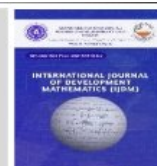




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Compartmental Model for Substance Abuse Dynamics: Threshold, Stability Analysis and Numerical Simulations

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Abstract

Substance abuse continues to pose a serious public health threat worldwide, fueling illness, premature death, crime, family breakdown, and heavy economic losses. To tackle this crisis effectively, we first need to understand how substance use takes root and persists across communities. With this in mind, we developed a deterministic compartmental model that tracks the movement of individuals through distinct stages from susceptible to initial use, dependence, treatment, recovery, and possible relapse. We ensured the model makes biological and mathematical sense by confirming that all solutions remain non-negative and bounded within a realistic region. We identified both drug-free and endemic steady states and computed the Threshold number, R_0 , using the standard next-generation matrix approach. Our threshold analysis shows that when $R_0 < 1$, the drug-free equilibrium is locally stable,

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meaning the problem can die out over time. But once $R_0 > 1$, this stability breaks down, and the condition can persist. We also explored conditions under which the endemic equilibrium remains stable. To pinpoint which factors matter most, we carried out a sensitivity analysis, revealing the parameters that drive the spread and endurance of substance abuse. Finally, we ran numerical simulations to back up our theoretical results and to visualize how changing key parameters alters the course of the epidemic. Taken together, our findings suggest that curbing the rate at which people start using substances, reducing peer pressure, expanding access to treatment, improving recovery success, and preventing relapse could significantly lighten the burden of substance abuse. The framework we offer here is not only a tool for deeper scientific inquiry but also a practical guide for policymakers and health professionals designing long-term prevention and response strategies.

1 Introduction

The American Psychological Association [APA \(2015\)](#) also recognizes substance abuse as a key risk factor for behavioral instability, impaired judgment, and psychiatric co-morbidities. These impacts are particularly noticeable in young people, who are more vulnerable to social pressures, peer pressure, unemployment, and emotional discomfort. Generally speaking, substance addiction is a chronic and relapsing disorder characterized by compulsive drug-seeking behavior and continued substance use despite experiencing adverse consequences [Volkow et al. \(2016\)](#). At the individual level, substance abuse can result in a multitude of serious outcomes, including mental health disorders such as anxiety, depression, and psychosis; severe physical complications like liver failure, cardiovascular disease, and neurological impairment; and an overall deterioration in quality of life. It is essential to distinguish between substance use, substance abuse, and substance addiction, as these terms are often misunderstood or used interchangeably. Substance use refers to the controlled and medically supervised consumption of drugs, particularly prescription medications, for therapeutic or preventive health purposes. However, when usage patterns deviate from medical guidelines such as taking higher doses, using substances for non-medical reasons, or engaging in prolonged unsupervised use it can progress into substance abuse. If this pattern becomes chronic and is accompanied by an inability to stop despite harmful effects, it evolves into substance addiction.

Addiction is further marked by both psychological dependence, which manifests as cravings and compulsive behaviors, and physical dependence, evidenced by withdrawal symptoms when the substance is reduced or discontinued (United Nations Office on Drug and Crime [UNODC \(2021\)](#)). These include illicit drugs such as cannabis, cocaine, heroin, and methamphetamine; prescription medications like opioids (e.g., oxycodone, tramadol), benzodiazepines (e.g., diazepam), and stimulants (e.g., amphetamines); as well as legal substances including alcohol, tobacco, and inhalants. The prevalence and patterns of abuse vary by region, socio-economic status, and age group, but collectively these substances contribute significantly to the global burden of disease, addiction, and mortality. Understanding the diversity of substances involved in community-level abuse

is essential for developing targeted prevention and intervention strategies.

Substance abuse is deeply intertwined with a host of societal challenges, exerting a destabilizing influence on social and economic structures. It is strongly linked to increased rates of criminal activity, domestic violence, unemployment, family disintegration, and declining productivity at the community and national levels [Parry et al. \(2004\)](#); [Biswas \(2012\)](#). These ripple effects extend far beyond the individual, placing a significant burden on public health systems, overstraining law enforcement resources, and undermining national development agendas particularly in resource-limited countries. In developing nations such as Bangladesh, Nigeria, and parts of South Africa, the substance abuse crisis is intensified by underlying structural and socio-political vulnerabilities. These include pervasive poverty, weak institutional governance, inadequate healthcare facility, and porous borders that facilitate the flow of illicit drugs [UNODC \(2019\)](#). Although these countries are not among the major producers of narcotics, their strategic geographic positioning renders them vital transit corridors for international drug trafficking networks. This transit role inadvertently promotes local availability and consumption of substances, particularly among youth populations, who are often the most socioeconomically vulnerable [De Andres \(2008\)](#).

The alarming increase in youth addiction is attributable to several interrelated factors: economic instability, high rates of unemployment, exposure to violence or trauma, weak family support systems, and limited access to preventive education and rehabilitative services [Mushayabasa & Tapedzesa \(2015\)](#). The lack of structured interventions and social safety nets leaves young people especially susceptible to the lure of drugs as a coping mechanism or means of social inclusion. Beyond its socio-economic dimensions, substance abuse is also compounded by its complex biological and psychological foundations. Scientific evidence underscores that addiction is not merely a matter of personal choice or moral failing, but rather a multi-factorial condition influenced by a combination of genetic predispositions, neuro-chemical imbalances, environmental stressors, and behavioral ills [Kumar & Sahu \(2016\)](#); [Volkow et al. \(2016\)](#). This intricate interplay of factors underscores the challenge of addressing substance abuse through a single, isolated approach. Instead, it demands a comprehensive, interdisciplinary response that integrates public health, mental health, law enforcement, education, and community engagement strategies to create sustainable and context-sensitive solutions.

In this light, mathematical modelling has emerged as a powerful tool for studying the complex dynamics of substance abuse, offering insights into how addiction spreads, how individuals transition between different states (e.g., non-user, occasional user, addict, relapsed), and how interventions might influence these transitions [White & Comiskey \(2007\)](#). These models can simulate real-world phenomena, enabling researchers to estimate the long-term effects of prevention campaigns, treatment availability, relapse rates, and enforcement efforts. For example, compartmental models using systems of ordinary differential equations have been employed to analyze heroin addiction dynamics, highlighting the pivotal role of relapse in sustaining the cycle of addiction [White & Comiskey \(2007\)](#). Similarly, [Ahmed et al. \(2002\)](#) used computational models to study the neurobiological mechanisms underlying substance-seeking behavior, while [Rossi2 \(2002\)](#) demonstrated how substance addiction contributes to the global rise of infectious

diseases through modeling techniques.

Mathematical modeling has become an essential tool in understanding the dynamics of substance abuse and in evaluating the potential effectiveness of various intervention strategies. These models allow for the systematic incorporation of real-world control measures such as public education campaigns, detoxification and rehabilitation programs, law enforcement actions, and harm reduction initiatives by representing them as control parameters within the model structure. These parameters influence key outcomes, including the rate of new addiction cases, relapse rates, and successful recovery rates [Nyabadza et al. \(2013\)](#). By applying sensitivity analysis and numerical simulations, researchers can assess the relative impact of different interventions, determine which combinations are most effective, and optimize their timing, duration, and intensity for maximum impact. Despite the insights gained from these scientific approaches, substance abuse continues to pose a persistent and evolving public health challenge. Global initiatives such as those led by the United Nations Office on Drugs and Crime (UNODC) aimed at curbing the production, trafficking, and consumption of illicit substances have encountered limited success, especially in regions plagued by entrenched socio-economic vulnerabilities and adaptive drug trafficking networks [UNODC \(2019\)](#). In areas like West Africa, Latin America, and Southern Africa, the crisis is further compounded by the deepening nexus between substance abuse and organized crime. For example, in South Africa, substance abuse has been strongly correlated with high levels of criminal activity, including theft, assault, and domestic violence. The ready availability of illicit drugs, combined with fragile social support systems, creates a self-reinforcing cycle where addiction fuels criminal behavior, which in turn exacerbates social disintegration and economic instability [Nyabadza et al. \(2013\)](#). Addressing this cycle requires not only robust public health interventions but also inter-sectoral cooperation involving law enforcement, education, community development, and healthcare systems.

In light of these difficulties, the aim of this research is to improve the comprehension of substance abuse by creating a mathematical model that incorporates the dynamics of substance abuse and takes into account different management techniques. By simulating these scenarios, the model will provide evidence-based insights to inform public health policy, resource allocation, and targeted intervention strategies. This study places itself at the nexus of applied mathematics, public health, and behavioral science. It seeks to comprehend the mechanisms that lead to substance abuse as well as assess how effective interventions can lessen its effects through the use of strong mathematical approaches.

2 Formulation of Substance Abuse Model

A population size $N(t)$ is divided into twelve subgroups and an independent compartment making a total of thirteen compartments: susceptible compartments; $S_u(t)$ (unenlightened individuals who are not substance users, but at a high risk of taking substances as they interact with $I_1(t)$ or $I_2(t)$) and $S_e(t)$ (enlightened individuals who are not substance users, but at a high risk of taking substances as they interact with $I_1(t)$ or $I_2(t)$). Exposed individuals $E(t)$ (those whose company and surroundings are harmful and might have begun using substances but have not proclaimed or showed symptoms to be classified as light or heavy substance users). Light substance abusers $I_1(t)$ and

heavy substance abusers $I_2(t)$ (substance users who have demonstrated symptoms and are capable of convincing another vulnerable individuals to indulge in substance abuse). Heavy substance users under detention $D(t)$ (Heavy substance users under detention by law enforcement agencies). Persons with substance-induced psychiatric disorders $M(t)$ (Persons that have psychiatric disorders due to substance abuse). Substance induced impoverished persons $Z(t)$ (Substance users who have become poor and financially reckless due to substance abuse). Persons with substance-induced physiological ill conditions $P(t)$ (Heavy substance users who have physiological health problems due to substance abuse). Substance users under rehabilitation $Q(t)$ (Substance users who have began treatment in rehabilitation class, referred to as patients). Temporary recovered substance abusers $R_T(t)$ (Substance users who are temporary recovered where they are assumed to be temporarily invulnerable to substance use before becoming exposed again). Permanent recovered substance abusers $R_P(t)$ (substance users that have permanently recovered from substance abuse and have moved out of the substance abuse dynamics) and Law Enforcement Agencies $L(t)$. Close relationships or frequent interactions $(\psi_1, \psi_2, \psi_3, \psi_4)$ between vulnerable humans and substance users correspondingly enhance the odds of becoming a substance abuser and being in a high risk subgroup $E(t)$ at a rate λ_1 or λ_2 . A proportion of $(1 - \theta)$ of enlightened susceptible individuals is transmitted to $E(t)$ individuals class. Extensive exposure to sensitive substance abuse, leads to light substance users $I_1(t)$ or $I_2(t)$ heavy substance user at a rate α_1 and a proportion of $(1 - \rho)$ or ρ respectively.

Light substance users may develop hook to substances and become a heavy substance user over time at a rate σ_1 . Also, light substance users in a proportion $\psi_3 L$ due to interaction with law enforcement agencies may decide to quit abusing substances and move to Temporary recovered compartment, $R_T(t)$. Otherwise, depending on the character of the family and the environment, the light substance users progress to rehabilitation class at the rate σ_2 . Heavy substance users due to addiction may suffer a variety of effects thereby, progress to substance induced psychiatric disorder class, substance induced impoverished class and substance induced physiological ill conditions compartment at the rates π_1 , π_2 and π_3 respectively. A proportion of heavy substance users as a result of interaction with the law enforcement agencies may also be progressed to detention compartment at a rate $\psi_6 L$. The substance addicts under detention move to rehabilitation class at the rate r_4 or die due to substance abuse at the rate μ_6 . Due to family intervention, $M(t)$, $Z(t)$ and $P(t)$, might be moved to rehabilitation class at the rates; r_1 , r_2 and r_3 . Otherwise, if not rehabilitated, they die as a result of substantial substance addiction at the rates; μ_3 , μ_4 and μ_5 respectively. Successful treated substance abusers under rehabilitation recover and move to temporary recovered class at a rate α_2 and others progress to permanent recovered class at the rate α_3 , otherwise die due to substance abuse at the rate μ_6 . The temporary recovered substance abusers are temporarily invulnerable to the substance use environment, thereby return to exposed compartment at a rate α_4 . Humans are recruited at a steady pace Λ_1 and Λ_2 , and die naturally at a rate μ_1 . The Law Enforcement Agencies, in the course of their interactions with the heavy substance users die at the rate μ_8 .

Table 1. Substance abuse model variables description.

Variable	Description
$S_u(t)$	Unenlightened susceptible at time t
$S_e(t)$	Enlightened susceptible at time t
$E(t)$	Exposed at time t
$I_1(t)$	Light substance users at time t
$I_2(t)$	Heavy substance users at time t
$M(t)$	Persons with substance-induced psychiatric disorders at time t
$Z(t)$	Substance induced impoverished persons at time t
$P(t)$	Persons with substance-induced physiological ill conditions at time t
$D(t)$	Heavy substance users under detention at time t
$Q(t)$	Substance users under rehabilitation at time t
$R_T(t)$	Temporary recovered substance abusers at time t
$R_P(t)$	Permanent recovered substance abusers at time t
$L(t)$	Law enforcement agencies at time t

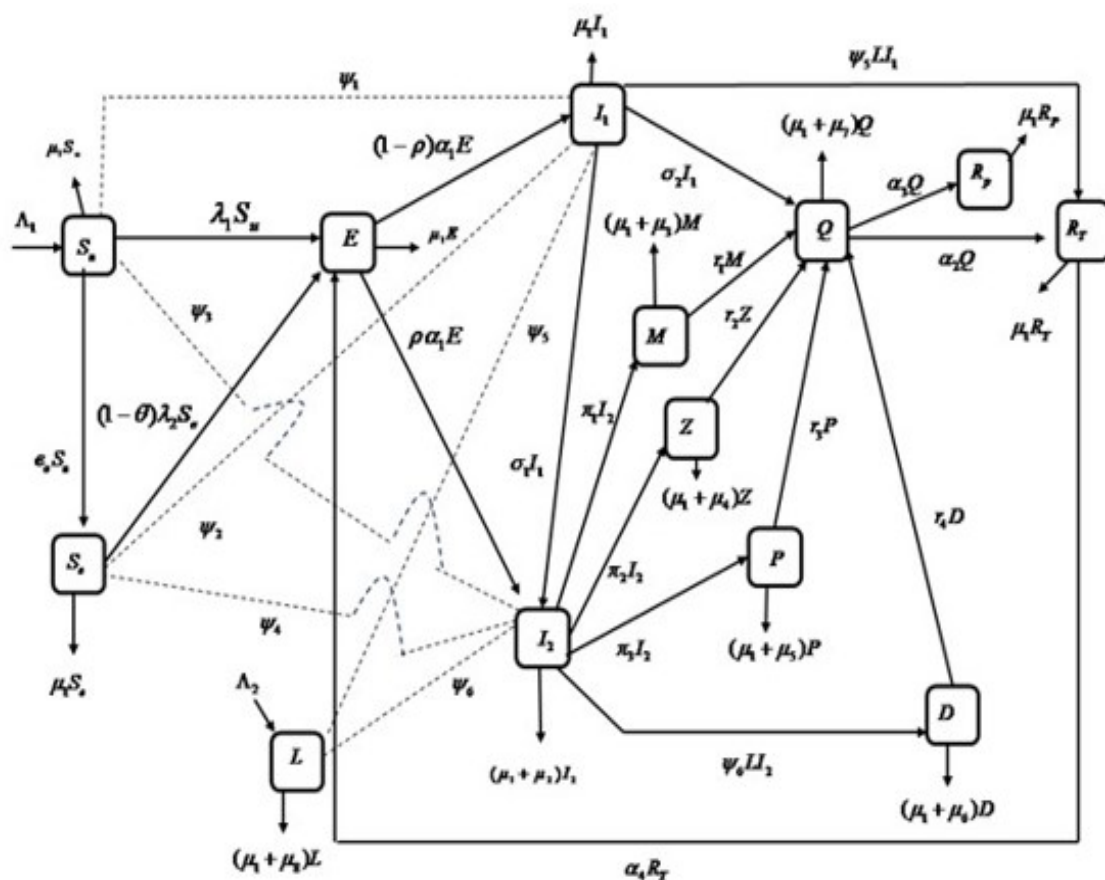
**Figure 1.** Schematic Substance Abuse Model Diagram.

Table 2: Substance Abuse Model Parameters

Parameters	Description
Λ_1	The recruitment rate of unenlightened susceptible
Λ_2	The recruitment rate of law enforcement agencies
ψ_1	The rate of interaction between unenlightened susceptible and light substance users
ψ_2	The rate of interaction between enlightened susceptible and light substance users
ψ_3	The rate of interaction between unenlightened susceptible and heavy substance users
ψ_4	The rate of interaction between enlightened susceptible and heavy substance users
ψ_5	The progression rate of light substance users to temporary recovered
ψ_6	The progression rate of heavy substance users to detention class
α_1	Progression rate of persons out of the exposed compartment
α_2	The progression rate of persons under rehabilitation to temporary
α_3	The progression rate of persons under rehabilitation to permanent recovered class
ρ	Proportion of exposed progressing to heavy substance users class
σ_1	Rate at which light substance users progress to heavy substance users class
π_1	Progression rate of heavy substance users to substance-induced psychiatric disorders class
π_2	Progression rate of heavy substance users to substance induced impoverished class
σ_2	Progression rate of light substance users to rehabilitation class
e_0	Enlightenment rate that result to enlightened susceptible class
π_3	Progression rate of heavy substance users to substance induced physiological ill conditions
μ_1	Natural death rate of beings
μ_2	Substance induced death rate of Heavy substance users
μ_3	Substance induced death rate of psychiatric disorder persons individual
μ_4	Substance induced death rate of impoverished persons
μ_5	Substance induced death rate of persons with physiological conditions
μ_6	Substance induced death rate of persons under detention
μ_7	Substance induced death rate of persons under rehabilitation
μ_8	Death rate of law enforcement agencies due to interaction with Heavy substance users
α_4	Rate at which temporary recovered persons return to exposed
θ	The measure of effectiveness of enlightenment on enlightened susceptible humans
λ_1	Progression rate of Unenlightened susceptible to Exposed class
λ_2	Progression rate of enlightened Susceptible to Exposed class
r_1	Progression rate to rehabilitation class
r_2	Progression rate to rehabilitation class
r_3	Progression rate of to Rehabilitation class
r_4	Progression rate to rehabilitation class
N	Total population

The substance abuse model equations are:

$$\left\{ \begin{array}{l} \frac{dS_u}{dt} = \Lambda_1 - (e_0 + \lambda_1 + \mu_1)S_u, \\ \frac{dS_e}{dt} = e_0S_u - ((1 - \theta)\lambda_2 + \mu_1)S_e, \\ \frac{dE}{dt} = \lambda_1S_u + (1 - \theta)\lambda_2S_e + \alpha_4R_T - (\alpha_1 + \mu_1)E, \\ \frac{dI_1}{dt} = (1 - \rho)\alpha_1E - (\sigma_1 + \sigma_2 + \psi_5L + \mu_1)I_1, \\ \frac{dI_2}{dt} = \rho\alpha_1E + \sigma_1I_1 - (\pi_1 + \pi_2 + \pi_3 + \psi_6L + \mu_1 + \mu_2)I_2, \\ \frac{dM}{dt} = \pi_1I_2 - (r_1 + \mu_1 + \mu_3)M, \\ \frac{dZ}{dt} = \pi_2I_2 - (r_2 + \mu_1 + \mu_4)Z, \\ \frac{dP}{dt} = \pi_3I_2 - (r_3 + \mu_1 + \mu_5)P, \\ \frac{dD}{dt} = \psi_6LI_2 - (r_4 + \mu_1 + \mu_6)D, \\ \frac{dQ}{dt} = \sigma_2I_1 + r_1M + r_2Z + r_3P + r_4D - (\alpha_2 + \alpha_3 + \mu_1 + \mu_7)Q, \\ \frac{dR_P}{dt} = \alpha_3Q - \mu_1R_P, \\ \frac{dR_T}{dt} = \alpha_2Q + \psi_5LI_1 - (\alpha_4 + \mu_1)R_T, \\ \frac{dL}{dt} = \Lambda_2 - (\mu_1 + \mu_8)L. \end{array} \right. \quad (1)$$

with the initial conditions

$$\left\{ \begin{array}{l} S_u(0) > 0, S_e(0) > 0, E(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, M(0) \geq 0, Z(0) \geq 0, \\ P(0) \geq 0, D(0) \geq 0, Q(0) \geq 0, R_P(0) \geq 0, R_T(0) \geq 0, L(0) > 0. \end{array} \right. \quad (2)$$

2.1 Basic Properties of Substance Abuse

Theorem 2.1. *The solution set of model equations (1) with non-negative initial conditions remains non-negative for all $t \geq 0$.*

Given that the initial conditions are non-negative, we rewrite equations (1) in the form:

$$\frac{dS_u}{dt} \geq -(e_0 + \lambda_1 + \mu_1)S_u \quad (3)$$

$$\frac{dS_e}{dt} \geq -(\beta\lambda_2 + \mu_1)S_e \quad (4)$$

$$\frac{dE}{dt} \geq -(\alpha_1 + \mu_1)E \quad (5)$$

$$\frac{dI_1}{dt} \geq -(\sigma_1 + \sigma_2 + \psi_5L + \mu_1)I_1 \quad (6)$$

$$\frac{dI_2}{dt} \geq -(\pi_1 + \pi_2 + \pi_3 + \psi_6L + \mu_1 + \mu_2)I_2 \quad (7)$$

$$\frac{dM}{dt} \geq -(r_1 + \mu_1 + \mu_3)M \quad (8)$$

$$\frac{dZ}{dt} \geq -(r_2 + \mu_1 + \mu_4)Z \quad (9)$$

$$\frac{dP}{dt} \geq -(r_3 + \mu_1 + \mu_5)P \quad (10)$$

$$\frac{dD}{dt} \geq -(r_4 + \mu_1 + \mu_6)D \quad (11)$$

$$\frac{dQ}{dt} \geq -(\alpha_2 + \alpha_3 + \mu_1 + \mu_7)Q \quad (12)$$

$$\frac{dR_P}{dt} \geq -\mu_1 R_P \quad (13)$$

$$\frac{dR_T}{dt} \geq -(\alpha_4 + \mu_1)R_T \quad (14)$$

$$\frac{dL}{dt} \geq -(\mu_1 + \mu_8)L \quad (15)$$

Solving the system using the method of separation of variables:

$$S_u(t) \geq S_u(0) e^{-(e_0 + \lambda_1 + \mu_1)t}, \quad \forall t > 0 \quad (16)$$

$$S_e(t) \geq S_e(0) e^{-(\beta \lambda_2 + \mu_1)t}, \quad \forall t > 0 \quad (17)$$

$$E(t) \geq E(0) e^{-(\alpha_1 + \mu_1)t}, \quad \forall t > 0 \quad (18)$$

$$I_1(t) \geq I_1(0) e^{-(\sigma_1 + \sigma_2 + \psi_5 L + \mu_1)t}, \quad \forall t > 0 \quad (19)$$

$$I_2(t) \geq I_2(0) e^{-(\pi_1 + \pi_2 + \pi_3 + \psi_6 L + \mu_1 + \mu_2)t}, \quad \forall t > 0 \quad (20)$$

$$M(t) \geq M(0) e^{-(r_1 + \mu_1 + \mu_3)t}, \quad \forall t > 0 \quad (21)$$

$$Z(t) \geq Z(0) e^{-(r_2 + \mu_1 + \mu_4)t}, \quad \forall t > 0 \quad (22)$$

$$P(t) \geq P(0) e^{-(r_3 + \mu_1 + \mu_5)t}, \quad \forall t > 0 \quad (23)$$

$$D(t) \geq D(0)e^{-(r_4+\mu_1+\mu_6)t}, \quad \forall t > 0 \quad (24)$$

$$Q(t) \geq Q(0)e^{-(\alpha_2+\alpha_3+\mu_1+\mu_7)t}, \quad \forall t > 0 \quad (25)$$

$$R_P(t) \geq R_P(0)e^{-\mu_1 t}, \quad \forall t > 0 \quad (26)$$

$$R_T(t) \geq R_T(0)e^{-(\alpha_4+\mu_1)t}, \quad \forall t > 0 \quad (27)$$

$$L(t) \geq L(0)e^{-(\mu_1+\mu_8)t}, \quad \forall t > 0 \quad (28)$$

Hence, the solutions remain non-negative for all $t \geq 0$.

Theorem 2.2. *The solution set of the model equations (1) is bounded in a positively invariant region Ω .*

The region:

$$\Omega = \left\{ (S_u(t), S_e(t), E(t), I_1(t), I_2(t), M(t), Z(t), P(t), D(t), Q(t), R_T(t), R_p, L(t)) \in \mathbb{R}_+^{13} \right\} :$$

$$0 < N_1 \leq \frac{\Lambda_1}{\mu_1}, 0 < N_2 \leq \frac{\Lambda_2}{\mu_1 + \mu_8} \quad (29)$$

(29) is positively invariant. in the absence of substance abuse induced death

$$\mu_2 = \mu_3 = \mu_4 = \mu_5 = \mu_6 = \mu_7 = 0$$

$$\frac{dN_1}{dt} = \Lambda_1 - \mu_1 N_1 \quad (30)$$

$$N_1 \leq \frac{\Lambda_1}{\mu_1} \quad (31)$$

Also,

$$\frac{dN_2}{dt} = \Lambda_2 - (\mu_1 - \mu_8)N_2 \quad (32)$$

$$N_2 \leq \frac{\Lambda_2}{\mu_1 + \mu_8} \quad (33)$$

The biological invariant region for the model (1) is given by

$$\Omega = \{(S_u(t), S_e(t), E(t), I_1(t), I_2(t), M(t), Z(t), P(t), D(t), Q(t), R_T(t), R_p, L(t)) \in \mathbb{R}_+^{13} \} :$$

$$0 < N_1 \leq \frac{\Lambda_1}{\mu_1}, 0 < N_2 \leq \frac{\Lambda_2}{\mu_1 + \mu_8} \}. \quad (34)$$

3 Substance Abuse Model Analysis

3.1 Substance Abuse Free Equilibrium State

$$\frac{ds_u}{dt} = \frac{ds_e}{dt} = \frac{dE}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dM}{dt} = \frac{dZ}{dt} = \frac{dP}{dt} = \frac{dD}{dt} = \frac{dQ}{dt} = \frac{dR_P}{dt} = \frac{dR_T}{dt} = \frac{dL}{dt} = 0 \quad (35)$$

At the Substance Abuse Free Equilibrium (SAFE), we set all infected compartments to zero:

That is: $E = I_1 = I_2 = M = Z = P = D = Q = R_P = R_T = 0$
solving the system at SAFE we obtained:

$$\therefore E^o = \left[\frac{\Lambda_1}{e_o + \mu_1}, \frac{e_o \Lambda_1}{\mu_1(e_o + \mu_1)}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_2}{\mu_1 + \mu_8} \right] \quad (36)$$

In the absence of substance abuse, system (36) admits a disease-free equilibrium (DFE), which characterizes the steady-state of the model in which no substance is being abused.

3.2 Substance Abuse Endemic Equilibrium State

$$\frac{ds_u}{dt} = \frac{ds_e}{dt} = \frac{dE}{dt} = \frac{dI_1}{dt} = \frac{dI_2}{dt} = \frac{dM}{dt} = \frac{dZ}{dt} = \frac{dP}{dt} = \frac{dD}{dt} = \frac{dQ}{dt} = \frac{dR_P}{dt} = \frac{dR_T}{dt} = \frac{dL}{dt} \neq 0 \quad (37)$$

At the Substance Abuse Endemic Equilibrium (S.A.E.E), we set all infected compartments $\neq 0$:

That is: $E \neq 0, I_1 \neq 0, I_2 \neq 0, M \neq 0, Z \neq 0, P \neq 0, D \neq 0, Q \neq 0, R_P \neq 0, R_T \neq 0$
solving the system (1) at S.A.E.E we obtained:

$$\left\{ \begin{array}{l} S_u^* = \frac{\Lambda_1}{e_o + \lambda_1 + \mu_1}, \\ S_e^* = \frac{e_o \Lambda_1}{(\beta_1 \lambda_2 + \mu_1)(e_o + \lambda_1 + \mu_1)}, \\ E^* = \tau_{10}, \\ I_1^* = \tau_2 \tau_{10}, \\ I_2^* = \tau_3 \tau_{10}, \\ M^* = \tau_4 \tau_{10}, \\ Z^* = \tau_3 \tau_5 \tau_{10}, \\ P^* = \tau_3 \tau_6 \tau_{10}, \\ D^* = \tau_1 \tau_3 \tau_{10}, \\ Q^* = \tau_7 \tau_{10}, \\ R_P^* = \tau_7 \tau_8 \tau_{10}, \\ R_T^* = \tau_9 \tau_{10}, \\ L^* = \frac{\Lambda_2}{\mu_1 + \mu_8}, \end{array} \right. \quad (38)$$

Therefore, (38) define the Substance Abuse Endemic Equilibrium state, representing the steady state at which substance are being abused in the society.

where,

$$\begin{aligned} \tau_{10} &= \frac{(\beta \lambda_2 + \mu_1) \lambda_1 \Lambda_1 + \beta \lambda_2 e_o \Lambda_1}{(\beta \lambda_2 + \mu_1)(e_o + \lambda_1 + \mu_1)[(\alpha_1 + \mu_1) - \alpha_4 \tau_9]}, \tau_9 = \frac{\psi_5 \Lambda_2 \tau_2 + (\mu_1 + \mu_8) \alpha_2 \tau_7}{(\mu_1 + \mu_8)(\alpha_4 + \mu_1)}, \tau_8 = \frac{\alpha_3}{\mu_3}, \tau_7 = \frac{(\sigma_2 \tau_2 + r_1 \tau_4 + (r_4 \tau_1 + r_2 \tau_5 + r_3 \tau_6) \tau_3)}{\alpha_2 + \alpha_3 + \mu_1 + \mu_7} \\ \tau_6 &= \frac{\pi_3}{r_3 + \mu_1 + \mu_5}, \tau_5 = \frac{\pi_2 \tau_3}{r_2 + \mu_1 + \mu_4}, \tau_4 = \frac{\pi_1 \tau_3}{r_1 + \mu_1 + \mu_8}, \tau_3 = \frac{(\mu_1 + \mu_8)(\rho \alpha_1 + \sigma_1 \tau_2)}{(\mu_1 + \mu_8)[(\pi_1 + \pi_2 + \pi_3) + (\mu_1 + \mu_2)] + \psi_6 \Lambda_2}, \\ \tau_2 &= \frac{(1 - \rho)(\mu_1 + \mu_8) \alpha_1}{(\sigma_1 + \sigma_2)(\mu_1 + \mu_8) + \psi_5 \Lambda_2 + \mu(\mu_1 + \mu_8)}, \tau_1 = \frac{\psi_6 \Lambda_2}{(\mu_1 + \mu_8)(r_4 + \mu_1 + \mu_6)} \end{aligned}$$

3.3 Threshold Number

We should note that, basic reproduction number is just the exact number of the secondary infection cases due to the introduction of a single infections agent into a population that is entirely susceptible. A generalized approach known as the next generation matrix technique was used. According to [Diekmann et al. \(2010\)](#). The threshold R_o is represented by $R_o = \rho(FV^{-1})$, ρ = the spectral radius. Given the infections classes as E, I_1, I_2, M, Z, P in (1) a vector F can be formed to represent the new infections. The components of the vector F can be obtained by considering the terms denoting new infections from the susceptible entering the exposed compartment.

$$F = \begin{bmatrix} E \\ I_1 \\ I_2 \\ M \\ Z \\ P \end{bmatrix} = \begin{bmatrix} \frac{\beta_1(\psi_1 I_1 + \psi_3 I_2) S_u}{N_1} + \frac{\beta_2 \beta (\psi_2 I_1 + \psi_4 I_2) S_e}{N_1} \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad (39)$$

Note: $\beta = (1 - \theta)$

$$F = \begin{pmatrix} \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I_1} & \frac{\partial f_1}{\partial I_2} & \frac{\partial f_1}{\partial M} & \frac{\partial f_1}{\partial Z} & \frac{\partial f_1}{\partial P} \\ \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I_1} & \frac{\partial f_2}{\partial I_2} & \frac{\partial f_2}{\partial M} & \frac{\partial f_2}{\partial Z} & \frac{\partial f_2}{\partial P} \\ \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I_1} & \frac{\partial f_3}{\partial I_2} & \frac{\partial f_3}{\partial M} & \frac{\partial f_3}{\partial Z} & \frac{\partial f_3}{\partial P} \\ \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I_1} & \frac{\partial f_4}{\partial I_2} & \frac{\partial f_4}{\partial M} & \frac{\partial f_4}{\partial Z} & \frac{\partial f_4}{\partial P} \\ \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I_1} & \frac{\partial f_5}{\partial I_2} & \frac{\partial f_5}{\partial M} & \frac{\partial f_5}{\partial Z} & \frac{\partial f_5}{\partial P} \\ \frac{\partial f_6}{\partial E} & \frac{\partial f_6}{\partial I_1} & \frac{\partial f_6}{\partial I_2} & \frac{\partial f_6}{\partial M} & \frac{\partial f_6}{\partial Z} & \frac{\partial f_6}{\partial P} \end{pmatrix} \quad (40)$$

$$F = \begin{pmatrix} 0 & \beta_1\psi_1S_u + \beta_2\beta\psi_2S_e & \beta_1\psi_3S_u + \beta_2\beta\psi_4S_e & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (41)$$

But $S_u = \frac{\Lambda_1}{e_o + \mu_1}$ and $S_e = \frac{e_o\Lambda_1}{\mu_1(e_o + \mu_1)}$, $N_1 = \frac{\Lambda_1}{\mu_1}$

$$F = \begin{pmatrix} 0 & \frac{\Lambda_1(\mu_1\beta_1\psi_1 + e_o\beta_2\beta\psi_2)}{\mu_1(e_o + \mu_1)} & \frac{\Lambda_1(\mu_1\beta_1\psi_3 + e_o\beta_2\beta\psi_4)}{\mu_1(e_o + \mu_1)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (42)$$

Let $\phi_1 = \frac{\mu_1\beta_1\psi_1 + \beta_2(1-\theta)\psi_2e_o}{e_o + \mu_1}$, $\phi_2 = \frac{\mu_1\beta_1\psi_3 + \beta_2(1-\theta)\psi_4e_o}{e_o + \mu_1}$

$$F = \begin{pmatrix} 0 & \phi_1 & \phi_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (43)$$

The matrix V , which denotes the net transfer terms between the infected compartments in equations (1), is also considered. It is given by:

$$V = \begin{pmatrix} \alpha_4 R_T - (\alpha_1 + \mu_1)E \\ (1 - \rho)\alpha_1 E - (\sigma_1 + \sigma_2 + \psi_5 L + \mu_1)I_1 \\ \rho\alpha_1 E + \sigma_1 I_1 - (\pi_1 + \pi_2 + \pi_3 + \psi_6 L + \mu_1 + \mu_2)I_2 \\ \pi_1 I_2 - (r_1 + \mu_1 + \mu_3)M \\ \pi_2 I_2 - (r_2 + \mu_1 + \mu_4)Z \\ \pi_3 I_2 - (r_3 + \mu_1 + \mu_5)P \end{pmatrix} \quad (44)$$

$$V = \begin{pmatrix} \frac{\partial g_1}{\partial E} & \frac{\partial g_1}{\partial I_1} & \frac{\partial g_1}{\partial I_2} & \frac{\partial g_1}{\partial M} & \frac{\partial g_1}{\partial Z} & \frac{\partial g_1}{\partial P} \\ \frac{\partial g_2}{\partial E} & \frac{\partial g_2}{\partial I_1} & \frac{\partial g_2}{\partial I_2} & \frac{\partial g_2}{\partial M} & \frac{\partial g_2}{\partial Z} & \frac{\partial g_2}{\partial P} \\ \frac{\partial g_3}{\partial E} & \frac{\partial g_3}{\partial I_1} & \frac{\partial g_3}{\partial I_2} & \frac{\partial g_3}{\partial M} & \frac{\partial g_3}{\partial Z} & \frac{\partial g_3}{\partial P} \\ \frac{\partial g_4}{\partial E} & \frac{\partial g_4}{\partial I_1} & \frac{\partial g_4}{\partial I_2} & \frac{\partial g_4}{\partial M} & \frac{\partial g_4}{\partial Z} & \frac{\partial g_4}{\partial P} \\ \frac{\partial g_5}{\partial E} & \frac{\partial g_5}{\partial I_1} & \frac{\partial g_5}{\partial I_2} & \frac{\partial g_5}{\partial M} & \frac{\partial g_5}{\partial Z} & \frac{\partial g_5}{\partial P} \\ \frac{\partial g_6}{\partial E} & \frac{\partial g_6}{\partial I_1} & \frac{\partial g_6}{\partial I_2} & \frac{\partial g_6}{\partial M} & \frac{\partial g_6}{\partial Z} & \frac{\partial g_6}{\partial P} \end{pmatrix} \quad (45)$$

$$V = \begin{pmatrix} (\alpha_1 + \mu_1) & 0 & 0 & 0 & 0 & 0 \\ -(1 - \rho)\alpha_1 & A_2 & 0 & 0 & 0 & 0 \\ -\rho\alpha_1 & -\sigma_1 & A_3 & 0 & 0 & 0 \\ 0 & 0 & -\pi_1 & (r_1 + \mu_1 + \mu_3) & 0 & 0 \\ 0 & 0 & -\pi_2 & 0 & (r_2 + \mu_1 + \mu_4) & 0 \\ 0 & 0 & -\pi_3 & 0 & 0 & (r_3 + \mu_1 + \mu_5) \end{pmatrix} \quad (46)$$

$$\text{Where } A_1 = (\alpha_1 + \mu_1), A_2 = \left(\sigma_1 + \sigma_2 + \frac{\psi_5 \Lambda_2}{\mu_1 + \mu_8} + \mu_1 \right), A_3 = \left(\pi_1 + \pi_2 + \pi_3 + \frac{\psi_6 \Lambda_2}{\mu_1 + \mu_8} + \mu_1 + \mu_2 \right)$$

$$A_4 = (r_1 + \mu_1 + \mu_3), A_5 = (r_2 + \mu_1 + \mu_4), A_6 = (r_3 + \mu_1 + \mu_5)$$

$$B_1 = -(1 - \rho)\alpha_1, B_2 = -\rho\alpha_1, B_3 = -\sigma_1, B_4 = -\pi_1, B_5 = -\pi_2, B_6 = -\pi_3$$

$$V = \begin{pmatrix} A_1 & 0 & 0 & 0 & 0 & 0 \\ -B_1 & A_2 & 0 & 0 & 0 & 0 \\ -B_2 & -B_3 & A_3 & 0 & 0 & 0 \\ 0 & 0 & -B_4 & A_4 & 0 & 0 \\ 0 & 0 & -B_5 & 0 & A_5 & 0 \\ 0 & 0 & -B_6 & 0 & 0 & A_6 \end{pmatrix} \quad (47)$$

$$V^{-1} = \begin{pmatrix} \frac{1}{A_1} & 0 & 0 & 0 & 0 & 0 \\ \frac{B_1}{A_1 A_2} & \frac{1}{A_2} & 0 & 0 & 0 & 0 \\ \frac{A_2 B_2 + B_1 B_3}{A_1 A_2 A_3} & \frac{B_3}{A_2 A_3} & \frac{1}{A_3} & 0 & 0 & 0 \\ \frac{A_2 B_2 B_4 + B_1 B_3 B_4}{A_1 A_2 A_3 A_4} & \frac{B_3 B_4}{A_2 A_3 A_4} & \frac{B_4}{A_3 A_4} & \frac{1}{A_4} & 0 & 0 \\ \frac{A_2 B_2 B_5 + B_1 B_3 B_5}{A_1 A_2 A_3 A_5} & \frac{B_3 B_5}{A_2 A_3 A_5} & \frac{B_5}{A_3 A_5} & 0 & \frac{1}{A_5} & 0 \\ \frac{A_2 B_2 B_6 + B_1 B_3 B_6}{A_1 A_2 A_3 A_6} & \frac{B_3 B_6}{A_2 A_3 A_6} & \frac{B_6}{A_3 A_6} & 0 & 0 & \frac{1}{A_6} \end{pmatrix} \quad (48)$$

$$FV^{-1} = \begin{pmatrix} \frac{\phi_1 A_2 A_3 B_1 + \phi_2 A_1 A_2 B_2 + \phi_1 B_1 B_3}{A_1 A_2 A_3} & \frac{\phi_1 A_3 + \phi_2 B_3}{A_2 A_3} & \frac{\phi_2}{A_3} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (49)$$

$$R_0 = \frac{\phi_1 A_3 B_1 + \phi_2 A_2 B_2 + \phi_1 B_1 B_3}{A_1 A_2 A_3} \quad (50)$$

$$R_0 = \frac{\left[\begin{aligned} &(\mu_1\beta_1\psi_1 + e_o\beta_2(1-\theta)\psi_2)(\rho-1)\alpha_1 \\ &\times \left\{ ((\sigma_1 + \sigma_2 + \mu_1)(\mu_1 + \mu_8) + \psi_5\Lambda_2) \right. \\ &\times [(\pi_1 + \pi_2 + \pi_3 + \mu_1 + \mu_2)(\mu_1 + \mu_8) + \psi_6\Lambda_2] - \sigma_1(\mu_1 + \mu_8)^2 \left. \right\} \\ &- \left\{ (\mu_1\beta_1\psi_3 + e_o\beta_2(1-\theta)\psi_4)(\sigma_1 + \sigma_2 + \mu_1)(\mu_1 + \mu_8) + \psi_5\Lambda_2 \right\} \\ &\times (\alpha_1 + \mu_1)(\mu_1 + \mu_8)\rho\alpha_1 \end{aligned} \right]}{\left[\begin{aligned} &(e_o + \mu_1)[(\sigma_1 + \sigma_2 + \mu_1)(\mu_1 + \mu_8) + \psi_5\Lambda_2] \\ &\times [(\pi_1 + \pi_2 + \pi_3 + \mu_1 + \mu_2)(\mu_1 + \mu_8) + \psi_6\Lambda_2](\alpha_1 + \mu_1) \end{aligned} \right]} \quad (51)$$

3.4 Global Stability for Substance Abuse-Free Equilibrium

Castillo *et al.* (2002) approach was used to analyzed substance abuse free steady state. For convenience in using the approach, equations (1) were are written as follows

$$\frac{dX}{dt} = H(X, Z) \quad (52)$$

$$\frac{dz}{dt} = G(X, Z), G(X, Z) = 0 \quad (53)$$

where:

$X \in \mathbb{R}^7$ and $X = (S_u, S_e, D, Q, R_P, R_T, L)$ representing the number of uninfected persons and

$Z \in \mathbb{R}^6$, $Z = (E, I_1, I_2, M, Z, P)$ representing the number of the infected persons.

The substance abuse free equilibrium of the system at this point becomes $E^0 = (X^0, 0)$. The GAS of the substance abuse free steady state is guaranteed once the following conditions are satisfied.

(a.) $\frac{dX}{dt} = H(X, 0), X^*$ is GAS

(b.) $\hat{G}(X, Z) = PZ - G(X, Z) \geq 0$ for $(X, Z) \in \Omega$

Here, $P = D_z G(X, 0)$ is an M-Matrix (The off diagonal elements are non-negative) and Ω is the invariant region of the model. If the two conditions (a) and (b) above are satisfied, it shows that the following theorem holds

Theorem 4 The substance abuse free equilibrium state of the model equations (a) and (b) above are satisfied.

Proof: Recall that

$$\begin{aligned} X &= (S_u, S_e, D, Q, R_P, R_T, L) \\ Z &= (E, I_1, I_2, M, Z, P)^T \end{aligned}$$

$$H(X, Z) = \begin{pmatrix} \Lambda_1 - e_o S_u - \lambda_1 S_u - \mu_1 S_u \\ e_o S_u - (1 - \theta) \lambda_2 S_e - \mu_1 S_e \\ \psi_6 L I_2 - (r_2 + \mu_1 + \mu_6) D \\ \sigma_2 I_1 + r_1 M + r_2 Z + r_3 P + r_4 D - (\alpha_2 + \alpha_3 + \mu_1 + \mu_7) Q \\ \alpha_3 Q - \mu_1 R_P \\ \alpha_2 Q + \psi_5 L I_1 - (\alpha_4 + \mu_1) R_T \\ \Lambda_2 - (\mu_1 + \mu_8) L \end{pmatrix} \quad (54)$$

$$H(X, 0) = \begin{pmatrix} \Lambda_1 - e_o S_u - \mu_1 S_u \\ e_o S_u - \mu_1 S_e \\ 0 \\ 0 \\ 0 \\ 0 \\ \Lambda_2 - (\mu_1 + \mu_8) L \end{pmatrix} \quad (55)$$

(55) was proven using Integrating factor method. Hence, condition (a) given above is satisfied.

Recall $\hat{G}(X, Z) = PZ - G(X, Z) \geq 0$, so:

$$G(X, Z) = \begin{pmatrix} \beta_1(\psi_1 I_1 + \psi_3 I_2) S_u + (1 - \theta) \beta_2(\psi_2 I_1 + \psi_4 I_2) S_e + \alpha_4 R_T - (\alpha_1 + \mu_1) E \\ (1 - \rho) \alpha_1 E - (\sigma_1 + \sigma_2 + \psi_5 L + \mu_1) I_1 \\ \rho \alpha_1 E + \sigma_1 I_1 - (\pi_1 + \pi_2 + \pi_3 + \psi_6 L + \mu_1 + \mu_2) I_2 \\ \pi_1 I_2 - (r_1 + \mu_1 + \mu_3) M \\ \pi_2 I_2 - (r_2 + \mu_1 + \mu_4) Z \\ \pi_3 I_2 - (r_3 + \mu_1 + \mu_5) P \end{pmatrix} \quad (56)$$

$$P = D_Z G(X, 0) \quad (57)$$

$$\hat{G}(X, Z) = PZ - G(X, Z) \quad (58)$$

$$P = \begin{pmatrix} -(\alpha_1 + \mu_1) & \beta_1\psi_1S_u^0 + \beta_2(1-\theta)\psi_2S_e^0 & \beta_1\psi_3S_u^0 + \beta_2(1-\theta)\psi_4S_e^0 & 0 & 0 & 0 \\ (1-\rho)\alpha_1 & -Q_1 & 0 & 0 & 0 & 0 \\ \rho\alpha_1 & \delta_1 & -Q_2 & 0 & 0 & 0 \\ 0 & 0 & \pi_1 & -Q_3 & 0 & 0 \\ 0 & 0 & \pi_2 & 0 & -Q_4 & 0 \\ 0 & 0 & \pi_3 & 0 & 0 & -Q_5 \end{pmatrix} \quad (59)$$

where $Q_1 = (\sigma_1 + \sigma_2 + \psi_5L^0 + \mu_1)$, $Q_2 = (\pi_1 + \pi_2 + \pi_3 + \psi_6L^0 + \mu_1 + \mu_2)$, $Q_3 = (r_1 + \mu_1 + \mu_3)$, $Q_4 = (r_2 + \mu_1 + \mu_4)$, $Q_5 = (r_3 + \mu_1 + \mu_5)$

$$PZ = \begin{pmatrix} -(\alpha_1 + \mu_1)E + (\beta_1\psi_1S_u^0 + \beta_2(1-\theta)\psi_2S_e^0)I_1 + (\beta_1\psi_3S_u^0 + \beta_2(1-\theta)\psi_4S_e^0)I_2 \\ (1-\rho)\alpha_1E - (\sigma_1 + \sigma_2 + \psi_5L^0 + \mu_1)I_1 \\ \rho\alpha_1E + \sigma_1I_1 - (\pi_1 + \pi_2 + \pi_3 + \psi_6L^0 + \mu_1 + \mu_2)I_2 \\ \pi_1I_2 - (r_1 + \mu_1 + \mu_3)M \\ \pi_2I_2 - (r_2 + \mu_1 + \mu_4)Z \\ \pi_3I_2 - (r_3 + \mu_1 + \mu_5)P \end{pmatrix} \quad (60)$$

$$\therefore \hat{G}(X, Z) = PZ - G(X, Z) \quad (61)$$

$$PZ - G(X, Z) = \begin{bmatrix} \beta_1(\psi_1I_1 + \psi_3I_2)(S_u^0 - S_u) + \alpha_4R_T \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad (62)$$

$$\therefore \hat{G}(X, Z) = \begin{bmatrix} \hat{G}_1(X, Z) \\ \hat{G}_2(X, Z) \\ \hat{G}_3(X, Z) \\ \hat{G}_4(X, Z) \\ \hat{G}_5(X, Z) \\ \hat{G}_6(X, Z) \end{bmatrix} = \begin{bmatrix} \beta_1(\psi_1I_1 + \psi_3I_2)(S_u^0 - S_u) + \alpha_4R_T \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad (63)$$

Therefore, when $S_u \leq S_u^0$ and $R_T \geq 0$ It implies,

$$\hat{G}_1(X, Z) \geq 0,$$

$$\hat{G}_2(X, Z) = 0$$

$$\hat{G}_3(X, Z) = 0$$

$$\hat{G}_4(X, Z) = 0$$

$$\hat{G}_5(X, Z) = 0$$

$$\hat{G}_6(X, Z) = 0$$

Hence, conditions (a) and (b) given above are satisfied. Therefore the system of equations (1) is Globally and Asymptotically Stable (G.A.S)

3.5 Sensitivity Analysis of Model Parameters

This section evaluates the influence of individual transmission parameters from the substance abuse model (1) on overall dynamics. By comparing how these parameters shift model outcomes, we can isolate the core drivers of the epidemic. Using the foundational equations, we computed the normalized forward sensitivity indices for the threshold number (R_0) to identify which parameters most heavily dictate substance abuse dynamics. The indices were calculated using the standard relative sensitivity formula:

$$\gamma_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0} \quad (64)$$

where;

R_0 is the threshold number and p represents the specific parameter under evaluation. The sensitivity analysis of the threshold number (R_0), which shows the relative influence of model parameters on the spread of substance abuse. The sensitivity analysis shows that β_1 (0.9970) is the strongest positive driver of substance-abuse transmission, indicating that increases in the primary transmission rate substantially increase the overall burden. ρ (0.6180) and ψ_1 (0.6069) are also important positive contributors, highlighting the roles of relapse and progression into high-risk states. Moderate positive effects are observed for ψ_3 (0.3899) and α_1 (0.3390). Conversely, σ_1 (-0.4562) has the strongest negative sensitivity, suggesting that strengthening recovery or treatment can substantially reduce the burden. μ_1 (-0.4148) also has a notable negative effect, while π_1 , π_2 , and π_3 provide smaller preventive effects. Parameters β_2 , ψ_2 , ψ_4 , ψ_5 , ψ_6 , σ_2 , e_0 , μ_8 , Λ_2 , and θ have sensitivity indices close to zero, indicating relatively little influence on the model outcome. In general, the study shows that reducing the initiation rate of substance use strengthened rehabilitation and recovery programs are the most effective approaches to reduce (R_0) and regulate substance misuse in the community.

Table 3: Sensitivity Indices for Substance Abuse Model R_0 .

Parameter	Baseline values	References	Sensitivity Index	Remarks
Λ_2	0.03	Micah <i>et al.</i> (2024)	-0.0050	Negative
e_0	0.03	Micah <i>et al.</i> (2024)	-0.0019	Negative
β_1	0.8	Assumed	0.9970	Positive
β_2	0.5	Assumed	0.0030	Positive
ψ_1	0.0001	Matonya & Kuznetsov (2021)	0.6069	Positive
ψ_2	0.0006	Assumed	0.0029	Positive
ψ_3	0.0005	Micah <i>et al.</i> (2024)	0.3899	Positive
ψ_4	0.0002	Assumed	0.0001	Positive
ψ_5	0.0009	Assumed	-0.0033	Negative
ψ_6	0.0007	Micah <i>et al.</i> (2024)	-0.0017	Negative
μ_1	0.02	Matonya & Kuznetsov (2021)	-0.4148	Negative
μ_2	0.059	Matonya & Kuznetsov (2021)	-0.0959	Negative
μ_8	0.0003	Assumed	0.0001	Positive
α_1	0.039	Matonya & Kuznetsov (2021)	0.3390	Positive
ρ	0.4	Matonya & Kuznetsov (2021)	0.6180	Positive
σ_1	0.0301	Matonya & Kuznetsov (2021)	-0.4562	Negative
σ_2	0.35	Matonya & Kuznetsov (2021)	-0.1003	Negative
θ	0.9	Micah <i>et al.</i> (2024)	-0.0273	Negative
π_1	0.07	Assumed	-0.1138	Negative
π_2	0.05	Assumed	-0.0813	Negative
π_3	0.04	Assumed	-0.0650	Negative

4 Numerical Simulations

In this section, the parameters were varied against time using the initial values

Table 4: Initial Values for the Variables.

Variables	Description	Values	Sources
$S_u(0)$	Unenlightened susceptible at time t	10,200	Micah <i>et al.</i> (2024)
$S_e(0)$	Enlightened susceptible at time t	1,500	Micah <i>et al.</i> (2024)
$E(0)$	Exposed at time t	350	Micah <i>et al.</i> (2024)
$I_1(0)$	Light substance users at time t	900	Matonya & Kuznetsov (2021)
$I_2(0)$	Heavy substance users at time t	340	Matonya & Kuznetsov (2021)
$D(0)$	Heavy substance users under detention at time t	150	Micah <i>et al.</i> (2024)
$M(0)$	Persons with substance-induced psychiatric disorders at time t	150	Assumed
$Z(0)$	Substance induced impoverished person at time t	150	Assumed
$P(0)$	Persons with substance-induced physiological ill conditions at time t	150	Assumed
$Q(0)$	Substance users under rehabilitation at time t	450	Assumed
$R_T(0)$	Temporary recovered substance abusers at time t	200	Matonya & Kuznetsov (2021)
$R_P(0)$	Permanent recovered substance abusers at time t	150	Matonya & Kuznetsov (2021)
$L(0)$	Law enforcement agencies at time t	100	Micah <i>et al.</i> (2024)

4.1 Experiment One: Varying Enlightenment Rate on Sub-Populations Over Time t

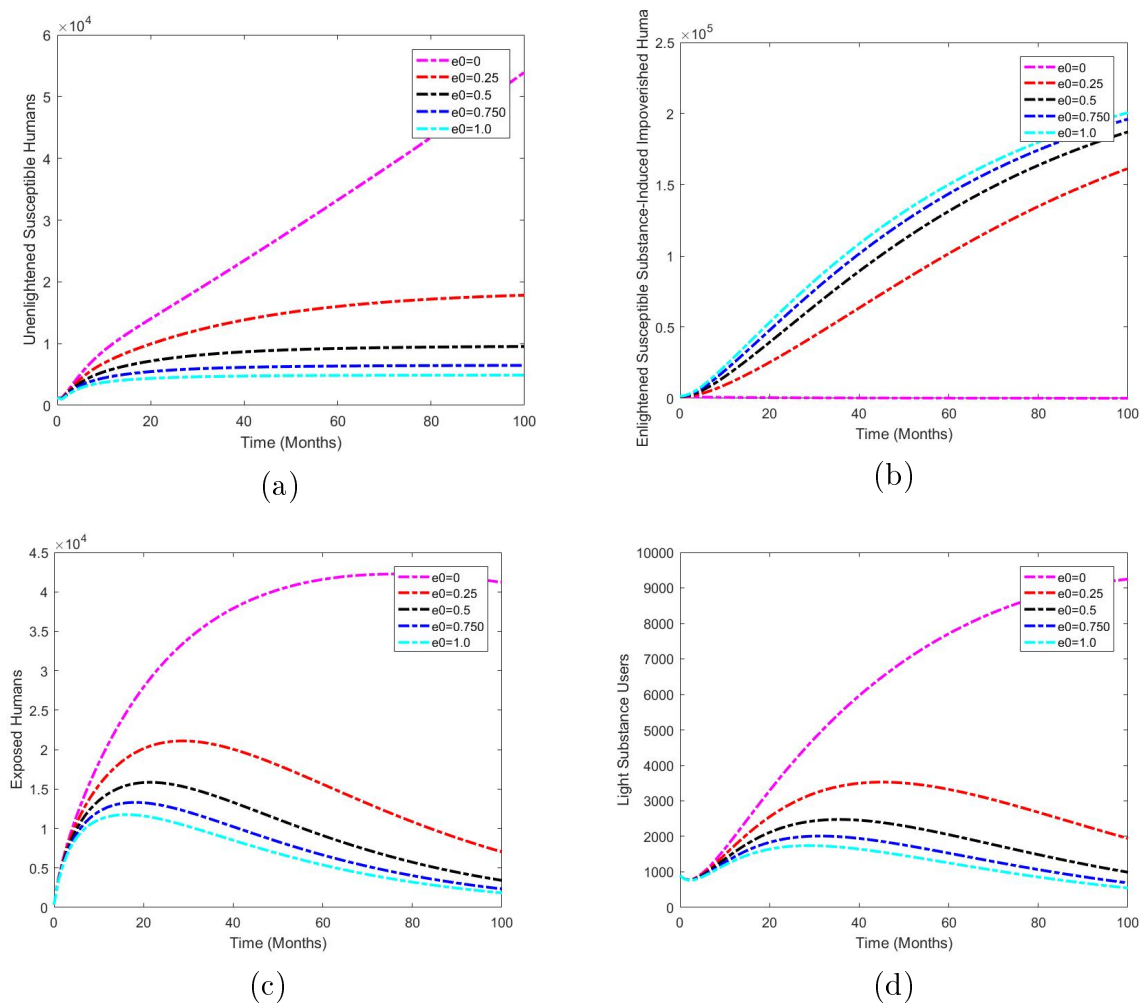


Figure 1: Impact of Enlightenment Rate (e_0) on Unenlightened Susceptible Humans, Enlightened Susceptible Humans, Exposed Humans, and Low Drug Users over time t .

As illustrated in Figures 1(a–d), increasing the enlightenment rate (e_0) exerts a significant regulatory influence across all evaluated sub-populations over time t . Specifically, Figure 1(a) demonstrates a marked and continuous depletion of the unenlightened susceptible compartment, as a higher transition rate e_0 rapidly shifts individuals out of the high-susceptibility state. Figure 1(b) highlights a rapid accumulation in the enlightened susceptible population, confirming that targeted public awareness effectively expands the proportion of informed individuals who possess adaptive behavioral safeguards. As shown in Figures 1(c) and 1(d), both the exposed cohort and the low-drug-user population experience substantial decreases in peak prevalence and overall burden as e_0 increases. By limiting the influx of naive individuals into exposure pathways, the higher enlightenment rate restricts the rate of primary initiation and subsequent progression toward active substance reliance. This finding is consistent with existing research on public health communication and behavioral interventions [Zhang et al. \(2021\)](#).

4.2 Experiment One Continues: Varying Enlightenment Rate on Sub-Populations Over Time t

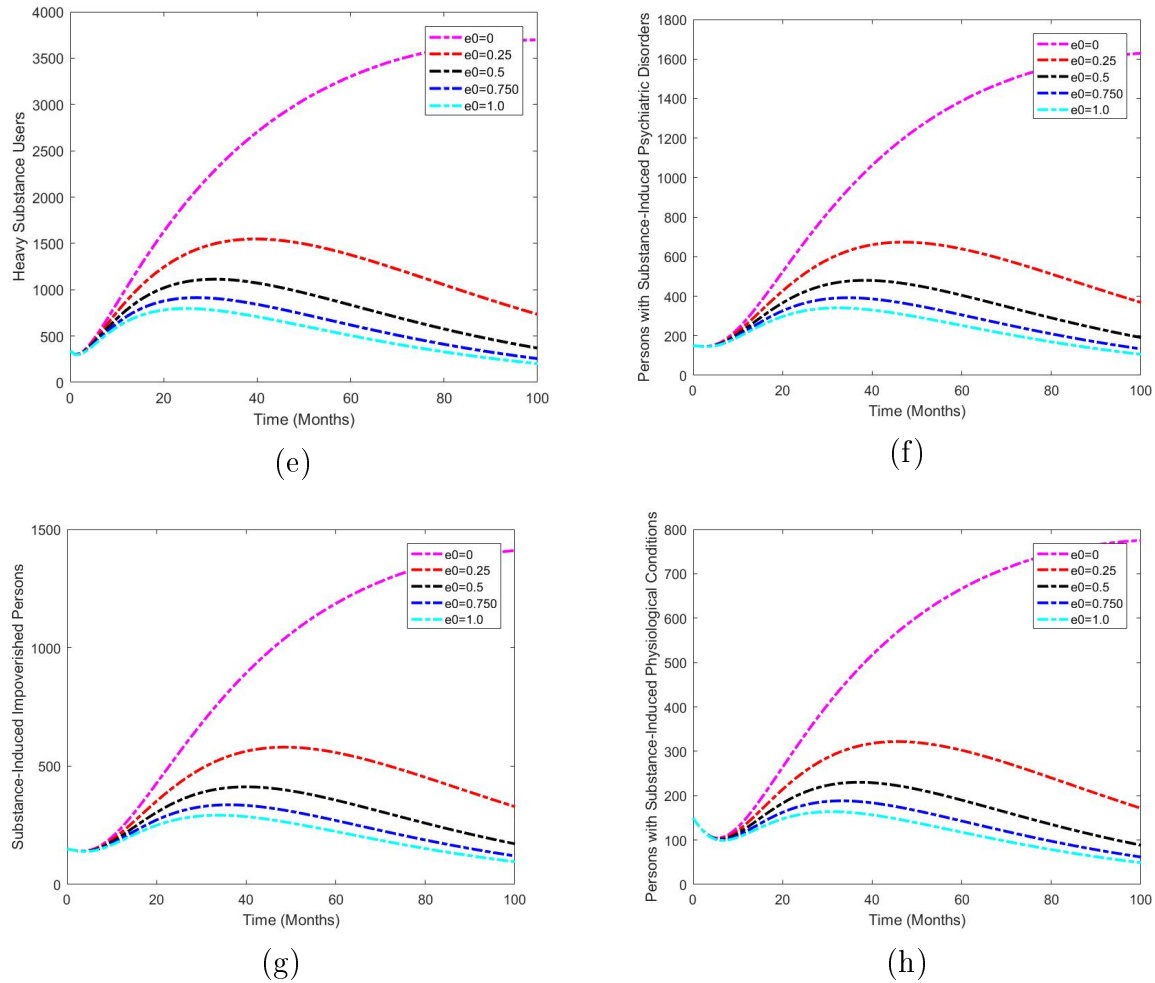


Figure 2: Impact of Enlightenment Rate (e_0) on Heavy Substance Users, Persons with Substance Induced psychiatric Disorders, Substance Induced Impoverished Persons and Persons with Substance Induced Physiological ill Health at time t

The long-term temporal trajectories of heavy substance users (I_2), individuals with substance-induced psychiatric disorders (M), individuals experiencing substance-induced impoverishment (Z), and those with substance-related physiological conditions (P) over a 100-month simulation window are detailed in Figures 2(e–h). All four cohorts experience substantial and sustained reductions, characterized by rapid initial drops followed by an asymptotic approach toward near-zero levels. These decreasing profiles demonstrate that the implemented intervention framework effectively disrupts substance dependence while mitigating its downstream psychological, economic, and systemic health impacts. These numerical outcomes concur with modeling studies by [Thompson et al. \(2022\)](#), who highlighted that integrated treatment and rehabilitation strategies significantly alleviate the long-term clinical and socioeconomic burdens of severe substance use. By capturing the concurrent suppression of medical, psychiatric, and financial indicators within a single system.

4.3 Experiment One Continues: Varying Enlightenment Rate on Sub-Populations Over Time t

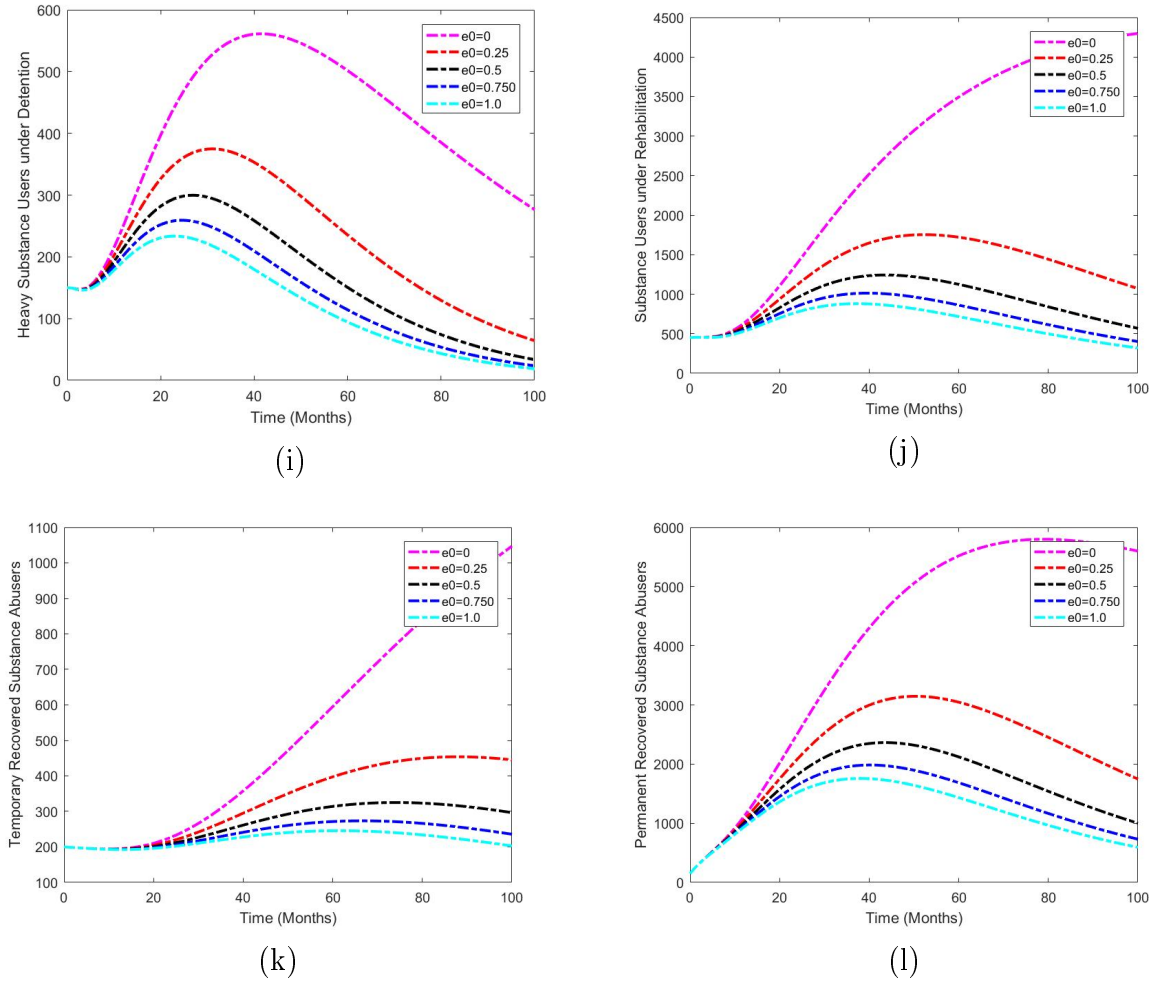


Figure 3: Impact of enlightenment rate (e_0) on Heavy substance users under detention, substance users under rehabilitation, temporary recovered substance abusers and permanent recovered substance abusers at time t

Figures 3(i–l) illustrate the 100-month simulation dynamics for individuals in detention (D), those undergoing active rehabilitation (Q), temporarily recovered individuals (R_T), and permanently recovered individuals (R_P). The detained cohort exhibits a steady decline toward zero, reflecting a reduced reliance on custodial measures as community-level transmission falls. Conversely, the rehabilitation compartment displays an initial influx as individuals enter clinical treatment, followed by a gradual decay as overall substance abuse prevalence contracts. Similarly, the temporarily recovered population steadily decreases, whereas the permanently recovered class experiences a steep initial surge before leveling off at a high sustained baseline. These results align with modeling research by [williams et al. \(2023\)](#), who established that coupling criminal justice diversions with robust rehabilitation and aftercare infrastructure significantly enhances long-term cessation and prevents recidivism. By explicitly tracking transitions between transient and permanent recovery states within a unified mathematical framework.

4.4 Experiment two: Varying Contact Rate β_1 on Sub-Populations over t

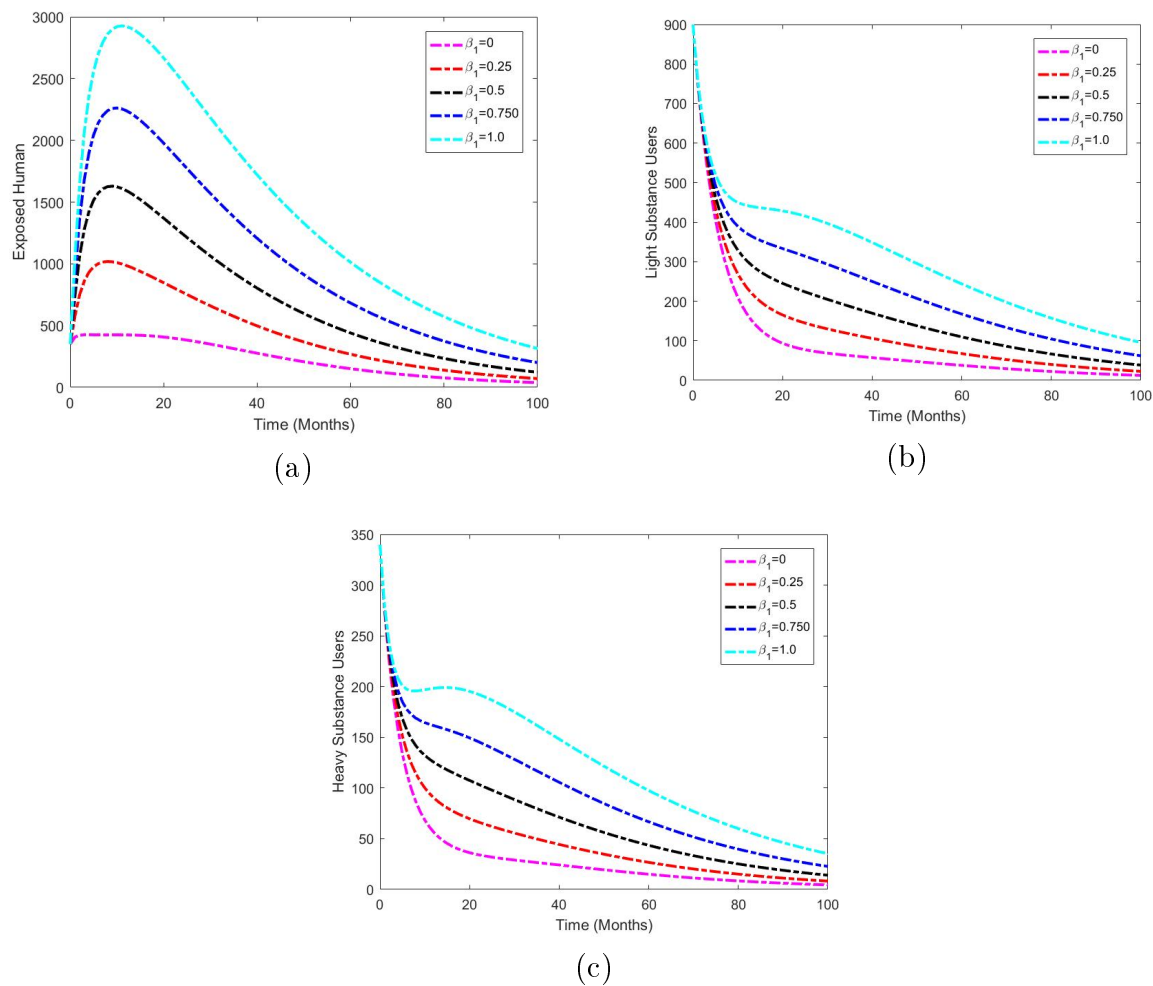


Figure 4: Impact of Contact Rate (β_1) on Exposed Human, Light Substance Users, and Heavy Substance Abusers at time t

Figures 4(a–c) illustrate the impact of varying the contact rate (β_1) on the exposed population, light substance users, and heavy substance users across a 100-month simulation horizon. Higher values of β_1 significantly elevate the exposed cohort while delaying the decline of both light and chronic substance users. Conversely, reducing the contact rate suppresses initial exposure and accelerates the clearance of active substance-using populations, demonstrating that contact intensity is a primary driver of behavioral transmission and disease persistence. These numerical outcomes align with epidemiological modeling by [Rodriguez et al. \(2024\)](#), who showed that peer interaction frequency strongly dictates initiation rates and recovery trajectories in substance use dynamics. By explicitly quantifying the threshold effects of β_1 across multiple progression stages within a single system, these findings reinforce the necessity of network-based interventions, peer-focused education, and environmental controls that minimize high-risk social contact.

4.5 Experiment three: Varying Interaction Rate (ψ_1) on Sub Populations over time t

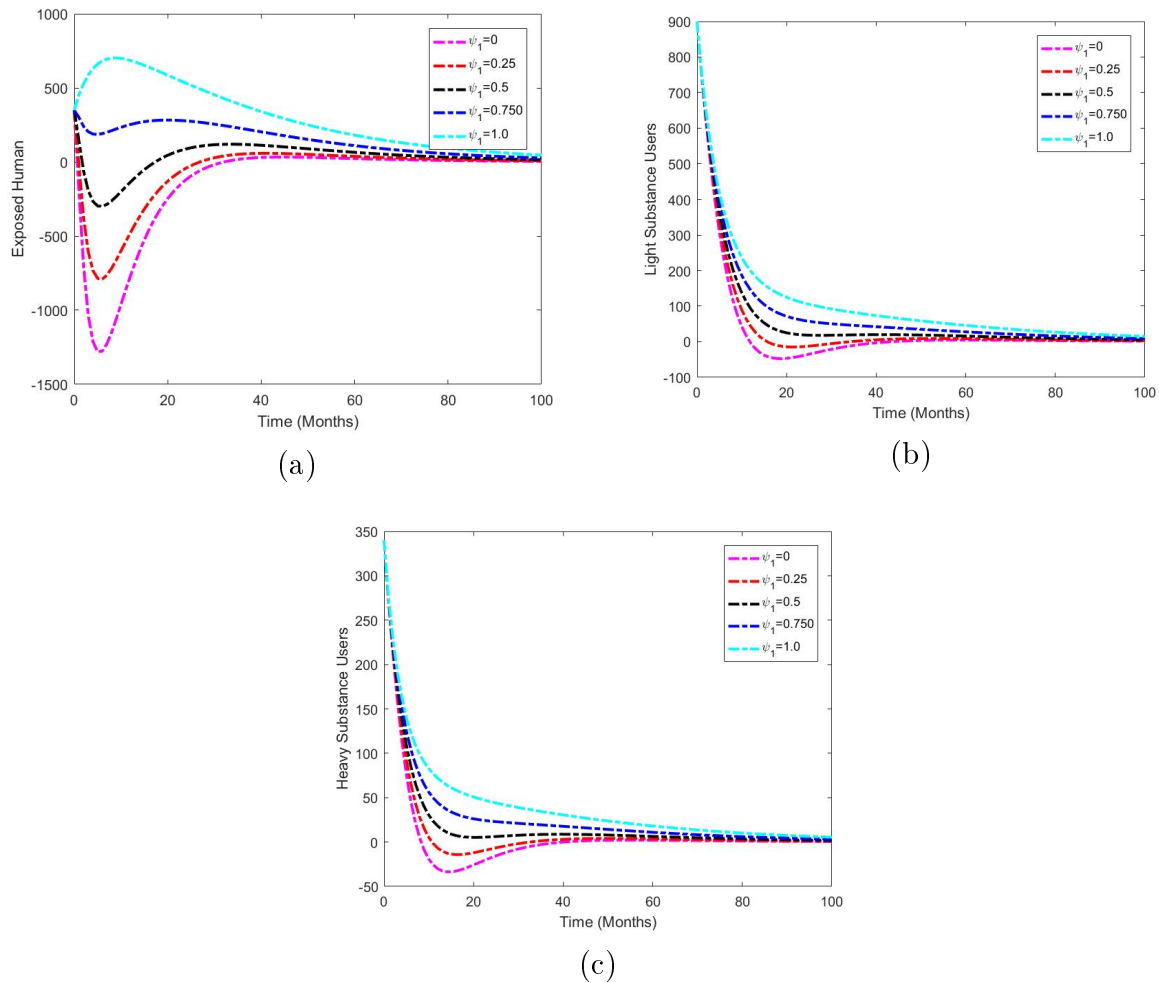


Figure 5: Impact of Interaction Rate (ψ_1) on Exposed Human, Light Substance Users, and Heavy Substance Abusers at time t

Figures 5(a–c) depict how variations in the interaction rate (ψ_1) affect the exposed, light substance-users, and heavy substance-users compartments over a 100-month simulation period. Higher values of ψ_1 expand the exposed cohort and retard the clearance of both light and chronic users, underscoring how intensified social contacts and peer pressure sustain substance abuse propagation. Conversely, reducing the interaction rate accelerates the decline across all three groups. Brief numerical excursions below zero in the plots represent transient computational artifacts rather than biologically meaningful population states. The simulated findings by Nakamura & Fernandes (2024), who established that network connectivity and interaction intensity strongly governed initiation rates and recovery delays. By quantifying the sensitivity of distinct usage compartments to variations in ψ_1 , these results highlight the strategic value of community-level interventions that weaken negative peer influences and cultivate supportive social environments.

5 Conclusion

This study evaluated the threshold dynamics and stability characteristics of a deterministic mathematical model governing substance abuse. Utilizing the next-generation matrix framework, the threshold number ($\mathcal{R}_0$) was formulated to establish the threshold conditions governing epidemic persistence versus suppression. Rigorous stability analysis employing the Castillo Chavez framework confirmed that the substance abuse free equilibrium is globally asymptotically stable whenever $\mathcal{R}_0 < 1$, proving that sustained eradication of substance abuse is achievable when the threshold number is constrained below unity. Furthermore, sensitivity evaluations pinpointed key transition parameters most notably public enlightenment, contact, and social interaction rates as the primary drivers of population-level transmission. Numerical experiments corroborated these theoretical proofs, demonstrating that expanding public education while curtailing high-risk contact and peer interaction significantly reduces overall exposure and suppresses progression toward light and heavy usage states. These theoretical and computational insights offer valuable epidemiological guidance for structuring targeted prevention, treatment, rehabilitation, and awareness campaigns to mitigate the burden of substance abuse.

Availability of data and material

Not Applicable.

Competing Interest

No competing interest is hereby declared by the authors.

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