Global Dynamics of Drug Use Model and Its Optimal Control Analysis

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Abstract

In this paper, we propose and analyze a mathematical model of drug use involving rehabilitation. With our model, we assume that the use of drugs can be initiated by three groups of people; light drug users, heavy drug users and drug users under rehabilitation. In this model, the nonnegativity and boundedness of solutions are verified, and two equilibrium points (drug-free and drug-endemic) are obtained. The basic reproduction number is calculated. We show that each equilibrium point is stable locally and globally under some conditions. Further, an optimal control problem is applied to the model by adding three control variables; awareness and educational program control, family and friends care control, and rehabilitation campaign control. The numerical simulation of this optimal control model is performed, and the results show that each control alone could already reduce the number of drug users for some certain amount, however, a combination of all three controls gives the best result in reducing overall drug use.

Keywords: drug use, awareness and educational program, family and friends care, basic reproduction number, optimal control

Introduction

Drug use remains one of the major health, psychosocial and socioeconomic problems globally. Drug misuse negatively impacts personal health, the economy, and society overall. The World Drug Report (World Drug Report, 2021), released by the United Nations Office on Drugs and Crime (UNODC), reported that there were about 275 million people using drugs in 2020 and over 36 million people suffered from drug use disorders (UNODC, 2021). Further, it is estimated that by 2030, the number of people using drugs will rise by 11% over 2021 Fig.s worldwide. For Thailand, in 2019 an estimated 3.75 million people aged 12-65 years had used drugs at some time and 1.97 million people had used drugs in 2018 (Kanato & Leyatikul, 2019). Unfortunately, the number of drug users seems to increase year by year.

To start using drugs, there needs to be motivation to do so, and drugs must be accessible. It is believed that having only motivation without any access to drugs cannot lead to drug use, whereas interaction with drug lords would provide easy access to drugs. Therefore, motivation plus access creates the situation of initiating drug abuse (Johnston, O'Malley, & Bachman, 1988).

Like infectious diseases, compartmental models have been used as a tool for better understanding drug use dynamics. Several research projects have been undertaken to study drug use dynamics. Some researchers focussed on specific drugs e.g., methamphetamine (Nyabadza & Hove-Musekwa, 2010), heroin (Rossi, 2004; White & Comiskey, 2007, Samanta, 2011), and cocaine (Everingham & Rydell, 1994; Caulkins, Gragnani, Feichtinger, & Trangler, 2006) etc. Elsewhere, some researchers focused on general drug use without specific consideration of particular drugs, which is also the focus of this paper. Some examples of mathematical models of general drug use include Njagarah and Nyabadza (2013) who proposed a drug use model by considering the role of drug lords in initiating drug use. They divided their model into five categories of susceptible individuals, light drug users, heavy drug users, drug users under rehabilitation, categories that could initiate drug use, and drug lords, who facilitate initial drug use. Mushayabasa and Tapedzesa (2015) proposed a model with five

subclasses of the population which were susceptible to drug use; light drug users, heavy drug users, mentally ill individuals, and known illicit drug users. In this model, they assumed that drug initiation was done by only light drug users and heavy drug users. They extended their model by applying the optimal control problem to their model with two controls; control by reducing the intensity of social influence between susceptible population and illicit drug users, and control by increasing the rate of detection and rehabilitation of illicit drug users. Kanyaa, Osman, and Wainaina (2018) presented a model of substance use specifically by commercial drivers and they defined four classes of population which were susceptible classes; all commercial drivers that are at risk of using any substance (drug), commercial drivers who use a substance, commercial drivers who abuse drug of any form, and drivers who had stopped using drugs. Li and Ma (2018) proposed a model involving the effects of family influence and public health education on drug transmission, where their model consisted of six categories of susceptible individuals, including those who do not accept the education, susceptible individuals who accept the education, light drug users, heavy drug users, drug users with treatment, and ex-drug users who had permanently quit. Similar to Li and Ma, 2018, Hafiruddin, Fatmawati, and Miswanto (2019) presented a drug transmission model that included the effect of criminal penalties. The model consisted of six subclasses; susceptible individuals, individuals vulnerable to being a narcotic addict but had received drug misuse education, mild drug users, heavy drug users, drug users who had been impacted by criminal law, and ex- drug users who stopped using drugs. They also applied rehabilitation control as an optimal control to their model. Islam and Biswas (2020) presented a model with five subclasses; susceptible individuals, light drug users, heavy drug users, drug users under treatment in rehabilitation, and ex- drug users who had quit. Here in this model, they assumed the relapse of heavy drug users from the rehabilitation group. They applied three controls to their model; awareness and educational program control, family-based care control, and rehabilitation-centred control. Olajide (2020) proposed a drug use model that incorporated the banditry component. Binuyo and Osuntokun (2021) proposed a model of addiction among students in Nigeria that comprised five variables; susceptible students, drug users, students who are exposed to the use of drug substances, students who are addicted to drug substances, and students who stopped using drug substance.

In this study, we propose a mathematical model for drug use by modifying the work of Islam and Biswas (2020) by considering the heavy drug users reverting to light drug users, and both light and heavy drug users being recruited into rehabilitation programs. Further, we also apply the optimal control problem into our model by adding three controls; awareness and educational program control, family and friends care control, and rehabilitation campaign control. We are seeking the best strategy for effective control of drug use. In our study, we consider general substance abuse, not the abuse of any specific substance.

Model Description

We propose a mathematical model to better understand the dynamics of drug use by considering four subclasses; susceptible individuals, which we code as (S), light drug users (L), heavy drug users (H), and drug users under rehabilitation (R). Following the work of Islam and Biswas (2020), we assume that (i) drug initiation can be done by three groups of people; light drug users, heavy drug users, and drug users under rehabilitation, (ii) heavy drug users can revert to being light drug users, and (iii) both light and heavy drug users can get some treatment under rehabilitation. The schematic diagram of the model is shown in Fig. 1.

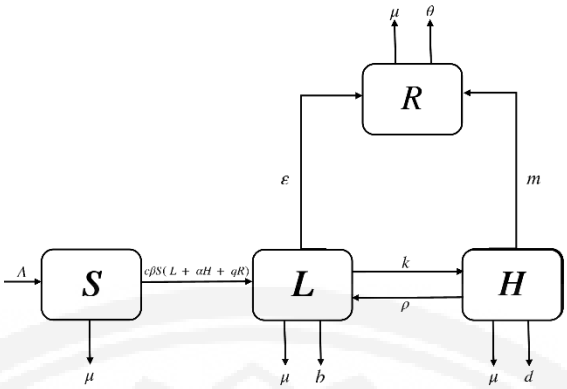

Figure 1 A schematic diagram of the drug use dynamics model.

From the above description, we can write our model as a system of equations as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - c\beta S\,(L + \alpha H + qR) - \mu S \\
\frac{dL}{dt} &= c\,\beta S\,(L + \alpha H + qR) + \rho H - (\epsilon + k + \mu + b)L \\
\frac{dH}{dt} &= k\,L - (\rho + d + m + \mu)H \\
\frac{dR}{dt} &= \epsilon L + m\,H - (\theta + \mu)R.
\end{aligned} \tag{1}$$

The corresponding differential equations are with initial conditions $S(0) \geq 0,\ L(0) \geq 0,\ H(0) \geq 0,\ R(0) \geq 0.$ The total population size is N where $N = S + L + H + R.$

In this model, the susceptible population is recruited at a rate Λ. The susceptible population can be initiated to become light drug users with a term $c\beta S(L+\alpha H+qR)$, where c represents the mean number of effective contacts between current drug users and susceptible individuals in the population, β represents the probability that contact between drug users and susceptible individuals results in the initiation of new drug users, α represents the ability of heavy drug users to initiate new drug users, and q represents drug users under rehabilitation who can initiate new drug users. All populations die naturally at rate μ. The light drug users can escalate to being heavy drug users at a rate k, and they die because of light drug use at a rate b, whereas the heavy drug users can revert to being light drug users at a rate ρ, and they die because of heavy drug use at a rate d. Both light and heavy drug users can be recruited to rehabilitation at a rate ε and m, respectively. Finally, those who are in rehabilitation can quit using drugs permanently at a rate θ.

Model Analysis

1. Nonnegativity and boundary of solutions

For any nonnegative initial conditions, consider the following,

$$\left.\frac{dS}{dt}\right|_{S=0} = \Lambda \geq 0,\ \left.\frac{dL}{dt}\right|_{L=0} = c\beta S(\alpha H + qR) + \rho H \geq 0,\ \left.\frac{dH}{dt}\right|_{H=0} = kL \geq 0,\ \text{and}\ \left.\frac{dR}{dt}\right|_{R=0} = \varepsilon L + mL \geq 0.$$

Hence, all solutions of (1) are nonnegative for all $t > 0$.

Next, the boundary of solutions of (1) is determined.

Since, $N = S + L + H + R$, then $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dL}{dt} + \frac{dH}{dt} + \frac{dR}{dt} = \Lambda - \mu N - bL - dH - \theta R.$ (2)

Then, $$\frac{dN}{dt} \leq \Lambda - \mu N\ .$$ (3)

We integrate equation (3) by using the integrating factor method, which gives

$$N \leq \frac{\Lambda}{\mu} + C_1 e^{-\mu t}.$$

Therefore when $t \to \infty$, then $N \to \frac{\Lambda}{\mu}$, implies that $0 \leq N \leq \frac{\Lambda}{\mu}$.

Thus, the considered region for this model is $\Omega = \{(S, L, H, R) \in \mathbb{R}^4_+ : N \leq \frac{\Lambda}{\mu}\}$.

All solutions of this model are bounded and enter the region Ω. Hence, Ω is positively invariant. That is, every solution of this model remains there for all $t \geq 0$.

2. Equilibrium points

Two equilibrium points are obtained in this model, and they are

(i) The drug-free equilibrium point (E_0)

It is the equilibrium point at which drug use is eradicated. From (1), the drug-free equilibrium point is as follows: $E_0 = (S_0, L_0, H_0, R_0) = (\frac{\Lambda}{\mu}, 0, 0, 0)$.

(ii) The drug endemic equilibrium point (E_1)

The drug endemic equilibrium point is denoted by $E_1 = (S^*, L^*, H^*, R^*)$, where

$$S^* = \frac{\Lambda}{c\beta\left(\frac{\rho + d + m + \mu}{k} + \alpha + \frac{q(\rho + d + m + \mu + km)}{k(\theta + \mu)}\right) H^* + \mu}, L^* = \frac{(\rho + d + m + \mu)H^*}{k},$$

$$H^* = \frac{\mu\rho + \Lambda c\beta\left[\frac{\rho + d + m + \mu}{k} + \alpha + \frac{q}{(\theta + \mu)}\left(\frac{\epsilon(\rho + d + m + \mu)}{k} + m\right)\right] - \frac{\mu(\epsilon + k + b + \mu)(\rho + d + m + \mu)}{k}}{\left(\frac{(\epsilon + k + b + \mu)(\rho + d + m + \mu)}{k} - \rho\right) \Lambda c\beta\left[\frac{\rho + d + m + \mu}{k} + \alpha + \frac{q}{(\theta + \mu)}\left(\frac{\epsilon(\rho + d + m + \mu)}{k} + m\right)\right]},$$

and $R^* = \frac{\left(\frac{\epsilon(\rho + d + m + \mu)H^*}{k}\right) + mH^*}{\theta + \mu}$.

3. The basic reproduction number (R_0)

The basic reproduction number (R_0) is the average number of secondary infections that are produced by a typical infective individual. We use the next-generation matrix method by van den Driessche and Watmough (2002) to calculate R_0. The next-generation matrix is the matrix FV^{-1} where F and V are the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$. Here $\mathcal{F}$ is the matrix of the rate of appearance of new addictions in compartments L, H and R and and $\mathcal{V}$ is the matrix of the transfer rate of individual addictions. Thus, we obtain

$$\mathcal{F} = \begin{bmatrix} c\beta S(L + \alpha H + qR) \\ 0 \\ 0 \end{bmatrix} \text{ and } \mathcal{V} = \begin{bmatrix} (\varepsilon + k + b + \mu)L - \rho H \\ (\rho + d + m + \mu)H - kL \\ (\theta + \mu)R - \varepsilon L - mH \end{bmatrix}$$

Then, the Jacobian matrices of the above matrices are

$$F = \begin{bmatrix} c\beta S & \alpha c\beta S & qc\beta S \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \varepsilon + k + b + \mu & -\rho & 0 \\ -k & \rho + d + m + \mu & 0 \\ -\varepsilon & -m & \theta + \mu \end{bmatrix}.$$

By substituting the drug-free equilibrium point in the Jacobian matrices above, we get

$$F(E_0) = \begin{bmatrix} \frac{c\beta\Lambda}{\mu} & \frac{\alpha c\beta\Lambda}{\mu} & \frac{qc\beta\Lambda}{\mu} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad V(E_0) = \begin{bmatrix} \varepsilon + k + b + \mu & -\rho & 0 \\ -k & \rho + d + m + \mu & 0 \\ -\varepsilon & -m & \theta + \mu \end{bmatrix}.$$

The next-generation matrix therefore is

$$FV^{-1} = \begin{bmatrix} \frac{c\beta\Lambda B_1}{\mu B_4} & \frac{c\beta\Lambda B_2}{\mu B_4} & \frac{c\beta\Lambda B_3}{\mu B_4} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

where,

$B_1 = (\theta + \mu)(\rho + d + m + \mu) + \alpha k(\theta + \mu) + q(km + (\rho + d + m + \mu)\epsilon)$
$B_2 = \rho(\theta + \mu) + \alpha(\theta + \mu)(\epsilon + k + b + \mu) + q((\epsilon + k + b + \mu)m + \rho\epsilon)$
$B_3 = ((\epsilon + k + b + \mu)(\rho + d + m + \mu) - \rho k)q$
$B_4 = (\theta + \mu)((\epsilon + k + b + \mu)(\rho + d + m + \mu) - \rho k).$

The basic reproduction number is the leading eigenvalue of FV^{-1}, then

$$R_0 = \frac{c\beta\Lambda\,((\theta+\mu)(\rho+d+m+\mu)+\alpha k(\theta+\mu)+q(km+(\rho+d+m+\mu)\epsilon))}{\mu\,((\theta+\mu)((\epsilon+k+b+\mu)(\rho+d+m+\mu)-\rho k))}. \quad (4)$$

4. Local stability of drug-free equilibrium point

Theorem 1 The drug-free equilibrium point (E_0) is locally asymptotically stable if $R_0 < 1$ and if it satisfies Routh-Hurwitz criteria. If $R_0 > 1$, then the drug-free equilibrium point (E_0) is unstable.

Proof The Jacobian matrix of (1) is

$$J(E) = \begin{bmatrix} -c\beta(L + \alpha H + qR) - \mu & -c\beta S & -\alpha c\beta S & -qc\beta S \\ c\beta(L + \alpha H + qR) & c\beta S - (\epsilon + k + b + \mu) & \frac{\alpha c\beta\Lambda}{\mu} + \rho & \frac{qc\beta\Lambda}{\mu} \\ 0 & k & -(\rho + d + m + \mu) & 0 \\ \alpha & \epsilon & m & -(\theta + \mu) \end{bmatrix}. \quad (5)$$

And at E_0, we have

$$J(E_0) = \begin{bmatrix} -\mu & -\frac{c\beta\Lambda}{\mu} & -\frac{\alpha c\beta\Lambda}{\mu} & -\frac{qc\beta\Lambda}{\mu} \\ 0 & \frac{c\beta\Lambda}{\mu} - (\epsilon + k + b + \mu) & \frac{\alpha c\beta\Lambda}{\mu} + \rho & \frac{qc\beta\Lambda}{\mu} \\ 0 & k & -(\rho + d + m + \mu) & 0 \\ \alpha & \epsilon & m & -(\theta + \mu) \end{bmatrix}. \quad (6)$$

From the Jacobian matrix above, we set $det(J(E_0) - \lambda I) = 0$ to find eigenvalues, and then we obtain

$$\det(J(E_0) - \lambda I) = (-\mu - \lambda)\left(\lambda^3 + \left[\theta + \mu + \rho + d + m + \mu + \epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right]\lambda^2 + \left[\left(\epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right)(\rho + d + m + \mu) + (\theta + \mu)(\epsilon + k + b + \mu) + (\theta + \mu)\left(\epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right) - \frac{\epsilon qc\beta\Lambda}{\mu} - \frac{\alpha kc\beta\Lambda}{\mu} - \rho k\right]\lambda - (\theta + \mu)\left(\frac{c\beta\Lambda}{\mu} - (\epsilon + k + b + \mu)\right)(\rho + d + m + \mu) - (\theta + \mu)\left(\frac{\alpha kc\beta\Lambda}{\mu} + \rho k\right) - \frac{qc\beta\Lambda}{\mu}(km + \epsilon(\rho + d + m + \mu))\right) = 0$$

Thus, $\lambda_1 = -\mu < 0$.

Next, we consider

$$\lambda^3 + \left[\theta + \mu + \rho + d + m + \mu + \epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right]\lambda^2 + \left[\left(\epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right)(\rho + d + m + \mu) + (\theta + \mu)(\epsilon + k + b + \mu) + (\theta + \mu)\left(\epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right) - \frac{\epsilon qc\beta\Lambda}{\mu} - \frac{\alpha kc\beta\Lambda}{\mu} - \rho k\right]\lambda - (\theta + \mu)\left(\frac{c\beta\Lambda}{\mu} - (\epsilon + k + b + \mu)\right)(\rho + d + m + \mu) - (\theta + \mu)\left(\frac{\alpha kc\beta\Lambda}{\mu} + \rho k\right) - \frac{qc\beta\Lambda}{\mu}(km + \epsilon(\rho + d + m + \mu)) = 0$$

which is considered in the form of $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0$. (7)

We have

$$a_1 = \theta + \mu + \rho + d + m + \mu + \epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu},$$

$$a_2 = \left(\epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right)(\rho + d + m + \mu) + (\theta + \mu)(\epsilon + k + b + \mu) + (\theta + \mu)\left(\epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right) - \frac{\epsilon qc\beta\Lambda}{\mu} - \frac{\alpha kc\beta\Lambda}{\mu} - \rho k,$$

$$a_3 = -(\theta + \mu)\left(\frac{c\beta\Lambda}{\mu} - (\epsilon + k + b + \mu)\right)(\rho + d + m + \mu) - (\theta + \mu)\left(\frac{\alpha k c\beta\Lambda}{\mu} + \rho k\right) - \frac{qc\beta\Lambda}{\mu}\left(km + \epsilon(\rho + d + m + \mu)\right).$$

Since, when $R_0 < 1$, we have $\frac{c\beta\Lambda\,(\,(\theta+\mu)(\rho+d+m+\mu)+\alpha k(\theta+\mu)+q(km+(\rho+d+m+\mu)\epsilon))}{\mu\,((\theta+\mu)((\epsilon+k+b+\mu)(\rho+d+m+\mu)-\rho k)\,)} < 1$, i.e. $c\beta\Lambda\left(\,(\theta + \mu)(\rho + d + m + \mu) + \alpha k(\theta + \mu) + q(km + (\rho + d + m + \mu)\epsilon)\right) < \mu\left((\theta + \mu)\left((\epsilon + k + b + \mu)(\rho + d + m + \mu) - \rho k\right)\right)$,

then we can derive to have $\frac{c\beta\Lambda}{\mu} < \epsilon + k + b + \mu$.

Consider $a_1 = \theta + \mu + \rho + d + m + \mu + \epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}$, since $\frac{c\beta\Lambda}{\mu} < \epsilon + k + b + \mu$. Therefore, $a_1 > 0$.

Next, we consider $a_3 = (-(\theta + \mu)\left(\frac{c\beta\Lambda}{\mu} - (\epsilon + k + b + \mu)\right)(\rho + d + m + \mu) - (\theta + \mu)\left(\frac{\alpha k c\beta\Lambda}{\mu} + \rho k\right) - \frac{qc\beta\Lambda}{\mu}\left(km + \epsilon(\rho + d + m + \mu)\right)$

$$= (\theta + \mu)\left((\epsilon + k + b + \mu)(\rho + d + m + \mu) - \rho k\right)(1 - R_0). \quad (8)$$

Therefore, $a_1 > 0$ and $a_3 > 0$ when $R_0 < 1$.

Finally, consider $a_1 a_2 > a_3$, we have

$$a_1 a_2 - a_3 = \left[-\theta + \mu + \rho + d + m + \mu + \epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right]\left[\left(\epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right)(\rho + d + m + \mu) + (\theta + \mu)(\epsilon + k + b + \mu) + (\theta + \mu)\left(\epsilon + k + b + \mu - \frac{c\beta\Lambda}{\mu}\right) - \frac{\epsilon qc\beta\Lambda}{\mu} - \frac{\alpha k c\beta\Lambda}{\mu} - \rho k\right] - \left[(\theta + \mu)\left(\frac{c\beta\Lambda}{\mu} - (\epsilon + k + b + \mu)\right)(\rho + d + m + \mu) - (\theta + \mu)\left(\frac{\alpha k c\beta\Lambda}{\mu} + \rho k\right) - \frac{qc\beta\Lambda}{\mu}\left(km + \epsilon(\rho + d + m + \mu)\right)\right].$$

From above, we cannot clarify that $a_1 a_2 - a_3$ is greater than zero.

Hence, by the criteria of Routh-Hurwitz ($a_1 > 0, a_3 > 0$ and $a_1 a_2 > a_3$), the drug-free equilibrium point is locally asymptotically stable when $R_0 < 1$ and $a_1 a_2 > a_3$. When $R_0 > 1$, E_0 is unstable. This completes the proof.

5. The global stability of the drug-free equilibrium point

Lamma 1 (Castillo-Chaves, Feng, & Huang, 2001) Consider a model system written in the form

$$\frac{dX_1}{dt} = F(X_1, X_2)$$

$$\frac{dX_2}{dt} = G(X_1, X_2), G(X_1, 0) = 0$$

where $X_1 \in R^m$ denotes (its components) the number of uninfected individuals and $X_2 \in R^m$ denotes (its components) the number of infected individuals including latent, infectious, etc; $X_0 = (x_1^*)$ denotes the disease-free equilibrium of the system. $F(X_1, X_2)$ and $G(X_1, X_2)$ are functions of X_1 and X_2. Also assume the conditions (H1) and (H2) below:

(H1) For $\frac{dX_1}{dt} = F(X_1, X_2)$, x_1^* is globally asymptotically stable,

(H2) $G(X_1, X_2) = AX_2 - \hat{G}(X_1, X_2)$, all elements in $\hat{G}(X_1, X_2)$ are nonnegative for $(X_1, X_2) \in \Omega$,

where $A = \frac{\partial G}{\partial X_2}\left(X_1^*, 0\right)$ is an M-matrix (the off-diagonal elements of A are nonnegative) and Ω is the region where the model makes biological sense.

The goal of the following theorem is to show the condition which indicates that, under the circumstance of model (1), drug use can be eradicated eventually, leading to global stability of a drug-free equilibrium point.

Theorem 2 If $R_0 < 1$ and $q = 0$, then the drug-free equilibrium point (E_0) is globally asymptotically stable.

Proof We use the conditions following Lemma 1 to determine global stability. To show that the conditions (H1) and (H2) hold when $R_0 < 1$. In our ODE system,

we let $X_1 = (S, R), X_2 = (L, H)$ and $x_1^* = \left(\frac{\Lambda}{\mu}, 0\right)$.

Therefore, the system has its solution which can be found as:

$$\frac{dX_1}{dt} = F(X_1, X_2) = \begin{bmatrix} \Lambda - c\beta S(L + \alpha H + qR) - \mu S \\ \epsilon L + mH - (\theta + \mu)R \end{bmatrix}.$$

We have

$$\frac{dX_1}{dt} = F(X_1, 0) = \begin{bmatrix} \Lambda - c\beta SqR - \mu S \\ -(\theta + \mu)R \end{bmatrix}.$$

From the above, we have $\frac{dR}{dt} = -(\theta + \mu)R$ and $\frac{dS}{dt} = \Lambda - \mu S$. By integrating factor, we consider when $t \to \infty$, then $R(t) \to 0$ and $S(t) \to \frac{\Lambda}{\mu}$. This show that (H1) holds.

Thus, $x_1^* = \left(\frac{\Lambda}{\mu}, 0\right)$ is the globally asymptotically stable equilibrium point for the reduced system model equation $\frac{dX_1}{dt} = F(X_1, 0)$.

Next, we consider

$$\frac{dX_2}{dt} = G(X_1, X_2) = \begin{bmatrix} c\beta S(L + \alpha H + qR) + \rho H - (\epsilon + k + \mu + b)L \\ kL - (\rho + d + m + \mu)H \end{bmatrix}.$$

We have

$$\frac{dX_2}{dt} = G(X_1, 0) = \begin{bmatrix} c\beta SqR \\ 0 \end{bmatrix}.$$

When $q = 0$, we have

$$\frac{dX_2}{dt} = G(X_1, 0) = \begin{bmatrix} 0 \\ 0 \end{bmatrix}.$$

Then,

$$\frac{\partial G}{\partial t}(x_1^*, 0) = \begin{bmatrix} \frac{c\beta\Lambda}{\mu} - (\epsilon + k + \mu + b) & \frac{\alpha c\beta\Lambda}{\mu} + \rho \\ k & -(\rho + d + m + \mu) \end{bmatrix} = A.$$

This is an M-matrix with non-negatives off the diagonal elements.

Next, $\hat{G}(X_1, X_2) = AX_2 - G(X_1, X_2)$,

$$= \begin{bmatrix} c\beta S - (\epsilon + k + \mu + b) & \alpha c\beta S + \rho \\ k & -(\rho + d + m + \mu) \end{bmatrix} \begin{bmatrix} L \\ H \end{bmatrix} - \begin{bmatrix} c\beta S(L + \alpha H + qR) + \rho H - (\epsilon + k + \mu + b)L \\ kL - (\rho + d + m + \mu)H \end{bmatrix}$$

$$\hat{G}(X_1, X_2) = \begin{bmatrix} c\beta L\left(\frac{\Lambda}{\mu} - S\right) + c\beta\alpha\left(\frac{\Lambda}{\mu} - S\right)H - c\beta SqR \\ 0 \end{bmatrix}.$$

Since, $0 \le S \le \frac{\Lambda}{\mu}$, then $\hat{G}(X_1, X_2) \ge 0$ when $q = 0$. This show that (H2) holds.

By Lemma 1, we can conclude that the drug-free equilibrium point is globally asymptotically stable under these extreme circumstances.

6. Local stability of the drug endemic equilibrium point

Theorem 3 When $R_0 > 1$, the drug endemic equilibrium point (E_1) exists and is stable if it satisfies the Routh-Hurwitz criteria.

Proof Consider the Jacobian matrix of drug endemic equilibrium point, we have

$$J(E_1) = \begin{bmatrix} -c\beta(L^* + \alpha H^* + qR^*) - \mu & -c\beta S^* & -\alpha c\beta S^* & -qc\beta S^* \\ c\beta(L^* + \alpha H^* + qR^*) & c\beta S^* - (\epsilon + k + b + \mu) & \alpha c\beta S^* + \rho & qc\beta S^* \\ 0 & k & -(\rho + d + m + \mu) & 0 \\ \alpha & \epsilon & m & -(\theta + \mu) \end{bmatrix}.$$

By setting $det(J(E_1^*) - \lambda I) = 0$, we have $\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0,$ (9)

where

$a_1 = C_1 + \mu + \theta + \mu - (C_2 - C_3 - C_4),$

$a_2 = (C_1 + \mu)(\theta + \mu) - q\epsilon C_2 - (C_1 + \mu + \theta + \mu)(C_2 - C_3 - C_4) - (C_2 - C_3)C_4 - k(\alpha C_2 + \rho) + C_1C_2,$

$a_3 = (C_1 + \mu + \theta + \mu)\big(-(C_2 - C_3)C_4 - k(\alpha C_2 + \rho)\big) - \mu q\epsilon - qC_2(km + \epsilon C_4) - (C_1 + \mu)(\theta + \mu)(C_2 - C_3 - C_4) + (\theta + \mu)C_1C_2 + C_1C_2C_4 + \alpha kC_1C_2,$

$a_4 = (C_1 + \mu)(\theta + \mu)\big(-(C_2 - C_3)C_4 - k(\alpha C_2 + \rho)\big) - \mu qC_2(km + \epsilon C_4) + (\theta + \mu)C_1C_2C_4 + (\theta + \mu)\alpha kC_1C_2,$

when $C_1 = \beta(L^* + \alpha H^* + qR^*)$, $C_2 = c\beta S^*$, $C_3 = \epsilon + k + b + \mu, C_4 = \rho + d + m + \mu.$

The drug endemic equilibrium point is locally asymptotically stable corresponding to the Routh–Hurwitz stability criteria if $a_1 > 1, a_2 > 1, a_4 > 1$ and $a_1a_2a_3 > a_3^2 + a_1^2a_4$. This completes the proof.

7. Global stability of the drug endemic equilibrium point

In this section, the geometric approach of Li and Muldowney (1993) and Li and Muldowney (1996) is used to analyze the global stability of the drug endemic equilibrium point. The concept of the geometric approach of Li and Muldowney is briefly explained below. Consider the autonomous dynamical system

$$\dot{x} = f(x), \quad (10)$$

where $f: \Omega \to \mathbb{R}^n, \Omega \subset \mathbb{R}^n$ open set and $f \in C^1(\Omega)$.

The following assumptions are made: (H1) Ω is simply connected; (H2) There exists a compact absorbing set $\Gamma \subset \Omega$; (H3) $\bar{x}$ is a unique equilibrium point of (10) in Ω. Here is the result due to Li and Muldowney (Li and Muldowney, 1993; Li and Muldowney, 1996).

Theorem 4 Under the assumptions (H1), (H2) and (H3), the unique equilibrium point $\bar{x}$ of (10) is globally asymptotically stable int Ω provided the quantity $\bar{q}_2 < 0$, where $\bar{q}_2 = \limsup_{t\to\infty} \sup_{x_0\in\Gamma} \frac{1}{t}\int_0^t v(B(x(s, x_0)))ds$. The matrix B is defined as $B = Q_fQ^{-1} + QJ^{[2]}Q^{-1}$, where Q_f is obtained by replacing the entry Q_{ij} of Q by its derivative in the direction of the solution of f and $J^{[2]}$ is the second additive compound matrix of Jacobian J of the system (10). Further, the $v(B)$ is the Lozinskii measure with respect to a vector norm $\|\cdot\|$ in $\mathbb{R}^n$, and $v(B) = \lim_{h\to 0^+} \frac{\|I+hB\|-1}{h}$.

Lemma 2 The system (1) is uniformly persistent in int Ω when $R_0 > 1$.

Proof We see that when $R_0 < 1$ and $q = 0, E_0$ is globally asymptotically and when $R_0 > 1, E_0$ is unstable. By the result of Freedman, Ruan and Tang (1994), and Butler, Freedman and Waltman (1986), we conclude that E_0 is unstable when $R_0 > 1$ and hence the system is uniformly persistent in the interior of Ω i.e., there exists a constant $w > 0$

$$\liminf_{t\to\infty} S(t) > w, \liminf_{t\to\infty} L(t) > w, \liminf_{t\to\infty} H(t) > w, \liminf_{t\to\infty} R(t) > w, \quad (11)$$

provided $(S(0), L(0), H(0), R(0)) \in \Omega$. The uniform persistence together with the boundedness of Ω is equivalent to the existence of a compact set, which is absorbing for our model (1) in the interior of Ω.

Theorem 5 The drug endemic equilibrium point E_1 is globally asymptotically stable in int Ω when $R_0 > 1$ and when $\bar{b} > 0$ where $\bar{b}$ is defined in the proof.

Proof From the above, the assumption (H1)- (H3) holds.

The Jacobian matrix of (1) is

$$\begin{bmatrix} -c\beta(L + \alpha H + qR) - \mu & -c\beta S & -\alpha c\beta S & -qc\beta S \\ c\beta(L + \alpha H + qR) & c\beta S - (\epsilon + k + b + \mu) & \frac{\alpha c\beta\Lambda}{\mu} + \rho & \frac{qc\beta\Lambda}{\mu} \\ 0 & k & -(\rho + d + m + \mu) & 0 \\ \alpha & \epsilon & m & -(\theta + \mu) \end{bmatrix} \quad (12)$$

Its corresponding second compound matrix $J^{[2]}$ is given by,

$$J^{[2]} = \begin{bmatrix} c\beta S - c\beta(L + \alpha H + qR) - \mu - (\epsilon + k + b + \mu) & \alpha c\beta S + \rho & \alpha c\beta S \\ k & -c\beta(L + \alpha H + qR) - \mu - (\rho + d + m + \mu) & -c\beta S \\ 0 & c\beta(L + \alpha H + qR) & c\beta S - (\epsilon + k + b + \mu) - (\rho + d + m + \mu) \end{bmatrix}. \quad (13)$$

We let $Q = Q(S, L, H) = \text{diag}(1, \frac{L}{H}, \frac{L}{H})$. Then we have $Q_f Q^{-1} = \text{diag}(0, \frac{L'}{L} - \frac{H'}{H}, \frac{L'}{L} - \frac{H'}{H})$. Next, we determine $B = Q_f Q^{-1} + QJ^{[2]}Q^{-1}$, i.e., $B =$

$$\begin{bmatrix} c\beta S - c\beta(L + \alpha H + qR) - \mu - (\epsilon + k + b + \mu) & \frac{(\alpha c\beta S + \rho)H}{L} & \frac{\alpha c\beta SH}{L} \\ \frac{kL}{H} & -c\beta(L + \alpha H + qR) - \mu - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H} & -c\beta S \\ 0 & c\beta(L + \alpha H + qR) & c\beta S - (\epsilon + k + b + \mu) - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H} \end{bmatrix} = \begin{bmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{bmatrix},$$

Here $B_{11} = [[c\beta S - c\beta(L + \alpha H + qR) - \mu - (\epsilon + k + b + \mu)], B_{12} = \begin{bmatrix} \frac{(\alpha c\beta S + \rho)H}{L} & \frac{\alpha c\beta SH}{L} \end{bmatrix}$,

$B_{21} = \begin{bmatrix} \frac{kL}{H} \\ 0 \end{bmatrix}$ and $B_{22} =$

$$\begin{bmatrix} -c\beta(L + \alpha H + qR) - \mu - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H} & -c\beta S \\ c\beta(L + \alpha H + qR) & c\beta S - (\epsilon + k + b + \mu) - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H} \end{bmatrix}$$

The Lozinskii measure of matrix B is defined as $v(B) \leq max\{g_1, g_2\}$, (14)

where $g_1 = v(B_{11}) + \|B_{12}\|$ and $g_2 = \|B_{21}\| + v(B_{22})$.

One can easily compute that $v(B_{11}) = c\beta S - c\beta(L + \alpha H + qR) - \mu - (\epsilon + k + b + \mu)$, $\|B_{12}\| = \frac{\alpha c\beta SH}{L}, \|B_{21}\| = \frac{kL}{H}$, *and* $v(B_{22}) = max\left\{-\mu - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H}, 2c\beta S - (\epsilon + k + b + \mu) - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H}\right\}$.

Therefore, we have

$$g_1 = v(B_{11}) + \|B_{12}\| = c\beta S - c\beta(L + \alpha H + qR) - \mu - (\epsilon + k + b + \mu) + \frac{(\alpha c\beta S + \rho)H}{L}, \quad (15)$$

$$g_2 = \|B_{21}\| + v(B_{22}) = \frac{kL}{H} + max\left\{-\mu - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H}, 2c\beta S - (\epsilon + k + b + \mu) - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H}\right\}. \quad (16)$$

From the system (1), we have

$$\frac{dL}{dt} = c\beta S(L + \alpha H + qR) + \rho H - (\epsilon + k + \mu + b)L$$

$$\frac{L'}{L} = \frac{c\beta S(L + \alpha H + qR) + \rho H}{L} - (\epsilon + k + \mu + b)$$

$$\frac{(\alpha c\beta S + \rho)H}{L} = \frac{L'}{L} - \frac{c\beta S(L + qR)}{L} + (\epsilon + k + \mu + b)$$

and

$$\frac{dH}{dt} = kL - (\rho + d + m + \mu)$$

$$\frac{H'}{H} = \frac{kL}{H} - (\rho + d + m + \mu)$$

$$\frac{kL}{H} = \frac{H'}{H} + (\rho + d + m + \mu).$$

We consider

$g_1 = c\beta S - c\beta(L + \alpha H + qR) - \mu - (\epsilon + k + b + \mu) + \frac{L'}{L} + \frac{c\beta S(L+qR)}{L} - (\epsilon + k + \mu + b) =$
$-c\beta(L + \alpha H + qR) - \mu + \frac{L'}{L} + \frac{qc\beta SR}{L},$

and

$g_2 = \frac{H'}{H} - (\rho + d + m + \mu)H + max\left\{-\mu - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H}, 2c\beta S -\right.$
$\left.(\epsilon + k + b + \mu) - (\rho + d + m + \mu) + \frac{L'}{L} - \frac{H'}{H}\right\} = -\mu + \frac{L'}{L} + sup\{0, 2c\beta S - (\epsilon + k + b)\}.$

Therefore,

$v(B) \leq \max\{g_1, g_2\}$

$$= max\left\{-c\beta(L + \alpha H + qR) - \mu + \frac{L'}{L} - \frac{qc\beta SR}{L}, -\mu + \frac{L'}{L} + sup\{0, 2c\beta S - (\epsilon + k + b)\}\right\}$$

$$= \frac{L'}{L} + max\left\{-c\beta(L + \alpha H + qR) - \mu - \frac{qc\beta SR}{L}, -\mu + sup\{0, 2c\beta S - (\epsilon + k + b)\}\right\}$$

$$= \frac{L'}{L} - min\{c\beta(L + \alpha H + qR) + \mu + \frac{(\alpha c\beta S + \rho)H}{L}, \mu - inf\{0, -2c\beta S + (\epsilon + k + b)\}\}.$$

Hence, we obtain $v(B) \leq \frac{L'}{L} - \bar{b}$, where

$$\bar{b} = min\{c\beta(L + \alpha H + qR) + \mu + \frac{(\alpha c\beta S + \rho)H}{L}, \mu - inf\{0, -2c\beta S + (\epsilon + k + b)\}\}.$$

Let us consider any solution $S(t), L(t), H(t)$ emanating from the compact absorbing set $\Gamma \subset \Omega$. Let $\bar{t}$ be large enough such that the system is persistent and $(S(t), L(t), H(t)) \subset \Gamma$ for all $t \geq \bar{t}$. Then along each solution $S(t), L(t), H(t)$ such that $(S(0), L(0), H(0)) \in \Gamma$, for $t \geq \bar{t}$, $\frac{1}{t}[ln\, L(t) - L(0)] < \frac{\bar{b}}{2}$.

As a result,

$\overline{q_2} = \frac{1}{t}\int_0^t v(B)ds \leq \frac{1}{t}\int_0^t (\frac{L'}{L} - \bar{b})ds = \frac{1}{t}((ln\, L(t) - ln\, L(0)) - \bar{b}t) = \frac{ln\, L(t) - ln\, L(0)}{t} - \bar{b} < -\frac{\bar{b}}{2}$, which implies $\overline{q_2} \leq -\frac{\bar{b}}{2} < 0$.

Hence, by Theorem 4, (S, L, H) is globally asymptotically stable in $int\Omega$ when $R_0 > 1$ and $\bar{b} > 0$.

Next, consider the fourth equation of the system (1),

$$\frac{dR}{dt} = \varepsilon L + mH - (\theta + \mu)R,$$

and its limit system is

$$\frac{dR}{dt} = \varepsilon L^* + mH^* - (\theta + \mu)R,$$

since $\varepsilon L^* + mH^* = (\theta + \mu)R^*$, we get

$$\frac{dR}{dt} = (\theta + \mu)(R^* - R).$$

Therefore, by integration we have

$$\int_0^t \frac{1}{(R^* - R)} dR = dt \int_0^t (\theta + \mu)dt$$
$$R(t) = R^* - (R^* - R(0))e^{-(\theta+\mu)t}.$$

By $t \to \infty$, we have

$$\lim_{t\to\infty} R(t) = R^*$$

Therefore E_1 is globally asymptotically stable when $R_0 > 1$. This completes the proof.

Optimal Control Model

We next extend model (1) by applying optimal control problem in the model, to seek the possible intervention strategies that help reduce drug use. The optimal control model includes three control variables defined as (i) $u_1(t)$ is the awareness and educational program control, (ii) $u_2(t)$ is the family and friend care control, and (iii) $u_3(t)$ is the rehabilitation campaign control. The model is written as follows:

$$\frac{dS}{dt} = \Lambda - c\beta S(L + \alpha H + qR) - (\mu + u_1(t))S + u_2(t)L$$
$$\frac{dL}{dt} = c\beta S(L + \alpha H + qR) + \rho H - (\epsilon + k + \mu + b + u_2(t) + u_3(t))L$$
$$\frac{dH}{dt} = kL - (\rho + d + m + \mu + u_3(t))H$$
$$\frac{dR}{dt} = (\epsilon + u_3(t))L + (m + u_3(t))H - (\theta + \mu)R. \quad (17)$$

The control set U is Lebesgue measurable and it is defined as $U = \{(u_1(t), u_2(t), u_3(t)): 0 \le u_1(t) \le u_{1max}(t) \le 1{,}0 \le u_2(t) \le u_{2max}(t) \le 1{,}0 \le u_3(t) \le u_{3max}(t) \le 1{,}0 \le t \le T\}$. We aim to reduce the population number of light drug users and the population number of heavy drug users.

The model is analyzed based on the theory of Pontryagin, Boltyanskii, Gamkrelidze, and Mishchenko (1986). For the optimal control model, the objective of the model is given by:

$$J(u_1^*, u_2^*, u_3^*) = min \int_0^T \left(W_1 L(t) + W_2 H(t) + \frac{1}{2}\left(W_3 {u_1}^2(t) + W_4 {u_2}^2(t) + W_5 {u_3}^2(t)\right)\right) dt \quad (18)$$

where W_1 and W_2 are weight constants and the terms $W_3 {u_1}^2(t), W_4 {u_2}^2(t)$ and $W_5 {u_3}^2(t)$ represent the costs associated with awareness and educational program control, family and friend care control and rehabilitation campaign control, respectively.

Next, by applying Pontryagin's Minimum Principle (PMP), we give the necessary conditions for an optimal control problem. Therefore, we obtain a Hamiltonian (M) function defined as:

$$M = L(L, H, u_1, u_2, u_3) + \lambda_S \frac{dS}{dt} + \lambda_L \frac{dL}{dt} + \lambda_H \frac{dH}{dt} + \lambda_R \frac{dR}{dt}. \quad (19)$$

Thus, we obtain a Hamiltonian function as follows

$$M = W_1 L(t) + W_2 H(t) + \frac{1}{2}\left(W_3 {u_1}^2(t) + W_4 {u_2}^2(t) + W_5 {u_3}^2(t)\right)$$
$$+\lambda_S(\Lambda - c\beta S(L + \alpha H + qR) - (\mu + u_1(t))S + u_2(t)L)$$
$$+\lambda_L(c\beta S(L + \alpha H + qR) + \rho H - (\epsilon + k + \mu + b + u_2(t) + u_3(t))L)$$
$$+\lambda_H(kL - (\rho + d + m + \mu + u_3(t))H)$$
$$+\lambda_R(\epsilon + u_3(t))L + (m + u_3(t))H - (\theta + \mu)R) \quad (20)$$

where $\lambda_S, \lambda_l, \lambda_H$ and λ_R are the adjoint variable functions to be determined suitably by applying Pontryagin's Minimum Principle of optimal control.

For an optimal control set u_1, u_2, u_3 that minimizes J over U, there are adjoint variables, $\lambda_S, \lambda_l, \lambda_H$ and λ_R such that:

$$\lambda_S' = -\frac{\partial M}{\partial S} = -\left[\lambda_S c\beta(\tilde{L} + \alpha\tilde{H} + q\tilde{R}) - \lambda_S(\mu + u_1(t)) + \lambda_L c\beta(\tilde{L} + \alpha\tilde{H} + q\tilde{R})\right]$$
$$\lambda_L' = -\frac{\partial M}{\partial L} = -[W_1 - \lambda_S c\beta\tilde{S} + \lambda_S u_2(t) + \lambda_L(\epsilon + k + \mu + b + u_2(t) + u_3(t)) + \lambda_H k + \lambda_R(\epsilon + u_3(t))]$$
$$\lambda_H' = -\frac{\partial M}{\partial H} = -[W_2 - \lambda_S \alpha c\beta\tilde{S} + \lambda_L \alpha c\beta\tilde{S} + \lambda_L \rho + \lambda_H(\rho + d + m + \mu + u_3(t)) + \lambda_R(\epsilon + u_3(t))]$$
$$\lambda_R' = -\frac{\partial M}{\partial R} = -\left[-\lambda_S q c\beta\tilde{S} + \lambda_L q c\beta\tilde{S} - \lambda_R(\theta + \mu)\right], \quad (21)$$

where the transversality conditions are $\lambda_S(T) = 0, \lambda_L(T) = 0, \lambda_H(T) = 0, \lambda_R(T) = 0$.

By the approach of Pontryagin, Boltyanskii, Gamkrelidze, and Mishchenko (1986), we solve the equation, $\frac{\partial H}{\partial u_i} = 0$ at u_i^*, for $i = 1,2,3$ and obtain:

$$\frac{\partial M}{\partial u_1} = W_1 u_3(t) + \lambda_S \tilde{S} = 0 \Rightarrow u_1 = \frac{\lambda_S \tilde{S}}{W_3}$$

$$\frac{\partial M}{\partial u_2} = W_4 u_2(t) + \lambda_S \tilde{L} - \lambda_L \tilde{S} = 0 \Rightarrow u_2 = \frac{\lambda_L \tilde{S} - \lambda_S \tilde{L}}{W_4}$$

$$\frac{\partial M}{\partial u_3} = W_5 u_3(t) - \lambda_H \tilde{H} + \lambda_R \tilde{L} + \lambda_R \tilde{H} - \lambda_L \tilde{S} = 0 \Rightarrow u_3 = \frac{\lambda_H H - \lambda_L \tilde{L} - \lambda_L (\tilde{L} + \tilde{H})}{M_5}.$$

Thus, we obtain the control set (u_1^*, u_2^*, u_3^*) characterized by

$u_1^*(t) = max\{0, min(\frac{\lambda_S \tilde{S}}{W_3}, u_{1max})\}.$

$u_2^*(t) = max\{0, min(\frac{\lambda_L \tilde{S} - \lambda_S \tilde{L}}{W_4}, u_{2max})\}.$

$u_3^*(t) = max\{0, min(\frac{\lambda_H H - \lambda_L \tilde{L} - \lambda_L (\tilde{L} + \tilde{H})}{M_5}, u_{3max})\}.$

Numerical Simulation

Numerical simulations are performed for the optimal control model (17). This is first done by solving $\lambda_S, \lambda_l, \lambda_H$ and λ_R in (21) and solving u_i^*, for $i = 1,2,3$, then substitute these u_i^* in model (17). Next, the forward-backwards sweep method is used to solve the optimality system numerically. The parameters used within this model are shown in Table 1 and we consider the entire period of $T = 15$ years. Four strategies are studied to seek the best strategy in reducing the number of drug use.

Table 1 Parameter values of the model used in the numerical study

Parameter	Description	Value	Ref
Λ	The constant recruitment rate from the population	2	assume
β	The probability that there is contact between susceptible individuals in the population and both levels of drug users and drug users under rehabilitation results in initiation.	0.105	Niagarah and Nyabadza. 2013
θ	The rate of drug users under rehabilitation permanently quit	0.2	Niagarah and Nyabadza. 2013
μ	The natural death rate within the population	0.013	Islam and Biswas 2020
ε	The rate at which light drug users are recruited into rehabilitation	0.1	Mushayabasa and Tapedzesa 2015
m	The rate at which heavy drug users are recruited into rehabilitation	0.233	Niagarah and Nyabadza. 2013
k	The rate at which light drug users escalate to heavy drug users	0.56	Niagarah and Nyabadza. 2013
ρ	The rate at which heavy drug users move back to light drug users	0.4	Niagarah and Nyabadza. 2013
b	Light drug use induced death rate	0.035	Mushayabasa and Tapedzesa 2015

Table 1 (Cont.)

Parameter	Description	Value	Ref
d	Heavy drug use induced death rate	0.14	Mushayabasa and Tapedzesa 2015
c	The mean number of effective contacts between drug users and susceptible population	0.04	assume
α	The ability of heavy drug users that can initiate new drug users	0.1	Niagarah and Nyabadza. 2013
q	The ability of drug users under rehabilitation that can initiate new drug users	1	Niagarah and Nyabadza. 2013

1. Strategy I: control with awareness and educational program only

Under this strategy, we use the control u_1 to optimize the objective function while u_2 and u_3 are set to zero. Fig. 2(a) shows that the number of susceptible in the control case reduces faster than in the non-control case in the first 6 years after which they are on the same level and, towards the 15^{th} year, are slightly higher than in the uncontrolled cases. The population of light drug users is shown in Fig. 2(b), in the control case the peak of light drug users is about 38 people/year which is significantly lower than non-control case. The number of light drug users in the control case is less than in the non- control case throughout 15 years and reaches a lower equilibrium value. Fig. 2(c) shows that, in the control case, the population of heavy drug users is also significantly lower than in the non-control case throughout 15 years. Fig. 2(d) shows that the number of drug users under rehabilitation in the control case is also lower than in the non-control case throughout 15 years. Fig. 2(e) shows the strategy of u_1 that we need to give u_1 at 70% for about 6 years and 5 months and decreases gradually towards zero in the 15^{th} year.

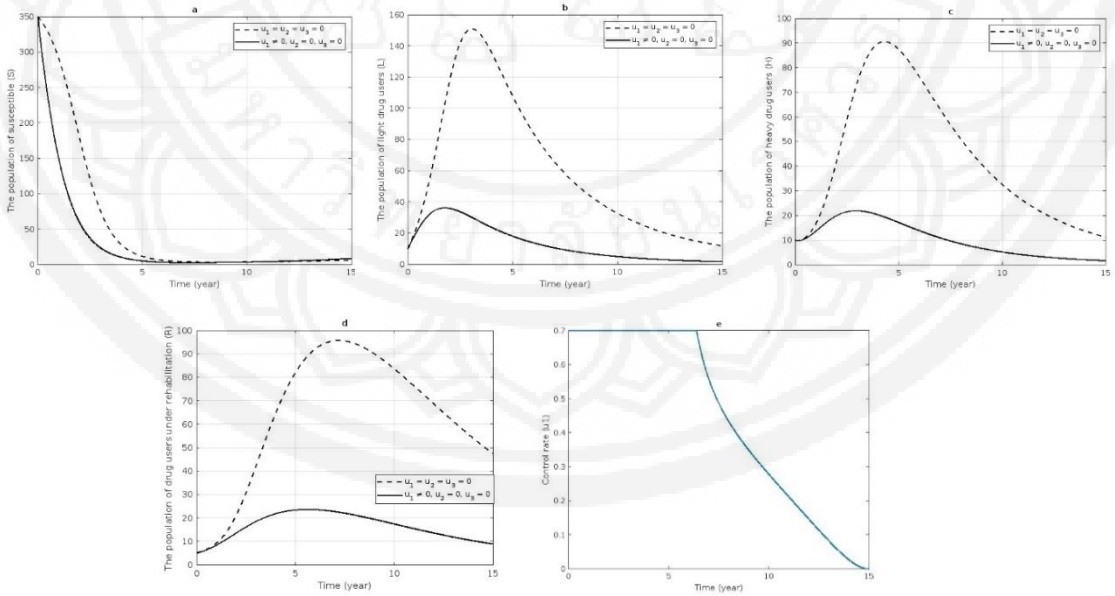

Figure 2 Numerical simulations of the optimal control model (17) with awareness and educational program control (u_1) only. (a) the population of susceptible, (b) the population of light drug users, (c) the population of heavy drug users, (d) the population of drug users under rehabilitation and (e) the strategic guideline of controls where $u_{1max} = 0.7, u_{2max} = 0, u_{3max} = 0$.

2 Strategy II: control with family and friend care only

Under this strategy, we use the control u_2 to optimize the objective function while u_1 and u_3 are set to zero. Fig. 3(a) shows that the population of susceptible individuals decreases at a significantly slower rate in the control case throughout 15 years and tends to reach a higher equilibrium value. Fig. 3(b) shows that the population of light drug users decreases in the control case with a peak of about 83 people/year, whereas it reaches a peak of more than 150 people/year in the non-control case. The time for the peak to occur in the control case is slightly slower than uncontrolled case. Similarly, Fig. 3(c) gives the same pattern as in Fig. 3(b) that in the control condition, the population of heavy drug users is lower than in the non-control case in the first 11 years and is a little higher than the uncontrolled case after that. Fig. 3(d) shows that the population of drug users under rehabilitation in the control case is also lower than in the non-control case throughout 15 years and in the control case the peak was increased to 64 people/year. It can be seen that a reduction of light drug users, heavy drug users and drug users under rehabilitation in the control case of Strategy I is lower than a reduction of them in Strategy II. Finally, Fig. 3(e) shows the strategy of u_2 that it has to be at the 70% for 14 years and 10 months and decreases sharply towards zero in the 15^{th} year.

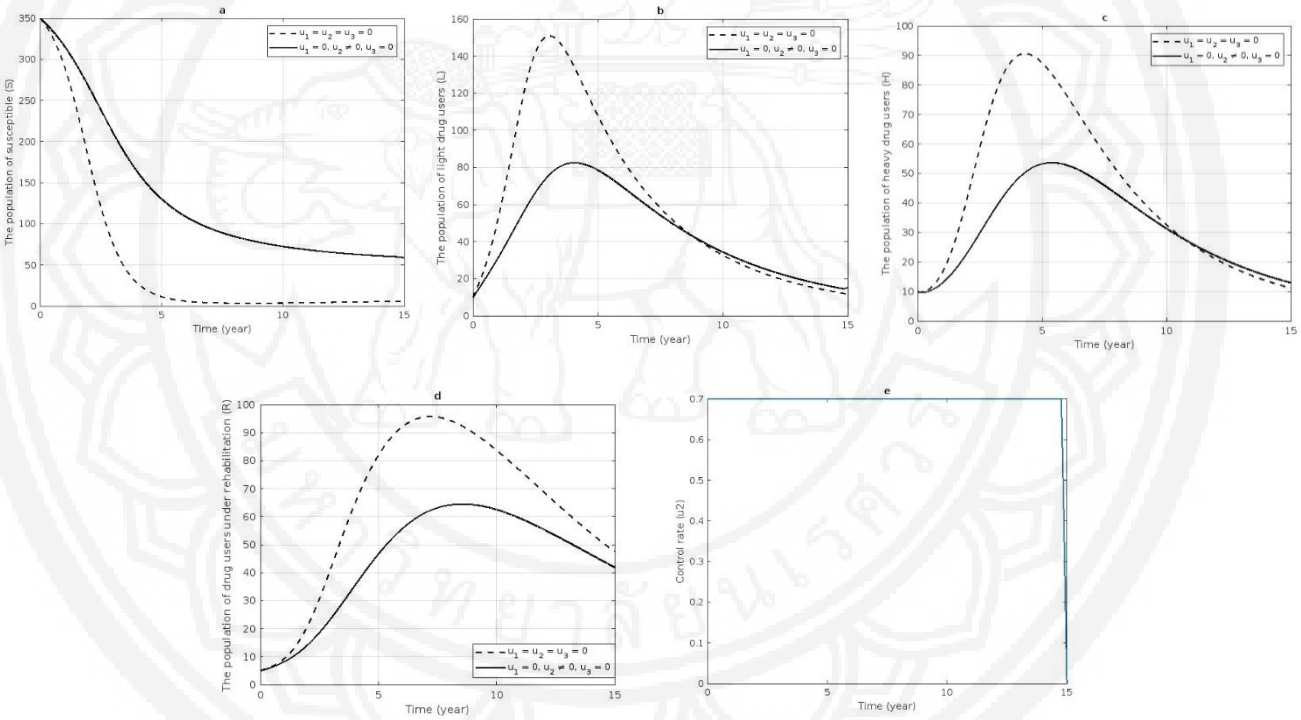

Figure 3 Numerical simulations of the optimal control model (17) with family and friend care control (u_2) only. (a) the population of susceptible, (b) the population of light drug users, (c) the population of heavy drug users, (d) the population of drug users under rehabilitation and (e) the strategic guideline of controls where $u_{1max} = 0, u_{2max} = 0.7, u_{3max} = 0$.

3. Strategy III: control with a rehabilitation campaign only

Under this strategy, we use the control u_3 to optimize the objective function while u_1 and u_2 are set to zero. Fig. 4(a) shows that the number of susceptible in the control case reduces more slowly than in the non-control case over 15 years. Interestingly, with control u_3 only, Fig.s 4(b) and Fig. 4(c) show that the number

of light drug users and heavy drug users significantly decreases and the time for the peak to occur in control is one year faster than in the non-control case. Fig. 4(d) shows that the number of drug users under rehabilitation in the control case is significantly greater than in the non-control case in the first 11 years and 6 months, whereas it then becomes lower than in the non-control case. Finally, Fig. 4(e) shows the strategy of u_3 has to be at the 70% for about 11 years and 3 months and gradually goes down to zero towards the 15^{th} year. These results demonstrate that rehabilitation campaign control could largely reduce the number of light drug users and significantly reduce the number of heavy drug users.

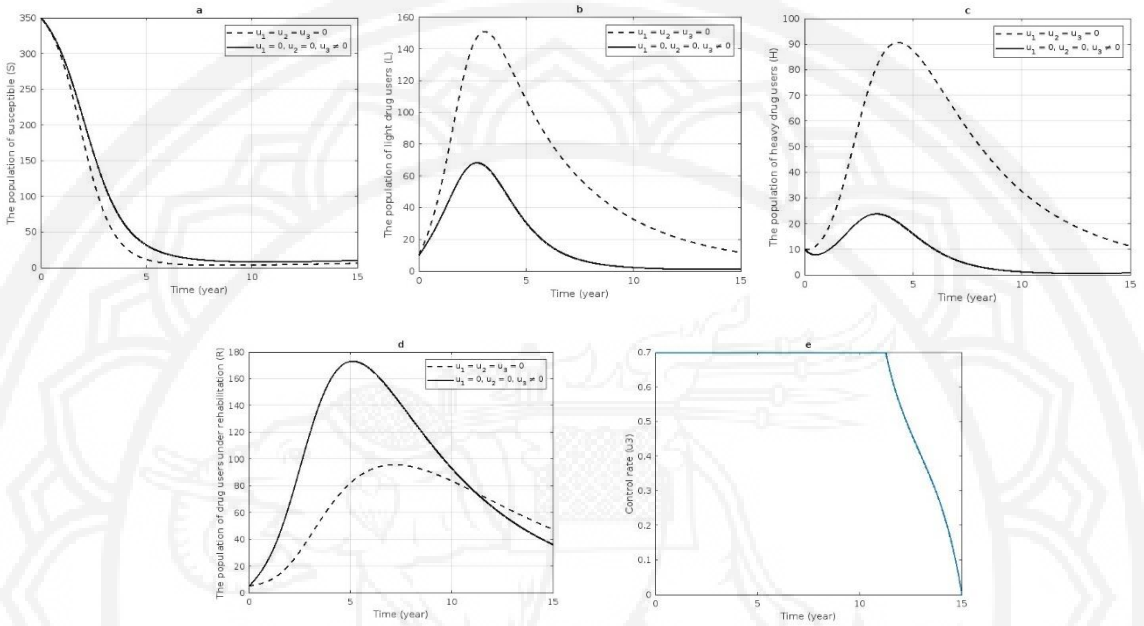

Figure 4 Numerical simulations of the optimal control model (17) with rehabilitation campaign control (u_3) only. (a) the population of susceptible, (b) the population of light drug users, (c) the population of heavy drug users, (d) the population of drug users under rehabilitation and (e) the strategic guideline of controls where $u_{1max} = 0, u_{2max} = 0, u_{3max} = 0.7$.

4. Strategy IV: control with awareness and educational program, family and friend care and rehabilitation campaign

Under this strategy, we use a combination of all three controls to optimize the objective function. Fig. 5(a) shows that the population of susceptible individuals also decreases slower in the control case over 15 years. Fig. 5(b) shows, in the control case, the number of light drug users is much lower than in the non-control case. It reaches zero in the 5^{th} year. Similarly, Fig. 5(c) shows, in the control case, that the number of heavy drug users is also clearly lower than in the non-control case. In both Fig.s 5(b) and (c), it can be seen that time for the peak to occur in the control case is faster than in the non-control case. Further, we showed that a reduction of both light drug users and heavy drug users in this strategy gives better results than Strategy I-III. Fig. 5(d) shows that the number of drug users under rehabilitation is larger than in the non-control case for the first 2 years and subsequently, it is below the number of drug users under rehabilitation in the non-control case. However, the number of drug users under rehabilitation overall in this strategy is lower than those in Fig. 5(d). Fig. 5(e) shows the strategy of u_1, that it has to be at the maximum rate of 70% for about just more than 4 years, then gradually drops towards zero in the 5^{th} year and dropped to the level of 4%, after that remains zero

towards the 15^{th} year. Fig. 5(f) shows the strategy of u_2 that it has to be at the 70% for about 4 years, then gradually drops towards zero in the 5^{th} year and increases slowly from the 5^{th} year to the level of about 1% on the 7^{th} year, then gradually drops towards zero in the 15^{th} year. Fig. 5(g) shows the strategy of u_3 that it has to be at the 70% for about 5 years and also dropped to the level of 3% and gradually goes down to zero in the 15^{th} year.

Overall, the results stated above are in line with the model reported in Islam and Biswas (2020) which we used in modified form as our model. Further, the results of Strategy IV in this study (a combination of all three controls), showed better results than those in the same strategy in the work of Islam and Biswas (2020) i.e., there are fewer light and heavy drug users in the control case in our study than in the work of Islam and Biswas (2020). This could be because of the different dynamics of control variables used.

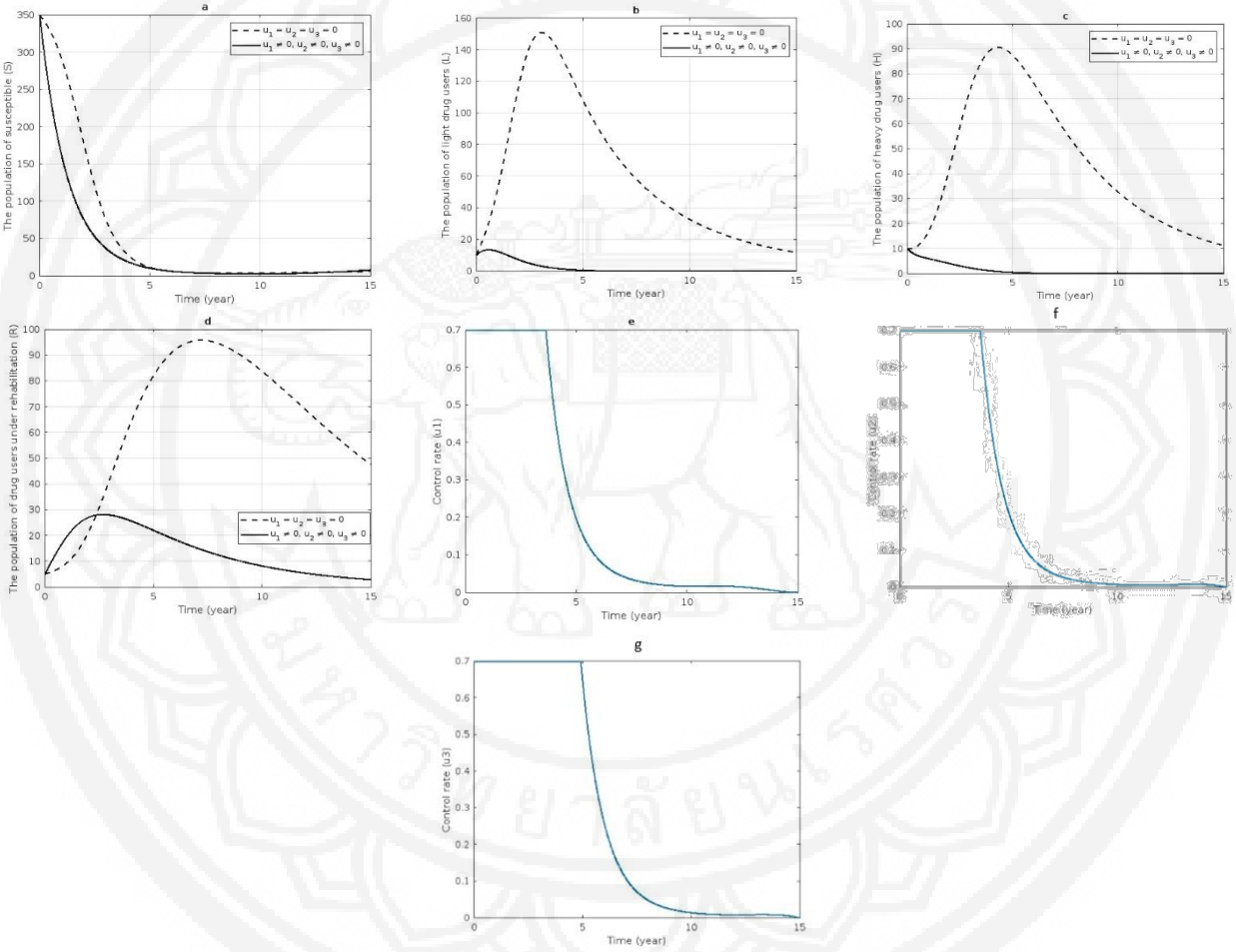

Figure 5 Numerical simulations of the optimal control model (17) with awareness and educational program control (u_1), family and friend care control (u_2) and rehabilitation campaign control (u_3). (a) the population of susceptible, (b) the population of light drug users, (c) the population of heavy drug users, (d) the population of drug users under rehabilitation and (e)–(g) the strategic guideline of controls u_1, u_2 and u_3, respectively where $u_{1max} = 0.7, u_{2max} = 0.7, u_{3max} = 0.7$.

Conclusions

In this model, a mathematical model of drug use is proposed to better understand the drug use dynamics. We are motivated by the work of Islam and Biswas (2020). The model comprises four subclasses within the population; susceptible individuals (S), light drug users (L), heavy drug users (H), and drug users under rehabilitation (R). We consider that new drug use can be initiated at a different rate by all three groups of drug users, light drug users, heavy drug users, and drug users under rehabilitation. Further, we assume that heavy drug users can revert to light drug users and finally both light and heavy drug users can be recruited for rehabilitation. In our model, we start by verifying that the solutions of the model are nonnegative and bounded. Two equilibrium points are obtained, and they are drug-free and drug-endemic. The basic reproduction number is calculated. From our theoretical results, we showed that the drug-free equilibrium point is globally stable when the basic reproduction number is less than one and when there is no initiation to new drug use by those who are in rehabilitation. Further, the drug-endemic persists when the basic reproduction number is greater than one and is globally stable when it meets certain conditions. In the later part of our paper, an optimal control problem is introduced into the model with three control variables; awareness and educational program control, family and friend care control, and rehabilitation campaign control. Numerical simulations of the optimal control model were performed, and the results showed the role of each control in helping to reduce the number of drug users by a certain amount. However, the best result in eventually reducing drug use was achieved with a combination of all three controls. Therefore, with the results from this study, all three of these controls should be encouraged in any society to lower the number of drug users overall. Further, more factors including drug sellers or drug lords as variables and the control by catching illicit drug users as optimal control could be added into the model for studying more realistically some specific interesting situations, and in society.

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