A SIMPLE MATHEMATICAL MODEL FOR HIV AND HCV CO-INFECTION

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ABSTRACT

We study a simple mathematical model for the co-infection of HIV and HCV. We study local stability of the disease-free equilibria for the full model and for the two submodels (HCV only and HIV only submodels). We observe that the model predicts four distinct equilibria, the disease free, the HIV only, the HCV only, and the full endemic equilibria. We conclude that the model is well presented epidemiologically and mathematically.

KEY WORDS

HIV/AIDS, HCV, co-infection, mathematical model

1 Introduction

Patients infected with the human immuno-deficiency syndrome (HIV) are commonly co-infected with other diseases, such as tuberculosis [8] or hepatitis viruses [7]. Thirty four to forty six million people worldwide are infected with HIV and about 4 to 5 millions are co-infected with Hepatites C virus (HCV) [15]. In the United States of America, 1.2 million people are estimated to be infected with HIV, 30% of which are co-infected with HCV. Additionally, more than 4 million are infected solely with hepatitis C [5]. In Portugal, it is estimated that approximately 25% to 40% of the 41086 people infected with HCV are co-infected with HCV [6]. This statistic puts Portugal among European countries with a highest rate of co-infection. Vertical transmission, that is transmission of HCV from mother to child, is three to four times higher if the mother is co-infected with HIV [6].

Usually coinfection is associated with more severity for the two diseases involved. In what concerns co-infection with HCV, HIV accelerates the progression of HCV. The risk of severe liver disease is greater if the count of CD4 cells falls below 200 cells/mm3 [6]. Moreover, there is a higher risk of cirrhosis, end-stage liver disease, hepatocarcinoma, and hepatic-related death [10, 2]. In Portugal, the co-infection is, after tuberculosis, the second leading cause of death for people with HIV [6].

Recent studies show, however, some success in treatments for HCV using combination drugs, in people co-infected with HIV. Moreover, the majority of people with HCV may be successfully treated for HIV [5]. Nevertheless, more studies are needed in order to show the effectiveness of new antiviral drugs for HCV in HIV co-infected people.

In [11], authors propose a dynamical model for HCV/HIV coinfection, aimed to evaluate the cost-effectiveness of needle and syringe programs for injecting drug users. A similar model was used to explore the low prevalence of HCV in Rawalpindi, in order to understand the future HIV/HCV epidemic and to estimate the impact of a generic intervention [12]. Recently, in [13], authors used a mathematical model to understand trends in the prevalence of HIV and HCV, determining the epidemiological profiles. In [3] authors concluded that HCV prevalence can be used as an indicator of risk for successful HIV introduction into an injecting drug user population.

In this paper, we study a mathematical model for HIV and HCV coinfection. The model includes treatment for both diseases. The paper is organized as follows. The model is described in Section 2. In Section 3, we compute the reproduction numbers and the stability of the disease-free equilibrium. Simulation results of the full model are presented in Section 4. In Section 5, we state the main conclusions of this work.

2 Description of model

In section, we describe the mathematical model for HIV and HCV coinfection.

The entire population consists of nine classes: the susceptible individuals, $S$, the infected with HIV individuals, $I_a$, the individuals showing symptoms of AIDS, $A_a$, the individuals infected with HCV, $I_c$, the chronic HCV infected individuals, $C_c$, the co-infected with HIV and HCV individuals, $I_aI_c$, the individuals infected with HIV and with chronic HCV infection, $I_aC_c$, the individuals showing symptoms of AIDS and co-infected with HCV, $A_aI_c$, and the individuals showing symptoms of AIDS and chronic HCV infection $A_aC_c$. The schematic diagram of the model is given in Figure 1. The population of size $N(t)$, at time $t$, has constant inflow.
of susceptibles $S$, $\Lambda N$, where $\Lambda$ is the recruitment rate. The natural mortality rate is $\mu$ in all classes.

Susceptible individuals, $S$, when in contact with individuals infected with HIV, move to the class $I_a$, at a rate $\lambda_H$, given by $\lambda_H = c_1 \beta_1 (I_a + A_a + \sigma (I_a L + I_a C) + \sigma_1 (A_a L + A_a C))$. Parameter $\beta_1$ is the sexual contact rate, and $c_1$ is the average number of sexual partners per unit of time.

Susceptible individuals move, at a rate $\lambda_C$, to the class $I_c$, where $\lambda_C = \frac{S_C (I_C + C) + \eta_1 (I_a L + A_a L) + \eta_2 (I_a C + A_a C)}{N}$. Parameter $\beta_C$ is the product of the probability of HCV transmission per sexual contact and the effective contact rate for HCV infection to occur. Parameters $\eta_1, \eta_2 > 1$ model the fact that dually infected individuals are more infectious than their corresponding counterparts.

A fraction of new born children are infected with HIV during birth and hence are directly recruited into the infectious class, $I_a$, at a rate $(1 - \epsilon) \theta$. Other children die at birth $(0 \leq \epsilon \leq 1)$, where $\epsilon$ is the fraction of newborns infected with HIV who dies immediately after birth, and $\theta$ is the rate of newborns infected with HIV. Infected individuals, $I_a$, move at a rate $\rho$ to the AIDS class, $A_a$. Individuals with AIDS are treated at a rate $\upsilon_1$, and die because of AIDS at a rate $d_a$.

The HIV infected individuals, $I_a$ are infected with HCV at a rate $\sigma \lambda_C$ and move to the class $I_a I_c$. The modification parameter $\sigma > 1$ accounts for the increased risk of getting HCV for someone already infected with HIV. Dually HIV and chronic HCV infected individuals, $I_a C_c$, may progress to AIDS at a rate $\rho$ and move to class $A_a C_c$.

The HCV infected individuals, $I_c$ move to the susceptible class, $S$, after treatment at a rate $r_1$. They can also progress to a chronic stage, $C_c$, at a rate $\rho_1 C_c$, where $\rho_1$ is the proportion of infected individuals who are chronic carriers and $\frac{1}{\theta}$ is the average time that an individual infected with HCV remains in a state of acute infection. The individuals with chronic HCV infection, $C_c$, die because of HCV at a rate $d_c$.

The HCV infected individuals, $I_c$ are infected with HIV at a rate $\delta \lambda_H$ and move to the class $I_a I_c$. Parameter $\delta > 1$ accounts for the increased susceptibility to HIV infection for HCV infected people, since HCV accelerates the decline of the immune function.

Individuals in class $I_a I_c$ recover from HCV infection at a rate $r_2$ and move to the class $I_a$. The HIV and HCV co-infected individuals, $I_a I_c$, progress to AIDS at a rate $\rho$ and move to class $A_a I_c$. Moreover, $I_a I_c$ individuals may become HCV chronic carriers, at a rate $\rho_2 C_c$, and move to class $I_a C_c$, where $\rho_2$ is the proportion of dually infected individuals who are chronic carriers.

AIDS individuals, $A_a$, are infected with HCV at a rate $\sigma \lambda_C$ and move to class $A_a I_c$. The individuals in class $A_a I_c$ are treated and recover from HCV infection at a rate $r_3$ and move to class $A_a$ or are treated and recover from HIV infection at a rate $\upsilon_2$ and move to class $I_a I_c$. The individuals in class $A_a I_c$ become HCV chronic carriers at a rate $\rho_3 C_c$ and move to class $A_a C_c$. Parameter $\rho_3$ is the proportion of individuals in $A_a I_c$ class who are chronic carriers. Dually AIDS and chronic HCV infected individuals are treated for HIV at a rate $\upsilon_3$.

The parameters and variables of the model are summarized in Table 1.

The system of nonlinear ordinary differential equations for the proposed model is given by:

$$
\begin{align*}
\frac{dS}{dt} &= \Lambda N - \lambda_H S - \lambda_C S + \upsilon_1 I_c - \mu S \\
\frac{dI_a}{dt} &= \lambda_H S - (\rho + \sigma \lambda_C + \mu) I_a + (1 - \epsilon) \theta I_a + r_2 I_a I_c + \upsilon_1 A_a \\
\frac{dI_c}{dt} &= \rho I_a - (\upsilon_1 + d_a + \mu) A_a + r_3 A_a I_c - \sigma \lambda_C A_a \\
\frac{dA_a}{dt} &= \lambda_C S - (r_1 + \delta \lambda_H + \mu) I_a \\
\frac{dI_a I_c}{dt} &= \rho_1 \sigma_1 I_a I_c - (\delta \lambda_H + \mu + d_c) I_a I_c \\
\frac{dI_a C_c}{dt} &= \delta \lambda_H I_a C_c + \frac{\lambda_C I_a C_c}{N} - (r_2 + \rho + \rho_2 \sigma_1 + \mu) I_a I_c \\
\frac{dA_a I_c}{dt} &= \rho_2 \sigma_1 I_a I_c + \delta \lambda_C I_a C_c + \frac{\lambda_C A_a C_c}{N} - (r_3 + \mu + d_a) I_a I_c \\
\frac{dI_a C_c}{dt} &= \sigma \lambda_C A_a + \rho I_a I_c - (\upsilon_3 + \upsilon_2 + \rho_3 C_c + \mu + d_c) A_a I_c \\
\frac{dA_a C_c}{dt} &= \rho_3 C_c A_a I_c + \rho I_a C_c - (\upsilon_3 + \rho_1 C_c + \mu + d_a) A_a C_c \\
\end{align*}
$$

The dynamics of the total population $N(t) = S(t) + I_a(t) + A_a(t) + I_c(t) + C_c(t) + I_a I_c(t) + A_a I_c(t) + A_a C_c(t)$ is given by:

$$
\frac{dN}{dt} = \Lambda N + (1 - \epsilon) \theta I_a - \mu N - d_a (A_a + A_a I_c + A_a C_c) - d_c (C_c + I_a C_c + A_a C_c)
$$

The variables can be normalized as follow $s = \frac{S}{N}$, $i_a = \frac{I_a}{N}$, $a_a = \frac{A_a}{N}$, $i_c = \frac{I_c}{N}$, $c_c = \frac{C_c}{N}$, $i_a i_c = \frac{I_a I_c}{N}$, $i_a c_c = \frac{I_a C_c}{N}$, $a_a i_c = \frac{A_a I_c}{N}$, and $a_a c_c = \frac{A_a C_c}{N}$. The normalized system is given by:

Figure 1. Flow chart of the model.
Table 1. Description of variables and parameters of model (2).

<table>
<thead>
<tr>
<th>Variable/Parameter</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$S$</td>
<td>Susceptible individuals</td>
</tr>
<tr>
<td>$I_a$</td>
<td>HIV infected individuals</td>
</tr>
<tr>
<td>$A_a$</td>
<td>AIDS individuals</td>
</tr>
<tr>
<td>$I_c$</td>
<td>HCV infected individuals</td>
</tr>
<tr>
<td>$C_c$</td>
<td>Chronic HCV infected individuals</td>
</tr>
<tr>
<td>$I_aC_c$</td>
<td>HIV/HCV coinfected individuals</td>
</tr>
<tr>
<td>$I_aI_c$</td>
<td>HIV infected individuals with chronic HCV infection</td>
</tr>
<tr>
<td>$A_aI_c$</td>
<td>AIDS and HCV infected individuals</td>
</tr>
<tr>
<td>$A_aC_c$</td>
<td>AIDS and chronic HCV infected individuals</td>
</tr>
</tbody>
</table>

$\lambda_H = c_1 \beta_1 (i_a + a_a + \sigma_1 i_a c_a + \sigma_2 a_a c_a)$, $\lambda_C = c_2 \rho_1 (i_c + c_c + \sigma_1 i_c c_c + \sigma_2 c_c c_a)$, $s + i_a + a_a + i_c + c_c + i_a c_a + i_c c_c + a_a c_c + a_a c_c = 1$ and $s(t) \geq 0$, $i_a(t) \geq 0$, $a_a(t) \geq 0$, $i_c(t) \geq 0$, $c_c(t) \geq 0$, $i_a c_a(t) \geq 0$, $i_a c_c(t) \geq 0$, $a_a c_c(t) \geq 0$ and $a_a c_c(t) \geq 0$.

3 Reproduction numbers and stability of disease-free equilibria

In this subsection, we compute the reproduction number of model (2), $R_0$. The basic reproduction number is defined as the number of secondary infections due to a single infection in a completely susceptible population.

We begin by considering two sub-models of model (2). Model (3) arises from model (2) by setting the variables concerning HCV dynamics ($i_c$, $c_c$, $i_a c_a$, $i_a c_c$, $a_a c_c$ and $a_a c_c$) to zero, and model (5) follows from model (2) by setting the variables concerning HIV dynamics ($i_a$, $i_c$, $i_a c_a$, $a_a c_a$ and $a_a c_c$) to zero.

We begin by computing the reproduction number of system (3), $R_{HIV}$. We use the next generation method to compute $R_{HIV}$ [4].

$$\frac{ds}{dt} = \lambda_H s - \lambda s(t) - (1 - \epsilon)\theta i_a - d_a a_a$$

$$\frac{di}{dt} = \lambda_H s - \rho i_a + (1 - \epsilon)\theta i_a + v_1 a_a - i_a(\Lambda + (1 - \epsilon)\theta i_a - d_a a_a)$$

$$\frac{da}{dt} = i_a - (v_1 + d_a) a_a - a_a(\Lambda + (1 - \epsilon)\theta i_a - d_a a_a)$$

where $\lambda_H = c_1 \beta_1 (i_a + a_a)$.

The disease-free equilibrium of model (3) is given by:

$$F_0 = \left(s_0, i_a 0, a_a 0, a_a 0, a_a 0\right) = (1, 0, 0)$$

Using the notation in [4] on system (3), matrices for the new infection terms, $F$, and the other terms, $V$, are given by:

$$F = \begin{bmatrix} c_1 \beta_1 & c_1 \beta_1 \\ 0 & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} \rho - (1 - \epsilon)\theta + \Lambda & -v_1 \\ -\rho & v_1 + d_a + \Lambda \end{bmatrix}$$

The associative basic reproduction number is given by:

$$R_{HIV} = \rho(FV^{-1}) = \frac{\beta c_1(\rho + A + d_a + v_1)}{\Lambda(d_a + \rho + v_1) - (1 - \epsilon)\theta(d_a + \rho + \Lambda) + d_a \rho + \Lambda^2}$$

where $\rho$ indicates the spectral radius of $FV^{-1}$. By Theorem 2 [4], we obtain the following lemma.
Lemma 1 The disease-free equilibrium $P_0^2$ is locally asymptotically stable if $R_{HIV} < 1$ and unstable if $R_{HIV} > 1$.

We proceed with the computation of the reproduction number of model (5) below, $R_{HCV}$.

\[ \frac{dHCV}{dt} = \Lambda - \lambda Cs + r_1ic - s(\Lambda - dc_c) \]

\[ \frac{di}{dt} = \lambda Cs - (r_1 + \rho_1\sigma c)i_c - ic(\Lambda - dc_c) \]  \hspace{1cm} (5)

\[ \frac{dc}{dt} = \rho_1\sigma c i_c - dc_c - c_c(\Lambda - dc_c) \]

where $\lambda_C = \beta_c(i_c + c_c)$.

The disease-free equilibrium state $P_0^2$ of model (5) is given by:

\[ P_0^2 = (s_0, i_0, c_0, c_0) = (1, 0, 0) \]

Using the notation in [4] on system (5), matrices for the new infection terms, $F$, and thee other terms, $V$, are given by:

\[ F = \begin{bmatrix} \beta_c & \beta_c \\ 0 & 0 \end{bmatrix} \]

\[ V = \begin{bmatrix} r_1 + \rho_1\sigma c + \Lambda & 0 \\ -\rho_1\sigma c & d_c + \Lambda \end{bmatrix} \]

The associative basic reproduction number is given by:

\[ R_{HCV} = \rho(FV)^{-1} = \frac{\beta_c(\Lambda + dc_c + \rho_1\sigma c)}{(d_c + \Lambda)(r_1 + \rho_1\sigma c + \Lambda)} \]  \hspace{1cm} (6)

where $\rho$ indicates the spectral radius of $FV^{-1}$. By Theorem 2 [4], we obtain the following lemma.

Lemma 2 The disease-free equilibrium $P_0^2$ is locally asymptotically stable if $R_{HIV} < 1$ and unstable if $R_{HIV} > 1$.

We now proceed with the calculation of the reproduction number of the full model (2), $R_0$. The disease-free equilibrium state $P_0$ of model (2) is given by:

\[ P_0 = (s_0^0, i_0^0, a_0^0, i_0^0 c_0^0, a_0^0 i_0^0, i_0^0 c_0^0, a_0^0 i_0^0 c_0^0) = (1, 0, 0, 0, 0, 0, 0) \]  \hspace{1cm} (7)

Using the notation in [4] on system (2), matrices for the new infection terms, $F$, and thee other terms, $V$, are given by:

\[ F = \begin{bmatrix} c_1\beta_1 & c_1\beta_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & c_1\beta_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & c_1\beta_3 & c_1\beta_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & c_1\beta_3 & c_1\beta_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & c_1\beta_3 & c_1\beta_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & c_1\beta_3 & c_1\beta_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & c_1\beta_3 & c_1\beta_3 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & c_1\beta_3 & c_1\beta_3 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & c_1\beta_3 & c_1\beta_3 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & c_1\beta_3 \end{bmatrix} \]

\[ V = \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix} \]

The associative basic reproduction number is given by:

\[ R_0 = \rho(FV)^{-1} = \max\{R_{HIV}, R_{HCV}\} \]  \hspace{1cm} (8)

In this section, we present numerical simulations of model (2). The parameter values used in the simulations can be found in Table 2 and the following initial conditions: $s(0) = 0.3$, $i_a(0) = 0.1$, $a(0) = 0.05$, $i_c(0) = 0.1$, $c(0) = 0.05$, $i_a i_c(0) = a a i_c(0) = a a c(0) = 0.1$.

In Figure 2-3, we plot the dynamics of the variables of system (2). We observe that, for the given parameters values and initial conditions, the model approaches asymptotically the stable disease free equilibrium.

In Figures 4-5, we plot the dynamics of the variables of system (2). We observe that, for the given parameters values and initial conditions, the model approaches asymptotically the stable two disease endemic equilibrium.

5 Conclusion

We study a simple mathematical model for the co-infection of HIV and HCV. We study local stability of the disease-free equilibrium for the full model and for the two submodels (HCV only and HIV only submodels). We observe that the model predicts four distinct equilibria, the disease free, the HIV only, the HCV only, and the full endemic equilibria. Future work is needed to study the behavior of the model with respect to some important parameters, such as $\beta_i$, the product of the probability of HCV transmission per sexual contact and the effective contact rate for HCV infection to occur, $c_1$, the average number of sexual partners per unit of time, and compare our results with real data. We conclude that the model is well presented epidemiologically and mathematically.

Acknowledgements

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Table 2. Parameters used in the numerical simulations of model (2).

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<th>Value</th>
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References


Figure 2. Disease-free equilibrium of system (2) for parameter values given in Table 4 and initial conditions ($R_{HIV} = 0.3895$, $R_{HCV} = 0.7258$, $R_0 = 0.7258$).

Figure 3. Disease-free equilibrium of system (2) for parameter values given in Table 4 and initial conditions ($R_{HIV} = 0.3895$, $R_{HCV} = 0.7258$, $R_0 = 0.7258$).
Figure 4. Stable two disease endemic equilibrium of system (2) for given parameter values in Table 4, except for $\beta_1 = 0.35$, $\sigma_C = 0.1667$ and $\beta_c = 0.8$, and initial conditions ($R_{HIV} = 1.3633$, $R_{HCV} = 1.1510$, $R_0 = 1.3633$).

Figure 5. Stable two disease endemic equilibrium of system (2) for given parameter values in Table 4, except for $\beta_1 = 0.35$, $\sigma_C = 0.1667$ and $\beta_c = 0.8$, and initial conditions ($R_{HIV} = 1.3633$, $R_{HCV} = 1.1510$, $R_0 = 1.3633$).