



Mathematical Analysis of Drugs and Substance Abuse in Kenya among the Adolescents

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Abstract

It is incontestable that the mortality rate among drugs and substance abusers is higher than that in the general population. The National Authority for the campaign against alcohol and substance abuse (NACADA) has painted a grim picture of the incessant rise in the number of youth becoming addicted. In this research, a deterministic model for drugs and substance abuse (DSA) driven by light drug abusers (LDA) and heavy drug abusers (HDA) was proposed. The basic reproduction number R_0 , the foundation upon which the model's stability analysis is established, was determined by utilizing the next-generation matrix (NGM) approach. The analysis showed that drug-free equilibrium (DFE) is locally asymptotically stable for $R_0 < 1$ and unstable if $R_0 > 1$. The global stability of both DFE and drugs endemic equilibrium (DEE) are explored by utilizing Lyapunov functions. The bifurcation analysis was carried out using the center manifold theorem, where the method utilized by Castillo-Chavez and Song was implemented and revealed that the rate of drug reinstitution drove backward bifurcation. The contribution of the important parameters to DSA are investigated, and results are presented graphically. Results from the simulation revealed that delayed exposure of the youth to drugs increased identification and treatment of the LDA and HDA, which would curtail DSA menace in Kenya.

Keywords: Drugs and substance abuse, Backward bifurcation, Stability and Numerical analysis.

1. Introduction

Substance abuse also known as drug abuse, is an influenced use of a drug where a user consumes a substance in amounts or with methods which are detrimental to themselves or to others [1]. DSA remains a problem globally with endless health consequences, high rates of suicide, crime and increased government spending. The NACADA reports have shown a significant increase in demand for drug abuse rehabilitation. In fact in the year 2015, there were 2.5 million people in need of rehab in Kenya.

In sub-Saharan Africa there are several studies that suggest a strong link between substance abuse and risky sex behaviour; such as having two or more sex partners, unprotected sex and engaging in commercial sex [2]. It is worth noting that addiction is a disease that requires multi-sectoral approach to deal with it effectively. Most studies on addiction reveal a similar pattern that start as a social activity before it mutates to severe mental illness or acute drugs dependency disorder.

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A research by [3], concluded that if one indulges in DSA at an early age, the likelihood of this habit continuing to adulthood is very high which ultimately leads to low productivity in the place of work and family breakups. In a 2011 report [4], NACADA declared that drugs and substance abuse was the major social problem in Kenya, with serious health ramification. NACADA estimates that half of all DSAs in Kenya to be between 10 and 19 years. According to the NACADA follow-up report [5], alcohol remains the most widely used substance of abuse in Kenya and the prevalence of cannabis use among youth aged (14-25) years has almost doubled over the last five years. This should be a wake-up call to the government to have concerted efforts toward reversing this trend. Other common drugs of abuse in Kenya include tobacco, khat, shisha and prescription drugs. It is thus indisputable that addiction of young adults robs the country of manpower to develop the nation, leads to increased dysfunctional families, risks of HIV infections and crimes, as well as premature deaths courtesy of increased road accidents and poisoning [6].

There is evidence that drugs and abuse spread like an infectious disease according to [7] and [8]. Hence, it can be formulated as a mathematical model. The infection and spreading of infectious diseases, similar to initiation into drugs and substance abuse spread, is a complex phenomenon with interacting factors, such as the environment in which individuals are situated. Whitey and Comiskey [9] modeled the heroin epidemics and treatment using ordinary differential equations in a similar way to modeling diseases. They aimed to identify parameters of interest for further study to inform and assist policy-makers in targeting prevention and treatment resources for maximum effectiveness. The results from this study revealed that prevention of drug initiation is better than treatment.

Alfiniyah et al. [10] presented a mathematical model of drugs and substance abuse reduction strategies that put into account the treatment type and risk level. In this research, the authors showed that anti-drug campaigns play a pivotal role as far as reduction in the number of DSAs is concerned, irrespective of whether one is under an outpatient or inpatient treatment scheme.

Sharma and Samanta [11], developed a mathematical model of alcohol abuse that has four compartments: moderate and occasions drinkers, heavy drinkers, drinkers in treatment and temporarily recovered class. Their aim was to develop alcohol abuse model by introducing a treatment program in the population and considering a possible relapse. The numerical findings were illustrated through computer simulations which indicate that the optimal control is efficient to reduce the spread of alcoholism.

Burattini et al. [12] modelled the dynamics of smoking of crack-cocaine (same mode of transmission is related to that of drug use). The structure of their model adapted from SIR model structure and they assumed that the population is divided into four classes namely, susceptibles, injecting drug users, crack-cocaine users and users of both crack-cocaine and injecting drugs. Their results suggested that the impact of the introduction of crack-cocaine use on the prevalence of HIV/AIDS depends on several factors and could result on the complex demographic interactions of dynamic system in the population of drug users and its relationship with the HIV/AIDS epidemic.

Nyabadza et al. [13] modelled the dynamics of crystal meth ('tik') abuse in the presence of drug-supply chains in South Africa. They considered a model for 'tik' use that accounts for rehabilitation, tracks drug-supply chains and amelioration for the addicted.

They considered both slow and fast dynamics in their model that were driven by drugs in the population and community respectively. Sensitivity analysis revealed that parameters with the most control over the epidemic are the quitting rate of light-drug users and the person-to-person contact rate between susceptible individuals and ‘tik’ users.

Our proposed model mirrors that studied by [11] but with inclusion of the exposed and mentally ill or acute drugs dependency compartments. Furthermore, this paper presents a modification of the model by Sharma et al. by considering the effects of re-initiation of DSA quitters as well as the impact of delayed initiation of the susceptible to drugs in the long term DSA dynamics.

This paper is organised as follows: We formulate deterministic model capturing the DSAs initiation dynamics. The section on model analysis encompasses the model’s well-posedness and the invariant region, derivation of equilibria, the reproduction number and the bifurcation analysis. In the numerical simulation section, projections on variation of paramount parameters are displayed.

2. Model formulation

A deterministic compartmental model with a bi-linear incidence rate that describes DSA initiation at any time t is developed and analyzed in this study. The whole population $N(t)$ is segmented into six (6) mutually exclusive compartments S , D_E , D_L , D_H , D_M and R which respectively represent the susceptible, exposed, light drugs abusers(LDAs), heavy drugs abusers(HDAs), mentally ill(MDAs) and Recovered individuals. The individuals get into the susceptible population at rate π . The recovered individuals are assumed to get re-initiated into drugs at the rate ω . The susceptible individuals progress to the exposed phase upon interaction with the light and heavy drugs abusers. The force of infection λ assumes the bilinear incidence and is expressed as:

$$\lambda = \beta(1 - \sigma_1)(D_L + \varphi D_H)$$

The exposed class exit this compartment at a rate k where a fraction r progress to HDA compartment while the fraction $(1 - r)$ become light drugs abusers (LDA). The parameter σ accounts for the rate of exit from LDA compartment where a fraction p of the youth exit drugs after having successfully been taught and counselled on the negative effects of DSA. Thus p accounts for efficacy of educational campaign against DSA. Besides natural death rate μ , the heavy drugs abusers(HDA) and persons with mental disorder are diminished by drugs induced death at the rates δ_1 and δ_2 respectively. The parameter γ_1 accounts for the successful treatment rate of the HDA while γ_2 represent the rate at which the HDA becomes mentally ill. Thus it follows that our model consist of the following system of non-linear differential equations:

$$\begin{cases} \frac{dS}{dt} = \pi + \omega R - \beta(1 - \sigma_1)(D_L + \varphi D_H)S - \mu S \\ \frac{dD_E}{dt} = \beta(1 - \sigma_1)(D_L + \varphi D_H)S - (k + \mu)D_E \\ \frac{dD_L}{dt} = (1 - r)kD_E - (\mu + \sigma)D_L \\ \frac{dD_H}{dt} = rkD_E + (1 - p)\sigma D_L - (\mu + \delta_1 + \gamma_1 + \gamma_2)D_H \\ \frac{dD_M}{dt} = \gamma_2 D_H - (\mu + \delta_2 + \varepsilon)D_M \\ \frac{dR}{dt} = \sigma p D_L + \gamma_1 D_H + \varepsilon D_M - (\mu + \omega)R \end{cases} \quad (2.1)$$

with initial conditions given by: $S(0) = S_0 \geq 0, D_E(0) = D_{E0} \geq 0, D_L(0) = D_{L0} \geq 0, D_H(0) = D_{H0} \geq 0, D_M(0) = D_{M0} \geq 0, R(0) = R_0 \geq 0$.

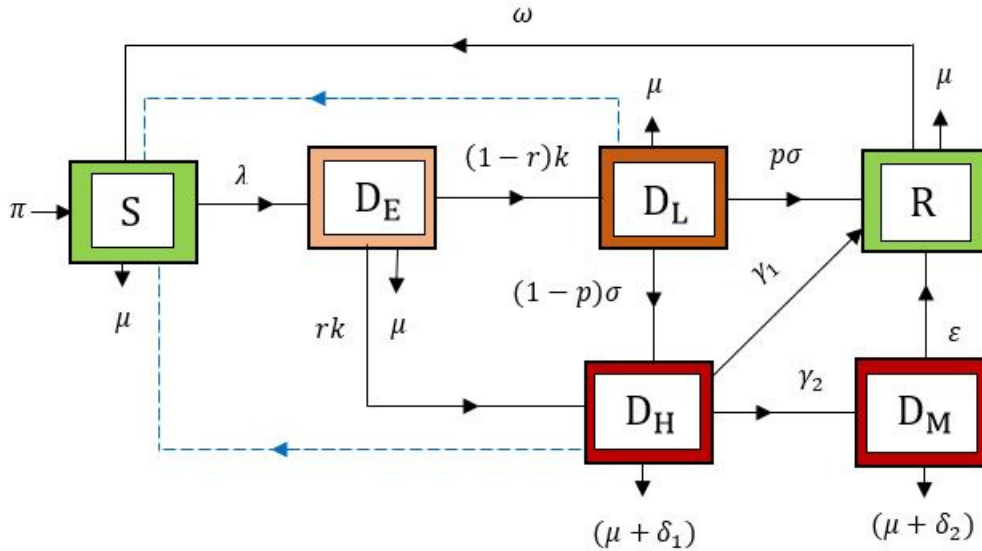


Figure 1: General drugs and substance flow model (2.1).

Table 1: Definition of state variables

Variable	Definition
S	Human population susceptible to Drugs and substance abuse
D_E	Individuals just initiated into drugs abuse
D_L	Light or moderate drugs users
D_H	Heavy drugs users
D_M	Mentally ill individuals
R	Recovered individuals

2.1. Model analysis

In this section, basic properties of the proposed model which include positivity of solution, feasible region, model’s equilibrium points as well as stability of the equilibrium points are discussed. In carrying out the model analysis, we consider a case where σ_1 in model (2.1) is zero.

2.2. Positivity and boundedness

Since the model in consideration entails human population, it is paramount for model (2.1) to be epidemiologically meaningful. We thus show that all state variables involved are non-negative for all time $t > 0$ and that the region.

$$\Omega = \{(S, D_E, D_L, D_H, D_M, R) \in \mathbb{R}_+^6 : S + D_E + D_L + D_H + D_M + R \leq N(0)\}$$

is bounded. We thus state explicitly the following theorem.

Table 2: Definition of model parameters

Variable	Definition
π	Recruitment rate of susceptible into the population
σ_1	DSA initiation control coefficient.
μ	Natural death rate
β	Drugs and substance abuse transmission rate.
φ	Modification factor for heavy drugs users.
r	Rate at which the exposed persons who become heavy drugs users.
k	Rate of exposed persons become drug users.
p	Recovery rate of the light drugs users.
σ	Rate at which individuals exit moderate drugs users compartment.
δ_1	Drugs induced death rate for heavy drugs users.
δ_2	Drugs induced death rate for the mentally ill persons.
ω	Rate at which the recovered drugs users become susceptible.
γ_1	Rate of recovery of heavy drugs users.
γ_2	Rate of movement from heavy drugs users to mentally ill persons.
ε	recovery of the mentally ill persons.

Theorem 2.1. *If $S(0) \geq 0, D_E(0) \geq 0, D_L(0) \geq 0, D_H(0) \geq 0, D_M(0) \geq 0$ and $R \geq 0$, then the solution $S \geq 0, D_E(0) \geq 0, D_L(0) \geq 0, D_H(0) \geq 0, D_M(0) \geq 0$ and $R \geq 0$ of model (2.1) are positive for all $t > 0$.*

Proof. According to Zenebe and Legese [14], Asha and Nyimvua [15], and Rabiou et.al. [16], we proof the positivity of model (2.1) by contradiction. Given the non-negative initial conditions for $S(0), D_E(0), D_L(0), D_H(0), D_M(0)$ and $R(0)$, the positivity of the system can be determined as follows: We make an assumption that there exists time t_i such that, $S > 0, S(t_i) = 0, S'(t_i) < 0, D_E(t) > 0, D_L(t) > 0, D_H(t) > 0, D_M(t) > 0, R(t) > 0$, for all $0 \leq t < t_i$.

In relation to our case and considering the first equation of the model (2.1), we have:

$$\frac{dS}{dt}(t_i) = \pi + \omega R(t_i) - \beta(D_L + \varphi D_H)S(t_i)$$

Based on our assumption, this equation implies that:

$$\frac{dS}{dt}(t_i) = \pi + \omega R(t_i)(t_i) > 0$$

which is a contradiction. This indicates that $S(t) > 0$ for all $t > 0$.

Based on the second equation of model (2.1),

$$\frac{dD_E}{dt} = \beta(D_L + \varphi D_H)S - (\mu + k)D_E \geq -(\mu + k)D_E$$

owing to the fact that $S(t)$ is non-negative for all $t > 0$. Solving this equation gives:

$$D_E(t) = D_E(0) \exp(-(\mu + k)t) \geq 0 \quad \forall t > 0$$

In the similar manner, we solve the remaining equations to get:

$$\begin{aligned} D_L(t) &= D_L(0) \exp(-(\mu + \sigma)t) \geq 0 \quad \forall t > 0 \\ D_H(t) &= D_H(0) \exp(-(\mu + \delta_1 + \gamma_1 + \gamma_2)t) \geq 0 \quad \forall t > 0 \\ D_M(t) &= D_M(0) \exp(-(\mu + \delta_2 + \varepsilon)t) \geq 0 \quad \forall t > 0 \\ R(t) &= R(0) \exp(-(\mu + \omega)R)t \geq 0 \quad \forall t > 0 \end{aligned}$$

This demonstrates that the solution of all state variables of the model (2.1) are non-negative. \square

Invariant region

A population is said to be meaningful in biological sense if its global solution lies within an invariant region Ω [17, 18].

Theorem 2.2. *The solution set*

$\{S(t), D_E(t), D_L(t), D_H(t), D_M(t), R(t)\}$ of the model (2.1) is confined to non-negative feasible region Ω .

Proof. Consider the feasible region:

$\Omega = \{S(t), D_E(t), D_L(t), D_H(t), D_M(t), R(t)\} \in \mathbb{R}_+^6 \quad \forall t \geq 0$. At any given time, model (2.1) gives the total population as:

$$N(t) = S(t) + D_E(t) + D_L(t) + D_H(t) + D_M(t) + R(t) \quad (2.2)$$

Differentiating equation (2.2) with respect to t gives:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dD_E}{dt} + \frac{dD_L}{dt} + \frac{dD_H}{dt} + \frac{dD_M}{dt} + \frac{dR}{dt} \quad (2.3)$$

We now substitute model (2.1) into (2.3) and further simplification gives:

$$\frac{dN}{dt} = \pi - \mu N - (\delta_1 D_H + \delta_2 D_M) \leq \pi - \mu N \quad (2.4)$$

Evaluation of the equation (2.4) yields $N(t) \leq A \exp(-\mu t) + \frac{\pi}{\mu}$ which upon application of initial conditions, the above equation becomes:

$$N(t) \leq \frac{\pi}{\mu} + \left(\frac{\pi - \mu N_0}{\mu} \right) \exp(-\mu t) \quad (2.5)$$

where $N_0 = N(0)$

This implies that:

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\pi}{\mu}$$

This shows that the positive solutions of the model (2.1) are bounded. Hence, the system under consideration is well posed mathematically and epidemiologically. \square

2.3. Drugs free equilibrium

The drugs-free equilibrium (DFE) denoted as B^0 is a steady-state solution in which there is no individuals engaged drugs and substance abuse in the community. Consequently, apart from the susceptible, all other compartments are equated to zero. Thus $D_E = D_L = D_H = D_M = R = 0$ and $\frac{dS^0}{dt} = 0$. The model (2.1) reduces to:

$$S^0 = \frac{\pi}{\mu}$$

Thus the DFE state, $(S^0, D_E^0, D_L^0, D_H^0, D_M^0, R^0)$ is given by:

$$B^0 = \left[\frac{\pi}{\mu}, 0, 0, 0, 0, 0 \right]$$

2.4. The basic reproduction number

The basic reproduction number is the number of cases that one drugs abuser generates on average over entire drugs abuser period in an uninfected population [19]. We utilize the next generation matrix method [20] in the derivation of R_0 for our dynamical system. In this method, the infected compartments are decomposed into two matrices F and V , where F represents the matrix containing the new drugs abuser recruits and V is the matrix containing elements with transmission terms. The model (2.1) can be rewritten as:

$$\frac{d\chi}{dt} = F(\chi) - V(\chi) \tag{2.6}$$

More explicitly, the above equation can be written as:

$$\begin{pmatrix} \dot{D}_E \\ \dot{D}_L \\ \dot{D}_H \\ \dot{D}_M \end{pmatrix} = \begin{pmatrix} \beta(D_L + \varphi D_H)S \\ 0 \\ 0 \\ 0 \end{pmatrix} - \begin{pmatrix} (\mu + \omega)D_E \\ -(1-r)kD_E + (\mu + \sigma)D_L \\ -(1-p)\sigma D_L + (\mu + \delta_1 + \gamma_1 + \gamma_2)D_H \\ -\gamma_2 D_H + (\mu + \delta_2 + \varepsilon)D_M \end{pmatrix}$$

Evaluation of the Jacobian of matrices F and V at the B_0 gives;

$$\mathcal{F} = \begin{pmatrix} 0 & \frac{\beta\pi}{\mu} & \frac{\beta\pi\varphi}{\mu} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$\mathcal{V} = \begin{pmatrix} (\mu + k) & 0 & 0 & 0 \\ -\kappa(1-r) & (\mu + \sigma) & 0 & 0 \\ -kr & -\sigma(1-\kappa) & (\mu + \gamma_1 + \gamma_2 + \delta_1) & 0 \\ 0 & 0 & -\gamma_2 & (\mu + \delta_2 + \varepsilon) \end{pmatrix}$$

The inverse of the above matrix gives:

$$\mathcal{V}^{-1} = \begin{pmatrix} \frac{1}{\Phi_1} & 0 & 0 & 0 \\ \frac{k(1-r)}{\Phi_1\Phi_2} & \frac{1}{\Phi_2} & 0 & 0 \\ \frac{k\sigma(1-r)(1-p)}{\Phi_1\Phi_2\Phi_3} & \frac{\sigma(1-p)}{\Phi_2\Phi_3} & \frac{1}{\Phi_3} & 0 \\ \frac{\gamma_2 k\sigma(1-r)(1-p)}{\Phi_1\Phi_2\Phi_3\Phi_4} & \frac{\gamma_2\sigma(1-p)}{\Phi_2\Phi_3\Phi_4} & \frac{\gamma_2}{\Phi_2\Phi_3} & \frac{1}{\Phi_4} \end{pmatrix} \tag{2.7}$$

Where $\Phi_1 = (\mu + k)$, $\Phi_2 = (\mu + \sigma)$, $\Phi_3 = (\mu + \delta_1 + \gamma_1 + \gamma_2)$ and $\Phi_4 = (\mu + \delta_2 + \varepsilon)$
 The basic reproduction number is the spectral radius (The largest eigenvalue) of the next generation matrix $R_0 = \rho(\mathcal{F}\mathcal{V}^{-1})$. This computation gives:

$$\mathcal{F}\mathcal{V}^{-1} = \begin{pmatrix} \Psi_{11} & \Psi_{12} & \Psi_{13} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

Where:

$$\begin{aligned}\Psi_{11} &= \frac{\pi\beta k\{\varphi[(r\mu + r\rho\sigma) + \sigma(1-p)] + (1-r)(\mu + \sigma_1 + \gamma_1 + \gamma_2)\}}{\mu(\mu + k)(\mu + \sigma)(\mu + \delta_1 + \gamma_1 + \gamma_2)} \\ \Psi_{12} &= \frac{\pi\beta\{\varphi\sigma(1-p) + (\mu + \sigma_1 + \gamma_1 + \gamma_2)\}}{\mu(\mu + \sigma)(\mu + \delta_1 + \gamma_1 + \gamma_2)} \\ \Psi_{13} &= \frac{\pi\beta\varphi}{\mu(\mu + \delta_1 + \gamma_1 + \gamma_2)}\end{aligned}$$

The basic reproduction number plays a pivotal role when analyzing any epidemiological model. The effective reproduction of our dynamical system is thus:

$$R_0 = \frac{\pi\beta k\{\varphi[(r\mu + r\rho\sigma) + \sigma(1-p)] + (1-r)(\mu + \sigma_1 + \gamma_1 + \gamma_2)\}}{\mu(\mu + k)(\mu + \sigma)(\mu + \delta_1 + \gamma_1 + \gamma_2)} \quad (2.8)$$

For clear interpretation of the above reproduction number, we can separate this expression of R_0 as follows:

$$R_0 = R_1 + R_2$$

Where

$$R_1 = \frac{\pi\beta k\varphi\{(r\mu + r\rho\sigma) + \sigma(1-p)\}}{\mu(\mu + k)(\mu + \sigma)(\mu + \delta_1 + \gamma_1 + \gamma_2)} \quad (2.9)$$

R_1 represents the probability of the total population becoming heavy drugs abusers upon exposition times the mean exposed, light and heavy drugs users.

$$R_2 = \frac{\pi\beta k(1-r)}{\mu(\mu + k)(\mu + \sigma)} \quad (2.10)$$

R_2 represents the probability of the total population becoming light drugs users upon exposition multiplied by mean exposed and moderate drugs users population.

The analytically generated R_0 from our model thus points to the fact that the presence of both light and heavy drugs users could drive the growth of drugs and substance abuse in the general population.

2.5. The stability of drugs free equilibrium (DFE)

In this section, we explore the local and global stability of model (2.1) at DFE by utilizing the following theorems:

Theorem 2.3. *The disease-free state, B_0 , of the model (2.1) is locally asymptotically stable(LAS) when $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. The local stability of the model is determined by first evaluating the Jacobian matrix at DFE state B_0 which gives:

$$\begin{pmatrix} -\mu & 0 & -\frac{\beta\pi}{\mu} & -\frac{\beta\pi\varphi}{\mu} & -\frac{\beta\pi\varphi}{\mu} & \omega \\ 0 & -(\mu + k) & \frac{\beta\pi}{\mu} & \frac{\beta\pi\varphi}{\mu} & 0 & 0 \\ 0 & (1-r)k & -(\mu + \sigma) & 0 & 0 & 0 \\ 0 & rk & (1-p)\sigma & -(\mu + \delta_1 + \gamma_1 + \gamma_2) & 0 & 0 \\ 0 & 0 & 0 & \gamma_2 & -(\mu + \delta_2 + \varepsilon) & 0 \\ 0 & 0 & \sigma & \gamma_1 & \varepsilon & -(\mu + \omega) \end{pmatrix} \quad (2.11)$$

From the Jacobian matrix equation (2.11), we make use of the trace-determinant method so as to proof the local stability of B_0 . For local asymptotic stability, the following Routh-Hurwitz conditions have to be satisfied:

- (i) $\text{Tr}J(B_0) < 0$
- (ii) $\text{Det}J(B_0) > 0$

Thus:

$$\text{Tr}J(B_0) = -(6\mu + k + \sigma + \delta_1 + \delta_2 + \gamma_1 + \gamma_2 + \varepsilon) < 0 \quad (2.12)$$

Due to the complexity of the matrix space involved, we utilize python programming language to determine the determinant of $J(B_0)$. This gives:

$$\left\{ \begin{aligned} \text{Det}J(B_0) = & -(\mu + \omega)(\mu + \delta_2 + \varepsilon)\{-\beta k\pi[(1-r)(\mu + \delta_1 + \gamma_1 + \gamma_2) + \varphi(r\mu + r\rho\sigma \\ & + (1-p)\sigma)] - \mu(\mu + k)(\mu + \sigma)(\mu + \delta_1 + \gamma_1 + \gamma_2)\} \end{aligned} \right. \quad (2.13)$$

Through further simplification and utilization of equation (2.8), equation (2.13) becomes:

$$\text{Det}J(B_0) = \mu(\mu + k)(\mu + \sigma)(\mu + \omega)(\mu + \delta_1 + \gamma_1 + \gamma_2)(\mu + \delta_2 + \varepsilon)(1 - R_0) \quad (2.14)$$

Where R_0 is the effective reproduction number.

For determinant $\text{Det}J(B_0) > 0$, it follows that $R_0 < 1$. Hence the drugs-free equilibrium (B_0) of model (2.1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. \square

The implication of the above theorem is that the extinction drugs and substance abusers (DSA) in population is possible for $R_0 < 1$, but if $R_0 > 1$, the ballooning of drugs abusers in the population is assured.

For the next theorem, we investigate the global stability of DFE by using Lyapunov function technique [21].

Theorem 2.4. *The drugs-free equilibrium point B_0 of model (2.1) is globally asymptotically stable in the region Ω if $R_0 \leq 1$ and unstable if $R_0 > 1$.*

Proof. Let L be a Lyapunov function with positive constants c_1, c_2, c_3 and c_4 such that:

$$\left\{ L = (S_h - S^0 - S^0 \ln \frac{S}{S^0}) + c_1 D_E + c_2 D_L + c_3 D_H + c_4 D_M + c_5 R \quad (2.15) \right.$$

Differentiating the above Lyapunov equation with respect to time gives:

$$\frac{dL}{dt} = (1 - \frac{S^0}{S}) \frac{dS}{dt} + c_1 \frac{dD_E}{dt} + c_2 \frac{dD_L}{dt} + c_3 \frac{dD_H}{dt} + c_4 \frac{dD_M}{dt} + c_5 \frac{dR}{dt} \quad (2.16)$$

Substituting model (2.1) into (2.16) yields:

$$\left\{ \begin{aligned} \frac{dL}{dt} = & (1 - \frac{S^0}{S})[\pi + \omega R - \beta(D_L + \varphi D_H)S - \mu S] \\ & + c_1[\beta(D_L + \varphi D_H)S - (\mu + k)D_E] \\ & + c_2[(1-r)kD_E - (\mu + \sigma)D_L] \\ & + c_3[(1-p)\sigma D_L + rkD_E - (\mu + \delta_1 + \gamma_1 + \gamma_2)D_H] \\ & + c_4[\gamma_2 D_H - (\mu + \delta_2 + \varepsilon)D_M] \\ & + c_5[p\sigma D_L + \gamma_1 D_H + \varepsilon D_H - (\mu + \omega)R] \end{aligned} \right. \quad (2.17)$$

Suppose $S \leq S^0 = \frac{\pi}{\mu}$ equation (2.17) can be expressed as:

$$\begin{cases} \frac{dL}{dt} \leq [-(\mu + k)c_1 + k(1 - r)c_2 + rk c_3]D_E \\ \quad + [\frac{\pi\beta}{\mu}c_1 - (\mu + \sigma)c_2 + (1 - p)\sigma c_3 + p\sigma c_5]D_L \\ \quad + [\frac{\pi\beta\varphi}{\mu}c_1 - (\mu + \delta_1 + \gamma_1 + \gamma_2)c_3 + \gamma_2 c_4 + \gamma_1 c_5]D_H \\ \quad + [-(\mu + \delta_2 + \varepsilon)c_4 + \varepsilon c_5]D_M + [-(\mu + \omega)]R \end{cases} \quad (2.18)$$

Equating the coefficients of D_E , D_L , D_H , D_M and R to zero gives:

$$\begin{aligned} c_1 &= (\mu + \delta_1 + \gamma_1 + \gamma_2), \quad c_2 = \frac{\pi\beta\{(\mu + \delta_1 + \gamma_1 + \gamma_2) + (1 - p)\sigma\varphi\}}{\mu(\mu + \sigma)} \\ c_3 &= \frac{\pi\beta\varphi}{\mu}, \quad c_4 = c_5 = 0 \end{aligned}$$

Upon simplification and substitution of equation (2.8), equation (2.18) becomes:

$$\frac{dL}{dt} \leq (\mu + k)(\mu + \delta_1 + \gamma_1 + \gamma_2)(R_0 - 1)D_E \quad (2.19)$$

Its worth noting that: $\frac{dL}{dt} \leq 0$, if $R_0 \leq 1$ and $\frac{dL}{dt} = 0$ iff $D_E = 0$. Thus, plugging $D_E = D_L = D_H = D_M = R = 0$ into model (2.1) points to the fact that $S(t) \rightarrow \frac{\pi}{\mu}$ as $t \rightarrow \infty$. Hence the largest compact invariant set in $\{(S, D_E, D_L, D_H, D_M, R) \in \Omega; \frac{dL}{dt} \leq 0\}$, is the singleton set B_0 . Thus from LaSalle's invariance principle [22], we make the conclusion that the drugs free equilibrium point is globally asymptotically stable in Ω if $R_0 < 1$. The explanation above points to the fact that DSA can be abased in the general population if and only if $R_0 < 1$. \square

2.6. Existence of an endemic equilibrium

We endeavor to investigate the existence of endemic equilibrium, B_e of the model (2.1) in this subsection.

Theorem 2.5. *If $R_0 > 1$, there exists a unique endemic equilibrium*

$B_e = (S^*, D_E^*, D_L^*, D_H^*, D_M^*, R^*)$ of the model (2.1). *The endemic equilibrium does not exist for $R_0 < 1$.*

Proof. For the existence of endemic equilibrium, we equate the right side of the model (2.1) to zero and then solve.

$$\begin{cases} \pi + \omega R^* - \beta(D_L^* + \varphi D_H^*)S^* - \mu S^* = 0 \\ \beta(D_L^* + \varphi D_H^*)S^* - (\mu + k)D_E^* = 0 \\ (1 - r)kD_E^* - (\mu + \sigma)D_L^* = 0 \\ r k D_E^* + (1 - P)\sigma D_L^* - (\mu + \delta_1 + \gamma_1 + \gamma_2)D_H^* = 0 \\ \gamma_2 D_H^* - (\mu + \delta_2 + \varepsilon)D_M^* = 0 \\ \sigma p D_L^* + \gamma_1 D_H^* + \varepsilon D_M^* - (\mu + \omega)R = 0 \end{cases} \quad (2.20)$$

Simplification of equation (2.20) and utilization of equation (2.8) yields:

$$\begin{aligned}
 S^* &= \frac{\pi}{\mu R_0} \\
 D_E^* &= \frac{\Phi_2 \Phi_3 \Phi_4 \Phi_5 (R_0 - 1) \pi}{\{\Phi_1 \Phi_2 \Phi_3 \Phi_4 \Phi_5 - \omega k (\Phi_2 \Phi_4 \sigma p (1 - r) + \vartheta_1 \vartheta_2)\} R_0} \\
 D_L^* &= \frac{(1 - r) k \Phi_3 \Phi_4 \Phi_5 (R_0 - 1) \pi}{\{\Phi_1 \Phi_2 \Phi_3 \Phi_4 \Phi_5 - \omega k (\Phi_2 \Phi_4 \sigma p (1 - r) + \vartheta_1 \vartheta_2)\} R_0} \\
 D_H^* &= \frac{k \vartheta_2 \Phi_4 \Phi_5 (R_0 - 1) \pi}{\{\Phi_1 \Phi_2 \Phi_3 \Phi_4 \Phi_5 - \omega k (\Phi_2 \Phi_4 \sigma p (1 - r) + \vartheta_1 \vartheta_2)\} R_0} \\
 D_M^* &= \frac{k \gamma_2 \vartheta_2 \Phi_5 (R_0 - 1) \pi}{\{\Phi_1 \Phi_2 \Phi_3 \Phi_4 \Phi_5 - \omega k (\Phi_2 \Phi_4 \sigma p (1 - r) + \vartheta_1 \vartheta_2)\} R_0} \\
 R^* &= \frac{\{\Phi_3 \Phi_4 \sigma p (1 - r) + \vartheta_1 \vartheta_2\} k (R_0 - 1) \pi}{\{\Phi_1 \Phi_2 \Phi_3 \Phi_4 \Phi_5 - \omega k (\Phi_2 \Phi_4 \sigma p (1 - r) + \vartheta_1 \vartheta_2)\} R_0}
 \end{aligned}$$

where $\Phi_5 = (\mu + \omega)$, $\vartheta_1 = \gamma_1 \Phi_4 + \varepsilon \gamma_2$ and $\vartheta_2 = r(\mu + \sigma p + (1 - p)\sigma)$. It can be observed that: $\{\Phi_1 \Phi_2 \Phi_3 \Phi_4 \Phi_5 - \omega k (\Phi_2 \Phi_4 \sigma p (1 - r) + \vartheta_1 \vartheta_2)\} > 0$

It is apparent that if $R_0 = 1$, we obtain the drugs free equilibrium point. The unique endemic equilibrium in Ω exists if $D_E^* > 0$, $D_L^* > 0$, $D_H^* > 0$, $D_M^* > 0$ and $R^* > 0$. This is actualized if and only if $R_0 > 1$ and no endemic equilibrium when $R_0 < 1$. \square

2.7. Bifurcation analysis

In this subsection we investigate the nature of bifurcation by utilizing Theorem 4.1 from [23] which is based on the center manifold theory [24]. In this theory, there are two important coefficients, say, a and b, that dictate the dynamics of the system on the center manifold. In particular, if $a < 0$ and $b > 0$, then the nature of bifurcation is forward while if $a > 0$ and $b > 0$, then the bifurcation is backward.

This is done by first letting the coefficient of transmission β to be the bifurcation parameter. This is done by solving $R_0 = 1$ to obtain:

$$\beta = \beta^* = \frac{\mu(\mu + k)(\mu + \sigma)(\mu + \delta_1 + \gamma_1 + \gamma_2)}{\pi k \{\varphi(r(\mu + p\sigma) + \sigma(1 - p)) + (1 - r)(\mu + \delta_1 + \gamma_1 + \gamma_2)\}}$$

Substituting the value of β in the DFE matrix (2.11) gives:

$$\begin{pmatrix}
 -\mu & 0 & -\frac{\Phi_1 \Phi_2 \Phi_3}{k\{\varphi \vartheta_2 + (1-r)\Phi_3\}} & -\frac{\varphi \Phi_1 \Phi_2 \Phi_3}{k\{\varphi \vartheta_2 + (1-r)\Phi_3\}} & 0 & \omega \\
 0 & -\Phi_1 & \frac{\Phi_1 \Phi_2 \Phi_3}{k\{\varphi \vartheta_2 + (1-r)\Phi_3\}} & \frac{\varphi \Phi_1 \Phi_2 \Phi_3}{k\{\varphi \vartheta_2 + (1-r)\Phi_3\}} & 0 & 0 \\
 0 & (1-r)k & -\Phi_2 & 0 & 0 & 0 \\
 0 & rk & (1-p)\sigma & -\Phi_3 & 0 & 0 \\
 0 & 0 & 0 & \gamma_2 & -\Phi_4 & 0 \\
 0 & 0 & \sigma & \gamma_1 & \varepsilon & -\Phi_5
 \end{pmatrix} \tag{2.21}$$

It can be shown that the Jacobian matrix (2.21) above has one of the eigenvalues being zero(0) while the other five eigenvalues have negative sign. This implies that B_0 is a non-hyperbolic equilibrium at $\beta = \beta^*$. The presence of a simple zero eigenvalues permits us to utilize center manifold theory to establish the local stability of endemic equilibrium

B_e . Evaluating the right eigenvector $W = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ and left eigenvector $V = (v_1, v_2, v_3, v_4, v_5, v_6)^T$ associated to the zero eigenvalues give:

$$\begin{cases} w_1 = -\frac{w_6}{\mu} \left(\frac{\Phi_1 \Phi_2}{w_6} - \omega \right), & w_2 = (\mu + \sigma), & w_3 = (1 - r)k, \\ w_4 = \frac{k\vartheta_2}{\Phi_3}, & w_5 = \frac{\gamma_2 k \vartheta_2}{\Phi_3 \Phi_4}, & w_6 = \frac{k\{\Phi_3 \Phi_4 \sigma(1-r) + \vartheta_1 \vartheta_2\}}{\Phi_3 \Phi_4 \Phi_5} \end{cases}$$

The transpose of matrix (2.21) has one of the eigenvalue being a simple zero(0). Evaluation of left eigenvectors yields:

$$v_1 = v_5 = v_6 = 0, \quad v_2 = k(\varphi\vartheta_2 + (1-r)\Phi_3), \quad v_3 = \frac{\Phi_1 \Phi_2 (\Phi_1 + \sigma(1-r))}{\Phi_3} \quad \text{and} \quad v_4 = \varphi\Phi_1 \Phi_2.$$

We now calculate the bifurcation constants a and b where:

$$a = \sum_{k,i,j=1}^6 v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (B_0, \beta^*)$$

$$b = \sum_{k,i,j=1}^6 v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta} (B_0, \beta^*)$$

For simplicity and ease in algebraic manipulations, we re-write the state variables of our model (2.1) as follows: $S_h = x_1$, $V_h = x_2$, $E_h = x_3$, $I_{hA} = x_4$, $I_h = x_5$ and $R_h = x_6$. We further define equation $\frac{dX}{dt} = F(x)$ where $X = (x_1, x_2, x_3, x_4, x_5, x_6)$ and $F = (f_1, f_2, f_3, f_4, f_5, f_6)$. We consider the nonzero second order partial derivatives which gives;

$$\begin{cases} v_2 w_1 w_4 \frac{\partial^2 f_2}{\partial x_1 \partial x_4} = -v_2 \frac{w_1 w_4}{\mu} \left\{ \frac{\Phi_1 \Phi_2}{w_6} - \omega \right\} \beta^* \varphi \\ v_2 w_4 w_1 \frac{\partial^2 f_2}{\partial x_4 \partial x_1} = -v_2 \frac{w_4 w_1}{\mu} \left\{ \frac{\Phi_1 \Phi_2}{w_6} - \omega \right\} \beta^* \varphi \\ v_2 w_1 w_3 \frac{\partial^2 f_2}{\partial x_1 \partial x_3} = -v_2 \frac{w_1 w_3}{\mu} \left\{ \frac{\Phi_1 \Phi_2}{w_6} - \omega \right\} \beta^* \\ v_2 w_3 w_1 \frac{\partial^2 f_2}{\partial x_3 \partial x_1} = -v_2 \frac{w_3 w_1}{\mu} \left\{ \frac{\Phi_1 \Phi_2}{w_6} - \omega \right\} \beta^* \end{cases} \quad (2.22)$$

The sum of the equations (2.22) gives the value of a as:

$$a = -2v_2 \frac{w_6}{\mu} \left\{ \frac{\Phi_1 \Phi_2}{w_6} - \omega \right\} (w_3 + \varphi w_4) \beta^* \quad (2.23)$$

In the same manner we determine the value of b as:

$$\begin{cases} v_2 w_3 \frac{\partial^2 f_2}{\partial x_3 \partial \beta^*} = \frac{\pi v_2 w_3}{\mu} \\ v_2 w_4 \frac{\partial^2 f_2}{\partial x_4 \partial \beta^*} = \frac{\pi v_2 w_3 \varphi}{\mu} \end{cases} \quad (2.24)$$

The sum of the equations in (2.24) gives the value of b as:

$$b = \frac{\pi v_2}{\mu} (w_3 + \varphi w_4) > 0 \quad (2.25)$$

None negativity of b implies that direction of bifurcation at $R_0 = 1$ is entirely determined by the sign of a which depends on the re-infection rate ω . For backward bifurcation,

$$\omega > \frac{(\mu + k)(\mu + \sigma)(\mu + \delta_1 + \gamma_1 + \gamma_2)(\mu + \delta_2 + \varepsilon)(\mu + \omega)}{\{(\mu + \delta_1 + \gamma_1 + \gamma_2)(\mu + \delta_2 + \varepsilon)\sigma(1-r) + \vartheta_1 \vartheta_2\}} \quad (2.26)$$

Theorem 2.6. *The model (2.1) undergoes backward bifurcation at $R_0 = 1$ whenever inequality (2.26) holds.*

The above theorem is in conformity with study by Kassa et.al.[25] that suggested that backward bifurcation is possible if individuals do not permanently quite substance and drugs abuse. Moreover, it is note worthy that for the case where the recovered persons acquires permanent immunity to COVID-19 infection (i.e $\omega = 0$), the bifurcation coefficient α becomes:

$$\alpha = -2\nu_3 \frac{(\mu + \kappa)(\mu + \sigma)}{\mu} \beta^* (w_4 + \varphi w_5) < 0$$

This rules out the occurrence of backward bifurcation but instead, forward bifurcation sets in. Hence we obtain the following conclusion:

Theorem 2.7. *(Local stability of endemic equilibrium). The unique endemic equilibrium B_e of model (2.1) is locally asymptotically stable (LAS) if $R_0 > 1$.*

2.8. Global stability of DSA endemic equilibrium point

Theorem 2.8. *The unique endemic equilibrium B_e of the model (2.1) is globally asymptotically stable in Ω whenever $R_0 > 1$.*

Proof. Let c_1, c_2, c_3, c_4, c_5 and c_6 be non-negative constants and with no loss of generality, we consider a special case of our system where $\omega = 0$. We consider a Lyapunov function defined as:

$$L = c_1 L_1 + c_2 L_2 + c_3 L_3 + c_4 L_4 + c_5 L_5 + c_6 L_6$$

where

$$\begin{cases} L_1 = S^{**} \cdot g\left(\frac{S}{S^{**}}\right), & L_2 = D_E^{**} \cdot g\left(\frac{D_E}{D_E^{**}}\right), & L_3 = D_L^{**} \cdot g\left(\frac{D_L}{D_L^{**}}\right), \\ L_4 = D_L^{**} \cdot g\left(\frac{D_H}{D_H^{**}}\right), & L_5 = D_M^{**} \cdot g\left(\frac{D_M}{D_M^{**}}\right), & L_6 = R^{**} \cdot g\left(\frac{R}{R^{**}}\right). \end{cases} \tag{2.27}$$

and that $g(x) = x - 1 - \ln x \geq g(1) = 0$ for any $x > 0$

Differentiating L with respect to t gives:

$$\left\{ \begin{aligned} \frac{dL}{dt} = & c_1 \left(1 - \frac{S^{**}}{S}\right) \left\{ \pi - \beta(D_L + \varphi D_H)S - \mu S \right\} \\ & + c_2 \left(1 - \frac{D_E^{**}}{D_E}\right) \left\{ \beta(D_L + \varphi D_H)S - (\mu + k)D_E \right\} \\ & + c_3 \left(1 - \frac{D_L^{**}}{D_L}\right) \left\{ (1-r)kD_E - (\mu + \sigma)D_L \right\} \\ & + c_4 \left(1 - \frac{D_H^{**}}{D_H}\right) \left\{ rkD_E + (1-p)\sigma D_L - (\mu + \delta_1 + \gamma_1 + \gamma_2)D_H \right\} \\ & + c_5 \left(1 - \frac{D_M^{**}}{D_M}\right) \left\{ \gamma_2 D_H - (\mu + \delta_2 + \varepsilon)D_M \right\} \\ & + c_6 \left(1 - \frac{R^{**}}{R}\right) \left\{ \sigma p D_L + \gamma_1 D_H + \varepsilon D_M - \mu R \right\} \end{aligned} \right. \tag{2.28}$$

At steady endemic equilibrium,

$$\begin{cases} \pi = \beta(D_L^{**} + \varphi D_H^{**})S^{**} + \mu S^{**}, & (\mu + k) = \frac{\beta(D_L^{**} + \varphi D_H^{**})}{D_E^{**}}, \\ (\mu + \sigma) = \frac{(1-r)kD_E^{**}}{D_L^{**}}, & (\mu + \delta_1 + \gamma_1 + \gamma_2) = \frac{krD_E^{**} + (1-p)\sigma D_L^{**}}{D_H^{**}}, \\ (\mu + \delta_2 + \varepsilon) = \frac{\gamma_1 D_H^{**}}{D_M^{**}}, & \mu = \frac{\sigma p D_L^{**} + \gamma_1 D_H^{**} + \varepsilon D_M^{**}}{R^{**}}. \end{cases} \tag{2.29}$$

Substituting (2.29) into (2.28) gives,

$$\left\{ \begin{aligned} \frac{dL}{dt} = & c_1 \left(1 - \frac{S^{**}}{S}\right) \left\{ \beta S^{**} D_L^{**} \left(1 - \frac{SD_L^{**}}{S^{**} D_L^{**}}\right) + \beta \varphi S^{**} D_H^{**} \left(1 - \frac{SD_H^{**}}{S^{**} D_H^{**}}\right) \right. \\ & + \mu S^{**} \left(1 - \frac{S}{S^{**}}\right) \left. \right\} + c_2 \left(1 - \frac{D_E^{**}}{D_E}\right) \left\{ \beta S^{**} D_L^{**} \left(\frac{SD_L^{**}}{S^{**} D_L^{**}} - \frac{D_E}{D_E^{**}}\right) \right. \\ & + \beta \varphi S^{**} D_H^{**} \left(\frac{SD_H^{**}}{S^{**} D_H^{**}} - \frac{D_E}{D_E^{**}}\right) \left. \right\} \\ & + c_3 \left(1 - \frac{D_L^{**}}{D_L}\right) \left\{ (1-r)kD_E^{**} \left(\frac{D_E}{D_E^{**}} - \frac{D_L}{D_L^{**}}\right) \right\} \\ & + c_4 \left(1 - \frac{D_H^{**}}{D_H}\right) \left\{ rkD_E^{**} \left(\frac{D_E}{D_E^{**}} - \frac{D_H}{D_H^{**}}\right) + (1-p)\sigma D_L^{**} \left(\frac{D_H}{D_H^{**}} - \frac{D_M}{D_M^{**}}\right) \right\} \\ & + c_5 \left(1 - \frac{D_M^{**}}{D_M}\right) \left\{ \gamma_2 D_H^{**} \left(\frac{D_H}{D_H^{**}} - \frac{D_M}{D_M^{**}}\right) \right\} + c_6 \left(1 - \frac{R^{**}}{R}\right) \\ & \left. \left\{ \sigma p D_L^{**} \left(\frac{D_L}{D_L^{**}} - \frac{R}{R^{**}}\right) + \gamma_1 D_H^{**} \left(\frac{D_H}{D_H^{**}} - \frac{R}{R^{**}}\right) + \varepsilon D_M^{**} \left(\frac{D_M}{D_M^{**}} - \frac{R}{R^{**}}\right) \right\} \right\} \end{aligned} \right. \quad (2.30)$$

Letting $c_1 = c_2$, $c_3 = \frac{\beta S^{**} D_L^{**}}{(1-r)kD_E^{**}} c_1$ and $c_4 = \frac{\beta S^{**} D_H^{**}}{rkD_E^{**}} c_1$ and simplifying equation (2.30) gives:

$$\left\{ \begin{aligned} \frac{dL}{dt} = & -\frac{c_1 S^{**} \mu}{S} \left(\frac{S}{S^{**}} - 1\right)^2 + c_1 \beta S^{**} D_L^{**} \left\{ 3 - \frac{S^{**}}{S} - \frac{D_E D_L^{**}}{D_E^{**} D_L} - \frac{SD_E^{**} D_L}{S^{**} D_E D_L^{**}} \right\} \\ & + c_1 \beta \varphi S^{**} D_H^{**} \left\{ 3 - \frac{S^{**}}{S} - \frac{D_E D_H^{**}}{D_E^{**} D_H} - \frac{SD_E^{**} D_H}{S^{**} D_E D_H^{**}} \right\} \\ & + c_3 (1-r)kD_E^{**} \left\{ 1 - \frac{D_L}{D_L^{**}} - \frac{D_E}{D_E^{**}} \left(\frac{D_L^{**}}{D_L} - 1\right) \right\} \\ & + c_4 rkD_E^{**} \left\{ 1 - \frac{D_H}{D_H^{**}} - \frac{D_E}{D_E^{**}} \left(\frac{D_H^{**}}{D_H} - 1\right) \right\} \\ & + c_4 (1-p)\sigma D_L^{**} \left\{ 1 - \frac{D_H}{D_H^{**}} - \frac{D_L}{D_L^{**}} \left(\frac{D_H^{**}}{D_H} - 1\right) \right\} \\ & + c_5 \gamma_2 D_H^{**} \left\{ 1 - \frac{D_M}{D_M^{**}} - \frac{D_H}{D_H^{**}} \left(\frac{D_M^{**}}{D_M} - 1\right) \right\} \\ & + c_6 \sigma p D_L^{**} \left\{ 1 - \frac{R}{R^{**}} - \frac{D_L}{D_L^{**}} \left(\frac{R^{**}}{R} - 1\right) \right\} \\ & + c_6 \gamma_1 D_H^{**} \left\{ 1 - \frac{R}{R^{**}} - \frac{D_H}{D_H^{**}} \left(\frac{R^{**}}{R} - 1\right) \right\} \\ & + c_6 \varepsilon D_M^{**} \left\{ 1 - \frac{R}{R^{**}} - \frac{D_M}{D_M^{**}} \left(\frac{R^{**}}{R} - 1\right) \right\} \end{aligned} \right. \quad (2.31)$$

Finally, since the arithmetic mean is greater or equal to the geometric mean it follows that:

$$\begin{aligned} \left(3 - \frac{S^{**}}{S} - \frac{D_E D_L^{**}}{D_E^{**} D_L} - \frac{SD_E^{**} D_L}{S^{**} D_E D_L^{**}} \right) &\leq 0, \quad \left(3 - \frac{S^{**}}{S} - \frac{D_E D_H^{**}}{D_E^{**} D_H} - \frac{SD_E^{**} D_H}{S^{**} D_E D_H^{**}} \right) \leq 0 \\ \left\{ 1 - \frac{D_L}{D_L^{**}} - \frac{D_E}{D_E^{**}} \left(\frac{D_L^{**}}{D_L} - 1\right) \right\} &\leq 0, \quad \left\{ 1 - \frac{D_H}{D_H^{**}} - \frac{D_E}{D_E^{**}} \left(\frac{D_H^{**}}{D_H} - 1\right) \right\} \leq 0 \\ \left\{ 1 - \frac{D_H}{D_H^{**}} - \frac{D_L}{D_L^{**}} \left(\frac{D_H^{**}}{D_H} - 1\right) \right\} &\leq 0, \quad \left\{ 1 - \frac{D_M}{D_M^{**}} - \frac{D_H}{D_H^{**}} \left(\frac{D_M^{**}}{D_M} - 1\right) \right\} \leq 0 \\ \left\{ 1 - \frac{R}{R^{**}} - \frac{D_L}{D_L^{**}} \left(\frac{R^{**}}{R} - 1\right) \right\} &\leq 0, \quad \left\{ 1 - \frac{R}{R^{**}} - \frac{D_H}{D_H^{**}} \left(\frac{R^{**}}{R} - 1\right) \right\} \leq 0 \\ \left\{ 1 - \frac{R}{R^{**}} - \frac{D_M}{D_M^{**}} \left(\frac{R^{**}}{R} - 1\right) \right\} &\leq 0 \end{aligned}$$

The non-negativity of the system parameters permits us to make conclusion that $\frac{dL}{dt} \leq 0$ for $R_0 > 1$. Moreover, the set where $\frac{dL}{dt} = 0$ is $\Omega = \{(S, D_E, D_L, D_H, D_M, R) : S = S^{**}, D_E = D_E^{**}, D_L = D_L^{**}, D_H = D_H^{**}, D_M = D_M^{**}, R = R^{**}\}$, and by LaSalle's Invariance Principle [26], the only compact invariant set of Ω is the singleton set B_e . Thus the endemic equilibrium B_e is globally asymptotically stable. This completes the proof. \square

3. Numerical analysis and simulation

This section presents parameter values, sensitivity analysis and numerical simulation of the model (2.1).

3.1. Parameter values

The values assigned to the parameters used in this study are gotten from the existing related researches and others are estimated. The parameter values are presented in the table (4) below:

Table 3: The model parameter values and source

Parameters	Value	Source(s)
π	$\frac{1000}{67.12 \times 12}$	[27, 28]
β	0.000201	Estimated
ω	0.0089	[29]
μ	$\frac{1}{67.21 \times 12}$	[27]
φ	1.02	Estimated
k	0.045	Estimated
r	0.301	Estimated
σ	0.5	Estimated
p	0.60	Estimated
δ_1	0.059	[30]
δ_2	0.093	Estimated
γ_1	0.051	Estimated
γ_2	0.065	Estimated

3.2. Sensitivity Analysis

Sensitivity analysis is an important tool in mathematical modeling as it aids in unearthing the extend of influence various parameters have to the disease transmission and prevalence. With the analytical expression of the model’s reproduction number, R_0 , its reasonable to utilize the normalized forward sensitivity index of R_0 that depends differentially on a parameter h_j , as defined by [31] and mathematically expressed as:

$$\Lambda_{h_j}^{R_0} = \frac{\partial R_0}{\partial h_j} \times \frac{h_j}{R_0},$$

where h_j are the various model parameters whose sensitivity on R_0 is to be obtained.

Table 4: The normalized forward sensitivity indices of R_0 to model parameters evaluated at the baseline parameters as displayed in the table (3).

Parameters	Sensitivity indices	Values
β	$\Lambda_{\beta}^{R_0}$	+1.00000
π	$\Lambda_{\pi}^{R_0}$	+1.00000
φ	$\Lambda_{\varphi}^{R_0}$	+0.52167
k	$\Lambda_k^{R_0}$	+0.02681
p	$\Lambda_p^{R_0}$	-0.41610
σ_1	$\Lambda_{\sigma_1}^{R_0}$	-0.26582
γ_1	$\Lambda_{\gamma_1}^{R_0}$	-0.08548
r	$\Lambda_r^{R_0}$	-0.04313

From table (4), it is evident that β, π, φ , and k have a positive effect on R_0 while the rest of the parameters have a negative impact. For instance, 10% increase(decrease) in φ causes a corresponding increase(decrease) in R_0 by 5.2167% while 10% increase(decrease) in p results in a corresponding decrease(increase) in R_0 by 4.1610%. It is apparent that $\beta, \pi, \varphi, p, \gamma_1$, and γ_2 , are the most sensitive to R_0 since small perturbations to these parameters lead to a significant change in R_0 .

In figure (2), panels (a) and (b) respectively show the effects of transmission coefficients β and the modification factor for heavy drugs abusers (HDA) φ to reproduction number dynamics. These parameters positively impact the value of R_0 in that an increase(decrease) in these parameters causes a corresponding increase(decrease) in R_0 . Moreover, from panels (c) and (d), we depict that early identification and treatment of HDA γ_1 significantly abates DSA in the community. It is also evident that increased educational campaigns against DSA and the provision of counseling services to youths who are LDA cause a substantial reduction of R_0 .

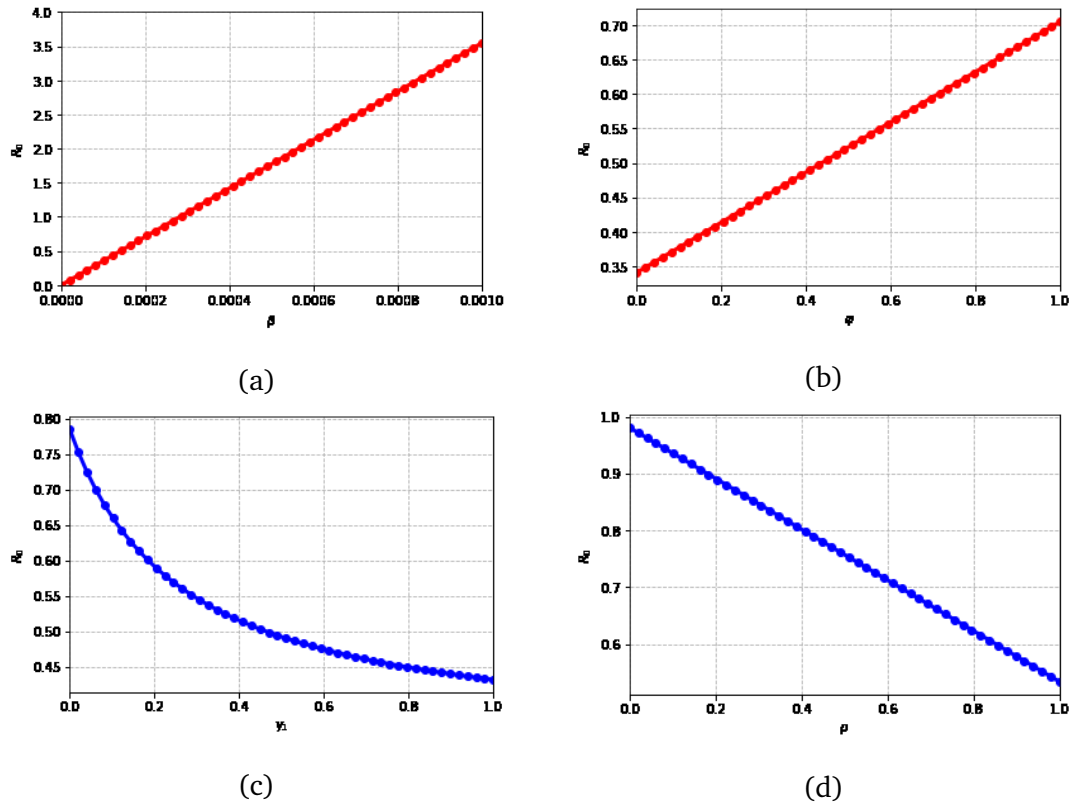


Figure 2: Variation of R_0 concerning (a) the coefficient of transmission β (b), modification factor φ , (c) the rate of detection and treatment of light drugs abusers γ_1 and (d) the rate of identification and treatment of heavy drugs users p . The parameter values used are those in the table (3).

3.3. Numerical results

In this section, we endeavor to discuss the behavior derived from the numerical simulations. The model parameters are listed in table (3). Figure (3) shows the temporal variations of different population compartments. From the projection, we can observe that a symmetrical aspect appears in the exposed and recovered sub-population.

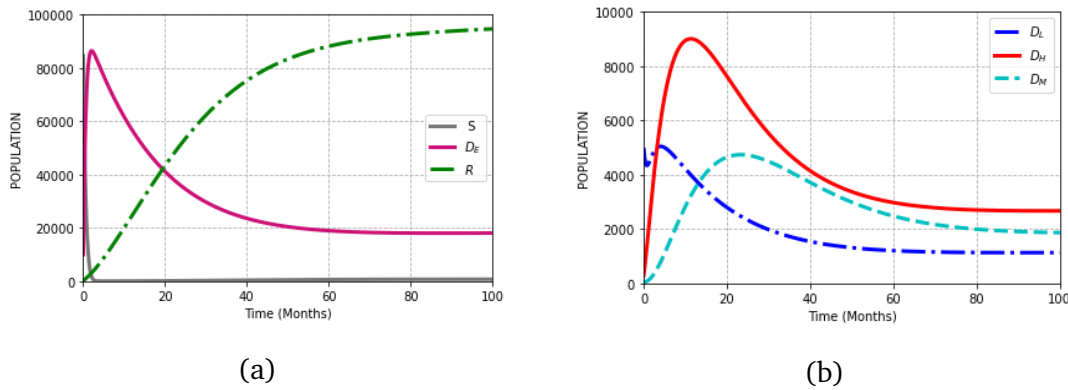


Figure 3: Time dependent variations for (a) Susceptible, Exposed, and Recovered (b) LDA, HDA, and MDA. All parameter values are given in the table (3).

In figure (4), the effect of the DSA re-initiation coefficient on the recovered population at different values is shown. The projection shows that the cumulative number of individuals becoming DSAs is greater for large values of ω and decreases when ω assumes low values. It is thus apparent that concerted efforts have to be put in place to monitor and engage recovered DSAs to deter them from slipping back into drugs.

Figure (5) explores the effects of the DSA initiation control coefficient σ_1 on the exposed LDUs, HDUs, and mentally ill population. The projections point to the fact that an increase in the value of σ_1 leads to a decrease in the number of drugs and substance abusers, which ultimately leads to a reduction in R_0 . This can be actualized through increased levels of campaigns against drugs and substances as well as providing the youth with emotional support through counseling sessions which will go a long way in decreasing drug and substance abusers in the country.

4. Discussion and conclusions

In this paper, we formulated and theoretically analyzed a non-linear deterministic model for DSA among the youth. We obtained the feasible region where the model has been proven well-posed. We utilized the next-generation matrix method to derive the reproduction number R_0 . Moreover, we verified the local stability of the DFE point by utilizing the Jacobian matrix and trace determinant method.

The analytical results point to the fact that DFE is locally and globally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. The global stability of DFE and DEE are investigated via the Lyapunov function and LaSalle's Invariance principle. We also proved the condition for backward bifurcation using the center manifold theory as applied by Castillo-Chavez and Song, where it was shown that the recovered individuals' re-initiation

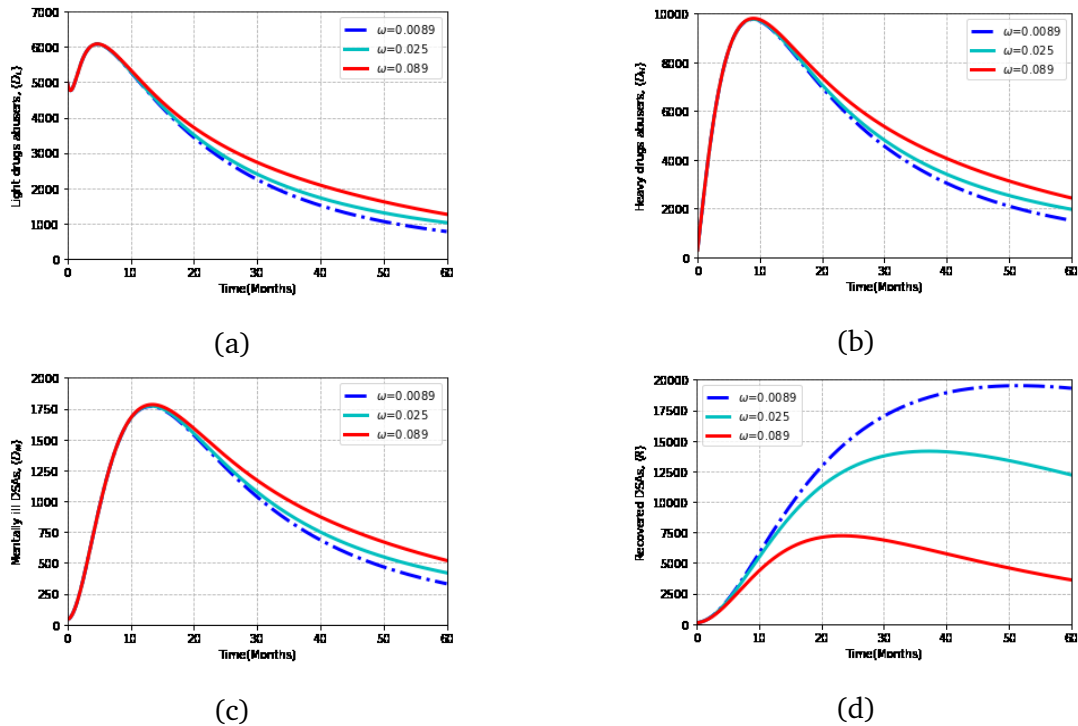


Figure 4: Projections of effects of DSA re-initiation coefficients for (a) Light drugs Abusers, (b) Heavy drugs abusers, (c) Mentally ill persons, and (d) Recovered DSAs at values of $\omega = 0.0089$, $\omega = 0.0250$ and $\omega = 0.0890$. All parameter values are given in table (3) except $\sigma_1 = 0.20$ so that $R_0 = 0.85348 < 1$.

rate drives this phenomenon. From an epidemiological viewpoint, the implication is the possible coexistence of both DFE and DEE points even after the classical requirement of reducing the reproduction number below the unit for disease eradication has been met. This would frustrate the government and the policymakers' efforts to reduce initiation into DSA.

In the sensitivity analysis, the normalized sensitivity indices of R_0 reveal that the most sensitive parameters are β , φ , and π with a positive sign while the parameters σ_1 , p and γ_1 are the most sensitive parameters with a negative sign. This implies that decreasing the effective contact rate between the susceptible and the DSAs through sensitization of the youth population on the harmful effects of DSA as well as identification and treatment of the light and heavy drugs abusers should be prioritized by public health policymakers since this leads to reduction in the number of new DSA recruits. The simulation shows that the relapse rate has to be reduced if the war against DSA has to be won. This underscores the pivotal role played by comprehensive and effective treatment schemes for the DSAs as well as strict adherence to relapse prevention programs for the recovered patients.

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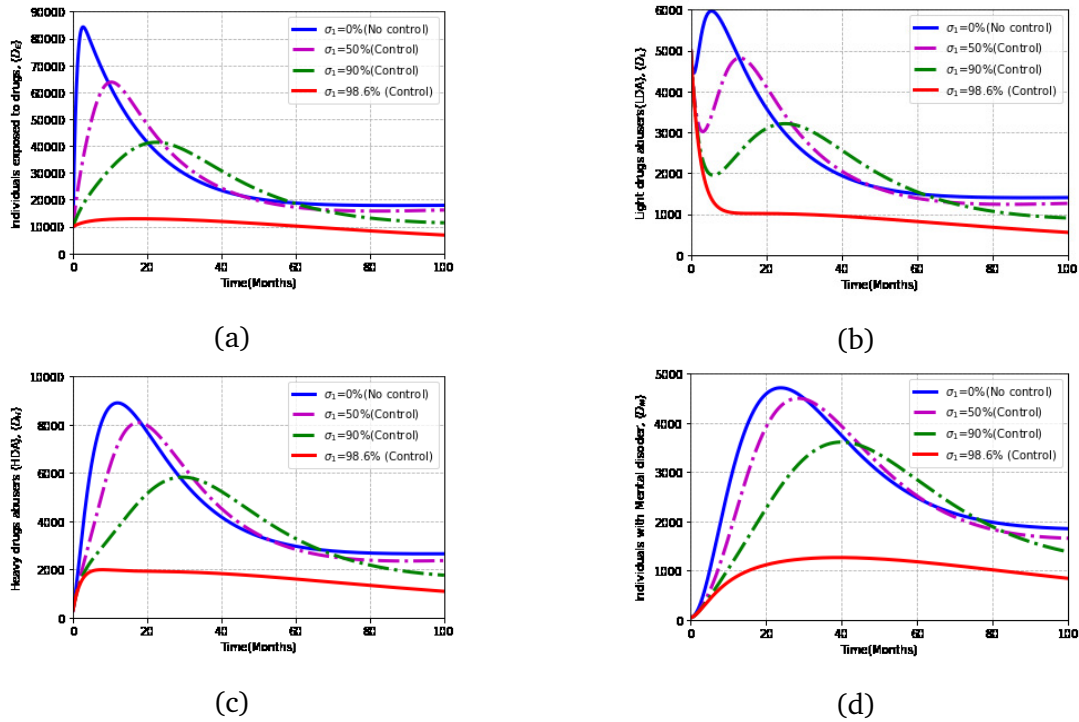


Figure 5: Projections with varying levels of DSA initiation control for (a) Individuals exposed to drugs, (b) Light drugs Abusers, (c) Heavy drugs abusers and (d) Mentally disordered persons at values of $\sigma_1 = 0.00$, $\sigma_1 = 0.50$ ($R_0 = 0.356208 < 1$), $\sigma_1 = 0.90$ ($R_0 = 0.071242 < 1$), $\sigma_1 = 0.98$ ($R_0 = 0.014248 < 1$). All parameter values are given in table (3).

Informed consent statement

Not applicable.

Declaration of competing interest

The author declares that there is no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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