Commun Nonlinear Sci Numer Simulat 17 (2012) 1714–1724

Contents lists available at SciVerse ScienceDirect

Commun Nonlinear Sci Numer Simulat

journal homepage: www.elsevier.com/locate/cnsns

Assessing the impact of educational campaigns on controlling HCV among women in prison settings

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ARTICLE INFO

Article history:
Received 7 March 2011
Received in revised form 28 May 2011
Accepted 16 August 2011
Available online 31 August 2011

Keywords:
HCV
Women prisoners
Educational campaigns
Reproductive number
Sensitivity analysis

ABSTRACT

Prior studies have shown that imprisonment is a major risk factor for hepatitis C infection, with the risk of infection directly proportional to the length of incarceration. Women are at least twice as likely as men to contract HCV as they have limited access to information, health services and safe intravenous drug injecting equipments. We develop a mathematical model to assess the impact of educational campaigns on controlling HCV among women in prison settings. Equilibria for the model are determined and their stability are examined. Population-level effects of increased educational campaigns to encourage safe injecting practices among women in prison are evaluated through numerical simulations. The results suggest that educating women prisoners about abstaining from intravenous drug misuse may significantly reduce HCV prevalence among women in prison settings. Targeted education campaigns, which are effective at stopping transmission of HCV more than 80% of the time, will be highly effective at controlling the disease among women in prisons.

1. Introduction

Globally, female prisoners represent about 5% of the total prison population, but this proportion is increasing rapidly, particularly in countries where levels of illicit substance use are high [1]. In 2005, on any given date, more than half a million women and girls were detained in prisons worldwide, either awaiting trial or serving sentences [1]. Most women in prison are from socially marginalized groups and are more likely to have engaged in sex work and/or drug use [2]. However, in many jurisdictions, a larger proportion of women than men are in prison for drug-related offences [3,4]. Many of these women will continue using and injecting drugs in prison [5], while women who have never used drugs may begin to do so while in prison. In the absence of sterile injecting equipment, women, like men, will inject with used needles or with homemade syringes [6]. Women who inject drugs are more likely to become infected with blood-transmitted infectious diseases such as HIV, hepatitis B or C than men who inject drugs, as they have limited access to information, health services and safe injecting equipment [2,7].

Hepatitis C Virus (HCV) is a bloodborne pathogen that affects an estimated 130 million to 170 million people, or 2.2–3.0% of the world's population. Current major risk factors for infection include Intravenous Drug Users (IDUs) (more than 80% of infections) and other procedures requiring skin penetration, such as nonsterile injections, tattooing and other body art [8,9]. In prison populations, HCV prevalence is much higher, 6.9–46%, due to the high proportion of IDUs which constitute 22–54% of the prison population [10–19].

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doi:10.1016/j.cnsns.2011.08.024

Prisons socially determine the transmission of HCV infection among inmates in several ways. First, a high proportion of inmates are addicted to illicit drugs that are injected, and some of the convicted illicit drug users manage to continue their habit during incarceration [20,21]. Many drug-addicted inmates turn to tobacco and illicit drug use to satisfy their addictive cravings, as well as to numb the pain of imprisonment, famously classified by Sykes as deprivations of liberty, goods and services, heterosexual relationships, autonomy, and security [22]. Second, as a high proportion of individuals in contact with the criminal justice system have already contracted HCV prior to incarceration, prison settings magnify the probability of HCV transmission among inmates who engage in IDU. For example, a 2004 survey of 612 Australian prison entrants indicated that 56% had a history of IDU and 39% had injected in the previous month [23]. Of 81 inmates surveyed as part of a HCV seroconversion study in New South Wales prisons, 29 (36%) gave a history of IDU and 13 (16%) self-reported drug use in prison [20]. Apart from IDU, other risk factors for HCV transmission, such as assault, body piercings and tattooing, are also commonly practiced by inmates [24–26]. Third, custodial policies and practices influence the likelihood of inmates contracting HCV. Prisons with lax or poorly implemented policies in relation to illicit drug use make inmates more vulnerable to contracting HCV. Lax policies include weak surveillance of drug and injecting-equipment trafficking, and inadequate sanctions meted to inmates or custodial workers found to be involved with drug trafficking. A study in Australia in the 1990s indicated that about half of all imprisoned IDUs injected drugs in prison, while non-random urine drug tests may reinforce and perpetuate the original reasons for drug use in prison [27].

Transmission of HCV in prison settings poses a threat to public health for two reasons. First, the risk of HCV infection evolving into liver cirrhosis is 10–20% for infected patients and the associated morbidity, mortality and healthcare costs are substantial. Second, the prison setting acts as an important reservoir of HCV, with up to 70% of IDUs sharing needles within the prison setting and after discharge, because of a high turnover rate [8,13,16]. Mathematical models have become invaluable management tools for epidemiologists, both shedding light on the mechanisms underlying the observed dynamics as well as making quantitative predictions on the effectiveness of different control measures. The literature and development of mathematical epidemiology is well documented [28–30]. Modelling the spread of infectious diseases prevalent in prison settings is an important and interesting topic for a lot of researchers [31–34]. Here, we evaluate the role of education as a method of controlling HCV among women in prison settings.

The structure of the paper is as follows. In Section 2, we formulate the model. In Section 3, we derive analytical results. In Section 4, we present numerical simulations. We conclude with a discussion in Section 5.

2. Model formulation

The host population is sub-divided in the following epidemiological classes or subgroups of female prisoners: susceptible IDUs (X_D), susceptible non-IDUs (X_N), latently infected individuals (L), and infectious individuals (I). Thus, the total population (N) is given by $N = X_D + X_N + L + I$. Prisoners are recruited at a constant rate Λ. We assume that a fraction π_0, π_1, π_2, and π_3 are recruited into X_D, X_N, L, and I, respectively. Susceptible IDUs are infected with HCV through sharing contaminated needles or syringes at a rate $\lambda = \frac{\beta I}{N}$, where β is the probability of getting infected whenever a susceptible individual uses a contaminated needle, syringe or any other tool that might be used to share intravenous drugs whilst in prison. Susceptible non-IDUs who become IDUs whilst in prison and are not infected on their first exposure to intravenous drugs join the X_D class at a constant rate α, while those who are infected on their first exposure to intravenous drugs move into the L class at a rate $\theta\lambda$, where θ $(0 < \theta < 1)$ models the reduced chances of a susceptible non-IDU individual to be infected on her first exposure to intravenous drugs compared to a susceptible IDU. Upon serving their sentence or amnesty, prisoners are assumed to leave the prison settings at a constant rate ω. The natural mortality rate μ is assumed to be constant in all classes. The role of health-education activities, such as counselling about safe injecting practices, is represented by κ. If $\kappa = 0$, then education campaigns are not effective; $\kappa = 1$ corresponds to completely effective education campaigns, while $0 < \kappa < 1$ implies that education campaigns about safe injecting practices in prison will be effective to some degree. The incubation rate is γ. We assume that education is broadly effective, reducing the initial IDU rate and the rate of infection on both first adoption and subsequently.

The model is thus

$$\begin{aligned} X_D' &= \Lambda\pi_0 - ((1-\kappa)\lambda + \mu + \omega)X_D + (1-\kappa)\alpha X_N, \\ X_N' &= \Lambda\pi_1 - ((1-\kappa)\alpha + (1-\kappa)\theta\lambda + \mu + \omega)X_N, \\ L' &= \Lambda\pi_2 + (1-\kappa)\lambda(X_D + \theta X_N) - (\gamma + \omega + \mu)L, \\ I' &= \Lambda\pi_3 + \gamma L - (\mu + \omega)I. \end{aligned} \tag{1}$$

The model flow diagram is depicted in Fig. 1.

For system (1), the first octant in the state space is positively invariant and attracting; that is, solutions that start where all the variables are non-negative and remain there. Thus, system (1) will be analyzed in a suitable region $\Phi \subset \mathbb{R}^4_{\geqslant 0}$. The region

$$\Phi = \left\{ (X_D, X_N, L, I) \in \mathbb{R}^4_{\geqslant 0} : N \leqslant \frac{\Lambda}{\mu + \omega} \right\} \tag{2}$$

is positively invariant and attracting. Existence, uniqueness and continuation results for system (1) hold in this region.

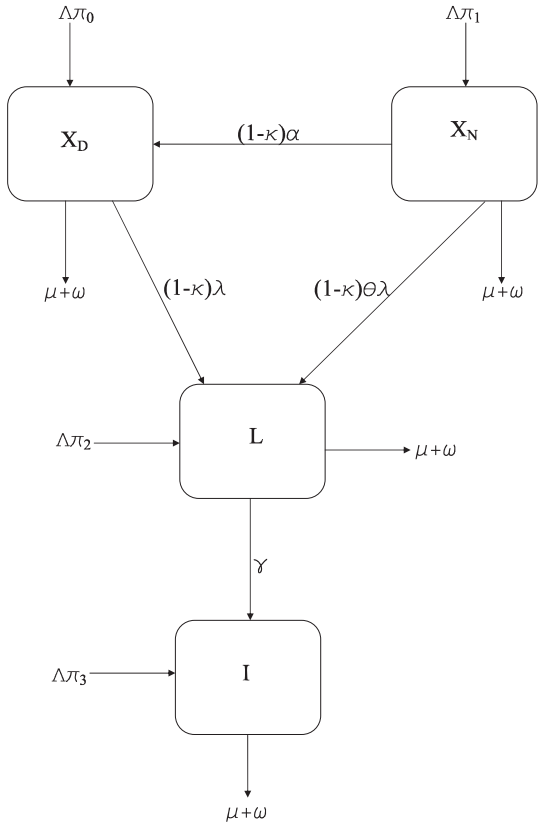

Fig. 1. Model flow diagram.

3. Analytical results

3.1. The disease-free equilibrium

Model system (1) has an evident disease-free equilibrium (DFE) given by

$$\mathcal{V}^0 = \left(X_D^0, X_N^0, L^0, I^0\right) = \left(\frac{\Lambda[\pi_0(\mu+\omega+\alpha(1-\kappa))+\alpha(1-\kappa)\pi_1]}{(\mu+\omega)(\mu+\omega+\alpha(1-\kappa))}, \frac{\Lambda\pi_1}{(\alpha(1-\kappa)+\mu+\omega)}, 0, 0\right). \tag{3}$$

The linear stability of $\mathcal{V}^0$ is obtained using the next-generation matrix [37,38] for system (1). Using the notation in [37], the nonnegative matrix F (representing the new infection terms) and the nonsingular matrix V (representing the remaining transfer terms) are given by

$$F = \begin{bmatrix} 0 & \frac{\beta(1-\kappa)[\pi_0(\alpha(1-\kappa)+\mu+\omega)+\pi_1(\alpha(1-\kappa)+\theta(\mu+\omega))]}{(\pi_0+\pi_1)(\alpha(1-\kappa)+\mu+\omega)} \\ 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} \gamma+\mu+\omega & 0 \\ -\gamma & \mu+\omega \end{bmatrix}.$$

Thus, the reproductive number for system (1) denoted by $\mathcal{R}_e$ is given by

$$\mathcal{R}_e = \frac{\beta(1-\kappa)\gamma[\pi_0(\alpha(1-\kappa)+\mu+\omega)+\pi_1(\alpha(1-\kappa)+\theta(\mu+\omega))]}{(\pi_0+\pi_1)(\mu+\omega)(\gamma+\mu+\omega)(\alpha(1-\kappa)+\mu+\omega)}. \tag{4}$$

$\mathcal{R}_e$ is a threshold for disease invasion or eradication, under suitable conditions, such as the absence of a backward bifurcation. See [39] for more discussion. Using Theorem 2 in [37], the following result is established.

Lemma 1. *The disease-free equilibrium $\mathcal{V}^0$ of system (1) is locally asymptotically stable if $\mathcal{R}_e \leqslant 1$ and unstable if $\mathcal{R}_e > 1$.*

The epidemiological implication of Lemma 1 is that HCV can be eliminated from the prison settings among women prisoners whenever $\mathcal{R}_e \leqslant 1$, provided the initial sizes of the sub-populations of the system (1) are in the basin of attraction of $\mathcal{V}^0$. In other words, an influx of a small number of infected women prisoners will not generate large outbreaks if $\mathcal{R}_e \leqslant 1$.

We claim the following result.

Lemma 2. *The disease-free equilibrium $(\mathcal{V}^0)$ of model system (1) is globally asymptotically stable (GAS) if $\mathcal{R}_e \leqslant 1$ and unstable if $\mathcal{R}_e > 1$.*

Proof. Following Castillo-Chavez et al. [41], we write system (4) in the form

$$\begin{aligned} X'(t) &= F(X,Y),\\ Y'(t) &= G(X,Y), \quad G(X,\mathbf{0}) = 0, \end{aligned} \tag{5}$$

where $X = (X_N, X_D)$ and $Y = (E, I)$. Here $X \in \mathbb{R}^2_+$ denotes the number of uninfected individuals and $Y \in \mathbb{R}^2_+$ denotes the number of infected individuals. The DFE is now denoted by $\mathcal{V}^0 = (X_0, \mathbf{0})$ where

$$X_0 = \left(\frac{\Lambda[\pi_0(\mu+\omega+\alpha(1-\kappa))+\alpha(1-\kappa)\pi_1]}{(\mu+\omega)(\mu+\omega+\alpha(1-\kappa))}, \frac{\Lambda\pi_1}{(\alpha(1-\kappa)+\mu+\omega)}\right).$$

We have to prove that the two conditions

$$\begin{aligned} &(H1)\ \text{For } X'(t) = F(X,0), && X \text{ is globally asymptotically stable},\\ &(H2)\ G(X,Y) = UY - \widehat{G}(X,Y), && \widehat{G}(X,Y) \geqslant 0 \text{ for } (X,Y) \in \Phi \end{aligned} \tag{6}$$

are satisfied, where Φ is a positively invariant attracting domain. Consider

$$F(X,0) = \begin{bmatrix} \Lambda\pi_0 - (\mu+\omega)X_D + (1-\kappa)X_N \\ \Lambda\pi_1 - (\mu+\omega)X_N - (1-\kappa)X_N \end{bmatrix},$$

$$U = \begin{bmatrix} -(\gamma+\mu+\omega) & \frac{\beta(1-\kappa)[\pi_0(\alpha(1-\kappa)+\mu+\omega)+\pi_1(\alpha(1-\kappa)+\theta(\mu+\omega))]}{(\pi_0+\pi_1)(\alpha(1-\kappa)+\mu+\omega)} \\ \gamma & -(\mu+\omega) \end{bmatrix}.$$

Thus

$$\widehat{G}(X,Y) = \begin{bmatrix} \widehat{G_1}(X,Y) \\ \widehat{G_2}(X,Y) \end{bmatrix} = \begin{bmatrix} \beta(1-\kappa)I\left(1-\frac{X_D+\theta X_N}{N}\right) \\ 0 \end{bmatrix}. \tag{7}$$

Since, $0 \leqslant \theta \leqslant 1$, and $0 \leqslant X_D + \theta X_N \leqslant X_D + X_N + L + I \leqslant N$, it follows that $\widehat{G}(X,Z) \geqslant 0$, and by Lemma 2, $\mathcal{V}^0$ is GAS. □

3.2. Endemic equilibrium

Model system (1) has an endemic equilibrium (EE) given by

$$\mathcal{V}^* = \left(X_D^*, X_N^*, L^*, I^*\right),$$

where

$$\begin{aligned} X_D^* &= \frac{\Lambda[\pi_0(\theta(1-\kappa)\lambda* + \mu+\omega) + \alpha(\pi_0+(1-\kappa)\pi_1)]}{((1-\kappa)\lambda^* + \mu+\omega)(\alpha+\theta(1-\kappa)\lambda* + \mu+\omega)},\\ X_N^* &= \frac{\Lambda\pi_1}{(\alpha+\theta(1-\kappa)\lambda* + \mu+\omega)},\\ L^* &= \frac{\Lambda\pi_2 + (1-\kappa)\lambda^*\left(X_D^* + \theta X_N^*\right)}{(\gamma+\omega+\mu)},\\ I^* &= \frac{\Lambda\pi_3(\gamma+\omega+\mu) + \gamma[\Lambda\pi_2 + (1-\kappa)\lambda^*\left(X_D^* + \theta X_N^*\right)]}{(\omega+\mu)(\gamma+\omega+\mu)}. \end{aligned} \tag{8}$$

The following theorem will be useful.

Theorem 1. *(See [36].) Consider the following general system of ordinary differential equations with a parameter ϕ,*

$$\frac{dx}{dt} = f(x,\phi), \quad f : \mathbb{R}^n \times \mathbb{R} \to \mathbb{R}^n \quad \text{and } f \in \mathbb{C}^2(\mathbb{R}^n \times \mathbb{R}). \tag{9}$$

Without loss of generality, it is assumed that 0 is an equilibrium for system (9) for all values of the parameter ϕ; that is, $f(0,\phi)=0$ for all ϕ. Assume

A1: $A = D_x f(0,0) = \left(\frac{\partial f_i}{\partial x_j}(0,0)\right)$ *is the linearisation of system (9) around the equilibrium 0 with ϕ evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts;*

A2: *Matrix A has a right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue.*

Let f_k be the kth component of f and

$$\begin{aligned} a &= \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(0,0), \\ b &= \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \phi}(0,0). \end{aligned} \tag{10}$$

The local dynamics of (9) around 0 are governed by a and b in the following manner:

i. *$a>0$, $b>0$, When $\phi<0$ with $|\phi| \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium; when $0<\phi \ll 1$, 0 is unstable and there exists a negative and locally asymptotically stable equilibrium;*
ii. *$a<0$, $b<0$. When $\phi<0$ with $|\phi| \ll 1$, 0 is unstable; when $0<\phi \ll 1$, 0 is locally asymptotically stable, and there exists a positive unstable equilibrium;*
iii. *$a>0$, $b<0$. When $\phi<0$ with $|\phi| \ll 1$, 0 is unstable, and there exists a locally asymptotically stable negative equilibrium; when $0<\phi \ll 1$, 0 is stable, and a positive unstable equilibrium appears;*
iv. *$a<0$, $b>0$. When ϕ changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly, a negative equilibrium becomes positive and locally asymptotically stable.*

In order to apply the Center Manifold Theory, we make the following change of variables. Set $X_D = x_1$, $X_N = x_2$, $L = x_3$, $I = x_4$, so that $N = \sum_{n=1}^{4} x_n$ and $\lambda = \frac{\beta x_4}{\sum_{n=1}^{4} x_n}$. Further, by using vector notation $\vec{x} = (x_1, x_2, x_3, x_4)^T$, model system (1) can be written in the form $\frac{d\vec{x}}{dt} = F(\vec{x})$, with $F = (f_1, f_2, f_3, f_4)^T$. That is:

$$\begin{aligned} x_1' &= f_1 = \Lambda\pi_0 - ((1-\kappa)\lambda + \mu + \omega)x_1 + (1-\kappa)\alpha x_2, \\ x_2' &= f_2 = \Lambda\pi_1 - ((1-\kappa)\alpha + (1-\kappa)\theta\lambda + \mu + \omega)x_2, \\ x_3' &= f_3 = \Lambda\pi_2 + (1-\kappa)\lambda(x_1 + \theta x_2) - (\gamma + \omega + \mu)x_3, \\ x_4' &= f_4 = \Lambda\pi_3 + \gamma x_3 - (\mu + \omega)x_4. \end{aligned} \tag{11}$$

The method entails evaluating the Jacobian of system (11) at $\mathcal{V}^0$, with $X_D^0 = x_1^0 = m$ and $X_N^0 = x_2^0 = n$. Thus,

$$J(\mathcal{V}^0) = \begin{bmatrix} -\mu-\omega & \alpha(1-\kappa) & 0 & -\frac{\beta(1-\kappa)m}{m+n} \\ 0 & -\alpha(1-\kappa)-\mu-\omega & 0 & -\frac{\beta(1-\kappa)\theta n}{m+n} \\ 0 & 0 & -\gamma-\mu-\omega & \frac{\beta(1-\kappa)(m+\theta n)}{m+n} \\ 0 & 0 & \gamma & -\mu-\omega \end{bmatrix} \tag{12}$$

from which it can be shown that

$$\mathcal{R}_e = \frac{\beta(1-\kappa)\gamma(m+\theta n)}{(m+n)(\mu+\omega)(\gamma+\mu+\omega)}. \tag{13}$$

Suppose β is chosen as a bifurcation parameter. Solving (13) for $\mathcal{R}_e = 1$, one gets

$$\beta = \beta^* = \frac{(m+n)(\mu+\omega)(\gamma+\mu+\omega)}{(1-\kappa)\gamma(m+\theta n)}. \tag{14}$$

It can be shown that the Jacobian $J(\mathcal{E}^0)$ of system (11) at $\beta = \beta^*$ has a right eigenvector (corresponding to the zero eigenvalue) given by $\vec{w} = (w_1, w_2, w_3, w_4)^T$, where

$$\begin{aligned} w_1 &= -\frac{\beta^*(1-\kappa)[\alpha\theta n + m(\gamma+\mu+\omega)]w_4}{(m+n)(\mu+\omega)(\gamma+\mu+\omega)}, \\ w_2 &= -\frac{\beta^*(1-\kappa)\theta n w_4}{(m+n)(\gamma+\mu+\omega)}, \\ w_3 &= -\frac{\beta^*(1-\kappa)(m+\theta n)w_4}{(m+n)(\gamma+\mu+\omega)}, \\ w_4 &> 0. \end{aligned} \tag{15}$$

Further, the Jacobian $J(\mathcal{V}^0)$ has a left eigenvector (associated with the zero eigenvalue) given by $\mathbf{v} = (v_1, v_2, v_3, v_4)^T$, where

$$v_1 = v_2 = 0, \quad v_3 = \frac{\gamma v_4}{(\gamma+\mu+\omega)}, \quad v_4 > 0. \tag{16}$$

The associated non-zero partial derivatives of F at the DFE are given by

$$\begin{aligned}
&\frac{\partial^2 f_3}{\partial x_1 \partial x_4} = \frac{\partial^2 f_3}{\partial x_4 \partial x_1} = \frac{\beta^*(1-\kappa)n(1-\theta)}{(m+n)^2},\\
&\frac{\partial^2 f_3}{\partial x_2 \partial x_4} = \frac{\partial^2 f_3}{\partial x_4 \partial x_2} = -\frac{\beta^*(1-\kappa)m(1-\theta)}{(m+n)^2},\\
&\frac{\partial^2 f_3}{\partial x_3 \partial x_4} = \frac{\partial^2 f_4}{\partial x_4 \partial x_3} = \frac{\partial^2 f_3}{\partial^2 x_4} = -\frac{2\beta^*(1-\kappa)(m+\theta n)}{(m+n)^2},\\
&\frac{\partial^2 f_3}{\partial x_4 \partial \beta^*} = \frac{(1-\kappa)(m+\theta n)}{m+n}.
\end{aligned} \tag{17}$$

From (17), it follows that

$$a = \frac{\gamma(\xi_1+\xi_2+\xi_3)v_4}{\gamma+\mu+\omega},$$

where

$$\begin{aligned}
\xi_1 &= -\frac{2\beta^{*2}(1-\kappa)^2 n(1-\theta)(\alpha\theta n + m(\gamma+\mu+\omega))w_4^2}{(m+n)^3(\mu+\omega)(\gamma+\mu+\omega)},\\
\xi_2 &= \frac{2\beta^{*2}(1-\kappa)^2 m\theta n(1-\theta)w_4^2}{(m+n)^3(\gamma+\mu+\omega)},\\
\xi_3 &= -\frac{2\beta^*(1-\kappa)(m+\theta n)((m+n)(\gamma+\mu+\omega)+\beta^*(1-\kappa)(m+\theta n))w_4^2}{(m+n)^3(\gamma+\mu+\omega)}
\end{aligned} \tag{18}$$

and

$$b = \frac{(1-\kappa)\gamma(m+\theta n)w_4 v_4}{(m+n)(\gamma+\mu+\omega)} > 0.$$

Results from (18) show that $a < 0$; hence, the following result is established according to Theorem 1 item (iv) above.

Lemma 3. *The endemic equilibrium $\mathcal{V}^*$ is locally asymptotically stable for $\mathcal{R}_e > 1$, but close to 1.*

Fig. 2 illustrates the possible bifurcation diagrams for the system. However, since $a < 0$ in the range of biologically reasonable parameters, no backward bifurcation occurs. By direct computations following Arriola and Hyman [40], it can be shown that $\mathcal{R}_e$ is most sensitive to β

$$\mathcal{U}_\beta = \frac{\beta}{\mathcal{R}_e}\frac{\partial \mathcal{R}_e}{\partial \beta} = 1, \tag{19}$$

such that an increase in β will bring about an increase of the same proportion in $\mathcal{R}_e$ and a decrease in β will result in a decrease in $\mathcal{R}_e$ with about an equivalent magnitude. Thus, more effort should be done on reducing β, in order to control HCV in prison settings. Using numerical simulations we now investigate the role of κ on $\mathcal{R}_e$.

Numerical results (Fig. 3) clearly show that an educational campaign may reduce HCV prevalence in prison. For instance if $\beta = 0.55$, $\kappa = 0.7$ then $\mathcal{R}_e = 0.799$, while if $\beta = 0.55$, $\kappa = 0.4$ then $\mathcal{R}_e = 1.63$.

4. Population-level effects

In order to illustrate the results of the foregoing analysis, we simulated model system (1) using the parameters in Table 1. Unfortunately, the scarcity and limited data on HCV in prison settings limits our ability to calibrate (for instance, to quantify the proportion of prisoners recruited either as latent or infectious HCV individuals would require analysis from the real demographic data); nevertheless, we assume some of the parameters in the realistic range for illustrative purpose. These parsimonious assumptions reflect the lack of information currently available on HCV in prison settings. Reliable data on HCV transmission in prison settings would enhance our understanding and aid in the possible interventions to be implemented.

Simulations in Fig. 4 present the dynamics of cumulative new HCV latent cases and cumulative HCV infectious cases. These illustrate that an education campaign about safe injecting practices among women in a prison setting may lead to

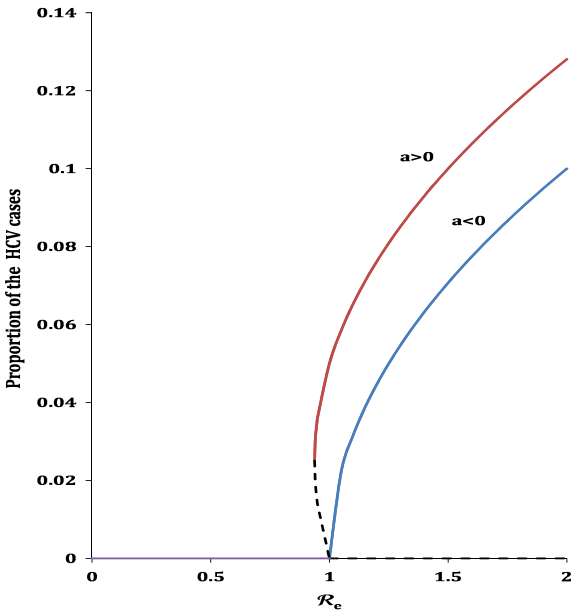

Fig. 2. Bifurcation diagram in the neighbourhood of $\mathcal{R}_e = 1$.

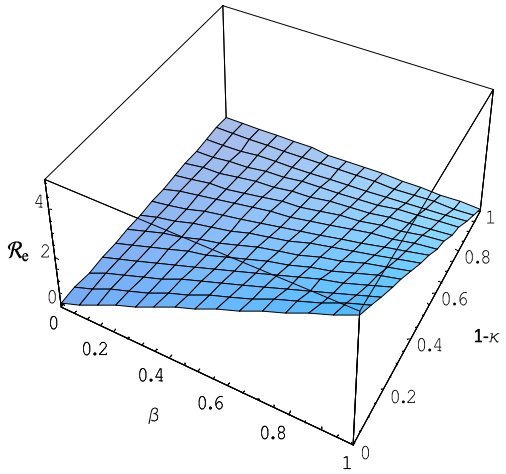

Fig. 3. The relationship between the reproductive number ($\mathcal{R}_e$), the probability of HCV infection (β) and education campaign κ.

Table 1
Model parameters and their interpretations.

Parameter	Symbol	Sample Value	Range	Source
Recruitment rate for female prisoners	Λ	123 per 100 000	N/A	[42]
Proportion of female prisoners recruited into X_D	π_0	0.3	0–0.3	Assumed
Proportion of female prisoners recruited into X_N	π_1	0.4	0–0.4	Assumed
Proportion of female prisoners recruited into L	π_2	0.15	N/A	Assumed
Proportion of female prisoners recruited into I	π_3	0.15	N/A	Assumed
Natural mortality rate	μ	0.0142 yr^{-1}	0.01–0.02	[43]
Transmissibility	β	0.9	0.11–0.95	[44]
Incubation rate	γ	7.3 yr^{-1}	2.433–24.333	[35]
Release rate	ω	0.2 yr^{-1}	0.33–0.125	[45]
IDU adoption rate	α	0.1 yr^{-1}	0–0.2	Assumed
Educational adjustment	κ	0.5	0.0–1.0	Assumed
Modification parameter	θ	0.8	N/A	Assumed

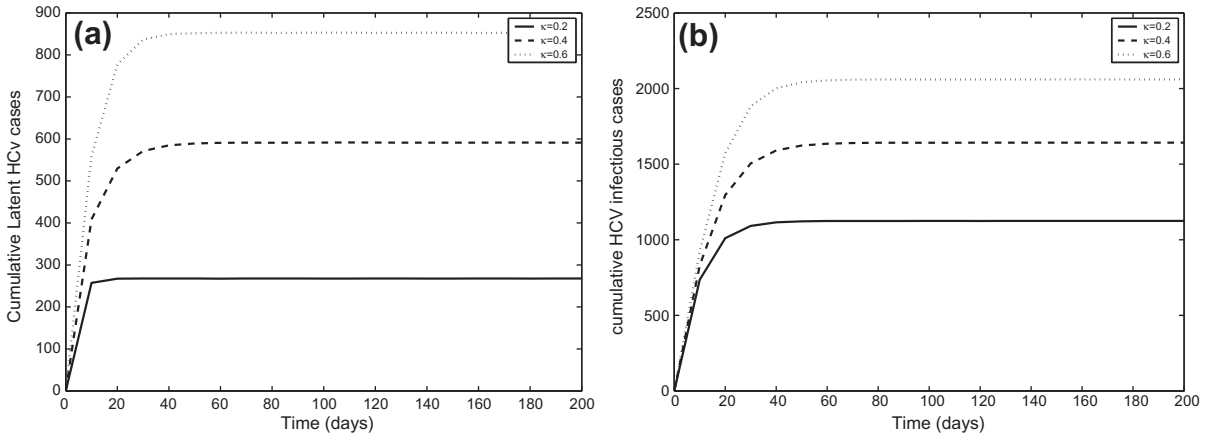

Fig. 4. The impact of educational campaigns (κ) on safe injecting practices among women in prison settings is demonstrated over a period of 200 days. The remaining parameters are fixed at their baseline values from Table 1, and the following initial conditions are used $X_D = 200$, $X_N = 200$, $L = 20$ and $I = 20$.

effective HCV control in the community. Numerical results in this study show that an increase in education campaigns about reducing or eliminating drug misuse will reduce the reproductive number. Thus, HCV may be controllable and the disease curtailed.

4.1. Sensitivity analysis

In many epidemiological models, the magnitude of the reproductive number is associated with the level of infection. The same is true in model (1). Sensitivity analysis assesses the amount and type of change inherent in the model as captured by the terms that define the reproductive number ($\mathcal{R}_e$). If $\mathcal{R}_e$ is very sensitive to a particular parameter, then a perturbation of the conditions that connect the dynamics to such a parameter may prove useful in identifying policies or intervention strategies that reduce epidemic prevalence.

Partial rank correlation coefficients (PRCCs) were calculated to estimate the degree of correlation between values of $\mathcal{R}_e$ and the nine model parameters across 1000 random draws from the empirical distribution of $\mathcal{R}_e$ and its associated parameters. A large PRCC is indicative of high sensitivity to parameter estimates (PRCCs > 0 will increase $\mathcal{R}_e$ when they are increased), while a small PRCC is reflects low sensitivity (PRCCs < 0 will decrease $\mathcal{R}_e$ when they are increased) [46–49]. Fig. 5 illustrates that $\mathcal{R}_e$ is most sensitive to β (the transmissibility) and κ (the educational adjustment parameter).

Since the educational adjustment parameter has a significant effect on $\mathcal{R}_e$, we examined the dependence of $\mathcal{R}_e$ in this parameter in more detail. We used Latin Hypercube Sampling and Monte Carlo simulations to run 1000 simulations, where all parameters were simultaneously drawn from across their ranges.

Fig. 6 illustrates the effect of educational adjustments on controlling HCV in prison settings. The results demonstrate that an increase in educational campaigns results in a decrease on the reproductive ratio. Thus, education campaigns will be an important intervention strategy for controlling HCV in prison settings, especially those in resource-limited settings where HCV treatment is limited or absent. If educational efforts can be effective 80% of the time or more, then the disease will be controlled.

Fig. 7 illustrates the effect of different strategies on controlling HCV. If the educational strategies vary across all possible values then the mean value of $\mathcal{R}_e$ will be below 1, but the upper quartile range is not. Thus, without specific targets, educational campaigns may not eliminate the disease. If educational strategies are 50–80% effective, then the mean and upper quartile range of $\mathcal{R}_e$ will be below one. However, there are still many outliers above one, suggesting that elimination may not always succeed. If educational strategies are more than 80% effective, then the disease is eliminated in almost all scenarios.

In resource-limited settings, education campaigns may not be comprehensive. Thus, we examined some substrategies for HCV educational control. Public health authorities may run a campaign warning against initial IDU use (thus reducing α, the IDU adoption rate). Conversely, they may target the consequences of unsafe needle practices (thus reducing the modification parameter θ). Fig. 8 demonstrates that targeting unsafe needle practices is significantly more effective than generalised messages discouraging injecting drug use. However, these strategies can be made vastly more effective when overall education is improved.

5. Discussion

Unsafe injecting practices, blood exchange, the use of non-sterile needles and other cutting instruments for tattooing are common in prison settings, resulting to a number of blood-transmitted infections. We formulated and analyzed a determin-

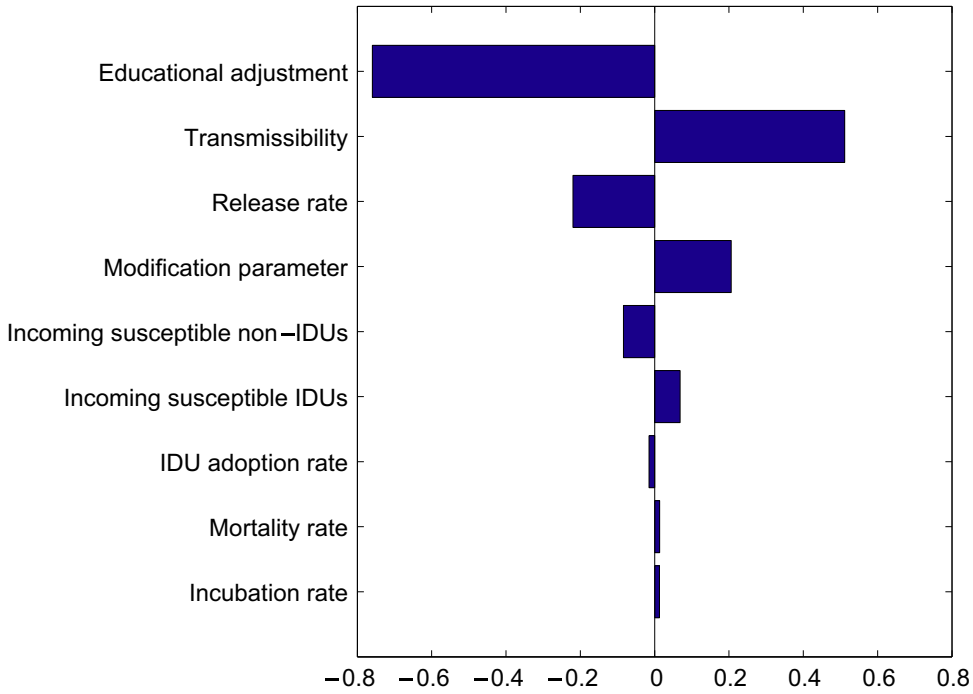

Fig. 5. Sensitivity of $\mathcal{R}_e$ to all parameters using partial rank correlation coefficients.

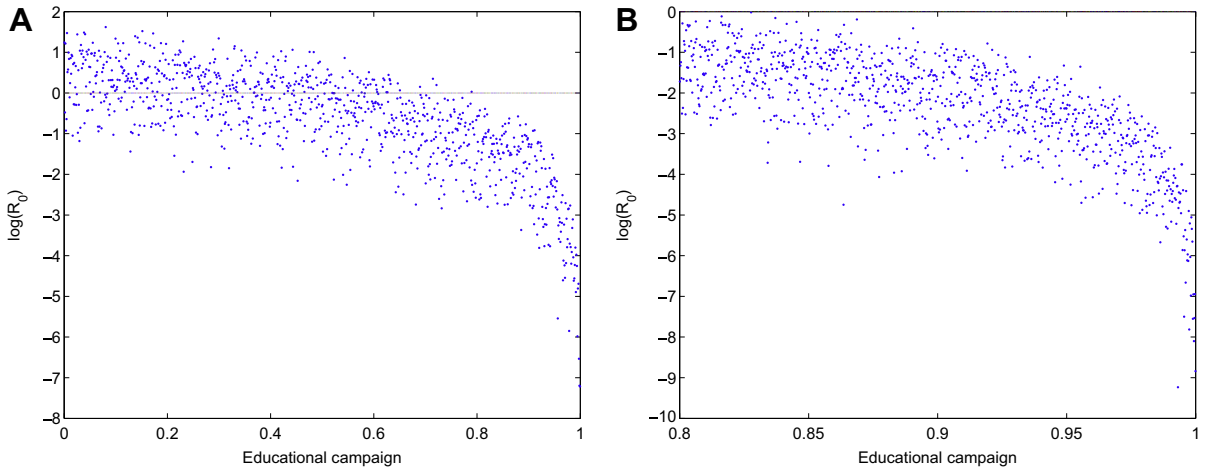

Fig. 6. Latin Hypercube Sampling for varying effectiveness of educational strategies. (A) Varying κ across all possible values. (B) If education is greater than 80% effective, then the disease can be controlled.

istic model to assess the impact of education campaigns about safe injecting practices among women in prison settings. Analytical results from the study have shown that the model has a globally stable disease-free equilibrium whenever the reproductive number is less than unity, and a locally asymptotically stable endemic equilibrium whenever the reproductive number is slightly larger than unity. Partial rank correlation coefficients (PRCCs) were calculated to estimate the correlation between values of $\mathcal{R}_e$ and the nine model parameters across 1000 random draws from the empirical distribution of $\mathcal{R}_e$ and its associated parameters. From Fig. 5, we note that the reproductive number is most sensitive to the transmissibility and the educational adjustment parameter. We have no control over the former, but the latter represents the effectiveness of our targeted strategies. Examining the effects of education more closely, we determined that education was highly likely to control the disease if it can be more than 80% effective (Fig. 7). In practice, however, this may be difficult or expensive to achieve. We note that education campaigns which are 50–80% effective will still control the disease more often than not. Even education campaigns which are less than 50% effective may nevertheless do some good. Furthermore, we noted that the

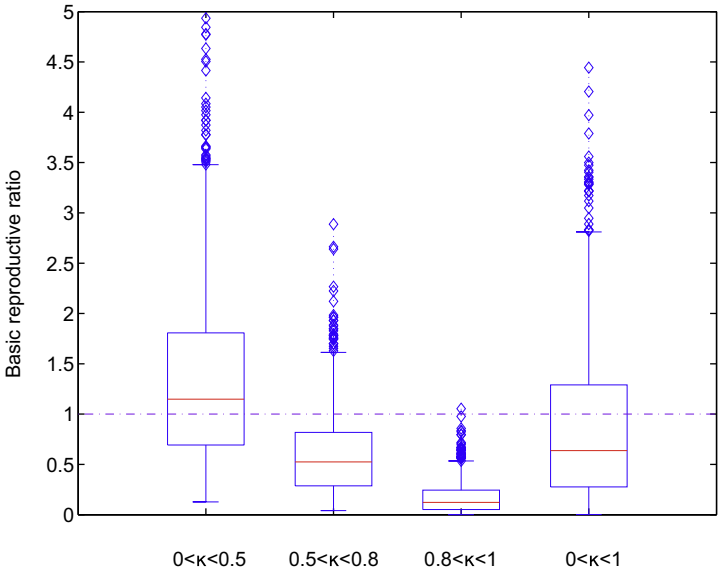

Fig. 7. Effect of different educational strategies of $\mathcal{R}_e$.

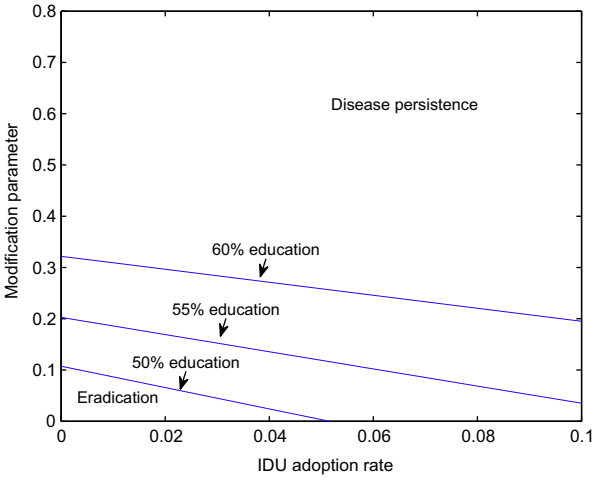

Fig. 8.

reproductive number decreases whenever the number of prisoners is dominated by non-IDUS, suggesting that screening for HCV might be important for controlling the disease in prison settings. This in-depth study of HCV among women in prison settings can be extended by including HCV treatment and screening.

Our model has several limitations, which should be acknowledged. The education parameter, κ, was applied uniformly, so that messages reached IDUs and non-IDUs equally. This may not occur, depending on the background of the prisoners. Furthermore, we assumed that this parameter was equally scaled whether reducing the transmission or affecting behaviour change. We also assumed a constant release rate, rather than staggered release rates.

In summary, we have illustrated the importance of education as an effective intervention tool. Although general education is never a bad thing, our sensitivity analysis shows that targeted education campaigns, which are effective at stopping transmission of HCV more than 80% of the time will be highly effective at controlling the disease among women in prisons.

Acknowledgments

The authors are grateful to the anonymous referee and the handling editor for their valuable comments and suggestions. RJS? is supported by an NSERC Discovery Grant, an Early Researcher Award and funding from MITACS. For citation purposes, note that the question mark in Smith? is part of his name.

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