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INTERPLAY BETWEEN HIV/AIDS EPIDEMICS AND DEMOGRAPHIC STRUCTURES BASED ON SEXUAL CONTACT NETWORKS

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In this article, we propose a network spreading model for HIV epidemics, wherein each individual is represented by a node of the transmission network and the edges are the connections between individuals along which the infection may spread. The sexual activity of each individual, measured by its degree, is not homogeneous but obeys a power-law distribution. Due to the heterogeneity of activity, the infection can persistently exist at a very low prevalence, which has been observed in the real data but cannot be illuminated by previous models with homogeneous mixing hypothesis. The model displays a clear picture of hierarchical spread: In the early stage the infection is adhered to these high-risk persons, and then, diffuses toward low-risk population. Furthermore, we find that to reduce the risky behaviors is much more effective in the fight against HIV/AIDS rather than the antiretroviral drug therapies. The prediction results show that the development of epidemics can be roughly categorized into three patterns for different countries, and the pattern of a given country is mainly determined by the average sex-activity and transmission probability per sexual partner. In most cases, the effect of HIV epidemics on demographic structure is very small. However, for some extremely countries, like Botswana, the number of sex-active people can be depressed to nearly a half by AIDS.

Keywords: HIV/AIDS epidemics; scale-free networks; mathematical modeling; demography.

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1. Introduction

AIDS (Acquired Immure Deficiency Syndrome), as one of the most dangerous diseases over human history, has been continuously spreading at an enormous speed

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with an extremely high rate of death (from the moment when the first infection case was confirmed). Now it has already spread to all the regions of the world and been a great threat not only to the human health but also to the human society due to its own epidemiologic characters which make the objection to AIDS an extremely complex and difficult task to address.

A high infection rate of population will cause catastrophe to the development of national economy for two reasons. On one hand, most infected people are in a group aged from 24–45 who do the main contributions to the country's productivity, thus the AIDS will cause a great decline of the social wealth. On the other hand, to carry out a widely covered treatment program on treating the HIV infected people will be a heavy burden for the government finance due to the high cost of expensive medicine as well as regularly and continuously implemented therapy. Therefore, to investigate the epidemic behaviors of HIV/AIDS appears not only of great theoretical interest in understanding the underlying spreading mechanism, but also necessary and urgent in practice.

The extensively investigated models for epidemics, such as the standard susceptible-infected-removed (SIR) and susceptible-infected-susceptible (SIS) models, often involve two hypotheses.[1−4] First, the population is assumed closed, that is, the population size is fixed. However, recent researches on the spread of HIV (especially in Africa and other worst-afflicted areas) indicate the existence of strongly interplay between HIV epidemics and age structures, thus the demographic impact cannot be neglected (see the review papers[5,6] and the references therein). Second, the epidemiological models are often established based on perfect and homogeneous mixing, that is to say, all individuals are able to infect all others and the infectivity of each individual is almost the same. To replace the perfect mixing assumption, one can introduce the *epidemic contact network*, wherein the nodes denote individuals and edges represent the connections between individuals along which the infection may spread (see the review papers[7,8] about network epidemics and the references therein). The infected individual can infect a susceptible one only if they are neighboring in the network. The homogeneous mixing assumption can be implemented by using epidemic contact networks with homogeneous degree distributions,[9] such as complete networks, random networks,[10] and so on. However, recent empirical data exhibit us that the real-world sexual contact patterns are far different from the homogeneous ones.[11−13] The corresponding networks, similar to many other real-life networks, display the so-called scale-free property,[14,15] that is, they are of power-law degree distributions. This power-law distribution falls off much more gradually than an exponential one, allowing a few nodes of very large degree to exist. These high-degree nodes are called *hub nodes* in network science[15−18] and *superspreaders* in epidemiological literatures.[19,20] Recent theoretical researches on epidemics show that the topology of epidemic contact networks will highly affect the dynamical behaviors,[21,22] and it is also demonstrated that the effect of the superspreaders on HIV epidemics cannot be ignored.[23,24] Therefore, the structural effect should be taken into account when modeling HIV/AIDS epidemics. In addition,

the introduction of Antiretroviral (ARV) drug therapy is also one of the important result-dependent factors.[25,26] No treatment is more efficient for HIV-infected individuals than the medicine, which combines two or three antiretroviral drugs in "cocktail" regimens. These regimens, known as highly active ARV therapy, have resulted in the reduction of HIV levels in the blood, often to undetectable levels, and have markedly improved the immune function of HIV-infected individuals. The advent and widespread application of ARV has dramatically changed the typical course of HIV infection and AIDS, especially in high-income countries.[27] On the other hand, however, in the low-income countries, the overwhelming proportion of HIV-infected persons have no access to ARV. In sub-Saharan Africa, for example, this lack of treatment access has transformed into rapidly escalating death rates. Although the usage of ARV appears effective in bating HIV, it will bring too heavy pressure in economy for developing countries. Therefore, the better understanding of the effect of ARV treatment may enlighten readers in allocating the financial resources.

Although the demographic structure[28,29] and sexual contact pattern,[30,31] respectively, has been taken into account in the previous HIV/AIDS epidemic models, there are few works simultaneously consider these two ingredients. In the present model, both the demographic impact and heterogeneity mixing effect are considered. And many important features of real-life HIV epidemics can be naturally generated by combining these two ingredients. This article is organized as follows: In Sec. 2, the model is presented in detail. In Sec. 3, the main properties of this model are shown. Then, in Sec. 4, we try to predict the HIV/AIDS epidemics by this model. Finally, we sum up this article and discuss the relevance of this model to the real world in Sec. 5.

2. Model

2.1. *Construction of epidemic contact networks*

The HIV is transmitted by body fluid through several main routes including sexual contacts, sharing injectors among drug users, perinatal transmissions, transfusion of contaminated blood products etc., which are closely related to human beings' social activities. In different regions the popularity of each route is variable according to the culture and social circumstance. In some areas the homosexual contacts and injecting drug usage play the main role in HIV epidemics, while the main track in HIV transmission is the heterosexual contacts in the global scope.[a] Therefore, in this model, only the heterosexual relationships are taken into account, thus the corresponding epidemic contact networks are bipartite graphs.[32,33]

The epidemic contact network starts with $N_0/2$ males and $N_0/2$ females, each of which is sex-active with age between 15 and 49, and only the heterosexual contacts

[a]See the reports on the global AIDS epidemic of *Joint United Nations Programme on HIV/AIDS* (UNAIDS) from the web site Http://www.unaids.org.

are permissive. Since men tend to over-report their number of partners whereas women tend to under-report, the total number of sexual partners of males and females are not equal in existing surveys.[11,29] However, for simplification, we assume the degree distribution for both male and female nodes are the same. Assign each male node's degree according to a given degree distribution $p(k) \sim k^{-\gamma}$ with minimal degree $k_{\min} = 1$. According to the empirical data in Sweden,[11] we set $\gamma = 3.5$. After obtaining the *degree sequence* of male nodes, we let the female nodes have the same degree sequence and randomly assign each female node's degree according to this sequence. Here the degree sequence means a set of all nodes' degrees, one can find a more detailed and strict definition in Ref. 34. The edges are generated randomly by using the mechanism of configuration model.[35] Note that, different from most previous studies about epidemics on static networks, the present network structure evolves with time according to some followed rules of HIV epidemic dynamics.

2.2. *Modeling spread of HIV/AIDS in networks*

We focus on the network SIR model in which individuals can be in three discrete states, susceptible, infected or removed (dead). The infected ones can be divided into two subclasses: The HIV-positive individuals and persons with AIDS. Since the median time from AIDS to death is very short (about 7 month for adults[36]) compared with the median incubation time for AIDS,[37] we assume that when an HIV-positive person becomes an AIDS-patient, she or he will immediately be in death (i.e., within one year). This model is implemented by computer simulation with a time step equal to one year when mimicking the reality. The simulation processes are as follows:

(1) Set all the nodes to be susceptible except one randomly selected infected one.

(2) At each time step for each susceptible node x, denote m_1 and m_2 the number of its neighboring infected nodes not in process of ARV treatment (non-ARV user) or contrary (ARV user), respectively. If the node x is male, then the probability that x will become infected in the next time step is

$$\pi_x = 1 - (1 - \beta_1)^{m_1}(1 - \beta_2)^{m_2} . \tag{1}$$

Here β is the transmission probability per sexual partner, which is considered as a more appropriate estimate than the transmission probability per sexual act,[38] and the subscript represents whether the corresponding HIV-positive person has taken the ARV treatment. Since the male-to-female transmission is about twice efficient as female-to-male transmission,[39] if x is female, the corresponding probability is

$$\pi_x = 1 - (1 - 2\beta_1)^{m_1}(1 - 2\beta_2)^{m_2} , \tag{2}$$

where β_1 and β_2 are restricted below 0.5. It has been estimated by an analysis of longitudinal cohort data that the antiretroviral therapy can reduce per-partnership infectivity by as much as 60%,[27] thus we set $\beta_2 = 0.4\beta_1$.

(3) At each time step, each infected node (except the newly infected ones) may die with probability either ζ_1 (for non-ARV user) or ζ_2 (for ARV user). According to the recent estimations,[40] we set $\zeta_1 = 0.15$ and $\zeta_2 = 0.08$. The dead individuals are removed from the population.

Repeat these processes for desired time. Note that, each newly infected node will be ARV user at probability ρ_{ARV}, and all the existing ARV users will keep using ARV.

2.3. *Demographic impact*

In this model, all the nodes (sex-active persons) are divided into 7 age-groups (labeled A1–A7): 15–19, 20–24, 25–29, 30–34, 35–39, 40–44, and 45–49. At the beginning, each node chooses to be in one age-group with probability according to the age structure in the year corresponding to time step zero. At each time step, each female individual may bear a child according to the corresponding age-specific fertility rates. If she is infected and has not taken ARV treatment, the perinatal transmission probability is ε_1. And it reduces to ε_2 if ARV treatment is taken. Based on some previous empirical studies,[28,41] we set $\varepsilon_1 = 0.4$ and $\varepsilon_2 = 0.2$. The infected elder persons (>49 year) may die with probability ζ_1 or ζ_2 during each time step, and the corresponding probability for perinatally infected children is 0.2.[41]

At the end of each time step, 1/5 randomly selected living persons in age-group A1–A6 will reach the elder group, and 1/5 randomly selected living persons in group A7 will be removed from this system. If the time step t is less than 15, we simply assume equal number (to the number of removal nodes in A1) of susceptible individuals will be added to group A1; else if $t \geq 15$, $b(t-15)$ individuals will be added to group A1, where $b(t)$ denotes the number of newborn babies without HIV at time t. Here we simply assume all the infected babies will die before 15 years old since the mortality per year for them is much higher than adults. All these newly added ones will join the epidemic contact network according to the rules of Sec. 2.1, that is, the female/male nodes will randomly choose sexual partners among all the young and old men/women according to their given degrees obeying the distribution $p(k)$.

See Appendix A for the source of all the population and demographic data.

3. Main Properties

There are three free parameters in the present model: the average degree $\langle k \rangle$ which determines the degree distribution when the power-law exponent $\gamma = 3.5$ is given, the transmission probability β_1, and the ARV-receiving rate ρ_{ARV}. The former two parameters are relative to the behaviors while the last one is partially dependent on financial conditions. In this section, we will show some simulation results and investigate the main properties about this model by adjusting the above parameters. Some previous works show that for most cases, the qualitative features of epidemic dynamics will not be affected by the slightly varying of population size

and age-structure,[5,28,29,42] thus in this section, the age-specific fertility rates are kept unchanged. We use the age-density and age-specific fertility rates of China in 2005 for initialization, with the age-specific fertility rates unchanged all through. The network size is $N = 10^6$. To guarantee the universality of the simulation results, we also have implemented the present simulations with the age-density and age-specific fertility rates obtained from some other countries (e.g., US), and the results are qualitative the same.

3.1. *Effect of the heterogeneous degree distribution*

Many infections including HIV/AIDS can persistently exist in population despite of a very low prevalence. This epidemiological phenomenon cannot be illuminated by previous models with homogeneous mixing hypothesis.[7,8] By using the epidemic contact network with power-law degree distribution, the present model can reproduce the above observed phenomenon, which is in accordance with some previous theoretical studies about SIS/SIR models on scale-free networks.[21,22,43,44] Note that, since there are newly added susceptible individuals at each time step, the dynamic behaviors of present model may be closer to SIS model than SIR model. Figure 1 reports a typical simulation result wherein the prevalence of HIV is only about 2×10^{-3}. However, the infections can persistently exist for thousands years. For comparison, we exhibit the situation under homogeneous mixing hypothesis in Fig. 2, where the three parameters $(\langle k \rangle, \beta_1, \rho_{\mathrm{ARV}})$ are the same but all the nodes have fixed degree 3. The prevalence increases in the early stage since only few HIV-positive persons die, and then dies out obeying a linear form.

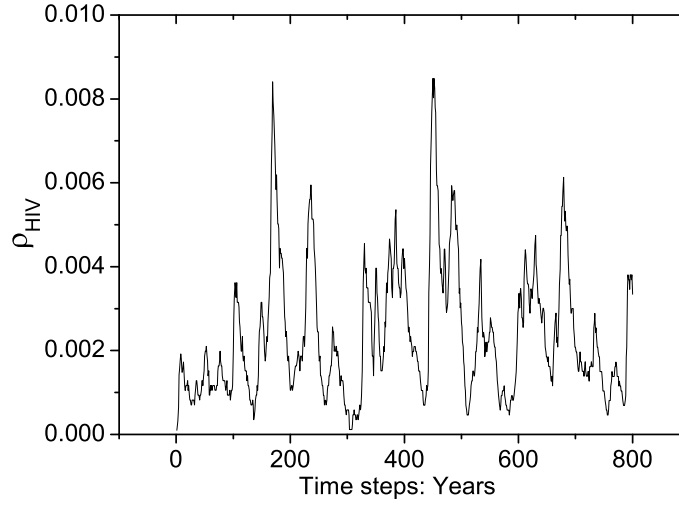


Fig. 1. The prevalence of HIV versus time. ρ_{ARV} denotes the ratio of HIV-positive individuals to the whole population of sex-active ones (i.e., the network size). The corresponding parameters are $(\langle k \rangle, \beta_1, \rho_{\mathrm{ARV}}) = (3.0, 0.10, 0)$.

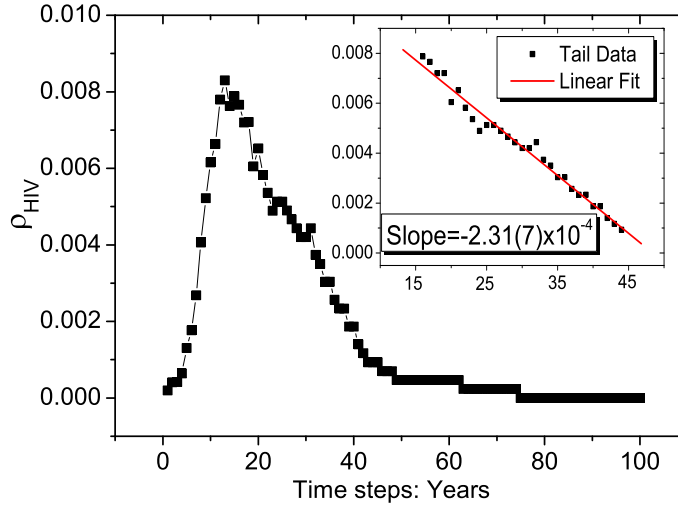


Fig. 2. The prevalence of HIV versus time in a homogeneous network. The inset shows the tail of ρ_{ARV} as a function of time step (from the peak point to nearly zero). The corresponding parameters are $(\langle k\rangle, \beta_1, \rho_{\mathrm{ARV}}) = (3.0, 0.10, 0)$.

In addition, this model displays oscillatory behaviors, which have been observed in the real world[45,46] and reproduced by some previous network epidemic models based on small-world networks[47–49] or scale-free networks.[50] One can see Refs. 51 and 52 for the concept of small-world networks. However, since the time from the first report about HIV cases to now is relatively short compared with the oscillatory period, we cannot make sure if the real-life HIV epidemics showing some kinds of oscillation.

3.2. *Effect of transmission probability*

The transmission probability β_1 not only depends on the pathological characters of HIV, but also can be managed by the government and other organizations. For example, the popularization of the usage of condoms will sharply reduce the transmission probability per sexual partner/act as observed in Thailand and Cambodia.[53,54] Figure 3 exhibits the $\rho_{\mathrm{ARV}} - t$ curves for different β_1: When β_1 is large, the prevalence fleetly increases until considerable ratio of the whole population get infected, while for smaller β_1, the infection either persistently exists in a low prevalence-level, or vanishes.

3.3. *Effect of average degree*

We have also investigated the effect of average degree $\langle k\rangle$ on the network epidemic behaviors. As shown in Fig. 4, the behaviors of this model are very sensitive to the mean degree. Clearly, larger mean degree will statistically enlarge the probability of coming into contact with infected individuals, thus leading to more serious situation.

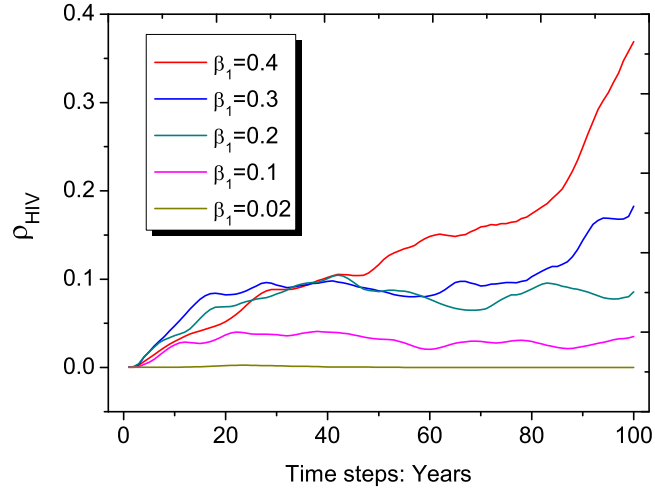


Fig. 3. The prevalence of HIV versus time for different transmission probability. The corresponding parameters are $(\langle k\rangle, \rho_{\mathrm{ARV}}) = (3.0, 0)$. All the data are obtained by averaging 10 independent realizations.

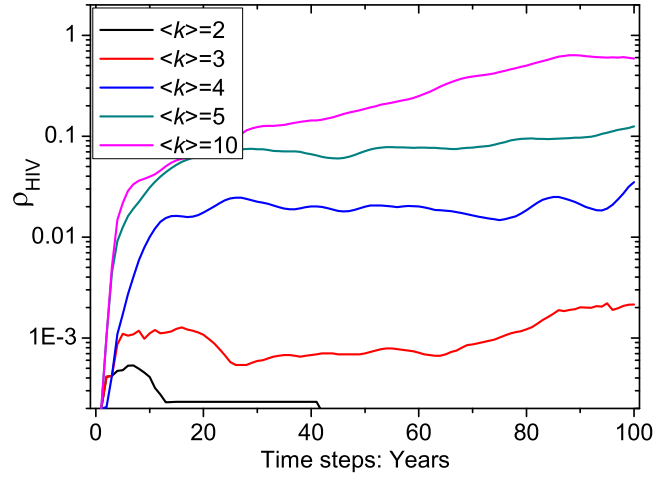


Fig. 4. The prevalence of HIV versus time for different average degrees of epidemic contact networks. The corresponding parameters are $(\beta_1, \rho_{\mathrm{ARV}}) = (0.10, 0)$. All the data are obtained by averaging 10 independent realizations.

Combine this result and that of Sec. 3.1, one will find that the epidemic behaviors are highly affected by the network topology.

3.4. *Impact of antiretroviral drug therapies*

The antiretroviral drug therapies have two opposite effects. On one hand, it will reduce the probabilities of both sexual transmission and perinatal transmission, thus

ought to be very helpful in controlling the epidemic spreads.[27,40] On the other hand, this treatment will increase the life expectancy for HIV-positive persons and these ARV users can infect more individuals if they do not stop their risky behaviors, thus this treatment may on the contrary increase the incidence of HIV/AIDS.[25,55] Here, we assume the usage of ARV will not change patients' behaviors, and in Fig. 5, one can find that this treatment can substantially reduce HIV epidemics, and even be possible to eradicate high prevalence HIV epidemics under certain ideal conditions. It is worthwhile to emphasize that, the simulation results in Fig. 5 strongly depend on the choices of some dubious and imprecise parameters[27,40] such as the ratios β_2/β_1, ζ_2/ζ_1, and $\varepsilon_2/\varepsilon_1$. Therefore, the corresponding results are not confessed.

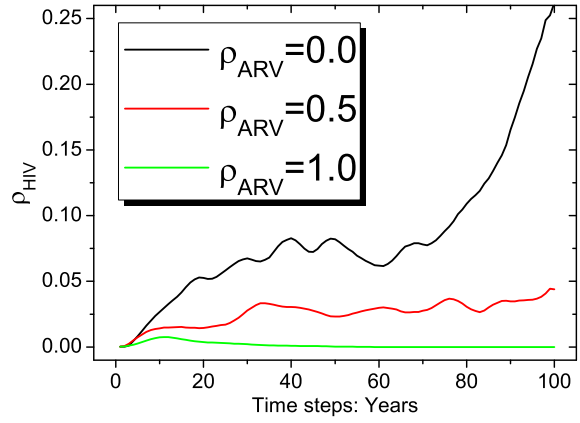


Fig. 5. The prevalence of HIV versus time for different ARV treatment levels. The corresponding parameters are $(\langle k\rangle, \beta_1) = (3, 0.23)$. This value of β_1 is chosen according to the case of northern Thailand.[56] All the data are obtained by averaging 10 independent realizations.

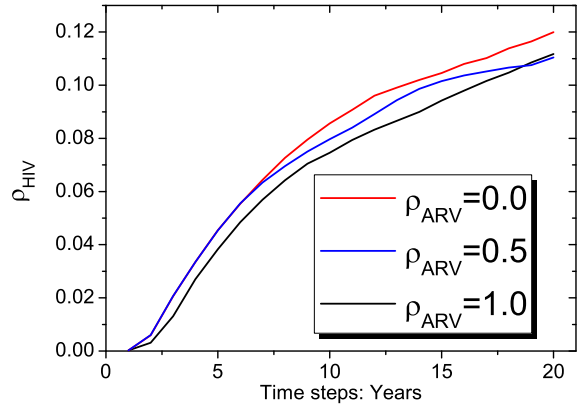


Fig. 6. The prevalence of HIV versus time for different ARV treatment levels. The corresponding parameters are $(\langle k\rangle, \beta_1) = (5, 0.23)$. All the data are obtained by averaging 10 independent realizations.

Maybe further empirical and experimental studies about antiretroviral drug therapies will lead to more accurate results. In addition, the behavior parameters $\langle k \rangle$ and β_1 are more important than ρ_{ARV}, and for very large $(\langle k \rangle, \beta_1)$ case, as shown in Fig. 6, the impact of antiretroviral drug therapies is very weak. Hence, to reduce the risky behaviors is much more effective in the fight against HIV/AIDS rather than ARV treatment, especially for the developing countries. The cases of Thailand[53] and Eastern Zimbabwe are very good examples.[57]

3.5. *Hierarchical spread of HIV epidemic outbreaks*

In the epidemic contact networks, the degree can reflect the susceptibility of individual to some extent, that is, the node with higher degree is easier to be infected statistically.[58] Here, we investigate the behavior of the average degree of the newly infected nodes in networks at time t, denoted by $k_{\mathrm{inf}}(t)$. We use the average of 10 realizations to reduce the fluctuations. As shown in Fig. 7, the dynamical spreading process is therefore clear: After the high-risk population are infected within a short time, the spread is going toward generic population (low-risk population). This hierarchical spread, has been recently reported in some previous pure theoretical studies on SI model,[59–63] but not been emphasized in previous HIV/AIDS epidemic models. However, this phenomenon has been observed in real-life HIV epidemics: In the early stage the infection is adhered to these high-risk persons, such as sex workers, injection drug users, men who have sex with men, and so on. And then, it diffuses to generic population. As a typical example, one can see the situation in China.[b]

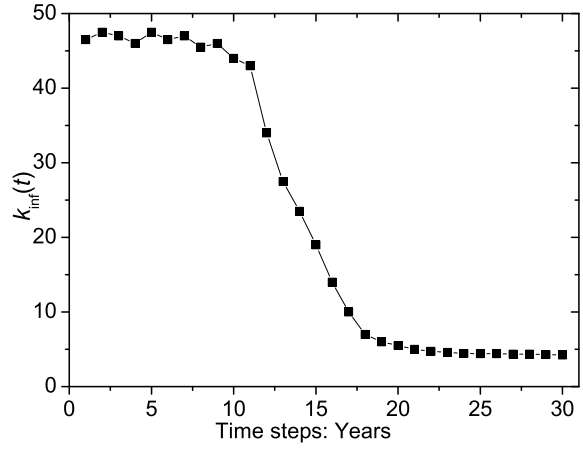


Fig. 7. Time behavior of the average degree of the newly infected nodes. All the data are the average over 10 realizations. The corresponding parameters are $(\langle k \rangle, \beta_1, \rho_{\mathrm{ARV}}) = (3.0, 0.10, 0)$.

[b]Annual reports on AIDS epidemic in China, from the web site http://www.chinaids.org.cn.

4. Relevance to the Real HIV/AIDS Epidemics

Previous studies on the prediction of the HIV/AIDS epidemics mainly concentrate on the data of the number of reported HIV-positive cases. These methods, like empirical Bayesian back-calculation,[64] can give a relatively accurate prediction within a short term. However, it cannot provide useful information about the underlying dynamic mechanism. Therefore, in this section, we will try to predict HIV/AIDS epidemics by using the present model.

The lack of comprehensive and authentic data is one of the most serious problems in evaluating and predicting HIV/AIDS epidemics. For example, in the year 2004, the Chinese Minister of Health reported that the number of living HIV-positive persons is about 8.6×10^5, but in the year 2006, it said that this number is completely incorrect due to the greatly overvaluing. Actually, the veracity of the reported HIV-positive numbers is dubious. From the web site of UNAIDS (see footnote a) except the data of HIV-positive numbers, one can also obtain the data about the number of AIDS-patients from national sentinel surveillances. These data are also dubious since the monitor policies are not professional especially in developing countries and some AIDS-patients do not want to report to the sentinel surveillances. However, the data from national sentinel surveillances do not involve external estimating algorithm, thus we believe they are at least more faithworthy than the HIV-positive numbers.

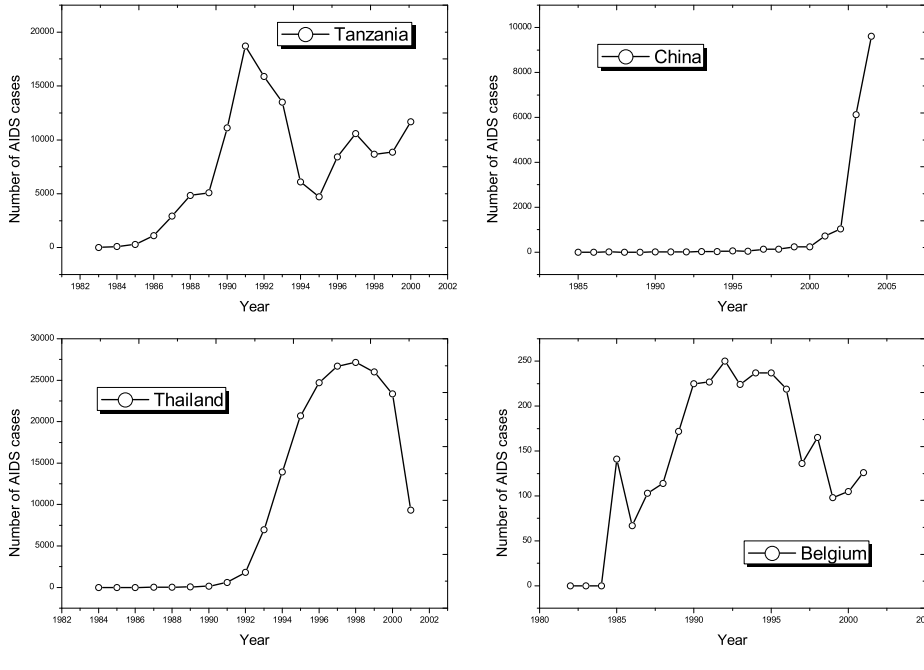


Fig. 8. The time series of AIDS-patients number from national sentinel surveillances in Tanzania, China, Thailand, and Belgium. These data are obtained from the web site of UNAIDS.

In Fig. 8, we show four typical forms of the time series of the number of AIDS cases. Although there are some other forms, the present fours are representative. The most serious country is Tanzania, wherein a considerable ratio of whole population is infected. Without impelling control policies, Tanzania will be completely destroyed before long. In China, the proportion of AIDS cases seems very small but the amount of AIDS is quite large as a result of the striking huge ensemble, and its quick and monotone increasing trend brings us heavy misgivings. Thailand is a successful example of external control. Once, Thailand, especially the northern Thailand, is the most serious country in Asia. Delightfully, the government is cognizant of this problem and forces all the sex workers using condoms. This policy leads to a sharply decreasing of HIV-positive and AIDS-patient numbers. Some other countries, like Brazil, have also achieved successful policies in controlling HIV/AIDS epidemics. However, these emergent external policies bring great challenges in predicting. The most optimistic situation is that of Belgium, where the HIV/AIDS persists in a very low prevalence level and no increasing trend is observed.

In our model, according the assumption in Sec. 2.2, we consider the mortality at time t as the number of newly monitored AIDS-patients. This quantity, denoted by $N_{\text{AIDS-NEW}}$, can be obtained from the model by combining the death rolls of children, adults, and old persons. Because of the computational limit, we can at most handle the epidemic contact network with size $N \sim 10^7$. However, the number of people aged from 15–49 in some countries is much larger than 10^7. In order to compare the time series generated by our model and those of real country, all the data are normalized by the population size aged from 15–49. The normalized number of AIDS cases is denoted by $\rho_{\text{AIDS-NEW}}$. In addition, we assume the number of AIDS cases $N_{\text{AIDS-NEW}}$ is proportional to the HIV-positive number N_{HIV} at a given time t. Denote the normalized data from sentinel surveillances by $x(0)$, $x(1), \ldots, x(T)$, and the data generated from our model by $y(0)$, $y(1), \ldots, y(T)$, the normalized average departure is defined as

$$e = \frac{1}{T+1} \sum_{t=0}^{T} \left(\frac{x(t) - y(t)}{x(t)} \right)^2 . \tag{3}$$

Since the parameter ρ_{ARV} is known after the country is selected (these data can also be found from UNAIDS), there are only two tunable parameter β_1 and $\langle k \rangle$. Hence this task degenerates to an optimal problem: Determine the proper value of β_1 and $\langle k \rangle$ to minimize the departure e. The optimal problem is carried out by searching all the values of $(\beta_1, \langle k \rangle)$ in the Cartesian product of sets $\Gamma = \{0.01, 0.02, \ldots, 0.50\} \times \{1.0, 1.1, \ldots, 10.0\}$. First, we average 100 independent realizations to obtain the mean value of error $\bar{e}$ in the parameter space Γ, and then choose the parameters, named *optimal parameters*, with the minimal mean error $\bar{e}$. The prediction result, as shown in the figures, corresponds to the best one (with the minimal error e) among the 100 simulations under the optimal parameters. The parameters will not change with time, that is to say, the present prediction is valid only for the cases

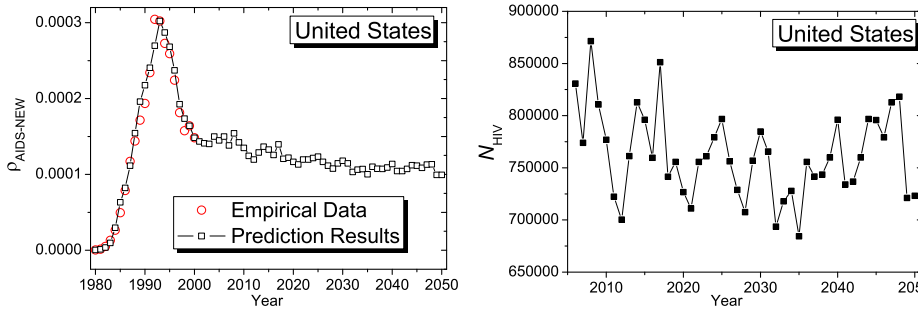


Fig. 9. The prediction results for US. The left plot shows the empirical data and the projections about AID cases reported annually, and the right plot is the corresponding HIV-positive numbers from year 2006–2050. In this case, $\rho_{\text{ARV}} = 0.65$, and the optimal parameters are $\beta_1 = 0.11$ and $\langle k \rangle = 4.3$.

with no additional interventions. A brief discussion about the robustness of the prediction results are given in Appendix B.

We have tried this prediction method for many representative countries, and found the cases can be roughly divided into three patterns. A typical example for the first pattern is the United States (US), wherein the curve of AIDS-patient number has an obvious peak before the year 2000, and then decreases to a relative low and stable level (Fig. 9). The similar behaviors have also been found for many other countries, such as Mexico, Spain, Australia, Belgium, Thailand, and so on. The common feature of these countries is that their transmission probabilities are all small. This may be because of the high popularization rates of condoms and disposable injectors.

China is a particular example, although the prevalence of AIDS cases are very low, it increases exponentially fast in the early stage with exponent $\approx 0.15 \pm 0.01$, that is, $\rho_{\text{AIDS-NEW}} \sim e^{0.15t}$ in the early stage. However, this velocity will be slowed down and the number of AIDS cases will get steady after the year 2025. This interesting behavior may due to the particular values of β_1 and $\langle k \rangle$ in China. Traditionally, Chinese women are not supposed to have sex with a man other than their future spouse, thus its mean degree $\langle k \rangle$ is very small compared to the "Western-style" society. However, since the popularization rate of the usage of condoms in China is very low ($< 20\%$), the transmission probability in China is much higher than these developed countries. In a word, although no efficient policies in controlling HIV epidemics have been implemented in Chinese government or other organizations, the traditional moral sense may protect China from suffering heavy AIDS (Fig. 10).

Note that, the shapes of $N_{\text{HIV}}(t)$ and $\rho_{\text{AIDS-NEW}}(t)$ are slightly different, which attributes to the fluctuation of population size. Although the numbers of HIV-positive dwellers seems high in some countries, such as US and China, their direct and indirect effects on the demographic structure of the whole population are very weak. If we fixed $\beta = 0$ for US and China from 2006–2050, then the population

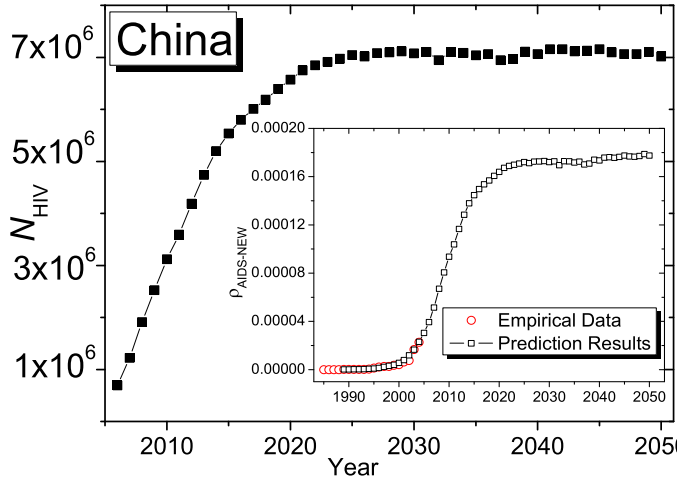


Fig. 10. The prediction results for China. The main plot shows the predicted HIV-positive numbers from year 2006–2050, and the inset exhibits the empirical data and the projections about AID cases reported annually. In this case, $\rho_{\mathrm{ARV}} = 0.05$, and the optimal parameters are $\beta_1 = 0.28$ and $\langle k \rangle = 2.1$.

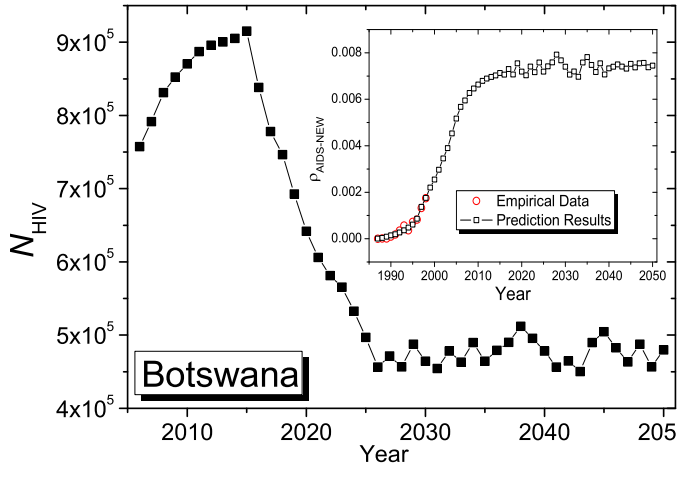


Fig. 11. The prediction results for Botswana. The main plot shows the predicted HIV-positive numbers from year 2006–2050, and the inset exhibits the empirical data and the projections about AID cases reported annually. In this case, $\rho_{\mathrm{ARV}} = 0.079$, and the optimal parameters are $\beta_1 = 0.31$ and $\langle k \rangle = 7.2$.

size (aged from 15–49) without HIV/AIDS will be almost the same as the original prediction results. Since the departures cannot be observed in plots, we have not shown here.

The most serious regions suffering AIDS are Africa (especially Sub-Saharan Africa)[65–67] as well as Latin America and Caribbean region.[68] A typical example

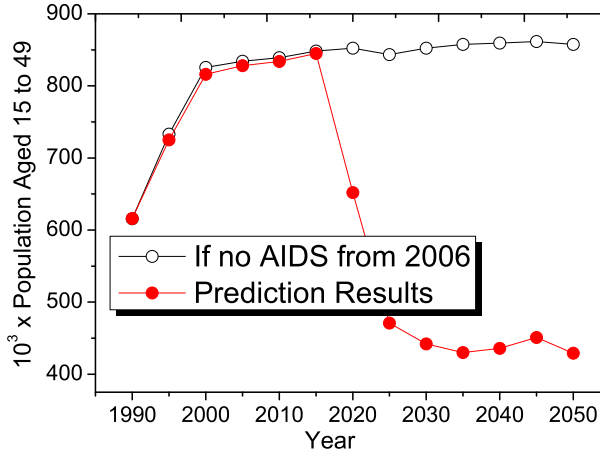
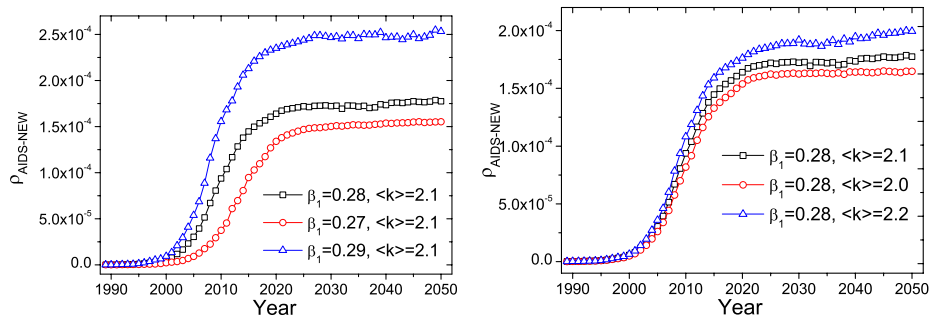


Fig. 12. Predicted population size aged 15–49 compared with the situation without AIDS.

is Botswana, which is a relative rich country in Africa but has the highest HIV prevalence all over the world (Fig. 11). If these is no additional interventions, the infection will kill more than 50% of persons in their prime of life. The ostensible stable behavior after 2020 is on account of the existence of some no-risk people: They adhere to monogamy and do not hit the pipe, thus will not be infected. In network language, these individuals belong to some isolated clusters. In network SIR model, these isolated clusters usually come into being as a result of the removal of some individuals.[69] Other than US and China, demographic impact of the HIV epidemic in Botswana is striking. In Fig. 12, we compare the predicted population size (aged from 15–49) with the no-AIDS case where we set $\beta_1 = 0$ from 2006–2050. After year 2015, the population size sharply declines, which is the very reason of the decline of N_{HIV} in Fig. 11. Only by 10 years, the population holds down to a half level. The demographic gets stable in this new level since there are a number of no-risk people. Finally, the age distribution in Botswana becomes sandglass-like since many people in their prime of life (aged 15–49) will be killed by AIDS. We are afraid that some other countries in Africa, such as Malawi, Tanzania, and Zambia, may face the same danger.[70,71]

5. Conclusion and Discussion

In this article, we propose a network epidemic model for HIV epidemics, wherein each individual is represented by a node and the edges are the connections between individuals along which the infection may spread. Motivated by some previous empirical studies on the pattern of sexual contact, we set the sexual activity of each individual, measured by its degree, is not homogeneous but obeys a power-law distribution.



(a) (b)

Fig. 13. Comparison of the simulation results of China around the predicted parameters β_1 and $\langle k\rangle$. The data of $(\beta_1, \langle k\rangle) = (0.28, 2.1)$ is the same as that plotted in Fig. 10. The left (a) and right (b) plots represent the comparisons for different β_1 and $\langle k\rangle$, respectively.

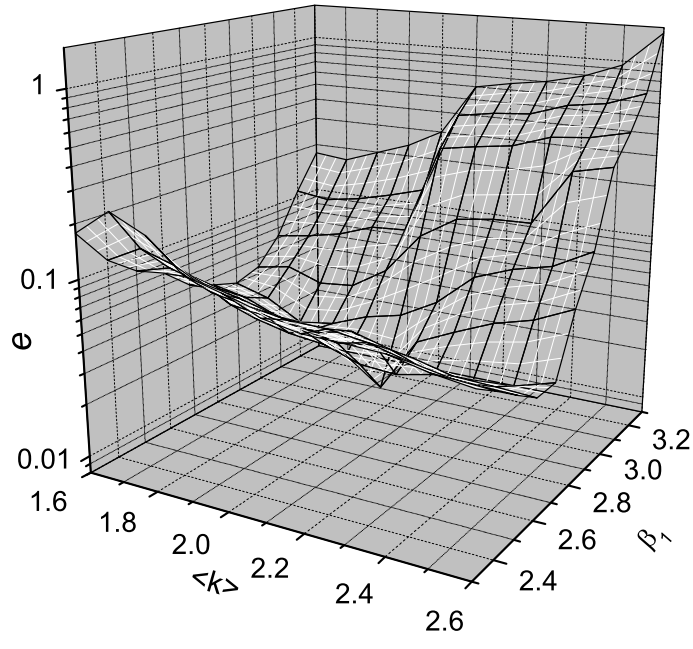


Fig. 14. The error-plane resulted from the prediction of China.

Many infections, including HIV/AIDS, can persist in populations at a very low prevalence. This epidemiological phenomenon cannot be illuminated by previous models with homogeneous mixing hypothesis, while our model can reproduce this feature due to the heterogeneity of activity. In addition, the model displays a clear picture of hierarchical spread: In the early stage the infection is adhered to these high-risk persons, and then, diffuses toward low-risk population.

We find that to reduce the risky behaviors is much more effective in the fight against HIV/AIDS rather than the antiretroviral drug therapies. It may result from the more risky brought by ARV treatment. In addition, the ARV treatment may generate epidemics of drug-resistant strains of HIV, which are much more terrible than the regular epidemics.[72] Therefore, the government should be cautious before working out the plan to generalize ARV treatment.

There are two main ingredients baffling the prediction obtained by dynamical model: The first is the lack of comprehensive and authentic data, and the second is the existence of unexpected interventions like the governmental action against HIV/AIDS epidemics. Right or wrong, we try to predict HIV epidemics by using the present model, and hope it will at least capture some qualitative features. The prediction results show that the development of epidemics can be roughly categorized into three patterns for different countries: persist in a stable and low level after a peak in the early stage (US), monotonously grow and then persist in a stable and low level (China), infect considerable ratio of population (Botswana). Which class the HIV epidemic of a given country finally belongs to is mainly determined by the corresponding behavior parameters $(\langle k\rangle, \beta_1)$. The interplay of demographic structure and HIV epidemics is also taken into account. In most cases, the effect of HIV epidemics on demographic structure is very weak, while for some extreme countries, like Botswana, the population size can be depressed to a half, and the age structure will become sandglass-like since many people in their prime of life (from aged 15–49) will be killed by AIDS.

We believe this work may have some contribution in understanding the underlying mechanism of HIV epidemic dynamics, since it can naturally reproduce some important observed characters in HIV spread that has not been emphasized in the previous models. However, is has many shortages which should be adverted to and may be considered in the future works. The first is the memory-limitation and time-complexity in simulation block the direct studies on huge systems. Therefore, we have to use the normalization method to mimic the real countries with huge population. This size effect may bring additional error in prediction. A recently proposed fast algorithm[73,74] may improve the situation if we have successfully modified some dynamical rules and translated this model into an equivalent rate-equation form. Secondly, this model considers only the heterosexual contacts and perinatal transmission, however, other transmission routes, especially the homosexual contacts[75,76] and sharing injectors among drug users,[77,78] are also significant in HIV epidemics. Finally, some details are ignored. For example, a fraction of people (e.g., $\sim 10\%$ in African communities) are infected but behave according to the normal statistical social data, a recent study[79] indicates the existence of large fertility differentials between HIV-infected and uninfected women, and some empirical studies show that the social networks have community structure,[80,81] which may affect the epidemic dynamics.[82] And in the reality, the individuals can move from one place to other place, which may also affect the epidemic behaviors.[83,84]

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The Population and Demographic Data

All the population and demographic data come from the United Nations and can be obtained by decompressing the .zip file from http://www.comap.com/undergraduate/contests/mcm/2006/problems/2006%20ICM.zip. The explanation about some used data in this article are as follows:

age_data.xls: These data give population (for both sexes, and by gender) by five-year groups, major area, region, and country, 1950–2050 ((a) Estimates for 1950–2005; (b) Projections for 2010–2050).

fertility_data.xls: These data give age-specific fertility rates by major area, region, and country, 1995–2050 ((a) Estimates for 1995–2005; (b) Projections for 2010–2050).

population_data.xls: These data give total population (both sexes combined) by major area, region, and country, annually for 1995–2050 ((a) Estimates for 1950–2005; (b) Projections for 2006–2050).

Robustness of the Prediction Results

In this section, we will give a brief discussion about the robustness of the prediction results, using the case of China as an example. As shown in Fig. 13, if one slightly varies the optimal parameters $(\beta_1, \langle k \rangle)$, the qualitative behavior will not change while the quantitative departures are clearly observed. And the simulation are more sensitive to the transmission probability than average degree. In addition, to check if there are more than one pairs of "optimal parameters" having almost the same $\bar{e}$, as shown in Fig. 14, we plot the error-plane resulted from the case of China. Since the error will sharply increase when the parameters' coordinates are far from the best point $(\beta_1, \langle k \rangle) = (0.28, 2.1)$, we just report the situation near this point. It can be clearly seen from the error-plane that there is only one best point having smallest error, and the points also having small errors are close to the best one, thus the results shown in Sec. 4 are robust. And the results shown in Fig. 14 also agree with the results in Fig. 13, that is, the parameter β_1 has more highly influence on the epidemic dynamics than $\langle k \rangle$'s.

The situations of another countries are qualitatively the same, thus are not shown here.

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