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DYNAMIC ANALYSIS OF A DRUG TRANSMISSION MODEL WITH ANTI-DRUG EDUCATION AND MEDIA COVERAGE*

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Abstract This paper is devoted to studying the dynamic behavior of drug transmission and the optimal strategy for controlling drug transmission. We constructed a six-dimensional drug transmission model with media coverage, family and public education, dividing the total population into six categories: high-risk susceptible individuals (S), low-risk susceptible individuals with protection awareness (P), psychological addicts (I_{PC}), physiological addicts (I_{PS}), drug addicts in community treatment (R_C), drug addicts in compulsory detoxification treatment (R_I). We first calculated the basic regeneration number and analyzed the existence and stability of the equilibrium point of the model. Then, sensitivity analysis and numerical simulations of the parameters are performed. Finally, we gained new insights that when $R_0 < 1$, controlling the contact between susceptible individuals and drug addicts is more effective than treatment; when $R_0 > 1$, anti-drug education and media coverage play a greater role, at which point prevention and treatment go hand in hand to control the drug epidemic in a more cost-effective and rapid manner.

Keywords Drug transmission model, basic regeneration number, sensitivity, numerical simulation.

MSC(2010) 34K20, 34D23.

1. Introduction

At present, drug abuse is still one of the biggest threats to global public safety, drug abuse refers to the use of heroin, cocaine, morphine, crystal meth and other psychoactive substances that are currently not approved for clinical use and psychoactive drugs that are not used in accordance with medical standards or are not produced by state-approved enterprises or are not circulated within the scope of relevant national regulations. According to the World Drug Report 2022, approximately 284 million people aged 15-64 used drugs worldwide in 2020, up 26% from the previous decade. Globally, 11.2 million people inject drugs. About half of them

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have hepatitis C, 1.4 million have HIV, and 1.2 million have both diseases [28]. As of December 2020, the total number of new psychoactive substances found in 126 countries reached 1,047, three times the number of internationally controlled substances. New psychoactive substances exhibit stronger psychological dependence than traditional drugs and are highly susceptible to overdose resulting in death [14]. In 2019, there were close to 500,000 drug-related fatalities, more than 36 million people with drug-related mental illnesses, and 18 million healthy lives lost to severe drug use disorders [28]. Although the number of drug abusers has decreased over the past two years, the numbers are still high. According to the 2019 China Drug Situation Report, there were 2.148 million drug users in China by the end of 2019, accounting for 0.16% percent of the country's total population [24]. Drugs not only threaten the lives and health of drug users, but also lead to criminal acts such as drug trafficking, drug production, kidnapping, and shootings [10]. In addition, compared to traditional drugs, teenagers make up the majority of victims of new psychotropic drugs [7]. Take China for example, there are 1.045 million drug users aged 18 to 35, accounting for 48.7% of current drug users [7,24]. The increasing pace of technological innovation has made it easier for people to access drugs through a variety of platforms and, as a result, has caused an accelerated paradigm shift in drug transmission, with significant implications for public health [3,25]. All the evidence above aroused that we must continue to devote the necessary resources and attention to addressing the global drug problem.

Existing papers have shown that mathematical models have been widely studied in controlling the spread of drug abuse [2,11]. In the 1970s, a comparison was made between drug abuse and infectious diseases, thereby confirming the significance and value of utilizing epidemiological methods to research heroin abuse [13]. White and Comiskey proposed a model in 2006 that divided drug users into those who are not in treatment (U_1) and those who are in treatment (U_2). They performed a series of analyses on the model and then obtained: prevention is better than treatment [29]. Based on the model of White and Comiskey, Reza Memarbashi and Elahe Sorouri considered the effect of receiving drug harm information on preventing drug epidemic, and divided the total population into susceptible individuals, infected individuals and responsive individuals. The responding individuals refer to those susceptible individuals who receive information about the harms and dangers of drugs [15]. In 2017, Mingju Ma established a drug model with psychological and physiological addiction. By analyzing the sensitivity of parameters, the results showed that reducing contact with drug users was more effective than treating them [16]. In the same year, Matintu introduced a new smoking model to analyze the spread of smoking. In their model, considering whether people smoke or not and the different levels of addiction to smoking, the population was divided into five categories: potential smokers, moderate smokers, heavy smokers, smokers who temporary quit smoking and smokers who permanently quit smoking [17]. In 2018, the authors formulate a six dimensional drug transmission model to study the effect of family education and public health education [12]. Puthur Thangaraj Sowndarrajan, developed a heroin model with prevention information and treatment and assuming that the individuals in preventive education were in a state of self-protection. According to the optimal control theory, the simultaneous implementation of preventive measures and treatment measures can not only reduce the cost, but also maximize the reduction of the number of heroin users [26]. Asaf Khan developed a heroin model with an age structure and considered medication

for addicts and risk education for susceptible individuals as two control measures in the model [9]. Haoxiang Tang has developed a drug misuse model in 2020. The model proposed that susceptible individuals start using drugs under the influence of treated drug users and hidden drug users. According to the simulation results, China's drug users will decline dramatically over the next ten years and increased investigation of drug users who are not in treatment is the most effective strategy to control the spread of drugs [27]. Njagarah and Nyabadza developed a mathematical model to assess the impact of rehabilitation and relapse on the prevalence of drug epidemics. Through the analysis of the model, they obtained that both prevention and treatment are necessary strategies for reduction of drug epidemics. At the end of the article they give some advice that preventive strategies should be directed toward reducing the contact rate and treatment should be combined with psychotherapy to accelerate quitting and reduce relapse [22]. Komal Bansal developed a fractional-order illicit drug transmission model. According to the numerical simulation, the most successful drug addiction reduction programs were those lasting 70 and 75 days [1].

It is well known that education and the media play an important role in controlling the spread of epidemics. Our goal is to simulate the pattern of drug transmission in today's society, consider the role that education and the media play in controlling the spread of drugs, and discuss in depth intervention strategies to control the spread of drugs. Our innovations are in the following three aspects. First, we consider the combined effect of anti-drug education and media coverage. Second, we allow for the possibility that someone who accidentally takes drugs due to curiosity, temptation from friends, etc. will voluntarily detoxify successfully. Finally, there are two treatment models, community-based treatment and compulsory detoxification treatment, were introduced. Based on the above three points, we will thoroughly study the preventive measures and treatment measures to provide new insights for controlling the spread of drugs.

This paper is organized as follows: In section 2, we develop a drug transmission model and discuss its basic properties. In section 3, the basic regeneration number of the model is calculated, and then the stability of the equilibrium point is analyzed qualitatively. In Section 4, we perform sensitivity analysis of the model parameters and obtain new insights for controlling the spread of drugs through numerical simulations. Finally, a summary and discussion are given.

2. Mathematical model

2.1. Drug transmission model

A questionnaire survey was developed and carried out at a male and female drug rehabilitation center in Shanxi, with 463 and 455 valid questionnaires returning, respectively. The purpose of the survey was to better understand the characteristics of drug users in today's drug market, the proportion of drug users using various types of drugs, and whether they use drugs out of curiosity. The survey results showed that only 3% of male drug users were over 60 years old and no female drug users were over 60 years old. Results of the survey are consistently with data from the National Drug Control Network (NDCN), which shows that the majority of drug users are concentrated in the 14-60 age group. So we will focus on the 14-60 age group in this paper. In general, adolescents between the ages of 14 and 18

acquire knowledge through family education, schooling education, and other public education. Also female questionnaire data shows that 43% of drug users between the age of 20 and 60 are more likely to obtain information from the Internet. Therefore, we considered adding media coverage, family education, and public education to the model. According to the director of the drug rehabilitation center, the drug addiction process can be divided into two stages. The first stage is psychological addiction, which mainly refers to the fact that most drug addicts start using drugs because they are curious, thrill-seeking or confused by their friends. In the questionnaire data, 53% of women and 71% of men use drugs out of curiosity. The second stage is physical addiction, which mainly means that as the frequency of drug use increases, the drug user becomes physically dependent [5]. According to [6, 8, 21], the main methods of drug rehabilitation are voluntary detoxification, community detoxification, community rehabilitation and compulsory isolation. Since the number of people who consider self-drug rehabilitation is very small, we mainly consider community treatment and compulsory detoxification treatment.

From the above, we propose a drug transmission model with anti-drug education and media coverage. In this model, we divide the total population into six compartments: high-risk susceptible individuals (S), low-risk susceptible individuals with protection awareness (P), psychological addicts (I_{PC}), physiological addicts (I_{PS}), drug addicts in community treatment (R_C), drug addicts in compulsory detoxification treatment (R_I). Then the model diagram is shown in Fig.1.

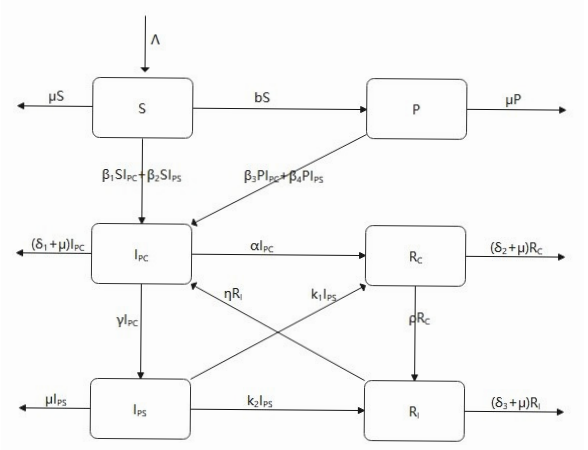

Figure 1. Compartmental diagram of drug transmission model with media coverage and anti-drug education

We suppose that the proportion b of high-risk susceptible individuals (S) are converted to low-risk susceptible individuals with protection awareness (P) through media coverage and education about the dangers of drugs. After contact with a drug addict, the susceptible person will first become a psychological addict. Psychological addicts who are caught will first be sent to the community for treatment, or if not caught and continue to use drugs they will become physiological addicts. Here we also take into account that some people who are accidental drug users due to mistaken use, compelled by friends, etc., will consciously avoid re-exposure to drugs and undergo voluntary detoxification. Referring to China's drug control model, community treatment generally lasts three years, and mandatory isolation

treatment lasts two years. Those who continue to use drugs during community treatment will be sent to a compulsory detoxification center for treatment. According to the management of the compulsory detoxification centers, there will be no physical dependency after compulsory detoxification treatment. Failure of compulsory detoxification treatment is usually a psychological failure, i.e. they become psychologically addicted again. We define a drug addict's relapse within 2 years of leaving a compulsory detoxification center as a failure of detoxification, or a permanent detoxification if the detoxification period exceeds 2 years.

2.2. Model equation

According to the model diagram, we have the following system:

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = \Lambda - \beta_1 S I_{PC} - \beta_2 S I_{PS} - bS - \mu S, \\ \dfrac{\mathrm{d}P}{\mathrm{d}t} = bS - \beta_3 P I_{PC} - \beta_4 P I_{PS} - \mu P, \\ \dfrac{\mathrm{d}I_{PC}}{\mathrm{d}t} = \beta_1 S I_{PC} + \beta_2 S I_{PS} + \beta_3 P I_{PC} + \beta_4 P I_{PS} + \eta R_I - (\alpha + \gamma + \mu + \delta_1) I_{PC}, \\ \dfrac{\mathrm{d}I_{PS}}{\mathrm{d}t} = \gamma I_{PC} - (k_1 + k_2 + \mu) I_{PS}, \\ \dfrac{\mathrm{d}R_C}{\mathrm{d}t} = \alpha I_{PC} + k_1 I_{PS} - (\rho + \mu + \delta_2) R_C, \\ \dfrac{\mathrm{d}R_I}{\mathrm{d}t} = \rho R_C + k_2 I_{PS} - (\eta + \mu + \delta_3) R_I. \end{cases} \tag{2.1}$$

In general, susceptible individuals are more likely to become addicts after contact with a physiological addict. Assuming that the effective contact rate of physiological addicts is q times that of psychological addicts, i.e., $\beta_2 = q\beta_1$. In addition, once someone is educated about drugs, they will reject drugs and consciously avoids interaction with drug users, so there will be a lower effective contact rate. Assuming that the reduction rate is ξ, i.e., $\beta_3 = \xi\beta_1$, $\beta_4 = \xi\beta_2$. Thereby $\beta_1 = \beta$, $\beta_2 = q\beta$, $\beta_3 = \xi\beta$, $\beta_4 = q\xi\beta$. Hence the system (1) transforms into the following form:

$$\begin{cases} \dfrac{\mathrm{d}S}{\mathrm{d}t} = \Lambda - \beta S I_{PC} - q\beta S I_{PS} - bS - \mu S, \\ \dfrac{\mathrm{d}P}{\mathrm{d}t} = bS - \xi\beta P I_{PC} - \xi q\beta P I_{PS} - \mu P, \\ \dfrac{\mathrm{d}I_{PC}}{\mathrm{d}t} = \beta S I_{PC} + q\beta S I_{PS} + \xi\beta P I_{PC} + \xi q\beta P I_{PS} + \eta R_I - m_1 I_{PC}, \\ \dfrac{\mathrm{d}I_{PS}}{\mathrm{d}t} = \gamma I_{PC} - m_2 I_{PS}, \\ \dfrac{\mathrm{d}R_C}{\mathrm{d}t} = \alpha I_{PC} + k_1 I_{PS} - m_3 R_C, \\ \dfrac{\mathrm{d}R_I}{\mathrm{d}t} = \rho R_C + k_2 I_{PS} - m_4 R_I, \end{cases} \tag{2.2}$$

where $S(0) > 0$, $P(0) > 0$, $I_{PC}(0) > 0$, $I_{PS}(0) > 0$, $R_C(0) > 0$, $R_I(0) > 0$,

$m_1 = \alpha + \gamma + \mu + \delta_1,\ m_2 = k_1 + k_2 + \mu,\ m_3 = \rho + \mu + \delta_2,\ m_4 = \eta + \mu + \delta_3$. The definitions of variables and parameters are shown in Table 1.

Table 1. Definition of variables and parameters

Variable Parameter	Description
Λ	Inflow rate into high-risk susceptible individuals
b	Conversion rate of high-risk susceptible individuals to low-risk susceptible individuals with protection awareness
β_1	Effective contact rate between psychological addicts and high-risk susceptible individuals
β_2	Effective contact rate between physiological addicts and high-risk susceptible individuals
β_3	Effective contact rate between psychological addicts and low-risk susceptible individuals with protection awareness
β_4	Effective contact rate between physiological addicts and low-risk susceptible individuals with protection awareness
γ	Rate of psychological addicts who continue to take drugs and become physiological addicts
α	Rate of psychological addicts entering community treatment
k_1	Rate of physiological addicts entering community treatment
k_2	Rate of physiological addicts entering compulsory detoxification treatment
ρ	Rate of addicts moving from community treatment to compulsory detoxification treatment
η	Failure rate of compulsory detoxification treatment
δ_1	Successful rate of voluntary detoxification
δ_2	Successful rate of community treatment
δ_3	Successful rate of compulsory detoxification treatment
μ	Natural death rate

3. Equilibrium and stability analysis of the model

3.1. Basic regeneration number

Firstly, we give a definition of the basic regeneration number in this paper.

Definition 3.1. The drug abuse reproduction number (R_0) is the number of people who converted from susceptible to psychological addiction by a single drug addict (including psychological addicts and physiological addicts) introduced into a totally susceptible groups (including high-risk susceptible individuals and low-risk susceptible individuals with protection awareness) during his/her effective addiction period.

Clearly, system (2) has a unique Drug-Free equilibrium $E_0 = (S_0, P_0, 0, 0, 0, 0)$, where $S_0 = \frac{\Lambda}{b+\mu}$, $P_0 = \frac{b\Lambda}{b\mu+\mu^2}$. Using method in [4], we can obtain

$$R_0 = \frac{\beta(S_0 + \xi P_0)m_2m_3m_4 + q\beta(S_0 + \xi P_0)\gamma m_3m_4}{A},$$

where $A = m_1m_2m_3m_4 - \eta\rho\gamma k_1 - \eta\gamma k_2m_3 - \eta\rho\alpha m_2$.

From this, the basic regeneration number R_0 can be regarded as the sum of two parts. The first part is: $\frac{\beta(S_0+\xi P_0)m_2m_3m_4}{A}$. This part represents the effect of psychological addicts in spreading drugs. And the second part is $\frac{q\beta(S_0+\xi P_0)\gamma m_3m_4}{A}$. This part represents the effect of physiological addicts in spreading drugs. To summarize, the basic reproduction number formula represents the superposition of the impacts of psychological and physiological addicts. In addition, the numerator of R_0 represents the number of new drug users generated by psychological and physical addicts, while the denominator represents the number of drug users reduced through community treatment, and compulsory detoxification treatment. Then drug abuse will spread, if the growth rate of the drug-using population (numerator of R_0) is larger than the reduction rate (denominator of R_0).

3.2. Existence of equilibrium

In this subsection, we will discuss the existence of the equilibrium point of the system (2).

(1). There has a drug-free equilibrium $E_0 = (S_0, P_0, 0, 0, 0, 0)$, where $S_0 = \frac{\Lambda}{b+\mu}$, $P_0 = \frac{b\Lambda}{b\mu+\mu^2}$;

(2). Drug-persistent equilibrium $E^* = (S^*, P^*, I^*_{PC}, I^*_{PS}, R^*_C, R^*_I)$. Let the right-hand side of the system (2) equation be equal to zero, we can obtain

$$
\begin{aligned}
&I^*_{PC} = \frac{m_2}{\gamma} I^*_{PS},\ R^*_C = \frac{\alpha m_2 + \gamma k_1}{\gamma m_3} I^*_{PS},\\
&R^*_I = BI^*_{PS}, S^* = \frac{\gamma\Lambda}{\beta(m_2+q\gamma)I^*_{PS} + (b+\mu)\gamma},\\
&P^* = \frac{b\gamma\Lambda}{\Big(\beta(m_2+q\gamma)I^*_{PS} + \gamma(b+\mu)\Big)\Big(\beta\xi(m_2+q\gamma)I^*_{PS} + \gamma\mu\Big)},
\end{aligned}
$$

and I^*_{PS} satisfies the equation $f(I^*_{PS}) = D(I^*_{PS})^2 + EI^*_{PS} + F = 0$, where:

$$
\begin{aligned}
B &= \frac{\rho\alpha m_2 + \rho\gamma k_1 + \gamma m_3 k_2}{\gamma m_3 m_4},\\
D &= (\eta B - \frac{m_1 m_2}{\gamma})\beta^2\xi(\frac{m_2}{\gamma} + q) = \frac{-A}{\gamma m_3 m_4}\beta^2\xi(\frac{m_2}{\gamma} + q),\\
E &= \beta^2\Lambda\xi(\frac{m_2}{\gamma} + q) + (\eta B - (\frac{m_1 m_2}{\gamma})(\beta\mu + \beta\xi b + \beta\xi\mu)(\frac{m_2}{\gamma} + q)\\
&= \frac{-A}{\gamma m_3 m_4}\Big((\beta\mu + \beta\xi b + \beta\xi\mu)(\frac{m_2}{\gamma} + q) - \beta^2\xi\Lambda(\frac{m_2}{\gamma} + q)\frac{\gamma m_3 m_4}{A}\Big),\\
F &= (\beta\mu\Lambda + \beta\xi b\Lambda)(\frac{m_2}{\gamma} + q) + \big(\eta B - (\frac{m_1 m_2}{\gamma}\mu(b+\mu))\big)\\
&= \frac{-A}{\gamma m_3 m_4}\big(\mu(b+\mu)(1-R_0)\big).
\end{aligned}
$$

Since $f(I^*_{PS})'' = 2D < 0$, the quadratic polynomial $f(I^*_{PS})$ is a concave parabola and has a maximum point: $\max I^*_{PS} = \dfrac{-E}{2D}$, $\max f(I^*_{PS}) = \dfrac{4DF - E^2}{4D}$.

If $R_0 > 1$, then we have $F > 0$ and $E^2 - 4DF > 0$, the equation $f(I^*_{PS})$ has a unique positive root. From this we can obtain that system (2) has a unique drug-persistent equilibrium, when $R_0 > 1$.

3.3. Backward Bifurcation

The above theorem shows that in the third case, system (2) has a backward branch around $R_0 = 1$. Next, we apply the central manifold theory to study the backward branching. First, we rewrite the variables of system (2) as $S = x_1, P = x_2, I_{PC} =$

$x_3, I_{PS} = x_4, R_C = x_5, R_I = x_6$ and $N = \sum_{k=1}^{6} x_n$,

$$\begin{cases} \dfrac{\mathrm{d}x_1}{\mathrm{d}t} = \Lambda - \beta S I_{PC} - q\beta S I_{PS} - bS - \mu S = f_1, \\ \dfrac{\mathrm{d}x_2}{\mathrm{d}t} = bS - \xi\beta P I_{PC} - \xi q\beta P I_{PS} - \mu P = f_2, \\ \dfrac{\mathrm{d}x_3}{\mathrm{d}t} = \beta S I_{PC} + q\beta S I_{PS} + \xi\beta P I_{PC} + \xi q\beta P I_{PS} + \eta R_I - m_1 I_{PC} = f_3, \\ \dfrac{\mathrm{d}x_4}{\mathrm{d}t} = \gamma I_{PC} - m_2 I_{PS} = f_4, \\ \dfrac{\mathrm{d}x_5}{\mathrm{d}t} = \alpha I_{PC} + k_1 I_{PS} - m_3 R_C = f_5, \\ \dfrac{\mathrm{d}x_6}{\mathrm{d}t} = \rho R_C + k_2 I_{PS} - m_4 R_I = f_6, \end{cases} \tag{3.1}$$

where β is the bifurcation parameter. Using the relationship [18], $R_0 = 1$ corresponds to $\beta^* = \dfrac{A}{(S_0 + \xi P_0)(m_2 + q\gamma)m_3 m_4}$. Thus the Jacobian matrix is:

$$J(\beta^*, E_0) = \begin{pmatrix} -(b+\mu) & 0 & -\beta^* S_0 & -q\beta^* S_0 & 0 & 0 \\ b & -\mu & -\xi\beta^* P_0 & -q\xi\beta^* P_0 & 0 & 0 \\ 0 & 0 & \beta^* S_0 + \xi\beta^* P_0 - m_1 & q\beta^* S_0 + q\xi\beta^* P_0 & 0 & \eta \\ 0 & 0 & \gamma & -m_2 & 0 & 0 \\ 0 & 0 & \alpha & k_1 & -m_3 & 0 \\ 0 & 0 & 0 & k_2 & \rho & -m_4 \end{pmatrix}.$$

The system (4) with $\beta = \beta^*$ has a simple eigenvalue, hence the center manifold theory can be used [18]. The right eigenvector corresponding to the eigenvalue zero is $u = (u_1, u_2, u_3, u_4, u_5, u_6)$ and the left eigenvector is $v = (v_1, v_2, v_3, v_4, v_5, v_6)$, where

$$\begin{cases} u_1 = -\dfrac{A\mu}{\gamma m_3 m_4(\mu + \xi b)(\mu + b)}, \\ u_2 = \dfrac{\mu\Lambda A(\mu + b) - A\Lambda(\mu + \xi b)}{\gamma\mu\Lambda m_3 m_4(\mu + \xi b)}, \\ u_3 = \dfrac{m_2}{\gamma}, \\ u_4 = 1, \\ u_5 = \dfrac{\alpha m_2 + \gamma k_1}{\gamma m_3}, \\ u_6 = \dfrac{\alpha\rho m_2 + \gamma\rho k_1 + \gamma k_2 m_3}{\gamma m_3 m_4}, \end{cases}$$

and

$$\begin{cases} v_1 = 0, \\ v_2 = 0, \\ v_3 = 1, \\ v_4 = \dfrac{-q\gamma\alpha\rho m_2 + \gamma\rho\eta k_1 + \gamma\eta k_2 m_3 + q\gamma m_1 m_4 m_3}{\gamma m_3 m_4 (m_2 + q\gamma)}, \\ v_5 = \dfrac{\rho\eta}{m_3 m_4}, \\ v_6 = \dfrac{\eta}{m_4}. \end{cases}$$

The bifurcation coefficients a and b are as follows

$$\begin{aligned} a &= \sum_{k,i,j=1}^{6} v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(S_0, P_0, 0, 0, 0) \\ &= u_1 u_3 \beta + u_1 u_4 q\beta + u_2 u_3 \xi\beta + u_2 u_4 q\xi\beta \\ &= \frac{A^2}{\big(\gamma m_3 m_4(\xi b + \mu)\big)^2 \Lambda}(-\xi\mu^2 - \xi\mu b + \xi^2 b + \xi\mu + \mu^2), \\ b &= \sum_{k,i=1}^{6} v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(S_0, P_0, 0, 0, 0) \\ &= v_3 u_3 (S_0 + \xi P_0) + v_3 u_4 q (S_0 + \xi P_0) \\ &= \frac{\Lambda(m_2 + \gamma q)(\mu + \xi b)}{\gamma\mu(\mu + b)}. \end{aligned}$$

Obviously $b > 0$, and if $\xi^2 b + \xi\mu + \mu^2 > \xi\mu^2 + \xi\mu b$, $a > 0$. Thus, it is proved that there is a backward branch in system (2) when $R_0 < 1$. The presence of branching implies that the drug persistence equilibrium point may still exist even if the basic regeneration number R_0 is reduced to less than one unit. This indicates that drug abuse is still prevalent and more attention should be paid to strengthening interventions to improve treatment rates and reduce the effective contact rate between susceptible individuals and drug addicts to effectively control drug transmission.

3.4. Stability of equilibrium

Theorem 3.1. *The drug-free equilibrium* (E_0) *of system (2) is locally asymptotically stable (LAS) when* $R_0 < 1$.

Proof. The Jacobian matrix of the system (2) at E_0 is given by:

$$J\,|_{(E_0)}=\begin{pmatrix} -(b+\mu) & 0 & -\beta S_0 & -q\beta S_0 & 0 & 0 \\ b & -\mu & -\xi\beta P_0 & -q\xi\beta P_0 & 0 & 0 \\ 0 & 0 & \beta S_0+\xi\beta P_0-m_1 & q\beta S_0+q\xi\beta P_0 & 0 & \eta \\ 0 & 0 & \gamma & -m_2 & 0 & 0 \\ 0 & 0 & \alpha & k_1 & -m_3 & 0 \\ 0 & 0 & 0 & k_2 & \rho & -m_4 \end{pmatrix}.$$

The eigenvalues of $J\,|_{(E_0)}$ are calculated as $\lambda_1=-(b+\mu),\ \lambda_2=-\mu$ and the other solutions of the following equation:

$$\lambda^4+C_1\lambda^3+C_2\lambda^2+C_3\lambda+C_4=0,$$

where

$$\begin{aligned} C_1 &= m_1+m_2+m_3+m_4-\beta S_0-\xi\beta P_0, \\ C_2 &= m_1m_3+m_1m_2+m_1m_4+m_3m_4+m_2m_3+m_2m_4 \\ &\quad -(m_2+m_3+m_4)(\beta S_0+\xi\beta P_0)-\gamma(q\beta S_0-q\xi\beta P_0), \\ C_3 &= m_1m_3m_4+m_2m_3m_4+m_1m_2(m_3+m_4)-m_2(m_3+m_4)(\beta S_0+\xi\beta P_0) \\ &\quad -m_3m_4(\beta S_0+\xi\beta P_0)-\gamma(m_3+m_4)(q\beta S_0-q\xi\beta P_0)+\gamma\eta k_2+\alpha\eta\rho, \\ C_4 &= m_1m_2m_3m_4-m_2m_3m_4(\beta S_0+\xi\beta P_0)-m_3m_4\gamma(q\beta S_0-q\xi\beta P_0) \\ &\quad +\gamma\eta\rho K_1+\gamma\eta k_2m_3+\alpha\rho\eta m_2. \end{aligned}$$

If $R_0<1$, then $m_2m_3m_4(\beta S_0+\xi\beta P_0)<m_1m_2m_3m_4$ and $m_3m_4\gamma(q\beta S_0-q\xi\beta P_0)<m_1m_2m_3m_4$, i.e. $\beta(S_0+\xi P_0)<m_1$ and $q\gamma\beta(S_0+\xi P_0)<m_1m_2$, we have $C_1>0,\ C_2>0,\ C_3>0,\ C_4>0$. According to Routh-Hurwitz criterion, all eigenvalues of $J\,|_{(E_0)}$ have negative parts when $C_3(C_1C_2-C_3)>C_1^2C_4$, which leads to the conclusion that E_0 of system (2) is locally asymptotically stable when $R_0<1$ and $C_3(C_1C_2-C_3)>C_1^2C_4$. □

Theorem 3.2. *The drug-free equilibrium (E_0) of system (2) is globally asymptotically stable (GAS) when $R_0<1$.*

Proof. Introducing the following Lyapunov function:

$$L=S-S_0-S_0\ln\frac{S}{S_0}+P-P_0-P_0\ln\frac{P}{P_0}+I_{PC}+D_1I_{PS}+D_2R_C+D_3R_I,$$

where

$$D_1=\frac{m_1m_3m_4-m_3m_4\beta(S_0+\xi P_0)-\alpha\rho\eta}{\gamma m_3m_4},\ D_2=\frac{\rho\eta}{m_3m_4},\ D_3=\frac{\eta}{m_4}.$$

The derivative of L is

$$\frac{dL}{dt}=(S-S_0)(\Lambda(\frac{1}{S}-\frac{1}{S_0})-\beta I_{PC}-q\beta I_{PS})$$

$$
\begin{aligned}
&+ (P - P_0)((\frac{1}{P} - \frac{1}{P_0}) + b(\frac{S}{P} - \frac{S_0}{P_0}) - \xi\beta I_{PC} - q\xi\beta I_{PS}) \\
&+ (\beta I_{PC} + q\beta I_{PS})((S - S_0) + \xi(P - P_0) + (S_0 + \xi P_0)) \\
&- m_1 I_{PC} + \eta R_I + D_1(\gamma I_{PC} - m_2 I_{PS}) \\
&+ D_2(\alpha I_{PC} + k_1 I_{PS} - m_3 R_C) + D_3(\rho R_C + k_2 I_{PS} - m_4 R_I) \\
&= \Lambda(2 - \frac{S}{S_0} - \frac{S_0}{S}) + bS_0(1 + \frac{S}{S_0} - \frac{P}{P_0} - \frac{SP_0}{PS_0}) + f(I_{PS}),
\end{aligned}
$$

where

$$
\begin{aligned}
f(I_{PS}) &= (q\beta(S_0 + \xi P_0) - D_1 m_2 + D_2 k_1 + D_3 k_2) I_{PS} \\
&= (\beta(S_0 + \xi P_0) \frac{m_2 m_3 m_4 + q\gamma m_3 m_4}{\gamma m_3 m_4} \\
&\quad - \frac{m_1 m_2 m_3 m_4 + \alpha\rho\eta m_2 - \gamma\rho\eta k_1 - \gamma\eta k_2 m_3}{\gamma m_3 m_4}) I_{PS}.
\end{aligned}
$$

If $R_0 < 1$, then $\beta(S_0 + \xi P_0)(m_2 m_3 m_4 + q\gamma m_3 m_4) < m_1 m_2 m_3 m_4 + \alpha\rho\eta m_2 - \gamma\rho\eta k_1 - \gamma\eta k_2 m_3$, $f(I_{PS}) < 0$. And according to the geometric inequality, we have $(2 - \frac{S}{S_0} - \frac{S_0)}{S}) \leq 0$, $(1 + \frac{S}{S_0} - \frac{P}{P_0} - \frac{SP_0}{PS_0}) \leq 0$. Then we can obtain that $\frac{dL}{dt} \leq 0$ for $S, P > 0$, and $\frac{dL}{dt} = 0$ if and only if $S = S_0$, $P = P_0$. By LaSalle's invariance principle, the unique drug-free equilibrium is globally asymptotically stable. It completes the proof. □

Theorem 3.3. *The drug-persistent equilibrium* (E^*) *of system (2) is GAS when* $R_0 > 1$.

Proof. Introducing $f(a) = 1 - a + \ln a$ as in [2]. It is easy to prove $f(a) = 1 - a + \ln a \leq 0$ for all $a > 0$. It follows that the derivative of V is

$$
V = V_1 + V_2 + V_3 + \frac{I^*_{PS}}{\gamma I^*_{PC}}(q\beta S^* + \xi q\beta P^*)V_4 + \frac{\eta R^*_I}{\alpha I^*_{PC} + k_1 I^*_{PS}} V_5 + \frac{\eta R I^*}{k_2 I^*_{PS} + \rho R^*_C} V_6,
$$

among which

$$
\begin{aligned}
&V_1 = S - S^* - S^* \ln \frac{S}{S^*}, \quad V_2 = P - P^* - P^* \ln \frac{P}{P^*}, \\
&V_3 = I_{PC} - I^*_{PC} - I^*_{PC} \ln \frac{I_{PC}}{I^*_{PC}}, \quad V_4 = I_{PS} - I^*_{PS} - I^*_{PS} \ln \frac{I_{PS}}{I^*_{PS}}, \\
&V_5 = R_C - R^*_C - R^*_C \ln \frac{R_C}{R^*_C}, \quad V_6 = R_I - R^*_I - R^*_I \ln \frac{R_I}{R^*_I}.
\end{aligned}
$$

Then we calculate the time derivative of V_4, V_5, V_6.

$$
\begin{aligned}
\frac{dV_4}{dt} &= \left(I_{PS} - I^*_{PS} - I^*_{PS} \ln \frac{I_{PS}}{I^*_{PS}}\right)' = \left(1 - \frac{I^*_{PS}}{I_{PS}}\right) \frac{dI_{PS}}{dt} \\
&= \left(1 - \frac{I^*_{PS}}{I_{PS}}\right) \left(\gamma I_{PC} - \gamma I^*_{PC} \frac{I_{PS}}{I^*_{PS}}\right) \\
&= \gamma I^*_{PC} \left(1 + \frac{I_{PC}}{I^*_{PC}} - \frac{I_{PS}}{I^*_{PS}} - \frac{I_{PC} I^*_{PS}}{I^*_{PC} I_{PS}}\right)
\end{aligned}
$$

$$
\begin{aligned}
&\leq \gamma I_{PC}^{*}\left(\frac{I_{PC}}{I_{PC}^{*}}-\ln\frac{I_{PC}}{I_{PC}^{*}}+\ln\frac{I_{PS}}{I_{PS}^{*}}-\frac{I_{PS}}{I_{PS}^{*}}\right),\\
\frac{dV_5}{dt}&=\left(R_C-R_C^{*}-R_C^{*}\ln\frac{R_C}{R_C^{*}}\right)'=\left(1-\frac{R_C^{*}}{R_C}\right)\frac{dR_C}{dt}\\
&=\left(1-\frac{R_C^{*}}{R_C}\right)\left(\alpha I_{PC}+k_1 I_{PS}-(\alpha I_{PC}^{*}+k_1 I_{PS}^{*})\frac{R_C}{R_C^{*}}\right)\\
&=\alpha I_{PC}^{*}\left(1+\frac{I_{PC}}{I_{PC}^{*}}-\frac{R_C}{R_C^{*}}-\frac{I_{PC}R_C^{*}}{I_{PC}^{*}R_C}\right)+k_1 I_{PS}^{*}\left(1+\frac{I_{PS}}{I_{PS}^{*}}-\frac{R_C}{R_C^{*}}-\frac{I_{PS}R_C^{*}}{I_{PS}^{*}R_C}\right)\\
&\leq \alpha I_{PC}^{*}\left(\frac{I_{PC}^{*}}{I_{PC}^{*}}-\ln\frac{I_{PC}}{I_{PC}^{*}}+\ln\frac{R_C}{R_C^{*}}-\frac{R_C}{R_C^{*}}\right)+k_1 I_{PS}^{*}\left(\frac{I_{PS}^{*}}{I_{PS}}-\ln\frac{I_{PS}}{I_{PS}^{*}}+\ln\frac{R_C}{R_C^{*}}-\frac{R_C}{R_C^{*}}\right),\\
\frac{dV_6}{dt}&=\left(R_I-R_I^{*}-R_I^{*}\ln\frac{R_I}{R_I^{*}}\right)'=\left(1-\frac{R_I^{*}}{R_I}\right)\frac{dR_I}{dt}\\
&=\left(1-\frac{R_I^{*}}{R_I}\right)\left(\rho R_C+k_2 I_{PS}-(\rho R_C^{*}+k_2 I_{PS}^{*})\frac{R_I}{R_I^{*}}\right)\\
&=\rho R_C^{*}\left(1+\frac{R_C}{R_C^{*}}-\frac{R_I}{R_I^{*}}-\frac{R_C R_I^{*}}{R_C^{*}R_I}\right)+k_2 I_{PS}^{*}\left(1+\frac{I_{PS}}{I_{PS}^{*}}-\frac{R_I}{R_I^{*}}-\frac{I_{PS}R_I^{*}}{I_{PS}^{*}R_I}\right)\\
&\leq \rho R_C^{*}\left(\frac{R_C}{R_C^{*}}-\ln\frac{R_C}{R_C^{*}}+\ln\frac{R_I}{R_I^{*}}-\frac{R_I}{R_I^{*}}\right)+k_2 I_{PS}^{*}\left(\frac{I_{PS}}{I_{PS}^{*}}-\ln\frac{I_{PS}}{I_{PS}^{*}}+\ln\frac{R_I}{R_I^{*}}-\frac{R_I}{R_I^{*}}\right).
\end{aligned}
$$

The derivative of $V_1+V_2+V_3$ along the solution of the system (2) is given by

$$
\begin{aligned}
&\frac{dV_1}{dt}+\frac{dV_2}{dt}+\frac{dV_3}{dt}\\
&=\left(S-S^{*}-S^{*}\ln\frac{S}{S^{*}}\right)'+\left(P-P^{*}-P^{*}\ln\frac{p}{p^{*}}\right)'\\
&\quad+\left(I_{PC}-I_{PC}^{*}-I_{PC}^{*}\ln\frac{I_{PC}}{I_{PC}^{*}}\right)'\\
&=\left(1-\frac{S^{*}}{S}\right)\frac{dS}{dt}+\left(1-\frac{P^{*}}{P}\right)\frac{dP}{dt}+\left(1-\frac{I_{PC}^{*}}{I_{PC}}\right)\frac{dI_{PC}}{dt}\\
&=bS^{*}\left(1-\frac{S}{S^{*}}-\frac{SP^{*}}{S^{*}P}+\frac{P^{*}}{P}\right)+\mu S^{*}\left(2-\frac{S^{*}}{S}-\frac{S}{S^{*}}\right)\\
&\quad+\mu P^{*}\left(2-\frac{P^{*}}{P}-\frac{P}{P^{*}}\right)+\beta S^{*}I_{PC}^{*}\left(2-\frac{S^{*}}{S}-\frac{S}{S^{*}}\right)\\
&\quad+\xi\beta P^{*}I_{PC}^{*}\left(2-\frac{P^{*}}{P}-\frac{P}{P^{*}}\right)+\eta R_I^{*}\left(1+\frac{R_I}{R_I^{*}}-\frac{I_{PC}}{I_{PC}^{*}}-\frac{R_I I_{PC}^{*}}{R_I^{*}I_{PC}}\right)\\
&\quad+q\beta S^{*}I_{PS}^{*}\left(2-\frac{S^{*}}{S}-\frac{I_{PC}}{I_{PC}^{*}}+\frac{I_{PS}}{I_{PS}^{*}}-\frac{SI_{PS}I_{PC}^{*}}{S^{*}I_{PS}^{*}I_{PC}}\right)\\
&\quad+\xi q\beta P^{*}I_{PS}^{*}\left(2-\frac{P^{*}}{P}-\frac{I_{PC}}{I_{PC}^{*}}+\frac{I_{PS}}{I_{PS}^{*}}-\frac{PI_{PS}I_{PC}^{*}}{P^{*}I_{PS}^{*}I_{PC}}\right)\\
&\leq bS^{*}\left(1-\frac{S}{S^{*}}-\frac{SP^{*}}{S^{*}P}+\frac{P^{*}}{P}\right)\\
&\quad+\mu S^{*}\left(2-\frac{S^{*}}{S}-\frac{S}{S^{*}}\right)+\beta S^{*}I_{PC}^{*}\left(2-\frac{S^{*}}{S}-\frac{S}{S^{*}}\right)
\end{aligned}
$$

$$
\begin{aligned}
&+\mu P^*\left(2-\frac{P^*}{P}-\frac{P}{P^*}\right)+\xi\beta P^* I_{PC}^*\left(2-\frac{P^*}{P}-\frac{P}{P^*}\right)\\
&+q\beta S^* I_{PS}^*\left(\frac{I_{PS}}{I_{PS}^*}-\ln\frac{I_{PS}}{I_{PS}^*}+\ln\frac{I_{PC}}{I_{PC}^*}-\frac{I_{PC}}{I_{PC}^*}\right)\\
&+\eta R_I^*\left(\frac{R_I}{R_I^*}-\ln\frac{R_I}{R_I^*}+\ln\frac{I_{PC}}{I_{PC}^*}-\frac{I_{PC}}{I_{PC}^*}\right)\\
&+\xi q\beta P^* I_{PS}^*\left(\frac{I_{PS}}{I_{PS}^*}-\ln\frac{I_{PS}}{I_{PS}^*}+\ln\frac{I_{PC}}{I_{PC}^*}-\frac{I_{PC}}{I_{PC}^*}\right).
\end{aligned}
$$

As a result, we obtain the time derivative of the Lyapunov function as follows

$$
\begin{aligned}
\frac{dV}{dt}=&\frac{dV_1}{dt}+\frac{dV_2}{dt}+\frac{dV_3}{dt}+\left(q\beta S^*+\xi q\beta P^*\right)\frac{I_{PS}^*}{\gamma I_{PC}^*}\frac{dV_4}{dt}\\
&+\frac{\eta R_I^*}{\alpha I_{PC}^*+k_1 I_{PS}^*}\frac{dV_5}{dt}+\frac{\eta R_I^*}{k_2 I_{PS}^*+\rho R_C^*}\frac{dV_6}{dt}\\
\le& bS^*\left(1+\frac{P^*}{P}-\frac{S^*}{S}-\frac{SP^*}{S^*P}\right)+\mu S^*\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)\\
&+\mu P^*\left(2-\frac{P^*}{P}-\frac{P}{P^*}\right)+\eta R_I^*\left(1+\frac{I_{PS}}{I_{PS}^*}-\frac{R_C}{R_C^*}-\frac{I_{PS}R_C^*}{I_{PS}^*R_C}\right)\\
&+\eta R_I^*\left(1+\frac{I_{PS}}{I_{PS}^*}-\frac{R_I}{R_I^*}-\frac{I_{PS}R_I^*}{I_{PS}^*R_I}\right).
\end{aligned}
$$

Hence, according to the geometric inequality

$$
\left(2-\frac{S}{S^*}-\frac{S^*}{S}\right)\le 0,\quad \left(2-\frac{P}{P^*}-\frac{P^*}{P}\right)\le 0,\quad \left(1+\frac{P^*}{P}-\frac{S^*}{S}-\frac{SP^*}{S^*P}\right)\le 0,
$$
$$
\left(1+\frac{I_{PS}}{I_{PS}^*}-\frac{R_C}{R_C^*}-\frac{I_{PS}R_C^*}{I_{PS}^*R_C}\right)\le 0,\quad \left(1+\frac{I_{PS}}{I_{PS}^*}-\frac{R_I}{R_I^*}-\frac{I_{PS}R_I^*}{I_{PS}^*R_I}\right)\le 0,
$$

$\frac{dV}{dt}\le 0$ holds whenever $R_0>1$. The $\frac{dV}{dt}=0$ can be satisfied only when $S=S^*$, $P=P^*$, $I_{PC}=I_{PC}^*$, $I_{PS}=I_{PS}^*$, $R_C=R_C^*$, $R_I=R_I^*$. By the LaSalle's invariance principle, we conclude that the drug-persistent equilibrium is globally asymptotically stable when $R_0>1$. □

4. Numerical Simulations

4.1. Numerical Simulations

This subsection gives some numerical simulations to illustrate stability of the equilibrium point. From Table 2, We fix $\Lambda=0.1$, $\mu=0.007$, $q=1.2$, $\gamma=0.55$, $\alpha=0.1$, $k_1=0.02$, $k_2=0.1$, $\rho=0.035$, $\eta=0.04$, $\delta_1=0.0001$, $\delta_2=0.05$, $\delta_3=0.1$. (The biological significance of each parameter is detailed in Table 1)

Let $b=0.027$, $\beta=0.03$, then $R_0=0.8136$. The dynamics of the system (2) with the different initial conditions were presented in Fig.2. It shows that the number of high-risk susceptible individuals (S) and low-risk susceptible individuals with protection awareness (P) will continue to exist, and the other compartments gradually converge to zero.

Table 2. Parameter value ranges for numerical simulation

Parameter	Range	Reference	Parameter	Range	Reference
Λ	[0.02,0.2]	[23]	k_1	[0.0,0.054]	[19]
μ	[0.0064,0.007]	[23]	k_2	[0.05,0.54]	[19]
q	[1.0,1.5]	[12]	ρ	[0.0,0.06012]	[19]
b	[0.01,0.5]	Estimated	η	[0.00002,0.9]	[19]
γ	[0.0015,0.6]	[12]	δ_1	[0.0001,1]	[19]
α	[0.0,0.3]	[12]	δ_2	[0.001,1]	[19]
β	[0.0,0.9399]	Estimated	δ_3	[0.01,1]	[19]

Let $b = 0.027$, $\beta = 0.3$, then $R_0 = 14.0973$. As can be seen in Fig.3 that all six populations in the drug dynamic model were present after about 80 days and the stability of the drug spread steady state is not dependent on the initial conditions. So there is a drug-persistent equilibrium point and it is asymptotically stable if $R_0 > 1$.

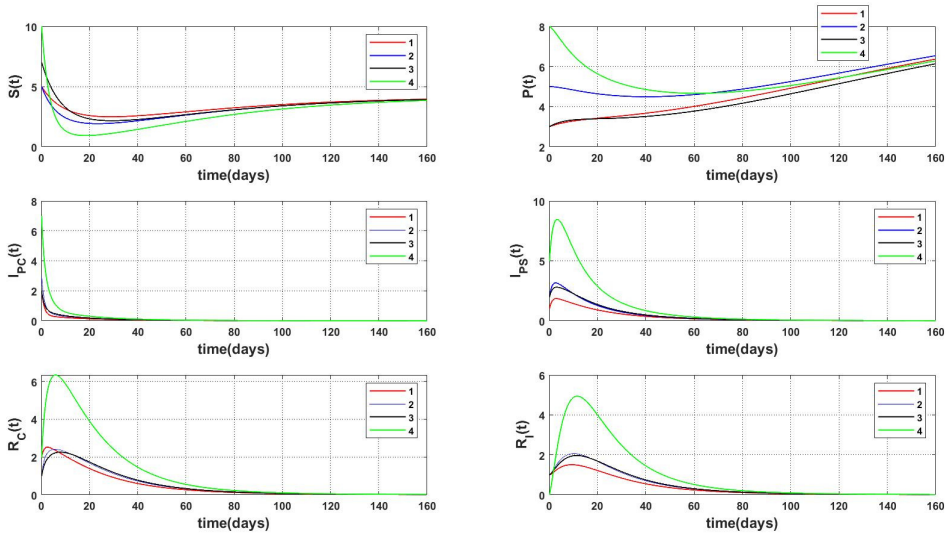

Figure 2. Numerical simulation of drug-free equilibrium under four different initial values

4.2. Sensitivity Analysis

In order to effectively control drug transmission, we will discuss the sensitivity of each parameter to R_0. As some parameters are beyond our control, we consider the sensitivity of eight parameters and calculate their partial derivatives :

$$\frac{\partial R_0}{\partial b} = \frac{\beta(m_2 + q\gamma)m_3m_4(\xi - 1)}{A(b+\mu)^2} < 0,$$

$$\frac{\partial R_0}{\partial \beta} = \frac{(S_0 + \xi P_0)(m_2 + q\gamma)m_3m_4}{A} > 0,$$

$$\frac{\partial R_0}{\partial \alpha} = \frac{\beta(S_0 + \xi P_0)(m_2 + q\gamma m_4m_3)(m_2m_3m_4 - \eta\rho m_2)}{A^2} < 0,$$

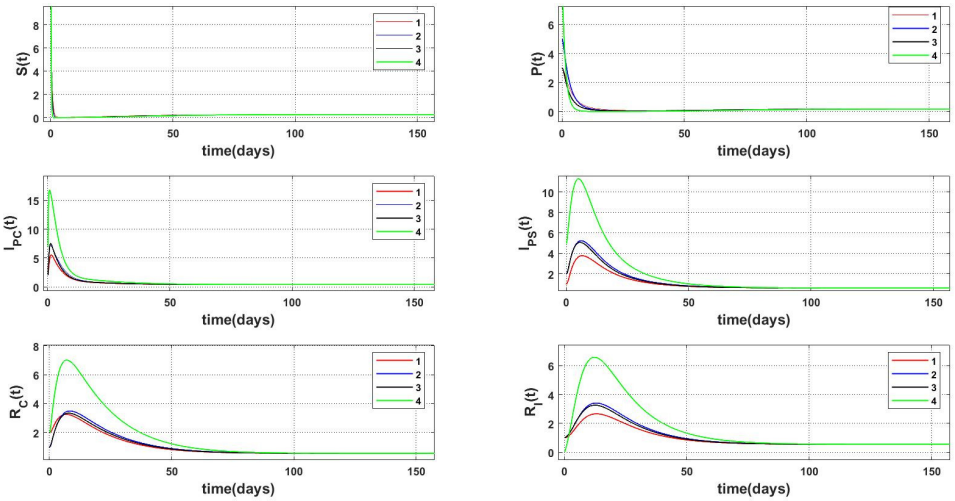

Figure 3. Numerical simulation of drug-persistent equilibrium under four different initial values

$$\frac{\partial R_0}{\partial \eta} = \frac{\beta(S_0+\xi P_0)(m_2+q\gamma)m_3(m_4-\eta)(\rho\gamma k_1+\gamma k_2 m_3+\rho\alpha m_2)}{A^2} > 0,$$
$$\frac{\partial R_0}{\partial \rho} = \frac{\beta(S_0+\xi P_0)(m_2+q\gamma)m_4(m_3-\rho)(\eta\gamma k_1+\eta\alpha m_2)}{A^2} > 0,$$
$$\frac{\partial R_0}{\partial \delta_1} = \frac{-\beta(S_0+\xi P_0)(m_2+q\gamma)m_2 m_3^2 m_4^2}{A^2} < 0,$$
$$\frac{\partial R_0}{\partial \delta_2} = \frac{-\beta(S_0+\xi P_0)(m_2+q\gamma)m_4(\eta\gamma\rho k_1+\eta\rho\alpha m_2)}{A^2} < 0,$$
$$\frac{\partial R_0}{\partial \delta_3} = \frac{-\beta(S_0+\xi P_0)(m_2+q\gamma)m_3(\eta\gamma\rho k_1+\eta\gamma k_2 m_3+\eta\rho\alpha m_2)}{A^2} < 0.$$

It can be seen that for all the parameters we considered, the results are as we expected. The results show that increasing the effective contact rate (β) increases R_0, and that increasing the failure rate (ρ and η) for both community-based treatment and compulsory detoxification treatment also increases R_0. Conversely, an increase in the treatment success rate, regardless of the treatment method used, is effective in reducing R_0.

According to the National Anti-Drug Network, 86% to 90% of people use drugs because of poor drug awareness and peer influence [20]. Next, we will simulate the power of media coverage, family education and public education in controlling the spread of drugs by drawing phase plane and contour maps (see Fig.4 and Fig.5).

From Fig.4 we can see that increasing b and decreasing β can both reduce R_0 to below unity. The rate that R_0 changes is accelerated by changing both β and b at the same time compared to simply changing one of the two. In addition, we can see from Fig.4(b) that the sensitivity of R_0 about β is much greater than that of b when $R_0 < 1$, but the sensitivity is gradually decreasing as R_0 increases. When R_0 is higher, the sensitivity of R_0 about b is larger than β. This suggests that in the early stages of controlling the spread of drugs, we should focus on strengthening the investigation of drug users and controlling the contact between susceptible people

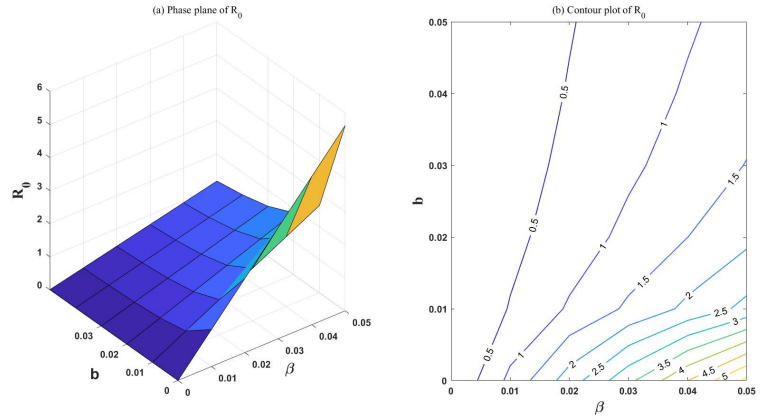

Figure 4. Sensitivity simulation of β (Effective contact rate between psychological addicts and high-risk susceptible individuals) and b (Conversion rate of high-risk susceptible individuals to low-risk susceptible individuals with protection awareness)

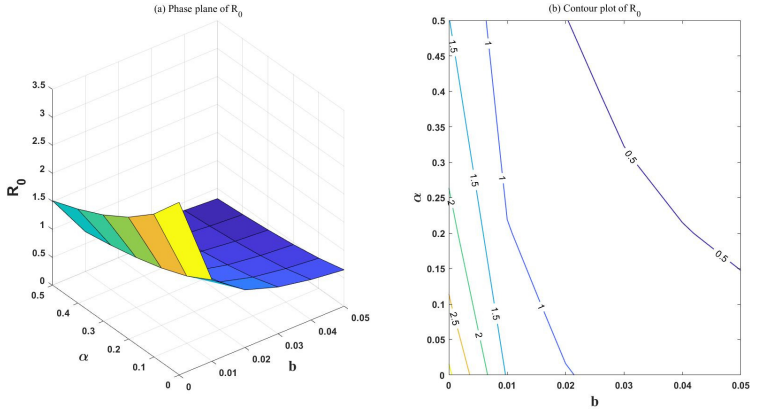

Figure 5. Sensitivity simulation of b (Conversion rate of high-risk susceptible individuals to low-risk susceptible individuals with protection awareness) and α (Rate of psychological addicts entering community treatment)

and drug addicts; When there is a drug epidemic trend, the focus should be shifted to strengthening anti-drug education and media coverage to transform more high-risk susceptible individuals into low-risk susceptible individuals. Given that in the actual implementation phase, resources for anti-drug reporting and education are not unlimited, the available police forces are also limited. Therefore, we must combine preventive measures with treatment measures to better control the spread of drugs. As can be seen in Fig.5, simultaneous increases in b and α, both of which need not be increased to a maximum, will make R_0 to fall rapidly below unity.

5. Conclusions

In this paper, a new six-dimensional drug transmission model is developed based on the main patterns of the drug epidemic in today's society. The total population is divided into six groups: high-risk susceptible individuals (S), low-risk susceptible in-

dividuals with protection awareness (P), psychological addicts (I_{PC}), physiological addicts (I_{PS}), drug addicts in community treatment (R_C), drug addicts in compulsory detoxification treatment (R_I). Then, through the analysis of the model, the results obtained from the paper are as follows:

(1). We have shown that drug-free equilibrium (E_0) of system (2) is globally asymptotically stable when $R_0 < 1$, and drug-persistent equilibrium (E^*) is globally asymptotically stable When $R_0 > 1$.

(2). We performed bifurcation analysis and proved that there is a backward branch in system (2) if $R_0 < 1$ and $\xi^2 b + \xi\mu + \mu^2 > \xi\mu^2 + \xi\mu b$. Sensitivity analysis was performed for parameters b, β, α, ρ and η, and then simulated the role played by media coverage, family education and public education in controlling the spread of drugs. The results show that initially the sensitivity of R_0 with respect to β is greater than that of b, but as R_0 increases, the sensitivity of R_0 with respect to b is gradually greater than that of β after $R_0 > 2$. This suggests that as the trend of drug spread increases, media coverage and anti-drug education play an increasingly important role in controlling the spread of drugs.

(3). Our study shows that the most effective way to control the spread of drugs is a combination of preventive and therapeutic measures. Considering the limited resources for anti-drug education and media coverage, as well as the limited police force, an appropriate proportion of preventive and treatment measures invested at different stages can be economical and effective in controlling the spread of drugs. When $R_0 < 1$, the focus is on surveys of drug users, along with moderate media outreach and education. In this case, it is more effective to control the contact between susceptible individuals and drug addicts than to treat them. When $R_0 > 1$, the focus should shift to significantly increasing anti-drug education and media coverage, while also bringing more psychological addicts into community treatment. In this situation, prevention and treatment go hand in hand and can control the drug pandemic economically and quickly.

Competing Interests

The authors declare that they have no competing interests.

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