.415
Control u_1 and u_2	1	1	2	1545.539

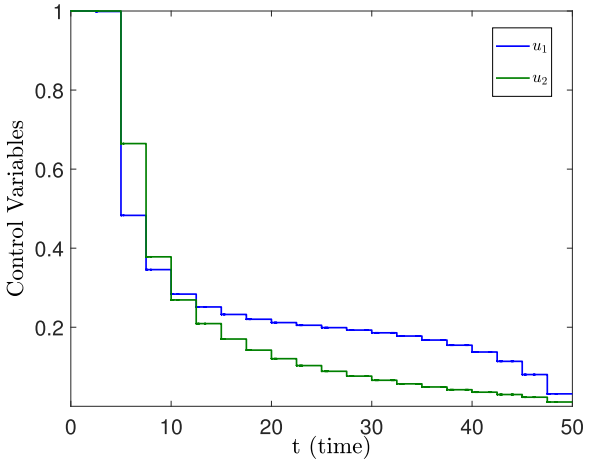

Fig. 6. Control profiles of u_1 and u_2.

The most effective scenario is represented by the red curve, where both controls u_1 and u_2 are applied together. This combination produces a synergistic effect, leading to a rapid and substantial reduction in M. By combining the strengths of both strategies, the impact of moderate alcoholism is minimized more quickly and efficiently. The graph highlights the importance of a multi-faceted approach to addressing moderate alcoholism. While individual controls (u_1 *or* u_2) are effective, their combination produces the most significant results, underscoring the need for comprehensive intervention strategies. Without any control measures, the problem persists much longer, causing prolonged negative consequences.

The Fig. 5b illustrates the numerical results of heavy drinking behavior under four different scenarios: no control, implementation of control u_1, implementation of control u_2, and the combined application of both controls (u_1 and u_2).

In the case without any control, depicted by the green curve, H starts at a high value of around 500 and decreases very slowly over time. This gradual decline indicates the persistence of heavy drinking behavior in the absence of interventions. It demonstrates that, without control measures, the problem resolves naturally but at a significantly slower rate, prolonging its adverse societal and individual impacts. The blue curve represents the scenario where control u_1 is implemented. This intervention results in a more rapid reduction in H compared to the uncontrolled case. Control u_1 might correspond to measures such as public education campaigns, early detection, or targeted support programs for heavy drinkers. While u_1 accelerates the decline, it is less effective than the combined control strategy in fully addressing the problem.

The yellow curve shows the results of applying control u_2. This strategy achieves a faster and more substantial reduction in H compared to control u_1. Control u_2 represent regulatory policies as legal enforcement measures targeting heavy drinking behavior. Despite its greater effectiveness, control u_2 alone still falls short of the impact achieved when combining u_1 and u_2. The red curve represents the most effective scenario, where both controls u_1 and u_2 are applied simultaneously. The combination of these strategies leads to a rapid and significant decline in H, reducing the number of heavy drinkers to nearly zero in the shortest possible time. This result underscores the synergistic effect of integrating multiple control measures, highlighting the importance of a comprehensive approach to combating heavy drinking behavior. The numerical results clearly show that the combined control strategy u_1 and u_2 is the most effective in mitigating heavy drinking. While individual controls can produce notable improvements, their combination yields the fastest and most significant impact. Without any intervention, the issue persists for a prolonged period, underscoring the necessity of proactive and coordinated strategies to address the challenges posed by heavy drinking.

To further illustrate the comparative effectiveness of each control strategy, Table 5 presents a comparison of the number of moderate and heavy alcohol drinkers at the end of the observation period for each strategy. This analysis provides valuable insights into the relative impact of different interventions on alcoholism transmission dynamics. Building upon this, Fig. 6 depicts the control profiles of u_1 (blue curve) and u_2 (green curve) over time (t), demonstrating the intensity of each control strategy during the observation

period from $t = 0$ to $t = 50$. Initially, both controls are implemented at their maximum values $(u_1 = u_2 = 1)$, ensuring full intervention at the outset. Over time, their intensities gradually decrease in a stepwise manner, reflecting a strategic reduction in control efforts while maintaining effectiveness. This figure further examines how the simulations application of u_1 and u_2 not only reduces alcoholism prevalence but also optimizes cost-effectiveness.

The figure highlights the coordinated application of both controls, where u_1 and u_2 are adjusted dynamically over time. This stepwise adjustment reflects the optimization of resource allocation, balancing effectiveness with cost considerations. By reducing the intensity of the controls as the situation improves, the strategies ensure both sustainability and efficiency in achieving the desired outcomes. These findings align with previous studies [19].

While the study provides valuable insights into the dynamics of alcohol consumption and the effectiveness of optimal control strategies, it is not without limitations. The model assumes constant parameters and does not account for time-dependent changes in drinking behavior or external socio-economic factors that could influence alcohol consumption trends [20]. Additionally, external influences such as peer pressure, cultural norms, and co-morbidities were not incorporated into the model [21].

Future research could build upon this work by exploring hierarchical relationships within the system and extending the analysis to other related systems [22]. Additionally, exploring the inclusion of socio-economic and psychological factors could provide deeper insights into how historical conditions shape current dynamics, creating a more comprehensive model that captures the interconnected evolution of the system over time [20]. Furthermore, extending the model to consider fractional-order dynamics could improve its ability to capture long-term behavioral effects and memory-dependent processes in alcohol consumption [23,24]. Additionally, the model could be further developed using partial differential equations (PDEs) to account for the spatial spread of alcoholism behavior across different regions or communities [25,26]. Such an approach would enable the analysis of how local interactions and geographic variations influence the dynamics of alcohol consumption, providing deeper insights into the mechanisms of behavioral spread. By refining these aspects, the model could offer a more comprehensive framework for guiding public health strategies aimed at reducing alcohol-related harm and promoting healthier communities.

Limitations

None

Ethics statements

The data used in this study is fully available within the manuscript and has been obtained from publicly available sources or previously published studies. All data sources have been appropriately cited in the References section. No additional ethical approval was required for this study, as no new data involving human or animal subjects were collected.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this article.

Data availability

No data was used for the research described in the article.

CRediT authorship contribution statement

Cicik Alfiniyah: Writing – review & editing, Writing – original draft, Validation, Supervision, Resources, Methodology, Funding acquisition, Conceptualization. **Tutik Utami:** Visualization, Software. **Miswanto:** Investigation, Formal analysis, Data curation, Conceptualization. **Nashrul Millah:** Validation, Formal analysis. **Reuben Iortyer Gweryina:** Writing – review & editing.

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