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MODELLING THE ROLE OF DRUG BARONS ON THE PREVALENCE OF DRUG EPIDEMICS

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Abstract. Substance abuse is a global menace with immeasurable consequences to the health of users, the quality of life and the economy of countries affected. Although the prominently known routes of initiation into drug use are; by contact between potential users and individuals already using the drugs and self initiation, the role played by a special class of individuals referred to as drug lords can not be ignored. We consider a simple but useful compartmental model of drug use that accounts for the contribution of contagion and drug lords to initiation into drug use and drug epidemics. We show that the model has a drug free equilibrium when the threshold parameter R_0 is less that unity and a drug persistent equilibrium when R_0 is greater than one. In our effort to ascertain the effect of policing in the control of drug epidemics, we include a term accounting for law enforcement. Our results indicate that increased law enforcement greatly reduces the prevalence of substance abuse. In addition, initiation resulting from presence of drugs in circulation can be as high as seven times higher that initiation due to contagion alone.

1. **Introduction.** Despite the international drug control system through the United Nations Office on Drug Control (UNODC) endeavouring to restrict the use of addictive drugs for medical purposes and stop the consumption of addictive drugs from spreading [39], drug lords play a totally contradictory or antagonistic role. They are at the heart of manufacturing, trafficking and ensuring distribution of illicit addictive drugs. Drug trafficking poses a security threat in many counties worldwide including many of the West African countries bordering the Atlantic ocean, Brazil, Mexico, South Africa due their porous borders, and Australia among others [10,38].

In South Africa for example, substance abuse accounts for most of the criminal offences in the townships [29]. The offences associated with alcohol and illicit drugs use include; possession and sale of illicit drugs, crime to obtain money to purchase drugs and quench their addiction, driving under the influence of alcohol and drugs, child abuse and domestic violence among others. Trafficking of drugs has made drugs easily accessible to combatants and the general population. The people using these drugs often work under their influence not acting rationally, committing crimes and posing serious security threats. With the existence of drug lords, it is

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beyond reasonable doubt that the drug epidemics will not only escalate but also remain prevalent at high consumption levels irrespective of the control strategies such as policing being in place. Technological advancement using Terahertz (THz) radiation and passenger profiling have been developed to curb drug trafficking through airports. The use of THz science is based on the fact that illicit drugs have got signatures different from those of pharmaceutical products in the THz range of the electromagnetic spectrum [36]. The major challenge is that it is expensive and limited mainly in airports.

For individuals to start using drugs, they often should have the motivation and access to drugs. It is believed that motivation without access to drugs can not result into drug use [23]. On the other hand, interaction with drug lords results in easy access to drugs and the motivation for potential users to get initiated into drug abuse.

Modelling of drug epidemics has been done previously, see for instance [26,28,42]. However, in all these references, initiation into drug use is based only on the contact between the susceptible population and the drug user. On the other hand, innovators exist where the initiation is based on the fact that the impetus to use substances is internal [2, 7]. At the heart of dynamics of illicit drug use patterns is a special class of individuals called drug lords. The drug lords, also commonly known as drug barons or kingpins, are individuals who command a sizeable network of individuals involved in illegal trading of drugs. They play a huge role in shaping drug use patterns over time. The patterns are also influenced by progression of users through different drug use states [7]. The drug use states are usually defined by a homogeneous drug use pattern in the population, for instance addicts form a distinct state when compared to those in rehabilitation or those in light drug use. These states allow us to compartmentalise any given population with each compartment comprising of individuals with the same drug use pattern. Compartmental models have been used to model drug epidemics, for example heroin [31,42], methamphetamine [28] and cocaine [7, 11, 13] among others. Once the population has been compartmentalised, ordinary differential equations can be used to describe the evolution of the size of each compartment over time.

In this paper, we present a compartmental model for a drug epidemic that is innovative in four regards. First, the role of drug lords is investigated as they play a central role in drug initiation, as we shall discuss later. Second, the essential aspect of initiation driven by interactions of drug users and the non-users is considered, similar to the past models [28,31,42]. Third, the model takes into account amelioration, i.e individuals are allowed to recover in stages. The model considers recovery, that leads to either an immediate susceptibility or permanent recovery. This may occur at any stage as an individual moves back to an earlier stage in the drug use cycle. Forth, the rise in the number of users can give rise to the increased number of drug barons. This means that the growth of drug barons is demand driven. Our aim is to determine the potential effects of drug lords, amelioration and policing on the dynamics of drug epidemics.

This paper is organised as arranged as follows; in section 2, we present the model formulation, followed by the model analysis in section 3. In section 4, we present the drug persistent equilibrium including its local stability analysis, followed by the numerical results in section 5. It is in this section that we carry out sensitivity analysis of the model output to input parameters and then conclude the paper in section 6.

2. **Model formulation.** We subdivide the total population into non-intersecting compartments. The individuals in the compartments homogeneously mix whereas the individual compartments are heterogeneous. These compartments include, the susceptible population S, comprising of individuals at the risk of using drugs, light drug users L, heavy drug users H and drug users under rehabilitation T. Assuming homogeneous mixing of the susceptible and drug users, the susceptible population gets initiated into substance abuse at a rate described by the function Γ, such that

$$\Gamma = \Lambda S + \alpha_1 SD \text{ where } \Lambda = c\beta\left(\frac{L + \eta_1 H + \eta_2 T}{N}\right). \tag{1}$$

The function Γ, describes the generation of new initiates. We also have a compartment of drug lords who may or may not be using drugs D. In our model, we assume that drug lords are not drug users themselves. This is based on the assumption that if they are to use substances, their levels of consciousness and cognition would be disturbed due to intoxication. As a result, they would end up giving out drugs free of charge as they may be under the influence of drugs themselves and this loss has a heavy price associated. We assume that the population of drug lords increases due to the increase in the number of drug users following the law of demand and supply. In the model, we suppose that law enforcement $h(r)D$ is proportional to the number of drug lords such that the proportionality constant is a constant removal rate r from the community. That is

$$h(r) = \begin{cases} r & \text{for} \quad D > 0 \\ 0 & \text{for} \quad D = 0 \end{cases} \tag{2}$$

Similar to the epidemic model in [41], $r > 0$ implies that law enforcement is kept in full force until the population of drug lords is remarkably reduced to such a value that substance abuse can be eliminated from the community. We endeavour to show that presence of drugs in the population is a major driving force of addiction and that law enforcement which is proportional to the population of drug lords/supplies has a significant effect on the reduction of drug epidemics. The flow of individuals between compartments is shown in the Figure 1 below. Based on the flow diagram,

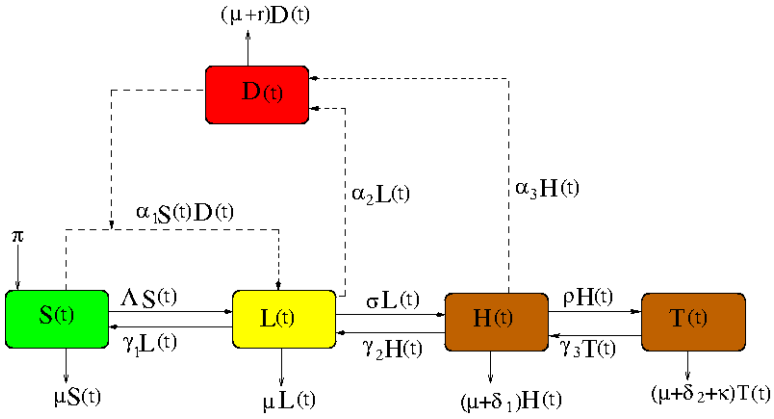


FIGURE 1. Flow diagram of the model of drug use in presence of drug lords.

assumptions and the parameter descriptions, the ordinary differential equations that

represent the compartmental model are given as

$$\begin{aligned}
\dot{S} &= \pi + \gamma_1 L - \Lambda S - \alpha_1 SD - \mu S,\\
\dot{L} &= \Lambda S + \alpha_1 SD + \gamma_2 H - Q_1 L,\\
\dot{H} &= \sigma L + \gamma_3 T - Q_2 H,\\
\dot{T} &= \rho H - Q_3 T,\\
\dot{D} &= \alpha_2 L + \alpha_3 H - Q_4 D.
\end{aligned} \tag{3}$$

where

$Q_1 = \mu + \sigma + \gamma_1,\ Q_2 = \mu + \rho + \gamma_2 + \delta_1,\ Q_3 = \mu + \gamma_3 + \delta_2 + k,\ Q_4 = (\mu + r),$ with initial conditions $x(0) = \{S_0, L_0, H_0, T_0\}$ such that $S_0 = S(0),\ L_0 = L(0),\ H_0 = H(0)$ and $T_0 = T(0)$.

In our model, we consider the epidemiological parameters to be constant. The parameters and their description is given in the Table 1.

TABLE 1. Symbols and description of parameters used in the model

Parameter	Description
π	Recruitment rate of individuals in the general population into the susceptible population
β	Probability that a contact between a drug users and S results into initiation
η_1	Ability of heavy drug users to initiate new drug users relative to light users
η_2	Ability of drug users under rehabilitation to initiate new drug users relative to light users
c	The mean number of effective contacts between drug users and the susceptible population
σ	Rate at which light drug users escalate into heavy drug use
ρ	Rate at which heavy drug users are recruited into rehabilitation
k	Rate drug users under rehabilitation permanently quit
μ	Per capita mortality rate of the general population
γ_1	Rate at which light users quit and become susceptible again.
γ_2	Rate at which heavy users move back to light using class. This constitutes amelioration
γ_3	Rate at which users under rehabilitation revert to heavy drug use
α_1	The effective contact rate between drug lords and the susceptible population.
α_2	Rate of escalation of drug lords due to presence of light drug users
α_3	Rate of escalation of drug lords due to presence of heavy drug users
r	Rate of removal of drug lords which constitutes mainly law enforcement
δ_1	Rate of removal of heavy drug users due to events related to drug abuse
δ_1	Rate of removal of drug users under rehabilitation due to events related to drug abuse

3. **Model analysis.**

3.1. **Positivity of solution.**

The system of equations (3), describes the dynamics of human population. Since the initials conditions are non negative, we must ensure that the solutions resulting from (3) are also non negative for all time $t > 0$. We therefore have the following lemma.

Lemma 3.1. *Given that the system (3) has non-negative initial conditions S_0, L_0, H_0, T_0 and $D_0 > 0$, the solution space (S, L, H, T, D) is non-negative for all $t > 0$.*

Proof. Suppose that

$$t^* = \sup\{t > 0 : S, L, H, T, D > 0\} \in [0, t]\,. \tag{4}$$

Then $t^* > 0$ and it follows from the first equation of system of equation (3) that

$$\frac{dS}{dt} \geq \pi - (\Lambda + \alpha_1 D + \mu)\, S. \tag{5}$$

The differential inequality (5) can be re-written as

$$\frac{dS}{dt} + (\Lambda + \alpha_1 D + \mu)\, S \geq \pi. \tag{6}$$

This differential equation can be solved using a suitable integrating factor obtaining

$$\frac{d}{dt}\left[S(t)\exp\{\mu t + \int_0^t (\Lambda(\omega) + \alpha_1 D(\omega))d\omega\}\right] \geq \pi \exp\left[\mu t + \int_0^t (\Lambda(\omega) + \alpha_1 D(\omega))d\omega\right].$$

Using the initial condition $S(0) = S_0$, at time t^* the solution is given by

$$S(t^*) \geq \exp\left[-\left(\mu t^* + \int_0^{t^*} (\Lambda(\omega) + \alpha_1 D(\omega))d\omega\right)\right] \times \tag{7}$$

$$\left[S_0 + \pi \int_0^{t^*} \exp\left(\mu t^* + \int_0^{t^*} (\Lambda(\omega) + \alpha_1 D(\omega))d\omega\right) dt^*\right] > 0. \tag{8}$$

Using the second equation of system (3), the equation of light users can written such that

$$\frac{dL}{dt} \geq -Q_1 L, \tag{9}$$

whose solutions t^* is given by

$$L(t^*) \geq L_0 \exp(-Q_1)t^* > 0. \tag{10}$$

Using the same approach on the rest of the equations on system (3), it can be easily shown that $H(t^*)$, $T(t^*)$ and $D(t^*) > 0$ for all $t^* > 0$. This completes the proof. □

3.2. **Feasible region.** In this section, we establish some facts relating to long term behaviour of the solution to the system of equations (3). We derive and investigate the stability of the equilibrium states; the drug free equilibrium (DFE) and the drug persistent equilibrium (DPE). In our analysis the phase space of (3) is given by

$$\Omega = \{S, L, H, T > 0 : S + L + H + T = N\}. \tag{11}$$

Lemma 3.2. *For Ω defined by (11), we let $x(t)$ denote the solution of (3) with initial conditions $x(0) \in \Omega$, then $x(t) \in \Omega$ for all $t > 0$.*

Proof. To show that the solution of the system (3) starting from any point in Ω remains in Ω, we use the total population of substance users $N = S + L + H + T$. The rate change of the total population is given by,

$$\frac{dN}{dt} = \pi - \mu N - \delta_1 H - \delta T \leq \pi - \mu N. \tag{12}$$

We now solve the differential inequality using a suitable integrating factor to obtain,

$$N \leq \frac{\pi}{\mu} + \left(N_0 - \frac{\pi}{\mu}\right)\exp(-\mu t) \quad \text{for} \quad t \geq 0. \tag{13}$$

If $N_0 \leq \frac{\pi}{\mu}$, the solution of the differential equation $\frac{dN}{dt} = \pi - \mu N$ is monotone increasing and bounded by $\frac{\pi}{\mu}$. Otherwise, if $N_0 > \frac{\pi}{\mu}$, the solutions of $\frac{dN}{dt} = \pi - \mu N$ are monotone decreasing and bounded below by $\frac{\pi}{\mu}$. Therefore, the phase space becomes

$$\Omega = \left\{S, L, H, T : S + L + H + T \leq \max\left\{N_0, \frac{\pi}{\mu}\right\}\right\}. \tag{14}$$

However, in either case, at limiting equilibrium $\lim_{t\to\infty} N = \frac{\pi}{\mu}$. Since all the phase space variables have been shown to be non negative, this means that the solution

trajectories of (3) do not go through the boundary of Ω forward in time. This condition is valid for all phase space variables. Thus, the phase space (14) is invariant and attracting system (3). □

3.3. **The basic reproduction number.** We define the basic reproduction number R_0 as the expected number of secondary initiations that result from introducing a single drug user and, or drug baron in a purely susceptible population. We use this R_0 as a fundamental quantity of our analysis to determine the situation when substance abuse can be wiped out or remain prevalent in the population. We use the next generation matrix approach described by van den Driesche and Watmough [40] to evaluate the basic reproduction number of the model. Then we have the matrices of initiation rates and transitions respectively given by,

$$F = \begin{pmatrix} \beta & \beta\eta_1 & \beta\eta_2 & \alpha_1\frac{\pi}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} Q_1 & -\gamma_2 & 0 & 0 \\ -\sigma & Q_2 & -\gamma_3 & 0 \\ 0 & -\rho & Q_3 & 0 \\ -\alpha_2 & -\alpha_3 & 0 & Q_4 \end{pmatrix}. \tag{15}$$

The basic reproduction number is given as the spectral radius of the next generation matrix FV^{-1} such that

$$R_{0UD} = \rho(FV^{-1}) = R_{0U} + R_{0D}, \tag{16}$$

Where

$$R_{0U} = \frac{\beta\left(Q_2Q_3(1-\Phi_1) + Q_3\eta_1\sigma + \rho\eta_2\sigma\right)}{Q_1Q_2Q_3\left(1-(\Phi_1+\Phi_2)\right)} \tag{17}$$

and

$$R_{0D} = \frac{\alpha_1\pi\left(\alpha_2Q_2(1-\Phi_1) + \sigma\alpha_3\right)}{\mu Q_1Q_2Q_4\left(1-(\Phi_1+\Phi_2)\right)}, \tag{18}$$

Where

$$\Phi_1 = \frac{\rho\gamma_3}{Q_2Q_3} \quad \text{and} \quad \Phi_2 = \frac{\sigma\gamma_2}{Q_1Q_2}.$$

The reproduction number R_0 is given in two parts indicating the contribution of two different groups in the drug initiation process. Therefore, the values R_{0U} and R_{0D} measure the average number of new drug users who may be initiated into drug use if a drug user or drug baron respectively is introduced in a purely susceptible population.

3.4. **Global stability of the DFE.** The model system (3), has a drug free equilibrium given by

$$DFE = \left(\frac{\pi}{\mu}, 0, 0, 0, 0\right). \tag{19}$$

The DFE exists whenever R_{0UD} is less than unity. This means that a small influx of drug users into the system does not lead to escalation of the population of drug users. The condition $R_{0UD} < 1$, means that the subsequent generation of the population of drug users is less than their predecessors. This Indicates that under favourable conditions and intervention measures, drug use can be eradicated. Therefore, the drug free equilibrium will be globally asymptotically stable whenever it exists and this leads to the following theorem.

Theorem 3.3. *The drug free equilibrium of model system (3) whenever it exists for $R_{0UD} < 1$, it is globally asymptotically stable.*

Proof. To show global stability of the DFE, we choose a suitable Lyapunov function

$$V = aL + bH + cT + dD,$$

where the Lyapunov coefficients are such that $a, b, c, d > 0$. The corresponding derivative of the Lyapunov function is given by

$$\begin{aligned}\frac{dV}{dt} &= a\frac{dL}{dt} + b\frac{dH}{dt} + c\frac{dT}{dt} + d\frac{dD}{dt},\\ &= a\pi S + (b\sigma - aQ_1 + d\alpha_2)L + (a\gamma_2 - bQ_2 + c\rho + d\alpha_3)H\\ &\quad + (b\gamma_3 - cQ_3)T + (a\alpha_1 S - Q_4 d)D.\end{aligned} \tag{20}$$

We linearise the Lyapunov derivative (20) at the drug free equilibrium. We note that near the DFE, $S \leq \frac{\pi}{\mu}$ and therefore, $\frac{S}{N} \leq 1$. Using this relation, we obtain

$$\begin{aligned}\frac{dV}{dt} &\leq (a\beta + b\sigma - aQ_1 + d\alpha_2)L + (a\beta\eta_1 + a\gamma_2 - bQ_2 + c\rho + d\alpha_3)H\\ &\quad + (a\beta\eta_2 + b\gamma_3 - cQ_3)T + \left(a\alpha_1\frac{\pi}{\mu} - Q_4 d\right)D.\end{aligned} \tag{21}$$

We choose the coefficients a, b, c, d such that the coefficients of H, T and D are zero. We thus obtain

$$a = \mu Q_2 Q_3 Q_4(1-\Phi_1), \qquad b = \mu Q_3 Q_4(\beta\eta_1 + \gamma_2) + \rho\mu Q_4\beta\eta_2 + \alpha_1\pi\alpha_3\mu Q_3,$$
$$c = \frac{\mu Q_2 Q_3 Q_4(1-\Phi_1)\beta\eta_2 + b\gamma_3}{Q_3}, \quad d = \alpha_1\pi\mu Q_2 Q_3(1-\Phi_1).$$

We now substitute the coefficients in (21) to obtain

$$\frac{dV}{dt} \leq \mu Q_1 Q_2 Q_3 Q_4(1-\Phi_1-\Phi_2)\left[R_{0UD} - 1\right]L.$$

Clearly, $\frac{dV}{dt} \leq 0$ whenever $R_{0UD} \leq 1$ with equality only when $R_{0UD} = 1$ or $L = 0$. Thus, according to the LaSalle Invariance principle [5], the DFE is globally asymptotically stable. This completes the proof. □

4. **Drug persistent equilibrium.** At the drug persistent equilibrium, the system satisfies

$$0 = \pi + \gamma_1 L^* - \Lambda^* S^* - \alpha_1 S^* D^* - \mu S^*, \tag{22}$$
$$0 = \Lambda^* S^* + \alpha_1 S^* D^* + \gamma_2 H^* - Q_1 L^*, \tag{23}$$
$$0 = \sigma L^* + \gamma_3 T^* - Q_2 H^*, \tag{24}$$
$$0 = \rho H^* - Q_3 T^*, \tag{25}$$
$$0 = \alpha_2 L^* + \alpha_3 H^* - Q_4 D^*. \tag{26}$$

From equations (24), (25) and (26) we have

$$H^* = \xi_1 L^*, \quad \text{where} \quad \xi_1 = \frac{\sigma}{Q_2(1-\Phi_1)}, \tag{27}$$
$$T^* = \xi_2 L^*, \quad \text{where} \quad \xi_2 = \frac{\rho}{Q_3}\xi_1, \tag{28}$$
$$D^* = \xi_3 L^* \quad \text{where} \quad \xi_3 = \frac{\alpha_2 Q_2(1-\Phi_1) + \alpha_3\sigma}{Q_2 Q_4(1-\Phi_1)}. \tag{29}$$

Using the resultant equations (27) and (28) and expression for the initiation function, equation (1), the initiation function at the endemic equilibrium can be given by

$$\Lambda^* = \Psi_1 \frac{L^*}{N^*} \quad \text{where} \quad \Psi_1 = \beta(1 + \eta_1\xi_1 + \eta_2\xi_2). \tag{30}$$

This can also be given in terms of the component R_{0U} of the reproduction number as

$$\Lambda^* = \Psi_2 R_{0U} \frac{L^*}{N^*} \quad \text{where} \quad \Psi_2 = \frac{Q_1\left[1 - (\Phi_1 + \Phi_2)\right]}{(1 - \Phi_1)}. \tag{31}$$

From equation (23)

$$\Psi_2 R_{0U} \frac{L^* S}{N^*} + \alpha_1\xi_3 S L^* + \gamma_2\xi_1 L^* - Q_1 L^* = 0,$$
$$L^*\left[\frac{\Psi_2 R_{0U} S^*}{N^*} + \alpha_1\xi_3 S^* + \gamma_2\xi_1 - Q_1\right] = 0.$$

Either $L^* = 0$ or

$$S^*\left(\frac{\Psi_2 R_{0U}}{N^*} + \frac{\mu\Psi_2 R_{0D}}{\pi}\right) = \Psi_2, \quad \Psi_2 > 0. \tag{32}$$

Note that the initiation function Λ^* is zero ($\Lambda^* = 0$) when

$$L^* = 0 \quad \Rightarrow H^* = T^* = 0, \quad \text{and} \quad S^* = \frac{\pi}{\mu}.$$

Consequently, the population of drug lords will reduce to low and consequently negligible values over time. This equilibrium is the drug free equilibrium as indicated in the expression (19). Using equations (22) and (23), is can easily be shown that

$$S^* = \frac{\pi}{\mu} - J_1 L^* \quad \text{where} \quad J_1 = \frac{1}{\mu}\left[(\mu + \sigma) - \frac{\sigma\gamma_2}{Q_2(1 - \Phi_1)}\right] > 0. \tag{33}$$

From the expressions presented, we note that the equilibrium point

$$S^* = \frac{\pi}{\mu} - J_1 L^*,\ H^* = \xi_1 L^*,\ T^* = \xi_2 L^*,\ D^* = \xi_3 L^*, \tag{34}$$

can be uniquely determined from the value of L^*. At the DPE, we assume that the total population has reached limiting equilibrium such that

$$\lim_{t\to\infty} N = \frac{\pi}{\mu} \approx N^*. \tag{35}$$

If we substitute for N^* in equation (32), we obtain $S^* = \frac{\pi}{\mu R_{0UD}}$.

Using equation (33), the expression for L^* will be given by

$$L^* = \frac{\pi}{J_1\mu}\left(\frac{R_{0UD} - 1}{R_{0UD}}\right). \tag{36}$$

Therefore, the DPE, is given by

$$S^* = \frac{\pi}{\mu R_{0UD}},\ L^* = \frac{\pi}{J_1\mu}\left(\frac{R_{0UD} - 1}{R_{0UD}}\right),\ H^* = \frac{\sigma\pi}{Q_2 J_1\mu(1 - \Phi_1)}\left(\frac{R_{0UD} - 1}{R_{0UD}}\right)$$
$$T^* = \frac{\xi_2\pi}{J_1\mu}\left(\frac{R_{0UD} - 1}{R_{0UD}}\right),\ D^* = \frac{\xi_3\pi}{J_1\mu}\left(\frac{R_{0UD} - 1}{R_{0UD}}\right).$$

Theorem 4.1. *The model system (3) has a unique drug persistent equilibrium whenever $R_{0UD} > 1$ and no drug persistent equilibrium otherwise.*

4.1. **Local stability of the drug persistence equilibrium.** We use the center manifold theory described in [6] to determine the local stability of the endemic steady state. Let us consider the system of equations (3) with the bifurcation parameter ϕ such that

$$\frac{dx}{dt} = f(x,\phi), f : \mathbb{R}^5 \times \mathbb{R} \to \mathbb{R} \quad \text{and} \quad f \in C^2(\mathbb{R}^5 \times \mathbb{R}). \tag{37}$$

Clearly, if 0 is the steady state of system (3), then $f(0,\phi) = 0$ for all ϕ. Let the linearisation matrix, $\mathbf{A}$

$$\mathbf{A} = D_x f(0,0), \tag{38}$$

have a left eigenvector denoted by y and the right eigenvector denoted by v. Then the local dynamics of the model around 0 is totally governed by $\mathbf{a}$ and $\mathbf{b}$ [6, 28], where

$$\mathbf{a} = \sum_{k,i,j=1} y_k v_i v_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \tag{39}$$

$$\mathbf{b} = \sum_{k,i,j=1} y_k v_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0). \tag{40}$$

Let us now define the terms (S, L, H, T) as (x_1, x_2, x_3, x_4). Then the system (3) can be rewritten as

$$f_1 = \pi + \gamma_1 x_2 - \beta \left(\frac{x_2 + \eta_1 x_3 + \eta_2 x_4}{\sum_{i=1}^4 x_i} \right) x_1 - \alpha_1 x_1 x_5 - \mu x_1, \tag{41}$$

$$f_2 = \beta \left(\frac{x_2 + \eta_1 x_3 + \eta_2 x_4}{\sum_{i=1}^4 x_i} \right) x_1 + \gamma_2 x_3 + \alpha_1 x_1 x_5 - Q_1 x_2, \tag{42}$$

$$f_3 = \sigma x_2 + \gamma_3 x_4 - Q_2 x_3, \tag{43}$$

$$f_4 = \rho x_3 - Q_3 x_4, \tag{44}$$

$$f_5 = \alpha_2 x_2 + \alpha_3 x_3 - Q_4 x_5. \tag{45}$$

We evaluate the bifurcation parameter ϕ by equating R_0 to one to obtain

$$\phi = \beta^* = \frac{\mu Q_1 Q_2 Q_3 Q_4 [1 - (\Phi_1 + \Phi_2)] - \alpha_1 \pi Q_3 (\alpha_2 Q_2 (1 - \Phi_1) + \sigma \alpha_3)}{\mu [Q_2 Q_3 Q_4 (1 - \Phi_1) + \eta_\sigma Q_3 + \eta_2 \rho \sigma]}. \tag{46}$$

We linearise the system of equations (3) at the drug free equilibrium and with the bifurcation parameter ϕ to obtain

$$J = \begin{pmatrix} -\mu & -\beta^* & -\beta^* \eta_1 & -\beta^* \eta_2 & -\alpha_1 \frac{\pi}{\mu} \\ 0 & \beta^* - Q_1 & \beta^* \eta_1 + \gamma_2 & \beta^* \eta_2 & \alpha_1 \frac{\pi}{\mu} \\ 0 & \sigma & -Q_2 & \gamma_3 & 0 \\ 0 & 0 & \rho & -Q_3 & 0 \\ 0 & \alpha_2 & \alpha_3 & 0 & -Q_4 \end{pmatrix}. \tag{47}$$

The matrix (47) has left eigenvectors $y = (y_1, y_2, y_3, y_4, y_5)$, where

$$\begin{aligned}
y_1 &= 0,\\
y_2 &= Q_2Q_3\mu Q_4(1-\Phi_1),\\
y_3 &= \mu^2\left[\beta^*\eta_1(Q_3+\rho)+Q_3\gamma_2\right]+\alpha_1\alpha_3\pi Q_3,\\
y_4 &= \beta^*\eta_2Q_2\mu^2(1-\Phi_1)+\frac{\gamma_3}{Q_3}y_3,\\
y_5 &= \alpha_1\pi Q_2Q_3(1-\Phi_1).
\end{aligned}$$

The right eigenvector associated with the zero eigenvalue of (47) is $v = (v_1, v_2, v_3, v_4)$ where

$$\begin{aligned}
&v_1 = Q_1Q_2Q_3Q_4(1-\Phi_1-\Phi_2)[R_c-R_{0UD}] \quad \text{for} \quad R_c = \frac{\gamma_1(1-\Phi_1)}{Q_1(1-\Phi_1-\Phi_2)},\\
&v_2 = \mu Q_2Q_3Q_4(1-\Phi_1), \qquad v_3 = Q_3Q_4\sigma\mu,\\
&v_4 = \sigma\mu\rho Q_4, \qquad v_5 = \alpha_2\mu Q_2Q_3(1-\Phi_1)+\alpha_3\mu Q_3\sigma.
\end{aligned}$$

We now evaluate the non-zero second order mixed derivatives of f_is with respect to the state variables to we obtain

$$\frac{\partial^2 f_1}{\partial x_2\partial x_3} = \frac{\partial^2 f_1}{\partial x_3\partial x_2} = \beta^*\frac{(1+\eta_1)\mu}{\pi}, \qquad \frac{\partial^2 f_1}{\partial x_2\partial x_4} = \frac{\partial^2 f_1}{\partial x_4\partial x_2} = \beta^*\frac{(1+\eta_2)\mu}{\pi}, \tag{48}$$

$$\frac{\partial^2 f_1}{\partial x_3\partial x_4} = \frac{\partial^2 f_1}{\partial x_4\partial x_3} = \beta^*\frac{(\eta_1+\eta_2)\mu}{\pi}, \qquad \frac{\partial^2 f_1}{\partial x_1\partial x_5} = \frac{\partial^2 f_1}{\partial x_5\partial x_1} = -\alpha_1, \tag{49}$$

$$\frac{\partial^2 f_2}{\partial x_2\partial x_3} = \frac{\partial^2 f_2}{\partial x_3\partial x_2} = -\beta^*\frac{(1+\eta_1)\mu}{\pi}, \qquad \frac{\partial^2 f_2}{\partial x_2\partial x_4} = \frac{\partial^2 f_2}{\partial x_4\partial x_2} = -\beta^*\frac{(1+\eta_2)\mu}{\pi}, \tag{50}$$

$$\frac{\partial^2 f_2}{\partial x_3\partial x_4} = \frac{\partial^2 f_2}{\partial x_4\partial x_3} = -\beta^*\frac{(\eta_1+\eta_2)\mu}{\pi}, \qquad \frac{\partial^2 f_2}{\partial x_1\partial x_5} = \frac{\partial^2 f_2}{\partial x_5\partial x_1} = \alpha_1. \tag{51}$$

The non-zero partial derivatives to used in calculating $\mathbf{b}$ are

$$\frac{\partial^2 f_1}{\partial x_2\partial\phi} = -, 1 \qquad \frac{\partial^2 f_2}{\partial x_2\partial\phi} = 1, \tag{52}$$

$$\frac{\partial^2 f_1}{\partial x_3\partial\phi} = -\eta_1, \qquad \frac{\partial^2 f_2}{\partial x_3\partial\phi} = \eta_1, \tag{53}$$

$$\frac{\partial^2 f_1}{\partial x_4\partial\phi} = -\eta_2, \qquad \frac{\partial^2 f_2}{\partial x_4\partial\phi} = \eta_2. \tag{54}$$

We now substitute the expressions (48)-(54) into (39) and (40) to obtain

$$\begin{aligned}
\mathbf{a} &= -2Q_2Q_3^2\beta^*(1-\Phi_1)\frac{\mu^3}{\sigma\pi}\left[Q_2Q_{3(1-\Phi_1)}(1+\eta_1)+Q_2(1-\Phi_1)(1+\eta_2)+\right.\\
&\quad \left.\sigma\rho(\eta_1+\eta_2)\right]-2\frac{Q_1Q_2^2Q_3^2(1-\Phi_1)\alpha_1}{\sigma}\left(\alpha_2Q_2(1-\Phi)+\alpha_3\sigma\right)(1-\Phi_1-\Phi_2)R_{0UD},\\
\mathbf{b} &= Q_2Q_3\mu^2(1-\Phi_1)\left[\frac{Q_2Q_3(1-\Phi_1)}{\sigma}+Q_3\eta_1+\rho\eta_2\right].
\end{aligned}$$

Clearly, we observe that $a < 0$ and $b > 0$. Thus, the drug persistent steady state is locally asymptotically stable when the reproduction number is close to 1. We thus have the following result,

Theorem 4.2. *The drug persistent steady state is locally asymptotically stable when* $R_{0UD} > 1$ *but only if* R_{0UD} *is close to 1.*

5. **Numerical results.** In this section we illustrate the theoretical results of the model by numerically integrating the model system (3). We note that drug use trends differ from one part of the world to the other. For example, the reasons that may prompt an individual to quite cannabis use in Amsterdam-Netherlands may be totally different from those of a quitting individual in Cape Town-South Africa. Therefore, although some of the parameters used may cut across borders, we limit most of them to South Africa. Some of these parameters include demographic parameters μ, δ_1 and δ_2. These are important regulators of the population of individuals susceptible to using drugs as well as drug users.

5.1. **Parameter estimation.** The current average life expectancy in South Africa is approximately 50 years [20]. Therefore, the corresponding mortality rate is $\mu = 0.02$ per year. The time individuals engage in high risk behaviour under the influence of drugs is not known, and varies between individuals depending on the type of drugs and amount they consume [19]. Therefore, precise estimation of mortality and removal rates related to substance abuse is difficult. In [4], mortality rates related to drugs among crack-cocaine users and injecting drug users are 0.018 and 0.008 respectively. According to [27], a man who stops smoking at 35 years of age can increase his life expectancy by 5 years. We assume a reduction of life expectancy by 14% due to general substance abuse since most of the drug users tend to be multiple drug users. With our estimated value for μ, the mortality rate of heavy users related to substance abuse δ_1 is $0.0014yr^{-1}$. Noting that treatment improves the quality of life, we assume that treatment reduces mortality rate related to drugs by at least 50%. Thus, we choose $\delta_2 = 0.003yr^{-1}$. In [7], the progression rate from light use to heavy use is 0.47%. This incorporates progressions from light use to moderate use and then escalation to heavy use. On the other hand, the value used was 0.024 in [1], and between $(0.003, 0.004)$ in [28] specifically for methamphetamine users. We choose a progression rate of 0.56 for general substance abuse. The observed treatment demand for cannabis users was in 17% [30]. There was an observed increase in cannabis use in the first two quarters of 2008 in South Africa, and including alcohol, the observed treatment demand for cannabis accounted for 23.5% of all substance abuse [38]. According to [37] treatment of drug users accounted for 20% of all Medicare hospitalisations globally. We assume that this value corresponds to the treatment demand. In [28], the upper limit of the treatment demand on the interval $(0.009, 0.3)$ for methamphetamine users corresponds to 30% treatment rate. In this paper, we choose the average treatment demand of 22.3% as the corresponding treatment rate of 0.223. The summary of parameter values (per year) used in model numerical integration is given in Table 2.

5.2. **Sensitivity analysis of the model to parameters.** To carry out sensitivity analysis of the model to the parameters, we use the change in R_{0UD} to variation of the input model parameters, using the Latin Hypercube Sampling scheme (LHS). This is an efficient method that enables us to analyse the uncertainty that occurs in ranges parameter values [3], taking into account the simultaneous combined variability in these input parameters [18]. We evaluate Partial Rank Correlation Coefficients (PRCCs) with 1000 simulations per run. In our uncertainty analysis and Monte Carlo simulation to generate data for the different input parameters,

TABLE 2. Nominal values of parameters used in the model

Parameter	nominal value	Range	Source
π	0.04	0.028-0.080	[28]
η_1	0.8	0-1	Estimated
η_2	0.6	0-1	Estimated
β	0.105	0.10-0.21	[28]
σ	0.56	0.40-0.70	Estimated
ρ	0.223	0.17-0.30	[28, 37, 38]
k	0.20	0.15-0.50	Estimated
μ	0.02	0.019-0.021	[20]
δ_1	0.0014	0.0-0.01	Estimated
δ_2	0.003	0.0-0.01	Estimated
γ_1	0.20	0.10-0.60	Estimated
γ_2	0.4	0.2-0.5	Estimated
γ_3	0.25	0.2-0.5	Estimated
α_1	0.4	0-1	Estimated
α_2	0.04	0-1	Estimated
α_3	0.08	0-1	Estimated
r	0.05	0-1	Estimated

we assume statistical independence of the parameters. The graphical display of the output of our simulation is indicated in the tornado plot, Figure 2.

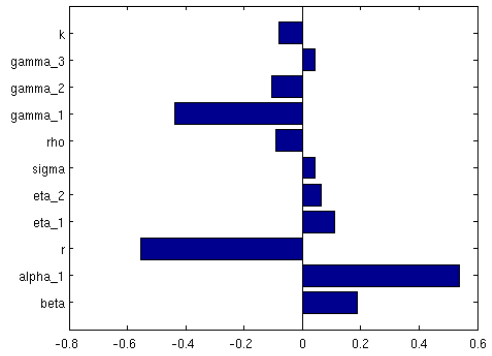


FIGURE 2. Tornado plot showing the Partial Rank Correlation Coefficients (PRCCs) for a selected range of model parameters in Table 2.

The parameter values β, α_1, η_1, η_2 and σ with positive PRCCs increase the value of R_{0UD} if their values are increased. In this case therefore, the parameters α_1 and β have the highest influence in increasing the value of R_{0UD} when they are increased.

Although, the parameters may have either positive or negative PRCCs, it is important to ascertain whether the monotone increasing or monotone decreasing trend is significant when such parameters are varied. To ascertain this for the

four parameters with the highest absolute values of the PRCCs, we produce their respective scatter plots. Our results are presented in Figure 3.

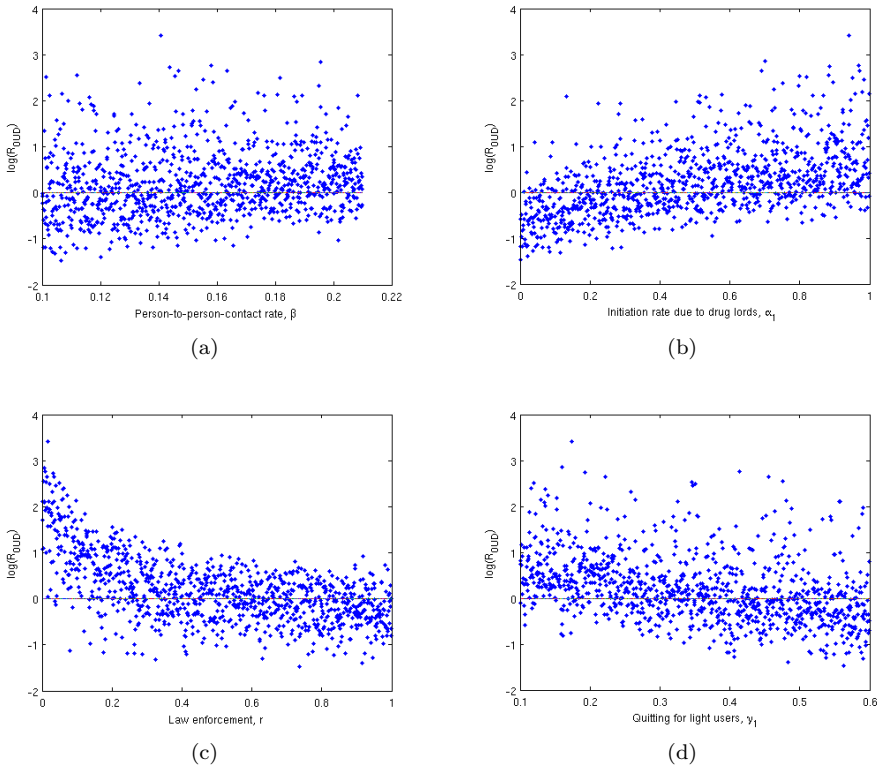


FIGURE 3. Monte Carlo simulations for the four parameters with the greatest absolute values of PRCC, obtained using parameter values in Table 2. In each run, 1000 simulations were used.

Although R_{0UD} is observed to be monotone increasing with increase in drug user-susceptible contact rate (β) and drug lord-susceptible contact rate, α_1, the trend with respect to α_1 is much stronger. This has strong implications regarding presence of drugs in the population with respect to accessibility and motivation to using them as opposed to simple initiation into drug use due to contagion.

Influence of law enforcement exhibits a strong monotone decrease of R_{0UD} when increased. This has strong influence in reducing the prevalence of drug epidemics. See also Figures 5 and 6. In the same way, quitting of light users is vital if drug epidemics are to be contained.

The scatter plot, Figure 3(d) shows a significant decrease in R_{0UD} for the selected range of γ_1 values. This has implications in that control measures should aim at targeting light users encouraging them to quit.

5.3. **Long term behaviour.** We consider the long term behaviour of the models using numerical simulations. The model is numerically integrated using the integration routine odeint in python scipy. We vary the initial population of light

drug users in our set of values of the initial population. Numerical results of our simulation are indicated in Figure 4.

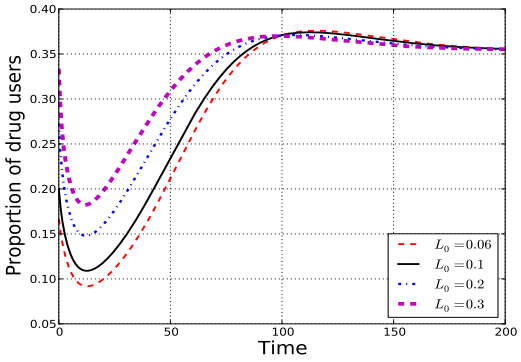


FIGURE 4. Prevalence of substance abuse with different initial population of light drug users

The long term behaviour of the model for differing initial conditions is characterised by convergence of the proportion of substance users in the population to a common equilibrium point. On the other hand, influence of parameters values is investigated assuming non-varying initial conditions. We however vary the parameter of interest to ascertain its influence while keeping the rest the parameters constant as indicated below.

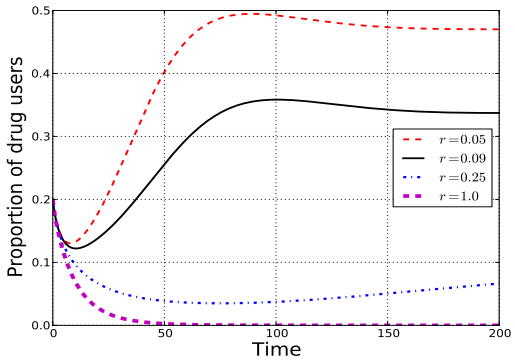
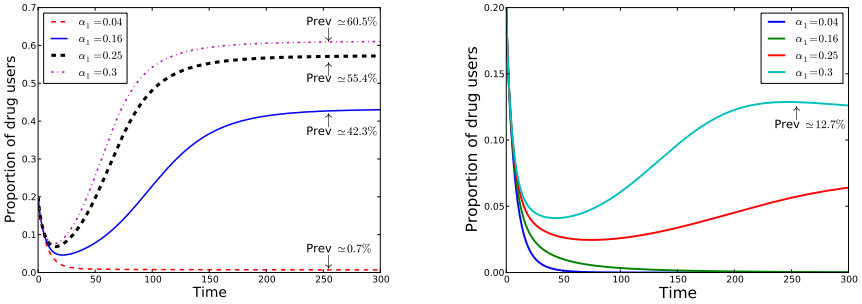


FIGURE 5. The impact of law enforcement on the prevalence of substance abuse

To investigate the relationship between law enforcement and contact between drug lords and potential initiates, we vary the contact rate α_1 between drug lords and drug users from 0.04 to 0.3 and present results in Figure 6. The prevalence is observed to increase from undetectable levels to 12.7% (Figure 6(a)). When compared with the case in absence of law enforcement but for similar contact rates between drug lords and drug users, the prevalence of substance abuse is observed to increase from 0.7% to 60.5% (Figure 6(b)). In the same way, we observe from Figures



(a) Prevalence of substance abuse without law enforcement, ($r = 0$).

(b) Prevalence of substance abuse with law enforcement ($r = 0.08$).

FIGURE 6. Shows that, given the same initiation potential by drug lords, effective policing and law enforcement, will always keep the prevalence of drug abuse lower than it would have been without law enforcement.

5 and 6 that, increasing law enforcement reduces prevalence. The reason for such a decrease is due to the fact that when the population of drug lords reduces, so does the supply of substances to potential users and the probability that a susceptible individual will meet a drug lord or acquire an addictive substance consequently reduces.

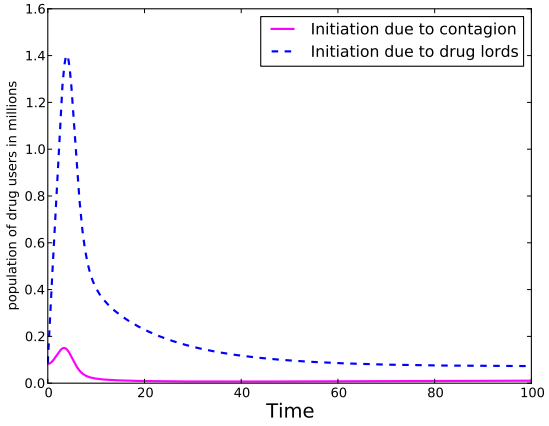


FIGURE 7. Comparison of the routes of initiation into drug use.

The heights of the peaks of the two initiation routes in Figure 7 indicate that initiation resulting from access to drugs and interaction with drug lords could be as high as 7 times higher than initiation due to contagion. This difference is observed at the initial stage of the epidemic. In the long run however, toward the equilibrium state, the difference in initiations between the two initiation routes reduces but the influence of drug lords and supply of drugs remains dominant. We observe therefore,

that the availability of drugs in the community, enhances initiation and hence the persistence of substance abuse. In this case, if substance abuse is to be contained, it is ideal to target drug lords.

6. **Conclusion.** We presented a compartmental model for general substance abuse showing the influence of both drug lords and person-to-person contact on the prevalence of substance abuse. Qualitative evaluation of the effectiveness of law enforcement as a means of intervention on general substance abuse epidemic was evaluated. The resulting plots on law enforcement are out of the motivation based on scenario analysis. In this process, we analysed the possible outcomes (trend of drug use prevalence) by considering alternative input values of law enforcement. The results indicate that stepping up law enforcement reduces the prevalence of substance abuse. Therefore, in the ideal case, if drugs were completely unavailable, there would be no drug addiction, see also [14]. In the same way reducing the number of drug lords/drug supplies reduces the probability of a susceptible individual being initiated into substance abuse by a drug lord and or accessing drugs. Since the growth of the drug barons is demand driven, reduction in the demand can actually be a control in the fight against any drug epidemic. Sensitivity analysis of the model output to input or predictor parameter values was done using the Latin Hypercube Sampling and the Partial Rank Correlation Coefficients (PRCCs) calculated. The results were graphically presented in a tornado plot and scatter plots. Sensitivity analysis indicates that the parameters r, (law enforcement) and γ_1, (quitting for light drug users) significantly reduce the drug epidemic if increased. On the other hand β (person-to-person contact rate) and α_1 (drug lord-potential drug user contact rate) worsen the drug epidemics if increased. It is therefore important to; step up law enforcement so as to reduce the supply of drugs hence reducing access by potential users; increase awareness targeting light drug users, encouraging them to quit so as to contain the epidemic.

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