The Role of Family on the Transmission Model of Methamphetamine

IOP Conf. Series: Journal of Physics: Conf. Series **1039** (2018) 012036 doi:10.1088/1742-6596/1039/1/012036

The Role of Family on the Transmission Model of Methamphetamine

Surapol Naowarat[1] and Nuengruedee Kumat[2]

[1] Department of Mathematics, Faculty of Science and Technology, Suratthani Rajabhat University, Surat Thani ,84100, Thailand.
[2]Department of Mathematics, Faculty of Science and Technology,Suratthani Rajabhat University, Surat Thani ,84100, Thailand.

Email: su1963n@gmail.com

Abstract: In this paper, we proposed and analyzed the mathematical model of methamphetamine epidemics with the role of family was taken into account. The objective of this research was to investigate the role of family on the transmission model of methamphetamine epidemics. The standard method of differential equations was used to analyze the proposed model, to find the drug free equilibrium points and drug present equilibrium point of the model and to determine the stability of the proposed model for both equilibrium points. The drug reproductive number (R_0) was found by applying the next generation matrix. The analytic solution and numerical solution were carried out. The results shown the proposed model which represented by five differential equations, consist of five subgroups that were the susceptible , light methamphetamine users, hard methamphetamine users, clients of health services in treatment and recovered individuals. We found that there were two equilibrium points, drug free equilibrium point and drug present equilibrium point. From the numerical simulation, when the effective of the role of family $k = 0.9$, the drug reproductive number $R_0 = 0.4928 < 1$, which mean that the methamphetamine epidemic was not occurred. Whenever the effective of the role of family $k = 0.1$, the drug reproductive number $R_0 = 3.7296 > 1$, which mean that the methamphetamine epidemic was occurred. Both drug free equilibrium points and drug present equilibrium points were local asymptotically stable. We concluded that if we increase the effective of the role of family (k) in the human population, then the methamphetamine users will decrease.

1. Introduction

In recent year, Western Cape Province of South Africa, there has been a dramatic increase in treatment demand for drugs such as dagga, mandrax, cocaine, heroin and methamphetamine (MA). MA patients defined heroin as drug users who used MA as a primary or secondary drug of abuse, increase from 121 to 376 patients from second half of 2003 to the first half of 2004. More than 50% of the patients were under the age of 20 [1]. MA is a highly addictive stimulant drug. The common effects of intoxication are increased energy and self-confidence, euphoria, heightened libido and appetite suppression. Prolonged use is usually characterized by severe weight loss, mood swing, violent behavior and body organ disorders [2]. Kalula and Nyabadza[3] extended the model by White and Comiskey [4] to model substance abuse in South Africa in order to qualitatively investigate the dynamics of substance abuse and predict drug abuse trends. Morris and Parry [5] reported that the high rates of increase of HIV infections in communities with high MA use. MA use has been linked to risky sexual behavior and

sexually transmitted infections. Families and friends play an incredibly critical role in motivation individuals with drug problems to enter treatment [6]. Rittisak and Naowarat [7] proposed and analyzed a mathematical model to study the dynamics of giving up smoking with effect of education campaign. It found that with an increase in the education campaign, the number of everyday smokers will decrease. Nyabadza, Njagarah and Smith [8] studied the modeling the dynamic of crystal meth abuse in the presence of drug-supply chains in South Africa by considering a model for MA use that tracks drug-supply chains, and accounts for rehabilitation and amelioration for the addicted. There was a unique drug free equilibrium. The sensitivity analysis is applied for this model. It found that the parameters with the most control over the epidemic are the quitting rate of light-drug users and the person-person contact rate between susceptible individuals and MA uses. This study is motivated by the recent work of [1] , by adding the parameter the role of family of [6,9] for those study. So, the objective for this study is to propose and analyze the MA epidemics with the role of family.

This paper is organized as follows. In section 2, the deterministic model is presented to describe the role of family. In section 3, the model analysis is carried out. In section 4, numerical results are illustrated our analytic results. The conclusion is presented in section 5.

2. Material and method

2.1 Material

To formulate the model , we consider the total human population at time t, denoted by N, is divided into five disjoint classes namely susceptible individuals at risk of using MA class S, light MA users class I_1, hard MA users class I_2, clients of health care services in treatment class T, and recovered individuals class, R. Hence, $S+I_1+I_2+T+R=N$. The possible changes in the life of a MA user can be tracked by the schematic representation in Fig. 1

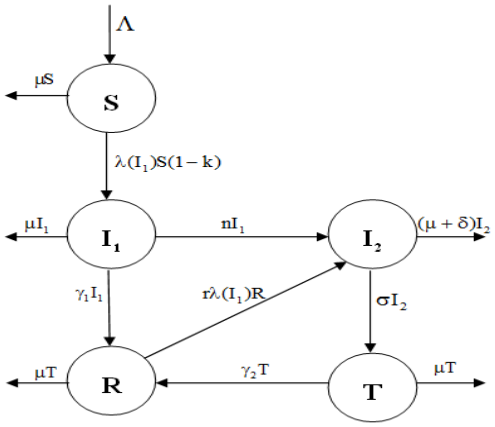

Fig. 1. Transfer diagram of the MA epidemic model.

Based on Fig. 1, the model is mathematically described by the following system of differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \frac{SI_1(1-k)}{a+I_1} - \mu S \\
\frac{dI_1}{dt} &= \frac{SI_1(1-k)}{a+I_1} - (\mu+\eta+\gamma_1)I_1 \\
\frac{dI_2}{dt} &= \eta I_1 + \frac{rRI_1}{a+I_1} - (\mu+\sigma+\delta)I_2 \\
\frac{dT}{dt} &= \sigma I_2 - (\mu+\gamma_2)T \\
\frac{dR}{dt} &= \gamma_1 I_1 + \gamma_2 T - r\frac{RI_1}{a+I_1} - \mu R
\end{aligned} \tag{1}$$

Where Λ is the recruitment rate of susceptible individuals, μ is the natural dead rate, η is the relative infectivity of I_2 when compare to I_1, r is the level of relapse to being a hard MA user, σ is the uptake rate into treatment programs, δ is the remove from the hard MA users' class that include drug related,

γ_1 is the recovery rate for light MA users, γ_2 is the recovery rate for MA users under treatment, a is the density of human population and k is the effective of the role of family.

2.2 Methods

From the proposed model, by setting the right-hand sides of the equations (1) to zero. We obtained two equilibrium points,

2.2.1 Drug Free Equilibrium(DFE) denoted by $E_0(S, I_1, I_2, T, R)$ **:** In this case when $I_1 = 0,$ we obtained

$$S = \frac{\Lambda}{\mu}, I_2 = 0, T = 0, R = 0, \text{ that is } E_0(\frac{\Lambda}{\mu}, 0, 0, 0).$$

2.2.2 Drug Present Equilibrium (DPE) denoted by $E_1(S^*, I_1^*, I_2^*, T^*, R^*)$ **:** In the case when $I_1^* > 0,$ we obtained

$S^* = \frac{\Lambda(a + I_1^*)}{A_1 I_1^* + a\mu}$, $I_2^* = \frac{B_1 I_1^{3*} + B_2 I_1^{2*} + naA_5 I_1^*}{B_3 I_1^{2*} + B_4 I_1^* + B_5}$, $T^* = \frac{\sigma I_1^{2*}}{\mu + \gamma_2}$, $R^* = \frac{A_2 I_1^{2*} + A_3 I_1^* + (\gamma_2 a\sigma + \gamma_2 \sigma I_1^*) I_2^*}{A_4 I_1^* + A_5}$ and $I_1^* = \frac{-C_2 \pm \sqrt{C_2^2 - 4C_1 C_3}}{2C_1}$,

Where $A_1 = (1-k) + \mu, A_2 = \gamma_1(\mu + \gamma_2),\ A_3 = a\gamma_1(\mu + \gamma_2),\ A_4 = (\mu + r)(\mu + \gamma_2),\ A_5 = a\mu(\mu + \gamma_2),\ B_1 = nA_4 + rA_2,\quad B_2 = nA_5 + rA_3 + anA_4,$

$B_3 = (\mu + \sigma + \delta)A_4 - \gamma_2 \sigma r,\ B_4 = (\mu a + \sigma a + \delta a)A_4 + (\mu + \sigma + \delta)A_5 - \gamma_2 a\sigma r,\ B_5 = (\mu + \sigma + \delta)aA_5, C_1 = (\mu + \eta + \gamma_1)A_1,$

$C_1 = (\mu + \eta + \gamma_1)A_1,\ C_2 = a(\mu + \eta + \gamma_1)A_1 + a\mu(\mu + \eta + \gamma_1) - \Lambda(1-k),\ C_3 = \mu a^2(\mu + \eta + \gamma_1) - \Lambda a(1-k).$

2.2.3 Drug Reproductive Number (R_0)

The drug reproductive number is defined as the number of secondary infections generated by a typical infected in an otherwise disease free population in his/her whole infectious period. The drug reproductive Number (R_0) for our model is computed using the method described in [10] and changing the notation as in [10] the matrices F and V are given by

$$F = \begin{bmatrix} 0 \\ \frac{I_1 S(1-k)}{a + I_1} \\ 0 \\ 0 \\ 0 \end{bmatrix}, V = \begin{bmatrix} -\Lambda + \mu S \\ (\mu + \eta + \gamma) I_1 \\ (\mu + \sigma + \delta) I_2 - \eta I_1 - r\lambda(I_1) R \\ -\sigma I_2 + (\mu + \gamma_2) T \\ -\gamma_1 I_1 - \gamma_2 T + r\lambda(I_1) R + \mu R \end{bmatrix}.$$

Now, the matrix F and V evaluated at disease free equilibrium point are given by

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\Lambda(1-k)}{\mu} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} \mu & \frac{\Lambda(1-k)}{a\mu} & 0 & 0 & 0 \\ 0 & \mu + \eta + \gamma & 0 & 0 & 0 \\ 0 & -\eta & \mu + \sigma + \delta & 0 & 0 \\ 0 & 0 & -\sigma & \mu + \gamma_2 & 0 \\ 0 & -\gamma_1 & 0 & -\gamma_2 & \mu \end{bmatrix}.$$

And the matrix FV^{-1} is given by

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\Lambda(1-k)}{a\mu(\mu + \eta + \gamma)} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

So, the drug reproduction number (R_0) which is the spectral radius of the matrix FV^{-1} is given by

$$R_0 = \frac{\Lambda(1-k)}{a\mu(\mu + \eta + \gamma_1)}.$$

2.2.4 The Stability of the Model

At the DFE, if all the eigenvalues of the Jacobean matrix of the system have negative real parts, then the DFE is locally asymptotically stable.

Theorem 1 The DFE for the system (1) is locally asymptotically if $R_0 < 1$.

Proof. The Jacobian matrix (J_0) of the model (1) at the DFE is given by

$$J_0=\begin{bmatrix} -\mu & \frac{\Lambda(1-k)}{a\mu} & 0 & 0 & 0 \\ 0 & \frac{\Lambda(1-k)}{a\mu}-(\mu+\eta+\gamma_1) & 0 & 0 & 0 \\ 0 & \eta & -(\mu+\sigma+\delta) & 0 & 0 \\ 0 & 0 & \sigma & -(\mu+\gamma_2) & 0 \\ 0 & \gamma_1 & 0 & \gamma_2 & -\mu \end{bmatrix}.$$

The eigenvalues of the Jacobian matrix are the solutions of the characteristic equation $|J_0-\lambda I|=0$,

$$\begin{vmatrix} -\mu-\lambda & \frac{\Lambda(1-k)}{a\mu} & 0 & 0 & 0 \\ 0 & \frac{\Lambda(1-k)}{a\mu}-(\mu+\eta+\gamma_1)-\lambda & 0 & 0 & 0 \\ 0 & \eta & -(\mu+\sigma+\delta)-\lambda & 0 & 0 \\ 0 & 0 & \sigma & -(\mu+\gamma_2)-\lambda & 0 \\ 0 & \gamma_1 & 0 & \gamma_2 & -\mu-\lambda \end{vmatrix}=0.$$

Expanding the determinant into a characteristic equation, we have

$$(\lambda+\mu)(\lambda+\mu)(\lambda+\mu+\gamma_2)(\lambda+\mu+\sigma+\delta)(\lambda+\frac{\Lambda(1-k)}{a\mu}-(\mu+\eta+\gamma_1))=0.$$

Thus, all the eigenvalues of the Jacobian matrix have negative real parts, implying that the DFE point is locally asymptotically stable $(R_0<1)$.

Theorem 2 The DPE for the system (1) is locally asymptotically if $R_0>1$

Proof. The Jacobian matrix (J_1) of the model (1) at the DPE is given by

$$J_1=\begin{bmatrix} -D_1 & D_3 & 0 & 0 & 0 \\ D_2 & -D_4 & 0 & 0 & 0 \\ 0 & D_5 & -D_7 & 0 & D_9 \\ 0 & 0 & \sigma & -D_8 & 0 \\ 0 & D_6 & 0 & \gamma_2 & -D_{10} \end{bmatrix}.$$

Where $D_1=\frac{I_1^*(1-k)}{a+I_1^*}+\mu,\ D_2=\frac{I_1^*(1-k)}{a+I_1^*},\ D_3=\frac{aS^*(1-k)}{(a+I_1^*)^2},\ D_4=\frac{aS^*(1-k)}{(a+I_1^*)^2}+(\mu+\eta+\gamma_1),\ D_5=\eta+\frac{arR^*}{(a+I_1^*)^2},$

$D_6=\gamma_1+\frac{arR^*}{(a+I_1^*)^2},\ D_7=\mu+\sigma+\delta,\ D_8=\mu+\gamma_2,\ D_9=\frac{rI_1^*}{a+I_1^*},\ D_{10}=\frac{rI_1^*}{a+I_1^*}+\mu.$

The eigenvalues of the Jacobian matrix are the solutions of the characteristic equation $|J_1-\lambda I|=0$,

$$\begin{vmatrix} -D_1-\lambda & D_3 & 0 & 0 & 0 \\ D_2 & -D_4-\lambda & 0 & 0 & 0 \\ 0 & D_5 & -D_7-\lambda & 0 & D_9 \\ 0 & 0 & \sigma & -D_8-\lambda & 0 \\ 0 & D_6 & 0 & \gamma_2 & -D_{10}-\lambda \end{vmatrix}=0.$$

Expanding the determinant into a characteristic equation, we have

$$\lambda^5+M_1\lambda^4+M_2\lambda^3+M_3\lambda^4+M_4\lambda+M_5=0.$$

Where $M_1=D_1+D_4+D_7+D_8+D_{10}$, $M_2=D_{10}(D_7+D_8)+(D_1+D_4)(D_7+D_8+D_{10}),\ M_5=\sigma\gamma_2D_9(D_1D_4+D_2D_3)$,

$M_3=D_7D_8D_{10}+D_7D_8+(D_1+D_4)(D_7D_8+D_7D_{10}+D_8D_{10})+\sigma\gamma_2D_9,\ M_4=(D_1+D_4)(D_7D_8D_{10}+\sigma\gamma_2D_9)$.

By applying Routh Hurwitz Criteria. All the eigenvalues of the Jacobian matrix have negative real parts,
if the coefficient of characteristic equation is satisfied this conditions:

1. $M_1>0,M_2>0,M_3>0,M_4>0,M_5>0.$
2. $M_1M_2M_3>M_3^2+M_1^2M_4$,
3. $(M_1M_4-M_5)(M_1M_2M_3-M_3^2-M_1^2M_4)>M_5(M_1M_2-M_3)^2+M_1M_2^5$,

So, it implied that the DPE is locally asymptotically stable $(R_0>1)$.

3. Results

From the numerical simulation, we presented the numerical results as shown in Fig. 2 and Fig. 3

3.1 Stability of drug free state: Using the values of parameters for simulation as followed, Λ = 0.028, μ =0.025, η = 0.008, r = 3.000, σ = 0.09, δ = 0.02, γ_1 = 0.01, γ_2 = 0.02, a = 0.01, k = 0.90.We obtained the values of eigenvalues and the drug reproductive number as follows

$$\lambda_1 = \lambda_2 = -0.025, \lambda_3 = -0.055, \lambda_4 = -0.04288, R_0 = 0.3696 < 1, E_0 = (1.12, 0, 0, 0, 0)\ .$$

Since all eigenvalues are to be negative and the drug reproductive number is less than one, the DFE, E_0 will be locally asymptotically stable, as shown in Fig. 2

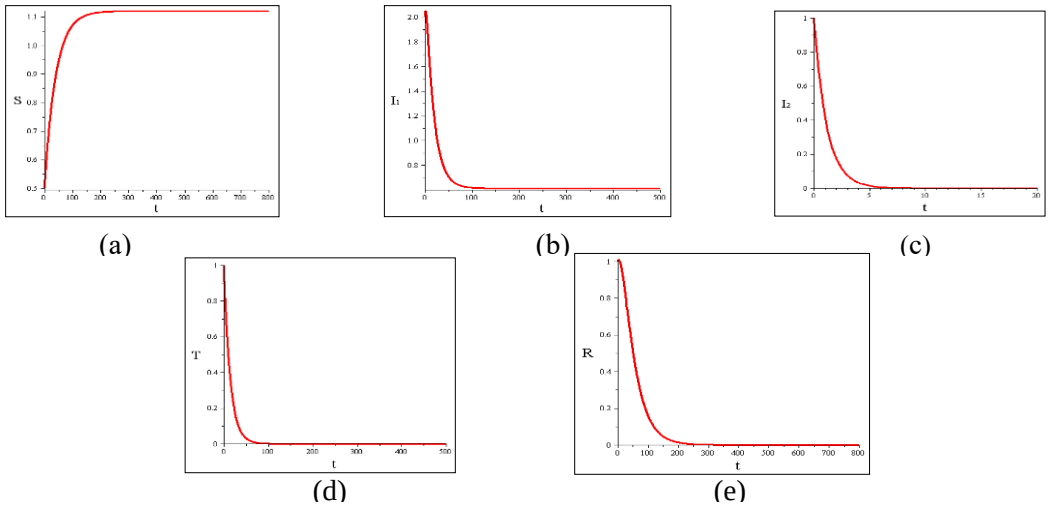

Fig. 2. The time series of (a) susceptible individuals at risk of using MA S, (b) light MA users I_1, (c) hard MA users I_2,(d) clients of health care services in treatment T, and recovered individuals, R with the values of parameters used from the text. We can see that the solutions converge to the DFE $(1.12, 0, 0, 0, 0)$

3.2 Stability of drug present state:

We changed the values of the effective of role family $k = 0.01$ and kept the other values of parameters to be those given in text. We obtained the values of eigenvalues and the drug reproductive number as follows

$$\lambda_1 = -0.0446964, \lambda_2 = -0.910689, \lambda_3 = -2.97826, \lambda_4 = -0.045205 - 0.03414i, \lambda_4 = -0.045205 + 0.03414i, R_0 = 3.7296 > 1,$$
$$E_1 = (0.0306070, 0.736034, 0.102036, 0.166967, 0.0026646).$$

Since all eigenvalues are to be negative and the drug reproductive number is greater than one, the DPE , E_1 will be locally asymptotically stable, as shown in Fig. 3

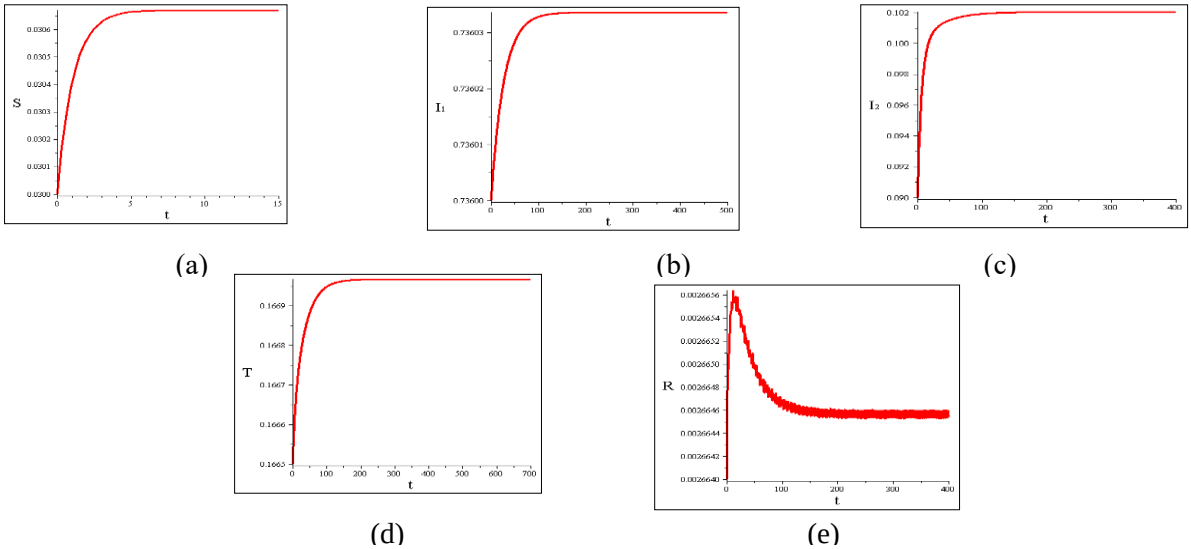

Fig. 3.The time series of (a) susceptible individuals at risk of using MA class S ,(b) light MA users I_1 ,(c) hard MA users I_2 ,(d) clients of health care services in treatment T , and recovered individuals , R with the values of parameters used from the text. We can see that the solutions converge to the DPE.

4. Conclusions

In this paper, we developed and analyzed a mathematical model for the MA epidemics. The role of family was taken into account. We analyzed the model by using the standard method. The drug reproductive is obtained through the use of next generation method and calculated the spectral radius of next generation matrix to obtain the drug reproductive number, $R_0 = \frac{\Lambda(1-k)}{a\mu(\mu+\eta+\gamma_1)}$. The drug reproductive number is the threshold condition for determining the stability of the model which are shown in Fig. 2and Fig. 3. Our simulation results shown that R_0 will increase when the effective of role of family decrease. We found the values of $R_0 = 0.3696,\ 3.7296$ when $k = 0.90,\ k = 0.01$, respectively. It seems that the light MA users and hard MA users will decrease when the effective of role of family is increased.

References

[1] Parry, C. 2005. *Substance abuse trends in the Western Cape: Summary Alcohol and Drug Abuse*. Medical Research Council, pp 195-213.

[2] Kapp, C. 2008. *Crysmeth boom adds to South Africa's health challenges* pp 193.

[3] Kalula, A. S. and Nyabadza, F. 2012. *A theoretical model for substance abuse in the presence of Treatment, S. Afr. J. Sci.* **108**(3-4), pp 1-12.

[4] White E and Comiskey C (2007)*Heroin epidemics treatment and ODE modelling*. Math. Biosci. **2007**(208), pp 312-324.

[5] Morris K and Parry C(2006) *South African methamphetamine boom could fuel further HIV* Lancet. **2006**(6) pp 471.

[6] Giord, S. 2017. *Family involvement is important in substance abuse treatment*, Psych Central. Jul 2017.

[7] Rittisak, T. and Naowarat, S. 2015. *Stability analysis and qualitative behavior of giving up smoking model with education campaign*, AJBAS, **2015**(9) pp 46-53.

[8] Nyabadza,F.Njagarah,J.B.H.and Smith,R.J. 2012. *Modelling the dynamic of crystal meth('Tik') abuse in the presence of drug-supply chains in South Africa***.** Bull. Math. **2012**(1) pp 24-48.

[9] Mushanyuzy, J. Nyabadza, F. Muchatibayaz, G. and Stewartz, A.G.R. 2016. *The role of family in initiating methamphetamine abuse treatment: insights through a mathematical model*. J. Appl Computat Math **2016**(5), pp 1-10.

[10] Driessche P V, and J Watmough J 2002 *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission***.** Math Biosci, **2002**(180), pp 29-48.

Acknowledgment

The authors would like to express their sincere thanks to the Department of Mathematics, Faculty of Science and Technology, Suratthani Rajabhat University, Thailand.