The analysis of HIV/AIDS drug-resistant on networks

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Received 30 June 2013
Accepted 30 September 2013
Published 22 January 2014

In this paper, we present an Human Immunodeficiency Virus (HIV)/Acquired Immune Deficiency Syndrome (AIDS) drug-resistant model using an ordinary differential equation (ODE) model on scale-free networks. We derive the threshold for the epidemic to be zero in infinite scale-free network. We also prove the stability of disease-free equilibrium (DFE) and persistence of HIV/AIDS infection. The effects of two immunization schemes, including proportional scheme and targeted vaccination, are studied and compared. We find that targeted strategy compare favorably to a proportional condom using has prominent effect to control HIV/AIDS spread on scale-free networks.

Keywords: HIV/AIDS; drug-resistant; networks; stability; immunization.

PACS Nos.: 89.75.Hc, 05.10.—a, 87.23.Ge.

1. Introduction

The Human Immunodeficiency Virus (HIV) is the causative agent of Acquired Immune Deficiency Syndrome in humans (AIDS). The transmission of HIV/AIDS is a serious problem for the health of the human. It is more important to study this disease theoretically through dynamic methods.\textsuperscript{1}

Many diseases such as tuberculosis, malaria, HIV/AIDS and influenza etc., drug-resistant strains emerge by the mutation of a wild strain. Misuse of drugs is a major cause of appearance of such stronger strains, thus antiviral drug resistance is an important concern in public health. In the past few years, many studies have focused on this topic.\textsuperscript{2–8} Some of these studies investigated drug resistance in influenza,\textsuperscript{3,4} HIV\textsuperscript{5,7} and malaria.\textsuperscript{6} Many of these work have emphasized the importance of mathematical models to gain further insight into the development of drug resistance.

In recent years, many mathematical models on the spread of the diseases on networks have been studied.\textsuperscript{9–20} As for epidemic with drug-resistant on network,
Smith et al.\textsuperscript{7} investigated dynamics of complex networks of HIV drug-resistant strains. However, there is no detail about the qualitative study. So in this paper, we analyze a deterministic model for monitoring the transmission dynamics in the presence of resistance to drugs on scale-free networks. We analyze the dynamics of the SIA model that is based on subdividing the whole population of an area or a country into four compartments namely: the susceptible ($S(t)$), the HIV infected with the wild strain ($I_1(t)$), and the HIV infected with the drug-resistant strain ($I_2(t)$) and the AIDS ($A(t)$), so that the total population size is

$$N(t) = S(t) + I_1(t) + I_2(t) + A(t).$$

The paper is organized as follows: The model is formulated in Sec. 2. Existence and stability of the equilibria of the model are investigated in Sec. 3. In Sec. 4, two immunization strategies are discussed. In Sec. 5, numerical simulations are illustrated.

2. The HIV/AIDS Model with Drug-Resistant

On the network, each site of $N$ is empty or occupied by only one individual. We give each site a number: 0, 1, 2, 3 or 4. Alternatively, we can interpret the five states as 0: vacant, 1: a healthy individual occupation, 2: an HIV infected individual with wild strain occupation, 3: an HIV infected individual with drug-resistant strain occupation, 4: an AIDS individual occupation. The state of the system at time $t$ can be described by a set of numbers $0, 1, 2, 3, 4$. That means if the system are in state $A$ and the site $x \in N$, then $A_t(x) \in \{0; 1; 2; 3; 4\}$. Each site can change its state with a certain rate. An empty site can give new individual to a healthy individual at rate $b$. A healthy individual can be infected by contact at the rate $\beta_1$ if there are infected individual with wild strain in its neighbors, or be infected by contact at the rate $\beta_2$ if there are infected individual with drug-resistant strain, or die at rate $\mu$ due to natural death. An infected with wild strain individual will become an infected with drug-resistant individual at rate $\xi$ due to using the drugs. The infected with wild strain individual and with drug-resistant individual will become an AIDS individual at rate $\delta$ due to the disease. If an individual dies, there is an empty site. Of course, each site can also maintain its state. The parameters $b, \beta_1, \beta_2, \xi, \sigma_1, \sigma_2, \delta > \mu$ are all non-negative. The state-transition rules of the contact process are schematically shown in Fig. 1.

2.1. The susceptible

The susceptible population is increased by recruitment of individuals (at a rate $b$) and decreased by natural death (at a rate $\mu$). In the presence of the disease, susceptible population also reduced by infected, which may be acquired via contact with infected individuals are either with the wild strain or the drug-resistant strain. The
parameters $\beta_1, \beta_2$ are the transmission probabilities of the wild and resistant strains, respectively. This gives:

$$\frac{dS_k(t)}{dt} = b(1 - S_k(t) - I_{1k}(t) - I_{2k}(t) - A_k(t)) - \beta_1 kS_k(t)\Theta_1(t) - \beta_2 kS_k(t)\Theta_2(t) - \mu S_k(t),$$

where $\Theta_1(t), \Theta_2(t)$ are defined as

$$\Theta_1(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^{\infty} kP(k)I_{1k}(t), \quad \Theta_2(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^{\infty} kP(k)I_{2k}(t)$$

and $\langle k \rangle = \sum_k kP(k)$, $P(k)$ is the connectivity distribution of the individuals.

### 2.2. The HIV infected with the wild strain

The HIV infected with the wild strain is increased by natural death (at a rate $\mu$), and by the infection of susceptible individuals with the wild type and diminished by resistance development to antimalarial drugs (at a rate $\xi$) and progression to the AIDS stage (at a rate $\sigma_1$). This gives

$$\frac{dI_{1k}(t)}{dt} = \beta_1 kS_k(t)\Theta_1(t) - \xi I_{1k}(t) - \sigma_1 I_{1k}(t) - \mu I_{1k}(t).$$

### 2.3. The HIV infected with the drug-resistant strain

The HIV infected with the drug-resistant strain is increased by natural death (at a rate $\mu$), and by the infection of susceptible humans with the resistant strain and by the development of resistance amongst treated individuals (at the rate $\xi$). It is diminished by progression to the AIDS stage (at a rate $\sigma_2$). This gives

$$\frac{dI_{2k}(t)}{dt} = \beta_2 kS_k(t)\Theta_2(t) + \xi I_{1k}(t) - \sigma_2 I_{2k}(t) - \mu I_{2k}(t).$$
2.4. The AIDS stage

The population is generated by the progression to the AIDS stage (at rates \( \sigma_1 \) and \( \sigma_2 \)) and disease-induced death (at a rate \( \delta \)). This gives

\[
\frac{dA_k(t)}{dt} = \sigma_1 I_{1k}(t) + \sigma_2 I_{2k}(t) - \delta A_k(t). \tag{4}
\]

3. The Model

The model is governed by (1)–(4). For each \( k \), adding above four equations gives

\[
\frac{dN_k(t)}{dt} \leq b - (b + \mu) N_k(t).
\]

Hence, \( \lim_{t \to \infty} \sup(S_k + I_{1k} + I_{2k} + A_k) \leq \frac{b}{b + \mu} \). Therefore, omega limit sets of system (1)–(4) are contained in the following bounded region in the non-negative cone of \( R^M \):

\[
\Omega = \left\{ (S_1, I_{11}, I_{21}, A_1, \ldots, S_M, I_{1M}, I_{2M}, A_M, S_k + I_{1k} + I_{2k} + A_k \leq \frac{b}{b + \mu}, 1 \leq k \leq M \right\},
\]

where \( M \) is the maximum number of contact in each individual. It can be verified that region \( \Omega \) is positively invariant. Consequently, the dynamics of the model would be considered in \( \Omega \). Since \( S_k = \frac{b}{b + \mu} - I_{1k} - I_{2k} - A_k \) at steady-state, it is sufficient to study the limiting system

\[
\frac{dI_{1k}(t)}{dt} = \beta_{1k}\left( \frac{b}{b + \mu} - I_{1k} - I_{2k} - A_k \right) \Theta_1(t) - \xi I_{1k}(t) - \sigma_1 I_{1k}(t) - \mu I_{1k}(t), \tag{5}
\]

\[
\frac{dI_{2k}(t)}{dt} = \beta_{2k}\left( \frac{b}{b + \mu} - I_{1k} - I_{2k} - A_k \right) \Theta_2(t) + \xi I_{1k}(t) - \sigma_2 I_{2k}(t) - \mu I_{2k}(t), \tag{6}
\]

\[
\frac{dA_k(t)}{dt} = \sigma_1 I_{1k}(t) + \sigma_2 I_{2k}(t) - \delta A_k(t), \tag{7}
\]

in the subspace

\[
\Omega_1 = \left\{ (I_{11}, I_{21}, A_1, \ldots, I_{1M}, I_{2M}, A_M, I_{1k} + I_{2k} + A_k \leq \frac{b}{b + \mu}, 1 \leq k \leq M \right\}.
\]

The model has the disease-free equilibrium (DFE) \( E_0 \). Using the next generation operator approach of Refs. 21 and 22, the non-negative matrix, \( F \), of the infection terms and the nonsingular \( M \)-matrix, \( V \), of the transition terms, are given by

\[
F = \begin{pmatrix} F_{11} & O & O \\ O & F_{22} & O \\ O & O & O \end{pmatrix}, \quad V = \begin{pmatrix} V_{11} & O & O \\ V_{21} & V_{22} & O \\ V_{31} & V_{32} & V_{33} \end{pmatrix}.
\]
where

\[
F_{11} = \frac{b\beta_1}{(b + \mu \langle k \rangle)} \begin{pmatrix} 1 \\ 2 \\ \vdots \\ M \end{pmatrix} (1 \times P(1)2 \times P(2) \cdots M \times P(M)),
\]

and

\[
F_2 = \frac{b\beta_2}{(b + \mu \langle k \rangle)} \begin{pmatrix} 1 \\ 2 \\ \vdots \\ M \end{pmatrix} (1 \times P(1)2 \times P(2) \cdots M \times P(M)),
\]

where \( V_{11} = (\xi + \sigma_1 + \mu)I, \ V_{21} = (-\xi)I, \ V_{22} = (\sigma_2 + \mu)I, \ V_{31} = (-\sigma_1)I, \ V_{32} = (-\sigma_2)I, \ V_{33} = \delta I, \) and \( I \) is identity matrix, \( O \) is zero matrix. The effective reproduction number, denoted by \( R_e \), are given by \( R_e = \rho(FV^{-1}) \), where \( \rho \) is the spectral radius. After some calculations, we get

\[
R_e = \max\{R_w, R_r\},
\]

where

\[
R_w = \frac{b\beta_1 \langle k^2 \rangle}{(b + \mu)(\xi + \sigma_1 + \mu)\langle k \rangle}, \quad R_r = \frac{b\beta_2 \langle k^2 \rangle}{(b + \mu)(\sigma_2 + \mu)\langle k \rangle}.
\]

Hence, using Theorem 2 of Ref. 22, the following result is established.

**Lemma 1.** The DFE \( E_{0} \) of the model (5)–(7) is locally asymptotically stable if \( R_e < 1 \), and unstable if \( R_e > 1 \).

The above lemma shows that the HIV/AIDS can be effectively controled if the initial population of the model are in the basis of attraction of the DFE. To eliminate the HIV/AIDS regardless of initial population sizes of different degree, we should consider the global stability proof for the DFE.

**Theorem 1.** The DFE of the model (5)–(7) is globally-asymptotically stable (GAS) in \( \Omega \), whenever \( R_e < 1 \).

**Proof.** Equations (5)–(7) can be written in terms of

\[
\begin{pmatrix}
\frac{dI_{1k}(t)}{dt} \\
\frac{dI_{2k}(t)}{dt} \\
\frac{dA_k(t)}{dt}
\end{pmatrix} = (F - V - U) \begin{pmatrix} I_{1k}(t) \\ I_{2k}(t) \\ A_k(t) \end{pmatrix},
\]

(8)
where the matrices $F$ and $Q$ are as defined above, and $U$ is a non-negative matrix given by

$$U = \begin{pmatrix}
\beta_1 k \Theta_1 & \beta_1 k \Theta_1 & \beta_1 k \Theta_1 \\
\beta_2 k \Theta_2 & \beta_2 k \Theta_2 & \beta_2 k \Theta_2 \\
0 & 0 & 0
\end{pmatrix}.$$  

Thus

$$\begin{pmatrix}
\frac{dI_{1k}(t)}{dt} \\
\frac{dI_{2k}(t)}{dt} \\
\frac{dA_k(t)}{dt}
\end{pmatrix} \leq (F - V) \begin{pmatrix}
I_{1k}(t) \\
I_{2k}(t) \\
A_k(t)
\end{pmatrix}. \quad (10)$$

If $R_e < 1$, then $\rho(FV^{-1}) < 1$, which is equivalent to $F - V$ having all its eigenvalues in the left half plane.\(^{21}\) It follows that the linearized differential inequality system (10) is stable whenever $R_e < 1$. Consequently, when $t \to \infty$, $I_{1k} \to 0$, $I_{2k} \to 0$, $A_k \to 0$, for this linear ordinary differential equations (ODE) system. Thus, Using a standard comparison theorem,\(^{22}\) $I_{1k} \to 0$, $I_{2k} \to 0$, $A_k \to 0$, for the nonlinear system (8) for $R_e < 1$, so that the DFE is GAS in $\Omega_1$ when $R_e < 1$. \(\square\)

The following we consider the existence of the endemic equilibrium. By letting the right-hand side of Eqs. (5)–(7) to be zero, we have

$$\beta_1 k \left(\frac{b}{b + \mu} - I_{1k} - I_{2k} - A_k\right) \Theta_1(t) - \xi I_{1k}(t) - \sigma_1 I_{1k}(t) - \mu I_{1k}(t) = 0,$$

$$\beta_2 k \left(\frac{b}{b + \mu} - I_{1k} - I_{2k} - A_k\right) \Theta_2(t) + \xi I_{1k}(t) - \sigma_2 I_{2k}(t) - \mu I_{2k}(t) = 0,$$

$$\sigma_1 I_{1k}(t) + \sigma_2 I_{2k}(t) - \delta A_k(t) = 0.$$

As far as the possibility of spreading is concerned, the following result holds.

**Lemma 2.** If and only if

$$\frac{b \beta_1 \langle k^2 \rangle}{(b + \mu)(\xi + \sigma_1 + \mu)\langle k \rangle} > 1,$$

there exists a unique endemic equilibrium $E^*$.\(^{23}\)

Wild strain-only boundary equilibrium is an equilibrium where only the wild strain is present. In this case the use of treatment does not lead to resistance
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and the model has a resistant strain-only boundary equilibrium if

\[ \frac{dS_k(t)}{dt} = b(1 - S_k(t) - I_{1k}(t) - A_k(t)) - \beta_1 k S_k(t) \Theta_1(t) - \mu S_k(t), \]
\[ \frac{dI_{1k}(t)}{dt} = \beta_1 k S_k(t) \Theta_1(t) - \sigma_1 I_{1k}(t) - \mu I_{1k}(t), \]
\[ \frac{dA_k(t)}{dt} = \sigma_1 I_{1k}(t) - \delta A_k(t). \]

In the case the use of treatment does not lead to resistance development, so that the model reduces to

\[ \frac{dS_k(t)}{dt} = b(1 - S_k(t) - I_{2k}(t) - A_k(t)) - \beta_2 k S_k(t) \Theta_2(t) - \mu S_k(t), \]
\[ \frac{dI_{2k}(t)}{dt} = \beta_2 k S_k(t) \Theta_2(t) - \sigma_2 I_{2k}(t) - \mu I_{2k}(t), \]
\[ \frac{dA_k(t)}{dt} = \sigma_2 I_{2k}(t) - \delta A_k(t). \]

After some analysis, we obtain the following result that is established.

**Theorem 2.** The model (1)–(4) has a wild strain-only boundary equilibrium if

\[ \frac{b \beta_1 \langle k^2 \rangle}{(b + \mu)(\sigma_1 + \mu)\langle k \rangle} > 1 \]

and the model has a resistant strain-only boundary equilibrium if

\[ \frac{b \beta_2 \langle k^2 \rangle}{(b + \mu)(\sigma_2 + \mu)\langle k \rangle} > 1. \]

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4.1. Proportional immunization

In this case, for fixed spreading rates \( \beta_1 \) and \( \beta_2 \), let \( \omega(0 < \omega < 1) \) is the density of immune nodes in the network. At the mean-field level, the presence of proportional immunity will effectively reduce the spreading rate \( \beta \) by a factor \( \beta(1 - \omega) \). Thus, we can approximatively use \( \beta_1(1 - \omega) \) to substitute \( \beta_1 \) and use \( \beta_2(1 - \omega) \) to substitute \( \beta_2 \), thus the system becomes:

\[ \frac{dS_k(t)}{dt} = b(1 - S_k(t) - I_{1k}(t) - I_{2k}(t) - A_k(t)) - \beta_1(1 - \omega) k S_k(t) \Theta_1(t) - \beta_2(1 - \omega) k S_k(t) \Theta_2(t) - \mu S_k(t), \]
\[ \frac{dI_{1k}(t)}{dt} = \beta_1(1 - \omega) k S_k(t) \Theta_1(t) - \xi I_{1k}(t) - \sigma_1 I_{1k}(t) - \mu I_{1k}(t), \]
\[ \frac{dI_{2k}(t)}{dt} = \beta_2(1 - \omega) k S_k(t) \Theta_2(t) + \xi I_{1k}(t) - \sigma_2 I_{2k}(t) - \mu I_{2k}(t), \]
\[ \frac{dA_k(t)}{dt} = \sigma_1 I_{1k}(t) + \sigma_2 I_{2k}(t) - \delta A_k(t). \]
Using the same method in Sec. 3, we obtain a self-consistency equation as follows:

\[
\Theta_1 = \frac{\sum_{k} k^2 P(k)(1 - \omega) b \delta(\sigma_2 + \mu) \beta_1 \Theta_1}{\langle k \rangle \Delta_1} \equiv \tilde{f}(\Theta_1),
\]

where \( \Delta_1 = [b \delta(\sigma_2 + \mu) k(1 - \omega) + bk\delta(\sigma_2 + \mu)] \beta_1 \Theta_1 + [b k(1 - \omega)(\sigma_2 \xi + \sigma_1 + \mu) + k(1 - \omega)\beta_2 \Theta_2 + \delta(\sigma_2 + \mu)(\xi + \sigma_1 + \mu) + \delta k(1 - \omega)(\sigma_2 + \mu)(\xi + \sigma_1 + \mu)] \beta_2 \Theta_2 + \delta(\sigma_2 + \mu)(\xi + \sigma_1 + \mu)(b + \mu). \) By arguments similar to those in Sec. 3, the epidemic threshold \( \tilde{\lambda}_c \) is determined by the following inequality:

\[
\left. \frac{d\tilde{f}(\Theta_1)}{d\Theta_1} \right|_{\Theta_1 = 0} > 1.
\]

Therefore, it can be shown that

\[
\tilde{\lambda}_c = \frac{\langle k \rangle (b + \mu)(\xi + \mu + \sigma_1)}{b(1 - \omega)\langle k^2 \rangle},
\]

i.e.

\[
\tilde{\lambda}_c = \frac{1}{1 - \omega} \lambda_c.
\]

When \( \omega = 0 \), i.e. if no immunization were done, then \( \tilde{\lambda}_c = \lambda_c \); when \( 0 < \omega < 1 \), \( \tilde{\lambda}_c > \lambda_c \), i.e. the immunization scheme is effective; while as \( \omega \to 1 \), \( \tilde{\lambda}_c \to +\infty \), that is, in the case of a full immunization, it would be impossible for the epidemic to spread in the network.

### 4.2. Targeted immunization

While proportional immunization schemes are effective, there may be more efficient schemes due to the heterogeneous nature of scale-free networks: they are robust to random attacks, but fragile to selective attacks. Accordingly, we can devise a targeted immunization scheme.\(^9\) We introduce an upper threshold \( \kappa \), such that all nodes with connectivity \( k > \kappa \) are immunized, i.e. we define the immunization rate \( \omega_k \) by

\[
\omega_k = \begin{cases} 
1, & k > \kappa, \\
0, & k < \kappa,
\end{cases}
\]

or
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where $0 < c < 1$ and $\sum_k \omega_k P(k) = \bar{\omega}$, where $\bar{\omega}$ is the average immunization rate. The epidemic dynamics model is

$$
\begin{align*}
\frac{dS_k(t)}{dt} &= b(1 - S_k(t) - I_{1k}(t) - I_{2k}(t) - A_k(t)) - \beta_1 k(1 - \omega_k)S_k(t)\Theta_1(t) - \beta_2 k(1 - \omega_k)S_k(t)\Theta_2(t) - \mu S_k(t), \\
\frac{dI_{1k}(t)}{dt} &= \beta_1 k(1 - \omega_k)S_k(t)\Theta_1(t) - \xi I_{1k}(t) - \sigma_1 I_{1k}(t) - \mu I_{1k}(t), \\
\frac{dI_{2k}(t)}{dt} &= \beta_2 k(1 - \omega_k)S_k(t)\Theta_2(t) + \xi I_{1k}(t) - \sigma_2 I_{2k}(t) - \mu I_{2k}(t), \\
\frac{dA_k(t)}{dt} &= \sigma_1 I_{1k}(t) + \sigma_2 I_{2k}(t) - \delta A_k(t).
\end{align*}
$$

This leads to

$$
\Theta_1 = \sum_k k^2 P(k)(1 - \omega_k)b\delta(\sigma_2 + \mu)\beta_1\Theta_1 \equiv \hat{f}(\Theta_1),
$$

where $\Delta = [b\delta(\sigma_2 + \mu)k(1 - \omega_k) + bk\delta \xi(1 - \omega_k) + bk(1 - \omega_k)(\sigma_2 \xi + \sigma_1 \omega_2 + \sigma_1 \mu) + \delta(\sigma_2 + \mu)(\xi + \sigma_1 + \mu)k(1 - \omega_k)]\beta_1 \Theta_1 + [b\delta k(1 - \omega_k)(\xi + \sigma_1 + \mu) + bk\sigma_2(1 - \omega_k)]\Theta_2 + \delta(\sigma_2 + \mu)(\xi + \sigma_1 + \mu)(b + \mu)\delta \Theta_2$. Therefore, the epidemic threshold

$$
\hat{\lambda}_c = \frac{\langle k \rangle(\xi + \sigma_1 + \mu)(b + \mu)}{b(\langle k^2 \rangle - \langle k^2 \omega_k \rangle)}.
$$

Note that $\langle k^2 \omega_k \rangle = \bar{\omega}\langle k^2 \rangle + \sigma'$, where $\sigma' = \langle(\omega_k - \bar{\omega}) \times [k^2 - \langle k^2 \rangle] \rangle$ is the covariance of $\omega_k$ and $k^2$. There may be $\kappa$ (usually big enough) where $\sigma' < 0$, but for appropriately small $\kappa$, $\omega_k - \bar{\omega}$ and $k^2 - \langle k^2 \rangle$ have the same signs except for some $k$'s where $\omega_k - \bar{\omega}$ and/or $k^2 - \langle k^2 \rangle$ is zero; therefore $\sigma' > 0$ for appropriate $\kappa$. Then

$$
\frac{1}{\hat{\lambda}_c} > \frac{1 - \omega}{1 - \bar{\omega}} \hat{\lambda}_c.
$$

If we set $\bar{\omega} = \omega$, then

$$
\hat{\lambda}_c > \hat{\lambda}_c(0 < \omega < 1),
$$

which means the targeted immunization scheme is more efficient than the proportional immunization scheme for the same average immunization rate.

5. Simulations

For an HIV/AIDS drug-resistant disease spreading with birth and death on scale-free networks, we have known that the stability of the DFE and the permanent of the disease. Here, we present numerical simulations to support the results obtained in previous sections. Our simulations are based on the Barabási–Albert (BA) network with $P(k) = (\gamma - 1)m^{\gamma - 1}k^{-\gamma}$. Parameters that are used in the simulations are listed
as follows: \( m = 2, \gamma = 3, b = 0.4, \beta_1 = 0.05, \beta_2 = 0.025, \xi = 0.01, \sigma_1 = 0.06, \sigma_2 = 0.04, \mu = 0.02, \delta = 0.05. \) Under the parameters given above, the basic reproduction number \( R_e = 1.82 > 1, \) which implies that the disease will persist without immunization.

Figure 2 shows the dynamics of nodes change as a function of time for nodes with different \( \xi, \) where \( \xi = 0.01, \xi = 0.03 \) and \( \xi = 0.05, \) respectively. And we can see that the smaller \( \xi \) is, the smaller the threshold \( R_e \) is. We can get a comparison in Fig. 3 for the total number of \( I_1 \) and \( I_2. \) Figure 3 shows that, targeted immunization has the absolute advantage to control the disease spread are compared with the proportional immunization. Every point of all curves is obtained under enough long time running of our programs which can guarantee that the system can infinitely near the equilibrium.

Fig. 2. (Color online) The time series of \( I_1 \) and \( I_2 \) with different \( \xi, \) where \( \xi = 0.01, \xi = 0.03 \) and \( \xi = 0.05. \)

Fig. 3. (Color online) The time series of \( I_1 \) and \( I_2 \) with different immunizations.
6. Conclusion

In this paper, we have discussed an HIV/AIDS dynamics on scale-free networks with drug-resistant. We derive the epidemic threshold $R_e$ dependent on the reproduction numbers, named the reproduction number with wild strain only and the reproduction number with resistant strain only. We analyzed the stability of the DFE and two boundary equilibria. It is shown that the development of drug-resistance can be controlled effectively by reducing the thresholds $R_w$ and $R_r$, and reducing the rate of acquisition of resistance. At the same time, we have also discussed proportional, targeted immunization schemes for network models. By comparing the thresholds for different immunization schemes, we have concluded that the targeted immunization scheme is more efficient than the proportional scheme.

Acknowledgments

The authors would like to acknowledge the support provided by the National Sciences Foundation of China (10901145) and the Top Young Academic Leaders of Higher Learning Institutions of Shanxi and the National Sciences Foundation of Shanxi (2012011002-1).

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