Int. J. Appl. Comput. Math (2018) 4:47
https://doi.org/10.1007/s40819-017-0477-3

ORIGINAL PAPER

Modelling Gender Differences in Drug Abuse Epidemics

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Published online: 11 January 2018

Abstract Drug abuse is an issue of considerable concern due to its association with numerous public health problems. Mathematical models developed to describe the spread of drug abuse have generally assumed that the dynamics of drug use and treatment are substantially the same for women as men. However, research has revealed that the dynamics of women's drug use and treatment are different in many ways from that of men's. Understanding gender differences in patterns of drug use is essential to identify the influences of gender on the trends of drug abuse in order to develop appropriate and effective prevention programs. We formulate a sex structured compartmental model for the spread of drug abuse using nonlinear ordinary differential equations. The least squares curve fit routine (lsqcurvefit) in Matlab with optimization is used to estimate the parameter values. The model is fitted to data on individuals under substance abuse treatment centres of the Western Cape Province of South Africa and parameter values that give the best fit chosen. The projections carried out the long term trends of proportions for male and female rehabilitants. The results show that the proportion of male drug abusers in Cape Town is expected to continue to decrease whereas that of female drug abusers shall continue to increase but steadily. The estimated proportion of female drug abusers in specialist treatment centres of Cape Town was observed to be approximately 34% by the year 2030.

Keywords Drug abuse · Gender differences · Abuse reproduction number · Least squares curve fitting

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Introduction

Historically, drug research has focused mostly on males as compared to females and drug treatment programs have been based upon the concerns of men [9]. Neglect of women in the area of drugs has been typified by the "too small numbers" argument, that is, too few women drug users to be a serious enough problem to warrant attention and/or justify special services [9]. From the onset, researchers and practitioners generally assumed that the dynamics of drug abuse and treatment were substantially the same for women as men, that is, it simply never occurred to some researchers that there might be variations on the basis of gender. Thus, the literature on gender differences in substance abuse treatment has been limited [30,39]. Drug abuse research is oriented almost exclusively to males [36]. In the United States of America, concerns for women drug users began to gain attention in the 1970s. In 1974, the National Institute on Drug Abuse (NIDA) developed a program for women's concerns and new training initiatives for drug treatment service providers were developed in 1985 [18]. In South Africa, data collected by the South African Community Epidemiology Network on Drug Use (SACENDU) [35] reveals women's widespread involvement in drugs making women drug use and treatment an issue of credible concern. Though the data collected by SACENDU indicate that the majority of clients in substance abuse treatment programs are men, the number of female clients appear to be rising steadily over the years. This fact gives rise to the need of more research on substance abuse treatment that includes women so that treatment programs are structured to suit the needs of both men and women. According to the Substance Abuse and Mental Health Services Administration (SAMHSA) [32], men are more likely than women to use almost all types of illicit drugs. The Treatment Episode Data Set (TEDS) [34], reveals that for most age groups, men have higher rates of use or dependence on illicit drugs and alcohol than do women. However, women are just as likely as men to become addicted [1].

In recent years, various mathematical models describing the spread of drug abuse have been proposed, see for example, [13,19–24,40]. In these models, the spread of misbehaviors is assumed to have mechanisms similar to epidemic diffusion. There are similarities between the spread of drug use and that of infectious diseases [6,16]. Compartmental models describing the spread of drug abuse have been formulated with an SIS or SIR structure or a combination of both. The compartments in these drug abuse models have not been split to consider male and female individuals separately. In other words, the basic assumption in all these models is that there are no gender differences with regards to susceptibility to drug abuse, levels of addiction and treatment uptake. This is not true. Several drug related data sources indicate significant gender imbalances for susceptibility to drug abuse, levels of addiction and treatment uptake [7,10,15,35]. Research has also revealed that the dynamics of women's drug use are different in many ways from that of men's [29,31]. Suffet and Brotman [33] reported, for example, that women and men are initiated differently into illicit drug use. Whereas men are usually introduced to drugs by male peers, in a study by [9], most women reported being turned on by males, often a boyfriend. In this paper, we formulate a more realistic compartmental model for drug abuse that takes into account gender differences in the dynamics of drug abuse. The model explicitly captures the contact pattern between males and females initiating drug abuse. Compared to previous mathematical models of drug abuse, a key novelty of our model is the inclusion of sex structured compartments to explore the impact of gender differences on the spread of drug abuse. Inclusion of such sex structured groups increases the realism of the model and allows trends of drug abuse to be examined in the respective sub groups. Detailed analysis of gender differences in the dynamics of drug abuse can yield important information

about changing lifestyles in relation to patterns of drug use and about the potential efficacy of drug prevention and treatment services for different groups.

The paper is arranged as follows; in "Model Formulation" section, we formulate and establish the basic properties of the model. The model is analysed for stability in "Model Analysis" section. In "Numerical Simulations" section, we carry out some numerical simulations. Parameter estimation is also presented in this section. The paper is concluded in "Conclusion" section.

Model Formulation

We propose a mathematical model that takes into account the spread of drug abuse amongst males and females. We include the class of individuals under treatment so as to assess the role of rehabilitation in controlling the spread of drug abuse. In this paper we assume that treatment is on an 'inpatient' basis and individuals are released from treatment centers when they have recovered. The human population is divided into eight sub-populations, $S_m(t), U_m(t), T_m(t), R_m(t), S_f(t), U_f(t), T_f(t)$ and $R_f(t)$. The class $S_m(t)/S_f(t)$ represents the male/female population at risk of being initiated into drug abuse, $U_m(t)/U_f(t)$ denotes males/females initiated into drug abuse together with relapsed male/female drug users. The class $T_m(t)/T_f(t)$ represents male/female clients of rehabilitation services in treatment and $R_m(t)/R_f(t)$ denotes the class of recovered males/females. It is upon this stage that a relapse can only occur. The total population is thus given by

$$N(t) = N_m(t) + N_f(t)$$

where

$$N_m(t) = S_m(t) + U_m(t) + T_m(t) + R_m(t) \text{ and } N_f(t) = S_f(t) + U_f(t) + T_f(t) + R_f(t)$$

with $N_m(t)$ and $N_f(t)$ being the total number of males and females respectively. The rate at which the general population enter the susceptible population, that is, the demographic process of individuals reaching age 15 years in the modelling time period is represented by Λ, a proportion p being males and the complementary proportion $(1-p)$ entering the female population. Susceptible males are initiated into drug abuse following contact with male drug users at a rate $\lambda_m = \frac{\beta_m U_m}{N_m}$ or upon contact with female drug users at a rate $\lambda_{f_m} = \frac{\beta_{fm} U_f}{N}$. The per capita contact rate β_m is a product of the effective number of contacts c_m, between male drug users and the susceptible male population, and the probability $\hat{\beta}_m$, that such a contact results into initiation into drug use, that is $\beta_m = c_m \hat{\beta}_m$. The per capita contact rate β_{f_m} is a product of the effective number of contacts c_{f_m}, between female drug users and the susceptible male population, and the probability $\hat{\beta}_{f_m}$, that such a contact results into initiation into drug use, that is $\beta_{f_m} = c_{f_m} \hat{\beta}_{f_m}$. Upon being initiated into drug use, a susceptible male moves into the compartment U_m, of male drug abusers. Susceptible females are initiated into drug abuse following contact with female drug users at a rate $\lambda_f = \frac{\beta_f U_f}{N_f}$ or upon contact with male drug users at a rate $\lambda_{m_f} = \frac{\beta_{m_f} U_m}{N}$. The per capita contact rate β_f is a product of the effective number of contacts c_f, between female drug users and the susceptible female population, and the probability $\hat{\beta}_f$, that such a contact results into initiation into drug use, that is $\beta_f = c_f \hat{\beta}_f$. The per capita contact rate β_{m_f} is a product of the effective number of contacts c_{m_f}, between male drug users and the susceptible female population, and the probability $\hat{\beta}_{m_f}$, that such a contact results into initiation into drug use, that is $\beta_{m_f} = c_{m_f} \hat{\beta}_{m_f}$. Upon being initiated into

drug use, a susceptible female moves into the compartment U_f, of female drug abusers. In this paper, we shall assume that male drug users have more contact with susceptible males as compared to susceptible females. Thus, we can safely assume that $\beta_{m_f} = \varepsilon_m \beta_m$ which corresponds to $\lambda_{m_f} = \varepsilon_m \lambda_m$ where $0 < \varepsilon_m < 1$. Similarly, we assume that female drug users have more contact with susceptible females as compared to susceptible males. Thus, we can also safely assume that $\beta_{f_m} = \varepsilon_f \beta_f$ which corresponds to $\lambda_{f_m} = \varepsilon_f \lambda_f$ where $0 < \varepsilon_f < 1$. Removal from the male/female drug users' class that include drug related death rate is represented by r_m/r_f. The natural recovery rate for male/female drug abusers is given by δ_m/δ_f. The rate at which male/female drug users are recruited into rehabilitation is given by σ_m/σ_f. Recovery rate for male/female drug users under treatment is given by γ_m/γ_f. The mean rate at which recovered males/females relapse into drug use is represented by ρ_m/ρ_f. Individuals experience natural death at a rate μ. The model involves two assumptions which are of critical importance and these are:

- Individuals in each compartment are indistinguishable and there is homogeneous mixing so that those at risk of drug use are equally susceptible. In practice, susceptibility to drug use varies. This is due to differences in behavioral, social and environmental factors.
- Drug users in treatment use drugs but cannot initiate non-drug users since they are completely immersed in the program and separated from the general population.

The schematic diagram below shows the movement of humans as their status with respect to drug use changes (Fig. 1).

Combining the parameters, assumptions and the schematic diagram, we obtain the following set of nonlinear ordinary differential equations:

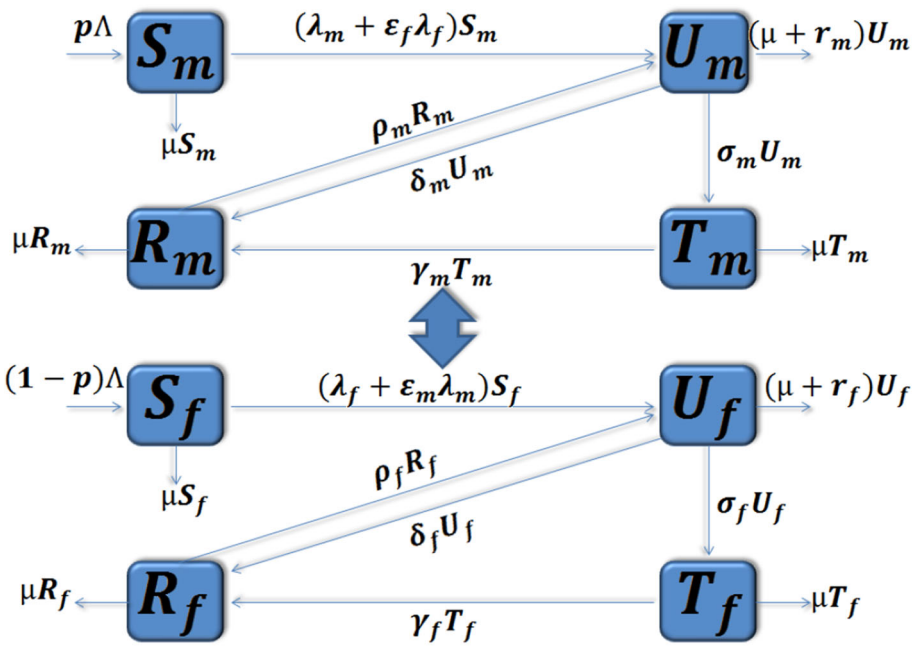

Fig. 1 Model flow diagram

$$\begin{cases} \dfrac{dS_m}{dt} = p\Lambda - \left(\lambda_m + \varepsilon_f \lambda_f\right) S_m - \mu S_m, \\ \dfrac{dU_m}{dt} = \left(\lambda_m + \varepsilon_f \lambda_f\right) S_m + \rho_m R_m - (\mu + r_m + \delta_m + \sigma_m) U_m, \\ \dfrac{dT_m}{dt} = \sigma_m U_m - (\mu + \gamma_m) T_m, \\ \dfrac{dR_m}{dt} = \gamma_m T_m + \delta_m U_m - (\mu + \rho_m) R_m, \\ \dfrac{dS_f}{dt} = (1-p)\Lambda - \left(\lambda_f + \varepsilon_m \lambda_m\right) S_f - \mu S_f, \\ \dfrac{dU_f}{dt} = \left(\lambda_f + \varepsilon_m \lambda_m\right) S_f + \rho_f R_f - (\mu + r_f + \delta_f + \sigma_f) U_f, \\ \dfrac{dT_f}{dt} = \sigma_f U_f - (\mu + \gamma_f) T_f, \\ \dfrac{dR_f}{dt} = \gamma_f T_f + \delta_f U_f - (\mu + \rho_f) R_f. \end{cases} \tag{1}$$

We assume that all the model parameters are positive and the initial conditions of the system (1) are given by

$$S_m(0) = S_{m0} > 0,\ U_m(0) = U_{m0} \geq 0,\ T_m(0) = T_{m0} \geq 0,\ R_m(0) = R_{m0} \geq 0,$$
$$S_f(0) = S_{f0} > 0,\ U_f(0) = U_{f0} \geq 0,\ T_f(0) = T_{f0} \geq 0,\ R_f(0) = R_{f0} \geq 0.$$

Model Analysis

Positivity of Solutions

We now consider the positivity of system (1). We prove that all the state variables remain non-negative and the solutions of system (1) with positive initial conditions will remain positive for all $t > 0$. We thus state the following theorem.

Theorem 1 *Given that the initial conditions of system* (1) *are* $S_m(0) > 0$, $U_m(0) > 0$, $T_m(0) > 0$, $R_m(0) > 0$, $S_f(0) > 0$, $U_f(0) > 0$, $T_f(0) > 0$ *and* $R_f(0) > 0$. *There exists* $(S_m(t), U_m(t), T_m(t), R_m(t), S_f(t), U_f(t), T_f(t), R_f(t)) : (0, \infty) \to (0, \infty)$ *which solve system* (1).

Proof Assume that

$$\hat{t} = \sup\{t >: S_m > 0,\ U_m > 0,\ T_m > 0,\ R_m > 0,\ S_f > 0, U_f > 0,\ T_f > 0,\ R_f > 0\} \in [0, t].$$

Thus $\hat{t} > 0$, and it follows from the first equation of system (1) that

$$\frac{dS_m}{dt} = p\Lambda - \left(\mu + \lambda_m + \varepsilon_f \lambda_f\right) S_m.$$

Thus

$$\frac{d}{dt}\left[S_m(t)\exp\left\{\mu t + \int_0^t \left(\lambda_m(s) + \varepsilon_f\lambda_f(s)\right) ds\right\}\right] = p\Lambda \exp\left[\mu t + \int_0^t \left(\lambda_m(s) + \varepsilon_f\lambda_f(s)\right) ds\right].$$

So

$$S_m(\hat{t})\exp\left[\mu\hat{t} + \int_0^{\hat{t}} \left(\lambda_m(s) + \varepsilon_f\lambda_f(s)\right) ds\right] - S_m(0) = \int_0^{\hat{t}} p\Lambda \exp\left[\mu\hat{t} + \int_0^{\hat{t}} \left(\lambda_m(\nu) + \varepsilon_f\lambda_f(\nu)\right) d\nu\right] d\hat{t},$$

giving

$$S_m(\hat{t}) = S_m(0)\exp\left[-\left(\mu\hat{t} + \int_0^{\hat{t}} \left(\lambda_m(s) + \varepsilon_f\lambda_f(s)\right) ds\right)\right] + \exp\left[-\left(\mu\hat{t} + \int_0^{\hat{t}} \left(\lambda_m(s) + \varepsilon_f\lambda_f(s)\right) ds\right)\right]\left[\int_0^{\hat{t}} p\Lambda \exp\left[\mu\hat{t} + \int_0^{\hat{t}} \left(\lambda_m(\nu) + \varepsilon_f\lambda_f(\nu)\right) d\nu\right] d\hat{t}\right] > 0.$$

From the second equation of system (1), we obtain

$$\begin{aligned} \frac{dU_m}{dt} &= \left(\lambda_m + \varepsilon_f\lambda_f\right) S_m + \rho_m R_m - (\mu + r_m + \delta_m + \sigma_m)U_m \\ &\geq -(\mu + r_m + \delta_m + \sigma_m)U_m, \\ &\Rightarrow U_m(\hat{t}) \geq U_{m0}e^{-(\mu+r_m+\delta_m+\sigma_m)\hat{t}} > 0. \end{aligned}$$

In a similar fashion, it can also be shown that $T_m(t) > 0$, $R_m(t) > 0$, $S_f(t) > 0$, $U_f(t) > 0$, $T_f(t) > 0$ and $R_f(t) > 0$ for all $t > 0$, and this completes the proof. □

Invariant Region

It follows from system (1) that

$$\frac{dN}{dt} = \Lambda - \mu N - r_m U_m - r_f U_f.$$

Note that $\frac{dN}{dt} \leq \Lambda - \mu N$. Using a theorem by Birkhoff and Rota [2] on differential inequality, it follows that $0 \leq N(t) \leq \frac{\Lambda}{\mu} - \frac{C}{\mu}e^{-\mu t}$, where C is a constant. Then, $\limsup_{t\to\infty} N \leq \frac{\Lambda}{\mu}$. Thus, the feasible region for system (1) is

$$\Omega = \left\{ (S_m, U_m, T_m, R_m, S_f, U_f, T_f, R_f) \in \mathbb{R}^8_+ \mid N \leq \frac{\Lambda}{\mu} \right\}. \tag{2}$$

It is easy to verify that the region Ω is positively invariant with respect to system (1).

Drug-Free Equilibrium and the Abuse Reproduction Number

The model has a drug-free equilibrium given by

$$\mathcal{G}_0 = \left(S_m^0, U_m^0, T_m^0, R_m^0, S_f^0, U_f^0, T_f^0, R_f^0 \right) = \left(\frac{p\Lambda}{\mu}, 0, 0, 0, \frac{(1-p)\Lambda}{\mu}, 0, 0, 0 \right),$$

a scenario depicting a drug-free state in the community or society. The abuse reproduction number $\mathcal{R}_a$ of the model, is defined herein in the drug-using context as the average number of people that each drug user will initiate to drug use during the drug-using career in a population of completely potential drug users. Usually, $\mathcal{R}_a < 1$ implies that drug abuse will die out, whereas $\mathcal{R}_a > 1$ implies that drug abuse will persist within a community and $\mathcal{R}_a = 1$ requires further investigation. The determination of $\mathcal{R}_a$ is done using the next generation matrix approach [37]. This method has been explored in many papers, see for instance [4,11,14,17,38]. Using this method we have

$$F = \begin{pmatrix} \beta_m & 0 & 0 & \frac{p\beta_f\epsilon_f}{1-p} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{(1-p)\beta_m\epsilon_m}{p} & 0 & 0 & \beta_f & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \text{ and}$$

$$V = \begin{pmatrix} g_{m_1} & 0 & -\rho_m & 0 & 0 & 0 \\ -\sigma_m & g_{m_2} & 0 & 0 & 0 & 0 \\ -\delta_m & -\gamma_m & g_{m_3} & 0 & 0 & 0 \\ 0 & 0 & 0 & g_{f_1} & 0 & -\rho_f \\ 0 & 0 & 0 & -\sigma_f & g_{f_2} & 0 \\ 0 & 0 & 0 & -\delta_f & -\gamma_f & g_{f_3} \end{pmatrix}$$

where

$$g_{m_1} = \mu + r_m + \delta_m + \sigma_m, \quad g_{m_2} = \mu + \gamma_m, \quad g_{m_3} = \mu + \rho_m,$$
$$g_{f_1} = \mu + r_f + \delta_f + \sigma_f, \quad g_{f_2} = \mu + \gamma_f, \quad g_{f_3} = \mu + \rho_f.$$

Thus, the abuse reproduction number is given by

$$\mathcal{R}_a = \frac{1}{2} \left(\mathcal{R}_m + \mathcal{R}_f + \sqrt{\left(\mathcal{R}_m - \mathcal{R}_f \right)^2 + 4\epsilon_f\epsilon_m\mathcal{R}_m\mathcal{R}_f} \right) \tag{3}$$

where

$$\mathcal{R}_m = \frac{\beta_m}{g_{m_1}(1-\Phi_m)} \text{ and } \mathcal{R}_f = \frac{\beta_f}{g_{f_1}\left(1-\Phi_f\right)} \tag{4}$$

with

$$\Phi_m = \frac{\rho_m \delta_m g_{m_2} + \rho_m \gamma_m \sigma_m}{g_{m_1} g_{m_2} g_{m_3}} \text{ and } \Phi_f = \frac{\rho_f \delta_f g_{f_2} + \rho_f \gamma_f \sigma_f}{g_{f_1} g_{f_2} g_{f_3}}. \tag{5}$$

We can clearly note that $\left(\rho_m \delta_m g_{m_2} + \rho_m \gamma_m \sigma_m\right) \leq \left(g_{m_1} g_{m_2} g_{m_3}\right)$ and $\left(\rho_f \delta_f g_{f_2} + \rho_f \gamma_f \sigma_f\right) \leq \left(g_{f_1} g_{f_2} g_{f_3}\right)$. Therefore, $\mathcal{R}_a$ is non-negative. The abuse reproduction number $\mathcal{R}_a$ of the model, is the average number of secondary cases generated by one drug user during his/her duration of drug use in a population of completely potential drug users. Here, $\mathcal{R}_a$ is a combination of two sub-reproduction numbers $\mathcal{R}_m$ and $\mathcal{R}_f$ representing the contributions of individuals in compartments U_m and U_f respectively. Theorem 2 follows from Driessche and Watmough [37] (Theorem 2).

Theorem 2 *The drug-free equilibrium point $\mathcal{G}_0$ of system (1) is locally asymptotically stable if $\mathcal{R}_a < 1$ and is unstable if $\mathcal{R}_a > 1$.*

Drug-Persistent Equilibrium

The drug-persistent equilibrium $\mathcal{G}^* = \left(S_m^*, U_m^*, T_m^*, R_m^*, S_f^*, U_f^*, T_f^*, R_f^*\right)$ always satisfies

$$\begin{cases} 0 = p\Lambda - \left(\lambda_m^* + \varepsilon_f \lambda_f^*\right) S_m^* - \mu S_m^*, \\ 0 = \left(\lambda_m^* + \varepsilon_f \lambda_f^*\right) S_m^* + \rho_m R_m^* - g_{m_1} U_m^*, \\ 0 = \sigma_m U_m^* - g_{m_2} T_m^*, \\ 0 = \gamma_m T_m^* + \delta_m U_m^* - g_{m_3} R_m^*, \\ 0 = (1-p)\Lambda - \left(\lambda_f^* + \varepsilon_m \lambda_m^*\right) S_f^* - \mu S_f^*, \\ 0 = \left(\lambda_f^* + \varepsilon_m \lambda_m^*\right) S_f^* + \rho_f R_f^* - g_{f_1} U_f^*, \\ 0 = \sigma_f U_f^* - g_{f_2} T_f^*, \\ 0 = \gamma_f T_f^* + \delta_f U_f^* - g_{f_3} R_f^*. \end{cases} \tag{6}$$

Here, the drug-persistent equilibrium $\mathcal{G}^*$ is given in terms of $\left(\lambda_m^*,\ \lambda_f^*\right)$ where

$$\begin{cases} S_m^* = \dfrac{p\Lambda}{\mu+\lambda_m^*+\epsilon_f\lambda_f^*}, \quad U_m^* = \dfrac{p\Lambda\left(\lambda_m^*+\epsilon_f\lambda_f^*\right)}{g_{m_1}\left(1-\Phi_m\right)\left(\mu+\lambda_m^*+\epsilon_f\lambda_f^*\right)}, \\ T_m^* = \dfrac{p\Lambda\sigma_m\left(\lambda_m^*+\epsilon_f\lambda_f^*\right)}{g_{m_1}g_{m_2}\left(1-\Phi_m\right)\left(\mu+\lambda_m^*+\epsilon_f\lambda_f^*\right)}, \\ R_m^* = \dfrac{p\Lambda\left(\lambda_m^*+\epsilon_f\lambda_f^*\right)\left(\gamma_m\sigma_m+\delta_m g_{m_2}\right)}{g_{m_1}g_{m_2}g_{m_3}\left(1-\Phi_m\right)\left(\mu+\lambda_m^*+\epsilon_f\lambda_f^*\right)}, \\ S_f^* = \dfrac{(1-p)\Lambda}{\mu+\lambda_f^*+\epsilon_m\lambda_m^*}, \quad U_f^* = \dfrac{(1-p)\Lambda\left(\lambda_f^*+\epsilon_m\lambda_m^*\right)}{g_{f_1}\left(1-\Phi_f\right)\left(\mu+\lambda_f^*+\epsilon_m\lambda_m^*\right)}, \\ T_f^* = \dfrac{(1-p)\Lambda\sigma_f\left(\lambda_f^*+\epsilon_m\lambda_m^*\right)}{g_{f_1}g_{f_2}\left(1-\Phi_f\right)\left(\mu+\lambda_f^*+\epsilon_m\lambda_m^*\right)}, \\ R_f^* = \dfrac{(1-p)\Lambda\left(\lambda_f^*+\epsilon_m\lambda_m^*\right)\left(\gamma_f\sigma_f+\delta_f g_{f_2}\right)}{g_{f_1}g_{f_2}g_{f_3}\left(1-\Phi_f\right)\left(\mu+\lambda_f^*+\epsilon_m\lambda_m^*\right)}. \end{cases} \tag{7}$$

Here,

$$\lambda_m^* = \frac{\beta_m U_m^*}{N_m^*} \text{ and } \lambda_f^* = \frac{\beta_f U_f^*}{N_f^*} \tag{8}$$

where $N^* = N_m^* + N_f^*$ with

$$N_m^* = \frac{p\Lambda\left(g_{m_1}g_{m_2}g_{m_3}\left(1-\Phi_m\right)+\left(\epsilon_f\lambda_f^*+\lambda_m^*\right)\left(\sigma_m\left(g_{m_3}+\gamma_m\right)+g_{m_2}\left(g_{m_3}+\delta_m\right)\right)\right)}{g_{m_1}g_{m_2}g_{m_3}\left(1-\Phi_m\right)\left(\mu+\epsilon_f\lambda_f^*+\lambda_m^*\right)} \tag{9}$$

and

$$N_f^* = \frac{(1-p)\Lambda\left(\left(\lambda_f^*+\epsilon_m\lambda_m^*\right)\left(\sigma_f\left(\gamma_f+g_{f_3}\right)+g_{f_2}\left(\delta_f+g_{f_3}\right)\right)+g_{f_1}g_{f_2}g_{f_3}\left(1-\Phi_f\right)\right)}{g_{f_1}g_{f_2}g_{f_3}\left(1-\Phi_f\right)\left(\mu+\lambda_f^*+\epsilon_m\lambda_m^*\right)}. \tag{10}$$

Using (7), (9) and expression for λ_m^* in (8) leads to the following equation

$$\begin{aligned} &g_{m_2}\left(g_{m_3}\left(\lambda_m^*\left(\lambda_f^*\epsilon_f+g_{m_1}\left(1-\Phi_m\right)+\lambda_m^*\right)-\beta_m\left(\lambda_f^*\epsilon_f+\lambda_m^*\right)\right)\right. \\ &\left.+\delta_m\lambda_m^*\left(\lambda_f^*\epsilon_f+\lambda_m^*\right)\right)+\lambda_m^*\sigma_m\left(g_{m_3}+\gamma_m\right)\left(\lambda_f^*\epsilon_f+\lambda_m^*\right)=0. \end{aligned} \tag{11}$$

Solving (11) for λ_f^* gives

$$\lambda_f^* = \frac{\lambda_m^* \left(g_{m_2}\left(g_{m_3}\left(\lambda_m^* - \beta_m\right) + \delta_m \lambda_m^*\right) + \lambda_m^* \sigma_m \left(g_{m_3} + \gamma_m\right) + g_{m_1} g_{m_2} g_{m_3} \left(1 - \Phi_m\right)\right)}{\epsilon_f \left(g_{m_2}\left(g_{m_3}\beta_m - \lambda_m^*\left(g_{m_3} + \delta_m\right)\right) - \lambda_m^* \sigma_m \left(g_{m_3} + \gamma_m\right)\right)}. \tag{12}$$

Similarly, using (7), (9) and expression for λ_f^* in (8) leads to the following equation

$$\begin{aligned}&\left(\lambda_f^* + \lambda_m \epsilon_m\right)\left(g_{f_2}\left(\delta_f \lambda_f^* + g_{f_3}\left(\lambda_f^* - \beta_f\right)\right) + \lambda_f^* \sigma_f \left(\gamma_f + g_{f_3}\right)\right)\\ &+ g_{f_1} g_{f_2} g_{f_3} \lambda_f^* \left(1 - \Phi_f\right) = 0.\end{aligned} \tag{13}$$

Solving (13) for λ_m^* gives

$$\lambda_m^* = \frac{\lambda_f^*\left(g_{f_2}\left(\delta_f \lambda_f^* + g_{f_3}\left(\lambda_f^* - \beta_f\right)\right) + \lambda_f^* \sigma_f \left(\gamma_f + g_{f_3}\right) + g_{f_1} g_{f_2} g_{f_3}\left(1 - \Phi_f\right)\right)}{\epsilon_m \left(g_{f_2}\left(\beta_f g_{f_3} - \lambda_f^*\left(\delta_f + g_{f_3}\right)\right) - \lambda_f^* \sigma_f \left(\gamma_f + g_{f_3}\right)\right)}. \tag{14}$$

Substituting (12) into (14) leads to the following fourth order polynomial equation of λ_m^*

$$\lambda_m^*\left(\xi_3 \lambda_m^{*3} + \xi_2 \lambda_m^{*2} + \xi_1 \lambda_m^* + \xi_0\right) = 0. \tag{15}$$

Solving (15) gives $\lambda_m^* = 0$ which corresponds to the drug-free equilibrium or

$$\xi_3 \lambda_m^{*3} + \xi_2 \lambda_m^{*2} + \xi_1 \lambda_m^* + \xi_0 = 0, \tag{16}$$

where

$$\begin{aligned}
\xi_0 &= g_{f_2} g_{f_3} g_{m_2}^2 g_{m_3}^2 \beta_m \epsilon_f (\beta_f g_{m_1}(1 - \Phi_m)((\epsilon_m \epsilon_f - 1)\mathcal{R}_m - 1)\\
&\quad + (1 - \Phi_m)(1 - \Phi_f) g_{f_1} g_{m_1}(\mathcal{R}_m - 1)),\\
\xi_1 &= -g_{m_2} g_{m_3}(g_{f_2}(g_{f_3}(-g_{m_1}(\Phi_m - 1)(\epsilon_f \sigma_m (g_{m_3} + \gamma_m)(\beta_f + g_{f_1}(\Phi_f - 1))\\
&\quad + g_{m_2}(\epsilon_f \delta_m (\beta_f + g_{f_1}(\Phi_f - 1))\\
&\quad + g_{m_3}(\epsilon_f(\beta_f + g_{f_1}(\Phi_f - 1)) + \beta_m(\epsilon_f \epsilon_m - 2)))) + \beta_m(2\epsilon_f \sigma_m (g_{m_3}\\
&\quad + \gamma_m)(\beta_f(\epsilon_f \epsilon_m - 1) - g_{f_1}(\Phi_f - 1))\\
&\quad + g_{m_2}(2\epsilon_f \delta_m(\beta_f(\epsilon_f \epsilon_m - 1) - g_{f_1}(\Phi_f - 1))\\
&\quad + g_{m_3}(2\epsilon_f(\beta_f(\epsilon_f \epsilon_m - 1) - g_{f_1}(\Phi_f - 1)) + \beta_m(1 - \epsilon_f \epsilon_m))))\\
&\quad + g_{m_1}^2 g_{m_2} g_{m_3}(\Phi_m - 1)^2) + \delta_f g_{m_2} g_{m_3}(g_{m_1}(\Phi_m - 1) + \beta_m)(\beta_m(1 - \epsilon_f \epsilon_m)\\
&\quad + g_{m_1}(\Phi_m - 1))) + \sigma_f g_{m_2} g_{m_3}(\gamma_f + g_{f_3})(g_{m_1}(\Phi_m - 1)\\
&\quad + \beta_m)(\beta_m(1 - \epsilon_f \epsilon_m) + g_{m_1}(\Phi_m - 1))),\\
\xi_2 &= -(\sigma_m(g_{m_3} + \gamma_m) + g_{m_2}(g_{m_3} + \delta_m))(g_{f_2}(g_{f_3}(\epsilon_f \sigma_m (g_{m_3}\\
&\quad + \gamma_m)(g_{f_1}(\Phi_f - 1) + \beta_f(1 - \epsilon_f \epsilon_m))\\
&\quad + g_{m_2}(\epsilon_f \delta_m(g_{f_1}(\Phi_f - 1) + \beta_f(1 - \epsilon_f \epsilon_m))\\
&\quad + g_{m_3}(\epsilon_f(g_{f_1}(\Phi_f - 1) + \beta_f(1 - \epsilon_f \epsilon_m))\\
&\quad + 2\beta_m(\epsilon_f \epsilon_m - 1))) + g_{m_1} g_{m_2} g_{m_3}(\Phi_m - 1)(\epsilon_f \epsilon_m - 2))\\
&\quad + \delta_f g_{m_2} g_{m_3}(g_{m_1}(\Phi_m - 1)(\epsilon_f \epsilon_m - 2)\\
&\quad + 2\beta_m(\epsilon_f \epsilon_m - 1))) + \sigma_f g_{m_2} g_{m_3}(\gamma_f + g_{f_3})(g_{m_1}(\Phi_m - 1)(\epsilon_f \epsilon_m - 2)
\end{aligned}$$

Table 1 Number of positive roots

	$\xi_3 > 0$							
	$\xi_2 > 0$				$\xi_2 < 0$			
	$\xi_1 > 0$		$\xi_1 < 0$		$\xi_1 > 0$		$\xi_1 < 0$	
	$\xi_0 > 0$	$\xi_0 < 0$	$\xi_0 > 0$	$\xi_0 < 0$	$\xi_0 > 0$	$\xi_0 < 0$	$\xi_0 > 0$	$\xi_0 < 0$
i^*	0	1	2	1	2	3	2	1

$$+ 2\beta_m(\epsilon_f\epsilon_m - 1))),$$

$$\xi_3 = \left(\epsilon_f\epsilon_m - 1\right)\left(\sigma_f\left(\gamma_f + g_{f_3}\right) + g_{f_2}\left(\delta_f + g_{f_3}\right)\right)\left(\sigma_m\left(g_{m_3} + \gamma_m\right) + g_{m_2}\left(g_{m_3} + \delta_m\right)\right)^2.$$

We now determine the number of possible positive real roots of polynomial (16) using the Descartes Rule of Signs. The possibilities can be tabulated as shown in Table 1 below.

From Table 1, we observe that system (1) can have a unique drug-persistent equilibrium. For this case, the bifurcation at $\mathcal{R}_a = 1$ is forward. However, system (1) can have multiple drug-persistent equilibrium. Hence, system (1) has a backward bifurcation at $\mathcal{R}_a = 1$ from the drug-free equilibrium to multiple drug-persistent equilibrium.

Conditions for the existence of backward bifurcation follow from Theorem 4.1 proven in [5]. We deliberately avoid rewriting the theorem and focus on its application. The theorem has been duplicated by many authors [3,8,23].

Let us make the following change of variables:

$S_m = x_1,\ U_m = x_2\ T_m = x_3,\ R_m = x_4,\ S_f = x_5,\ U_f = x_6\ T_f = x_7,\ R_f = x_8$, so that $\mathrm{N} = \sum_{n=1}^{8} x_n$. We now use the vector notation $X = (x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8)^T$. Then, system (1) can be written in the form $\dfrac{dX}{dt} = F(t, x(t)) = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8)^T$, where

$$\begin{cases} x_1^{'}(t) = p\Lambda - \dfrac{\beta_m(x_2 + \theta\varepsilon_f x_6)x_1}{N} - \mu x_1 = f_1, \\ x_2^{'}(t) = \dfrac{\beta_m(x_2 + \theta\varepsilon_f x_6)x_1}{N} + \rho_m x_4 - g_{m_1}x_2 = f_2, \\ x_3^{'}(t) = \sigma_m x_2 - g_{m_2}x_3 = f_3, \\ x_4^{'}(t) = \gamma_m x_3 + \delta_m x_2 - g_{m_3}x_4 = f_4, \\ x_5^{'}(t) = (1-p)\Lambda - \dfrac{\beta_m(\varepsilon_m x_2 + \theta x_6)x_5}{N} - \mu x_5 = f_5, \\ x_6^{'}(t) = \dfrac{\beta_m(\varepsilon_m x_2 + \theta x_6)x_5}{N} + \rho_f x_8 - g_{f_1}x_6 = f_6, \\ x_7^{'}(t) = \sigma_f x_6 - g_{f_2}x_7 = f_7, \\ x_8^{'}(t) = \gamma_f x_7 + \delta_f x_6 - g_{f_3}x_8 = f_8. \end{cases} \tag{17}$$

We now define

$$\beta_f = \theta\beta_m \tag{18}$$

with $\theta = 1$ signifying that the chance of initiating drug abuse habit upon contact with a male drug user or upon contact with a female drug user is the same, $\theta \in (0, 1)$ signifying a reduced chance of initiating drug abuse habit upon contact with a female drug user as compared to a male drug user, $\theta > 1$ signifies an increased rate of initiating drug abuse habit upon contact with a female drug user as compared to a male drug user.

Let β_m be the bifurcation parameter, $\mathcal{R}_a = 1$ corresponds to

$$\beta_m = \beta_m^* = \frac{g_{f_1}\left(\Phi_f - 1\right)}{2\theta\left(\epsilon_f\epsilon_m - 1\right)} + \frac{\theta g_{m_1}\left(\Phi_m - 1\right)}{2\theta\left(\epsilon_f\epsilon_m - 1\right)} + \frac{\sqrt{\left(g_{f_1}\left(\Phi_f - 1\right) + \theta g_{m_1}\left(\Phi_m - 1\right)\right)^2 + 4\theta g_{f_1}\left(\Phi_f - 1\right) g_{m_1}\left(\Phi_m - 1\right)\left(\epsilon_f\epsilon_m - 1\right)}}{2\theta\left(\epsilon_f\epsilon_m - 1\right)}.$$

The Jacobian matrix of system (1) at $\mathcal{G}_0$ when $\beta_m = \beta_m^*$ is given by

$$J^*(\mathcal{G}_0) = \begin{pmatrix} -\mu & -p\beta_m^* & 0 & 0 & 0 & -p\theta\beta_m^*\epsilon_f & 0 & 0 \\ 0 & p\beta_m^* - g_{m_1} & 0 & \rho_m & 0 & p\theta\beta_m^*\epsilon_f & 0 & 0 \\ 0 & \sigma_m & -g_{m_2} & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta_m & \gamma_m & -g_{m_3} & 0 & 0 & 0 & 0 \\ 0 & -(1-p)\beta_m^*\epsilon_m & 0 & 0 & -\mu & -(1-p)\theta\beta_m^* & 0 & 0 \\ 0 & (1-p)\beta_m^*\epsilon_m & 0 & 0 & 0 & (1-p)\theta\beta_m^* - g_{f_1} & 0 & \rho_f \\ 0 & 0 & 0 & 0 & 0 & \sigma_f & -g_{f_2} & 0 \\ 0 & 0 & 0 & 0 & 0 & \delta_f & \gamma_f & -g_{f_3} \end{pmatrix}.$$

System (17), with $\beta_m = \beta_m^*$ has a simple eigenvalue, hence the center manifold theory can be used to analyse the dynamics of system (1) near $\beta_m = \beta_m^*$. It can be shown that $J^*(\mathcal{G}_0)$, has a right eigenvector given by $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8)^T$. Further, the left eigenvector of $J^*(\mathcal{G}_0)$, associated with the zero eigenvalue at $\beta_m = \beta_m^*$ is given by $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8)^T$.

The computations of **a** and **b** are necessary in order to apply Theorem 4.1 in Castillo-Chavez and Song [5]. For system (17), the associated non-zero partial derivatives of F at the drug-free equilibrium are given below. We make use of the following notation:

$$\frac{\partial^2 f_k}{\partial x_i \partial x_j} = f_{k(i,j)} \text{ and } \frac{\partial^2 f_k}{\partial x_i \partial \beta_m^*} = f_{k(i,\beta_m^*)},$$
$$\text{with } f_{k(i,j)} = f_{k(j,i)}, \quad i, j, k = 1, 2, \ldots, 8.$$

We now write the associated non-zero partial derivatives in terms of the following two non-zero partial derivatives:

$$f_{1(1,2)} = \frac{-(1-p)\mu\beta_m}{\Lambda} \text{ and } f_{1(2,3)} = \frac{p\mu\beta_m}{\Lambda}. \tag{19}$$

Thus, the remaining associated non-zero partial derivatives are as follows:

$$f_{1(1,6)} = \theta\varepsilon_f f_{1(1,2)}, \quad f_{2(1,2)} = -f_{1(1,2)}, \quad f_{2(1,6)} = -\theta\varepsilon_f f_{1(1,2)},$$
$$f_{5(1,2)} = f_{5(2,3)} = f_{5(2,4)} = f_{5(2,7)} = f_{5(2,8)} = -\varepsilon_m f_{1(1,2)}$$
$$f_{5(2,2)} = -2\varepsilon_m f_{1(1,2)}, \quad f_{5(2,6)} = -(\theta + \varepsilon_m) f_{1(1,2)}, \quad f_{5(6,6)} = -2\theta f_{1(1,2)},$$
$$f_{5(3,6)} = f_{5(1,6)} = f_{5(4,6)} = f_{5(6,7)} = f_{5(6,8)} = -\theta f_{1(1,2)},$$
$$f_{6(1,2)} = f_{6(2,3)} = f_{6(2,4)} = f_{6(2,7)} = f_{6(2,8)} = \varepsilon_m f_{1(1,2)}$$
$$f_{6(2,2)} = 2\varepsilon_m f_{1(1,2)}, \quad f_{6(2,6)} = (\theta + \varepsilon_m) f_{1(1,2)}, \quad f_{6(6,6)} = 2\theta f_{1(1,2)},$$

$$
\begin{aligned}
&f_{6(3,6)} = f_{6(1,6)} = f_{6(4,6)} = f_{6(6,7)} = f_{6(6,8)} = \theta f_{1(1,2)},\\
&f_{1(2,4)} = f_{1(2,5)} = f_{1(2,7)} = f_{1(2,8)} = f_{1(2,3)},\\
&f_{1(2,2)} = 2f_{1(2,3)},\quad f_{1(2,6)} = (1+\theta\varepsilon_f) f_{1(2,3)},\quad f_{1(6,6)} = 2\theta\varepsilon_f f_{1(2,3)},\\
&f_{1(3,6)} = f_{1(4,6)} = f_{1(5,6)} = f_{1(6,7)} = f_{1(6,8)} = \theta\varepsilon_f f_{1(2,3)},\\
&f_{2(2,2)} = -2f_{1(2,3)},\quad f_{2(2,6)} = -(1+\theta\varepsilon_f) f_{1(2,3)},\quad f_{2(6,6)} = -2\theta\varepsilon_f f_{1(2,3)},\\
&f_{2(2,3)} = f_{2(2,4)} = f_{2(2,5)} = f_{2(2,7)} = f_{2(2,8)} = -f_{1(2,3)},\\
&f_{2(3,6)} = f_{2(4,6)} = f_{2(5,6)} = f_{2(6,7)} = f_{2(6,8)} = -\theta\varepsilon_f f_{1(2,3)},\\
&f_{5(2,5)} = -\varepsilon_m f_{1(2,3)},\quad f_{5(5,6)} = -\theta f_{1(2,3)},\quad f_{6(2,5)} = \varepsilon_m f_{1(2,3)},\\
&f_{6(5,6)} = \theta f_{1(2,3)},\quad f_{1(2,\beta_m^*)} = -p,\quad f_{1(6,\beta_m^*)} = -p\theta\varepsilon_f,\\
&f_{2(2,\beta_m^*)} = p,\quad f_{2(6,\beta_m^*)} = p\theta\varepsilon_f,\quad f_{5(2,\beta_m^*)} = -(1-p)\varepsilon_m,\\
&f_{5(6,\beta_m^*)} = -(1-p)\theta,\quad f_{6(2,\beta_m^*)} = (1-p)\varepsilon_m,\quad f_{6(6,\beta_m^*)} = (1-p)\theta.
\end{aligned}
$$

It thus follows that

$$\mathbf{a} = \sum v_k w_i w_j f_{k(i,j)},\ i, j, k = 1, 2, 3, \ldots, 8 \tag{20}$$

and

$$\mathbf{b} = \sum v_k w_i f_{k(i,\beta_m^*)},\ i, k = 1, 2, 3, \ldots, 8.$$

We thus have the following result

Theorem 3 *If $\boldsymbol{a} > 0$ and $\boldsymbol{b} > 0$, then system (1) has a backward bifurcation at $\mathcal{R}_a = 1$, otherwise if $\boldsymbol{a} < 0$ and $\boldsymbol{b} > 0$ then the drug-persistent equilibrium is locally asymptotically stable for $\mathcal{R}_a > 1$ but close to one.*

Numerical Simulations

A Case Study for the Western Cape Province of South Africa

We present an application of our model to a case study of Cape Town of South Africa. We focus mainly on the trends of drug abuse amongst males and females. The 2011 United Nations World Drug Report identified South Africa as one of the countries still experiencing 'some increase' in the use of 'undefined amphetamines'. The major contributor to an increase in the use of amphetamine-group substances has been an increase in consumption of methamphetamine in Cape Town and the surrounding Western Cape Province. This information has been documented via admissions to treatment centres, see [25–28]. Data collected by the Medical Research Council (MRC) from specialist substance abuse treatment centres in Cape Town (as part of the South African Community Epidemiology Network on Drug Use-SACENDU) show that males continue to consistently dominate patient intake.

Parameter Estimation

We make use of Matlab to estimate model parameters used in our numerical simulations through curve fitting, which is a process that allows us to quantitatively estimate the trend of the outcomes. The curve fitting process fits equations of approximating curves to the raw field data. Nevertheless, for a given set of data, the fitting curves of a given type are generally

Table 2 Male and female substance abuse patients in the Western Cape Province of South Africa for the period 1998a–2015b (%)

Year	1998a	1998b	1999a	1999b	2000a	2000b	2001a	2001b
Male (%)	81	81	80	83	84	82	80	84
Female (%)	19	19	20	17	16	18	20	16
Year	2002a	2002b	2003a	2003b	2004a	2004b	2005a	2005b
Male (%)	83	80	81	82	80	78	78	75
Female (%)	17	20	19	18	20	22	22	25
Year	2006a	2006b	2007a	2007b	2008a	2008b	2009a	2009b
Male (%)	74	75	76	76	74	75	74	74
Female (%)	26	25	24	24	26	25	26	26
Year	2010a	2010b	2011a	2011b	2012a	2012b	2013a	2013b
Male (%)	74	76	74	76	76	75	76	75
Female (%)	26	24	26	24	24	25	24	25
Year	2014a	2014b	2015a					
Male (%)	77	73	76					
Female (%)	23	27	24					

Letter 'a' represents the first six months of the year and 'b' represents the last six months of the year

not unique. Thus, a curve with a minimal deviation from all data points is desired. This best-fitting curve can be obtained by the method of least squares. The least squares curve fit routine (lsqcurvefit) in Matlab with optimization is used to estimate the parameter values. Many parameters are known to lie within some intervals. During the estimation of parameter values, unknown parameter values are given a lower and upper bound from which the set of parameter values that provide the best fit are obtained. The parameters obtained from curve fitting and intervals used are given in Tables 3 and 4. We present an application of system (1) through fitting the model to data from the Medical Research Council's (MRC's), South African Community Epidemiology Network on Drug Use (SACENDU) project. We fit the system (1) to data on male and female patients who are primary substance abusers in Cape Town of South Africa. We use data in Table 2 which was collected by the South African Community Epidemiology Network on Drug Use (SACENDU) [35].

Results

Figure 2 shows the trends in the proportion of male substance abusers in treatment centres of Cape Town. As can be seen in Fig. 2, the model fits well with the data from Table 2. The results show that the proportion of male substance abusers in Cape Town shall continue to steadily decrease over the years. However, male substance abusers continue to dominate their female counterparts. Parameter values estimated using data for male substance abusers in Cape Town are shown in Table 3. This estimation assumes that the dynamics remain the same over the entire period.

Figure 3 shows the trends in the proportion of female substance abusers in treatment centres of Cape Town. The results show that the proportion of female substance abusers in Cape Town shall steadily increase over the years. However, female substance abusers continue to be dominated by their male counterparts. Parameter values estimated using data

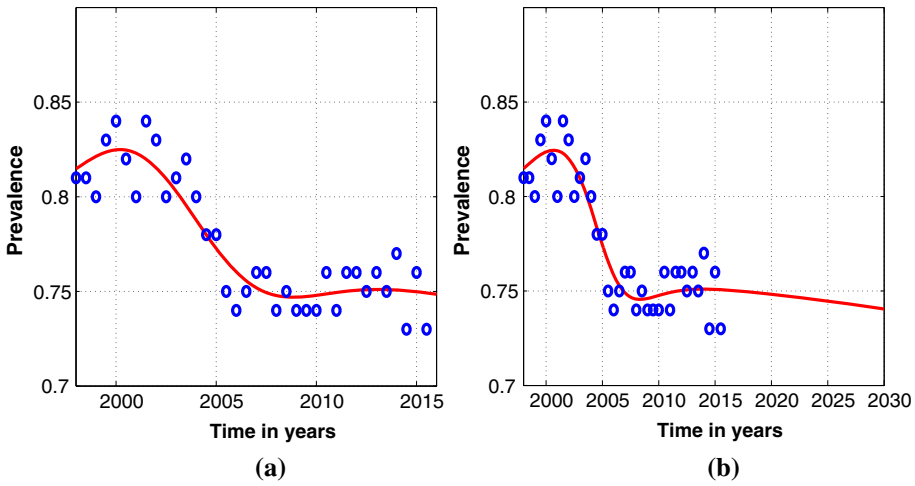

Fig. 2 System (1) fitted to data for substance abuse male patients (**a**) and projected for more years (**b**) in Cape Town. The blue circles indicate the actual data and the solid red line indicates the model fit to the data

Table 3 Parameter values and ranges obtained from data fitting using data for male substance abusers in Cape Town

Parameter	Range	Value	Source
p	0–1	0.0393	Estimated
β_m	0–1	0.6877	Estimated
β_f	0–1	0.1207	Estimated
ϵ_m	0–1	0.1623	Estimated
ϵ_f	0–1	0.9999	Estimated
σ_m	0–1	7.9161×10^{-4}	Estimated
σ_f	0–1	0.0572	Estimated
r_m	0–1	0.4742	Estimated
r_f	0–1	0.4985	Estimated
δ_m	0–1	2.3432×10^{-6}	Estimated
δ_f	0–1	0.0010	Estimated
ρ_m	0–1	0.8093	Estimated
ρ_f	0–1	0.7096	Estimated
γ_m	0–1	1.2395×10^{-4}	Estimated
γ_f	0–1	0.0147	Estimated
Λ	0.028–0.080	0.0324	Estimated
μ	0.019–0.021	0.020	Ref. [12]

for female substance abusers in Cape Town are shown in Table 4. This estimation assumes that the dynamics remain the same over the entire period.

Numerical Results

We carry out detailed numerical simulations using matlab programming language to support our theoretical findings. Numerical solutions of a model depend on the values of all its

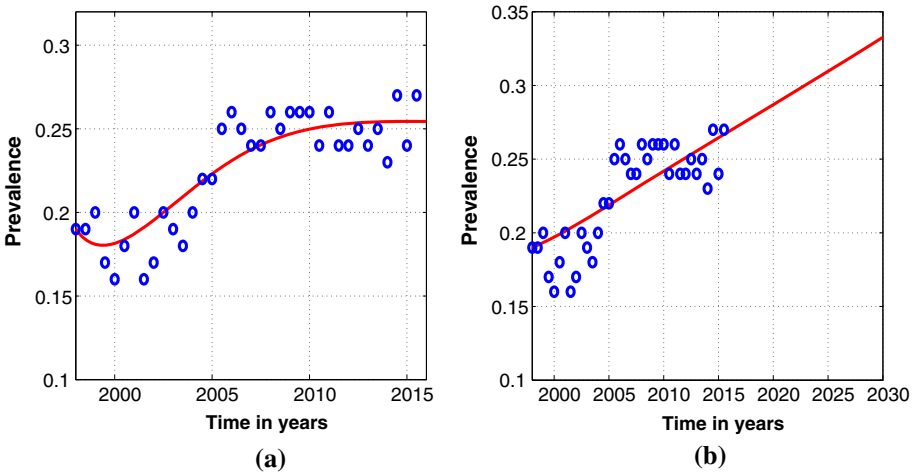

Fig. 3 System (1) fitted to data for substance abuse female patients (**a**) and projected for more years (**b**) in Cape Town. The blue circles indicate the actual data and the solid red line indicates the model fit to the data

Table 4 Parameter values and ranges obtained from data fitting using data for female substance abusers in Cape Town

Parameter	Range	Value	Source
p	0–1	4.2×10^{-5}	Estimated
β_m	0–1	0.6015	Estimated
β_f	0–1	2.1535×10^{-6}	Estimated
ϵ_m	0–1	1.8173×10^{-5}	Estimated
ϵ_f	0–1	3.0700×10^{-6}	Estimated
σ_m	0–1	0.1132	Estimated
σ_f	0–1	2.1447×10^{-6}	Estimated
r_m	0–1	0.3747	Estimated
r_f	0–1	0.9999	Estimated
δ_m	0–1	5.3434×10^{-5}	Estimated
δ_f	0–1	3.8806×10^{-6}	Estimated
ρ_m	0–1	0.3528	Estimated
ρ_f	0–1	0.2285	Estimated
γ_m	0–1	1.9912×10^{-6}	Estimated
γ_f	0–1	0.1367	Estimated
Λ	0.028–0.080	0.04	Estimated
μ	0.019–0.021	0.020	Ref. [12]

parameters. The initial conditions used are: $S_m(0) = 450$, $U_m(0) = 10$, $T_m(0) = 0$, $R_m(0) = 0$, $S_f(0) = 350$, $U_f(0) = 5$, $T_f(0) = 0$, $R_f(0) = 0$.

Figure 4 illustrates the effect of varying parameters β_m, β_f, δ_m and δ_f on the prevalence of drug abuse. Figure 4a shows that increasing the contact rate β_m lead to an increase in the prevalence of both male and female drug abusers. Though the contact rate β_m as defined in "Model Formulation" section, accounts for the interaction between males only, we observe that increasing β_m also results in an increase in the prevalence of female drug abusers. Thus,

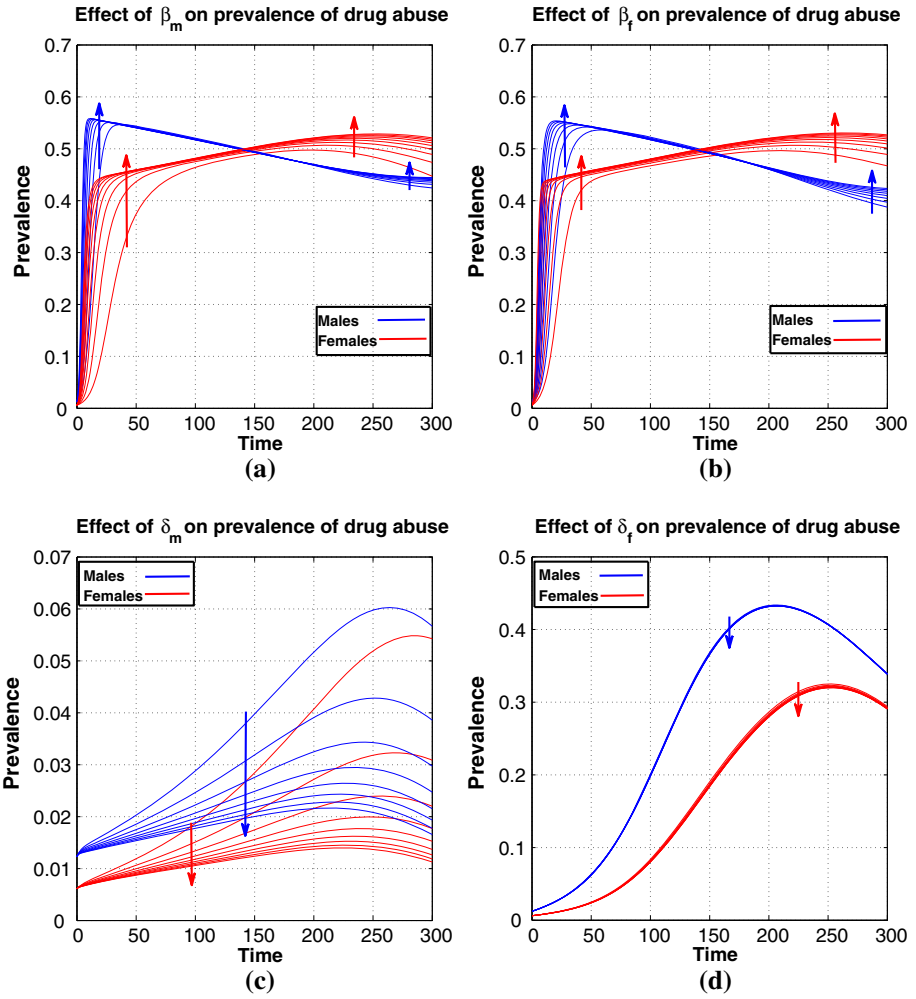

Fig. 4 Effects of varying β_m, (**a**), β_f, (**b**), δ_m, (**c**) and δ_f, (**d**) on the prevalence of drug abuse, starting from 0.2 up to 1.0 with a step size of 0.1 across all the parameters

we can deduce that reducing the magnitude of β_m results in a decrease in the prevalence of female drug users. The same can also be observed for the parameter β_f. Figure 4b shows that increasing the contact rate β_f lead to an increase in the prevalence of both male and female drug abusers. As given in "Model Formulation" section, parameter β_f accounts for the interaction between females only, but we observe that its increase also results in an increase in the prevalence of male drug abusers. Thus, we can deduce that reducing the magnitude of β_f results in a decrease in the prevalence of male drug users. Figure 4c, d illustrate the effect of parameters δ_m and δ_f on the prevalence of drug abuse for both males and females. We observe that parameter δ_m has more impact on decreasing prevalence of drug abuse for both males and females as compared to δ_f. Encouraging male drug abusers to quit drug abuse is of significant importance in controlling drug abuse amongst both males and females. Note also that encouraging female drug abusers to quit drug abuse is of importance in controlling

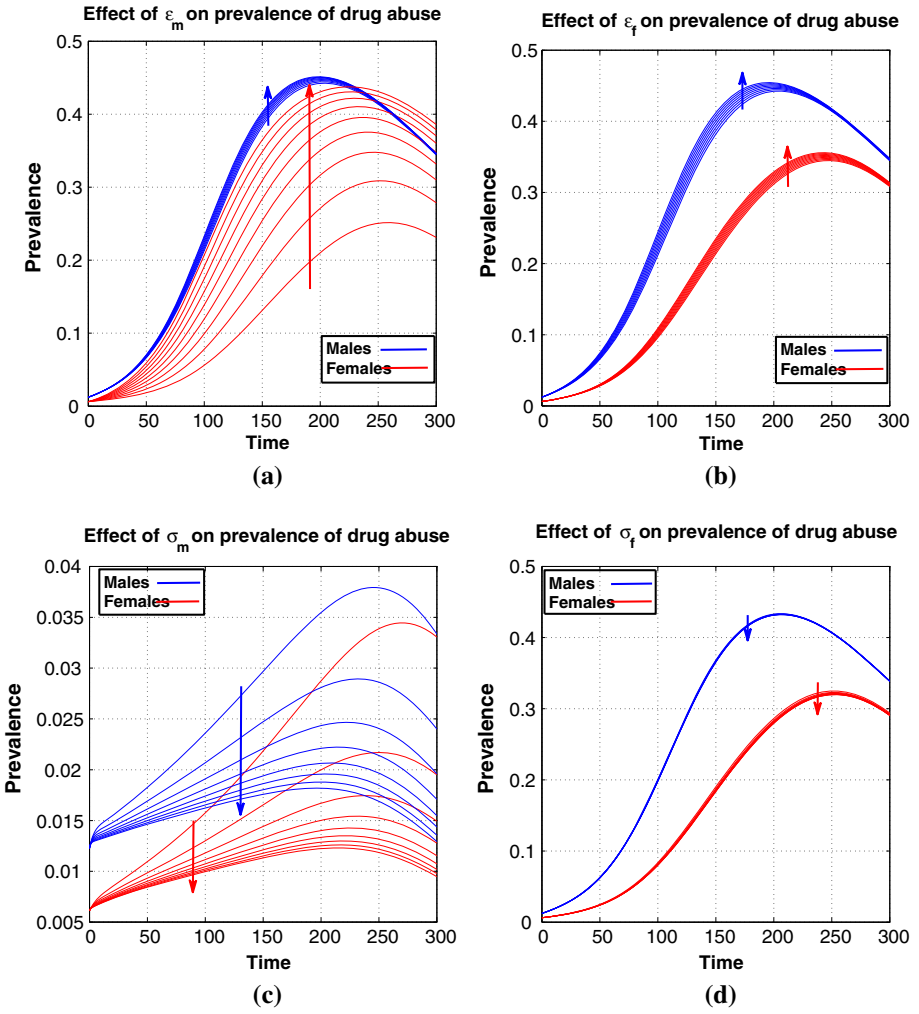

Fig. 5 Effects of varying ε_m, (**a**), ε_f, (**b**), σ_m, (**c**) and σ_f, (**d**) on the prevalence of drug abuse, starting from 0.2 up to 1.0 with a step size of 0.1 across all the parameters

drug abuse, but more effort should be directed to male drug abusers as they are currently the main contributors to most cases given their increased number.

Figure 5 illustrates the effect of varying parameters ε_m, ε_f, σ_m and σ_f on the prevalence of drug abuse. Figure 5a show that increasing the parameter ε_m results in an increase in the prevalence of both males and females, however with a proportionally higher increase in female drug abusers. Thus, efforts targeted at reducing the prevalence of female drug abusers should be directed towards reducing parameter ε_m. Figure 5b show the effect of parameter ε_f on the prevalence of male and female drug abusers. We observe that increasing ε_f results in an increase in the prevalence of both males and females, however with a proportionally smaller increase for both sexes. Figure 5c, d illustrate the effect of parameters σ_m and σ_f on the prevalence of drug abuse for both males and females. We observe that parameter σ_m has more impact on decreasing prevalence of drug abuse for both males and females as compared

to σ_f. Encouraging male drug abusers in need of help for overcoming the drug problem to seek treatment can be of great help in the fight against drug abuse amongst both males and females. Note also that encouraging female drug abusers in need of help to overcome the drug problem is also of great importance in controlling drug abuse, but more effort should be directed towards encouraging more male drug abusers as they are currently more of them who might be in need of help to overcome the drug problem given their dominance over females.

Conclusion

The goal of this paper is to model gender differences in the spread of drug abuse. The sex structured model developed in this paper explicitly captures the drug abuse dynamics amongst male and female individuals. Compared to previous drug abuse models, the model differentiates the human population in terms of sex and considers possible interactions within respective classes not considered in past drug abuse models. The model is fitted to data on drug abusers reporting in the treatment centres of Cape Town. The least squares curve fit routine (lsqcurvefit) in Matlab with optimization has been used to fit the model to data on male and female drug abusers reporting in treatment centres of Cape Town of South Africa. The model was observed to fit well with this data and parameters from model fitting were obtained. Performing a visual predictive check on the figures from data fitting suggests that the proportion of male drug abusers in Cape Town is expected to continue to decrease whereas that of female drug abusers shall continue to increase but steadily. The estimated proportion of female drug abusers in specialist treatment centres of Cape Town was observed to be approximately 34% by the year 2030.

Numerical results from this study have lead to other important insights in the dynamics of drug abuse. The identification of parameters that have more impact on the prevalence of drug abuse for males and females will be of priceless help for policy makers in coming up with relevant policies for the control of this dynamic social epidemic. Some investigation on the impact of some parameters on the prevalence of drug abuse was performed via numerical simulations. It was observed that parameters β_m and β_f though linked to a specific gender, have a direct effect on the prevalence of drug abuse for both sexes. Intuitively, one might regard the control of parameter β_m to be of effect to males only and that of β_f to be of effect to females only. But as observed, increasing/decreasing β_m or β_f leads to an increase/decrease in the prevalence of drug abuse for both males and females. It was also noted that increasing the magnitude of parameters δ_m and σ_m result in a significant decrease in the prevalence of drug abuse for both sexes as compared to increasing parameters δ_f and σ_f. This was seen to be the case since males currently dominate females in drug use and treatment. Thus control efforts targeted to males tend to have more impact as compared to efforts directed to females. This however should not be taken to mean that control measures should do away with females, rather the results suggest that more effort should be directed to males to achieve enhanced positive results. The study presented here is not exhaustive, it can be extended to include contextual dynamics, such as drug supply chains or changes in interdiction. Incorporating these processes will undoubtedly facilitate in the understanding of drug abuse dynamics.

Acknowledgements J. Mushanyu, P. Mafuta and E.T Nkarakana-Gwasira authors acknowledge, with thanks, the support of the Department of Mathematics, University of Zimbabwe. F. Nyabadza acknowledges with gratitude the support from National Research Foundation and Stellenbosch University for the production of this manuscript.

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