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A CA-based epidemic model for HIV/AIDS transmission with heterogeneity

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Abstract The complex dynamics of HIV transmission and subsequent progression to AIDS make the mathematical analysis untraceable and problematic. In this paper, we develop an extended CA simulation model to study the dynamical behaviors of HIV/AIDS transmission. The model incorporates heterogeneity into agents' behaviors. Agents have various attributes such as infectivity and susceptibility, varying degrees of influence on their neighbors and different mobilities. Additional, we divide the post-infection process of AIDS disease into several sub-stages in order to facilitate the study of the dynamics in different development stages of epidemics. These features make the dynamics more complicated. We find that the epidemic in our model can generally end up in one of the two states: extinction and persistence, which is consistent with other researchers' work. Higher population density, higher mobility, higher number of infection source, and greater neighborhood are more likely to result in high levels of infections and in persistence. Finally, we show in four-class agent scenario, variation in susceptibility (or infectivity) and various fractions of four classes also complicates the dynamics, and some of the results are contradictory and needed for further research.

Keywords HIV/AIDS transmission · Epidemic dynamics · Heterogeneity · Cellular automata

1 Introduction

Since the first case was reported in 1981, AIDS has been considered as the most devastating epidemic disease. For effective control and prevention of this epidemic, many researchers

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focus on HIV/AIDS transmission among human groups in order to better understand its dynamical behavior.

In epidemic modeling (see, e.g., Bailey 1975; Anderson and May 1991; Murray 2005), there are two frequently used methodologies: mathematical and simulation methods. For mathematical approaches, a cohort of people is often classified into susceptibles, infectives, and recovereds with (without) immunity (see, e.g., Kermack and McKendrick 1927). Systems of differential equations are used to describe the linear (nonlinear) dynamics of epidemics. Macroscopically, mathematical models can reveal the relationship among primary factors and describe their effects to epidemic spreading under certain assumptions. As to HIV/AIDS epidemic, many models have been proposed (May and Anderson 1987; May et al. 1988; Hyman et al. 1999; Brauer and Driessche 2001; Wu and Tan 2000, etc.). However, mathematical approaches have some serious drawbacks due to its intractability and the complexity of epidemics. Moreover, the complicated nature of HIV/AIDS transmission makes it even harder to obtain analytical solutions and difficult to study them. In the early 1990s, some researchers started to apply simulation approaches to this field. There is a large literature that addresses the computer simulation of epidemic dynamics (see, e.g., Leslie and Brunham 1990; Atkinson 1996; Rhodes and Anderson 1996; Rhodes and Anderson 1997; Ahmed and Agiza 1998; Benyoussef et al. 2003; Tarwater and Martin 2001). Particularly, cellular automata (CA) method (some literature refers to this as a lattice-based method) has been widely used in modeling complex adaptive systems. Despite of its simple structure, CA is well suited to describing the propagation phenomena, such as rumor spreading, particle percolation, innovation propagation, and disease spreading. For instance, in epidemic modeling, Fuentes and Kuperman (1999) propose two CA models corresponding to the classical mathematical SIS model and SIS model respectively. Ahmed and Agiza (1998) develop a CA model that takes into consideration the latency and incubation period of epidemics and allow each individual (agent) to have distinctive susceptibility. Gao et al. (2006) put forward a CA model for SARS spreading which takes account of social influence. More Recently, other methods such as agent-based modeling and system dynamics are introduced to this field (see, e.g., Gordan 2003; Bagni et al. 2002). Our paper contributes to this filed by developing an extended CA simulation model. We then use the new CA model to investigate some issues in HIV/AIDS epidemics.

Most models, including the foregoing CA models, have some limitations that fail to consider the peculiarities of HIV/AIDS epidemics and are thereby incapable of describing the epidemic accurately and completely (see Frauenthal 1980 for more discussion). First, most of the models assume that there is no latent (or incubation) period. However, for some epidemics, especially AIDS, there are variously lasting periods of latency and incubation as well as behavior-varying infectivity (or susceptibility) during these periods. In fact, the development of AIDS involves a few stages in which an infected individual can exhibit different behaviors. Those diversified behaviors, in turn, have some ignorable effects on the dynamics of HIV/AIDS. In light of this, we extend the conventional division of epidemic process (i.e., susceptible, infection, and removed) by dividing the infection period into three sub-stages, each corresponding to the clinical stage occurring in the course of AIDS development. Due to the inability of classical CA approaches to accommodate those newly added state transitions events, we also borrow some ideas from discrete-event simulation techniques and make one agent's stage transitions being time-triggered instead of using some state-based transition rules.

Secondly, it is commonly assumed that individuals in the population are homogenous in the sense that they have equal infectivity and susceptibility, or they can exert the same influence on each other, etc. This assumption may be satisfied in commonly observed epidemics

but not consistent with the HIV/AIDS epidemic. As we know, susceptibility and infectivity heavily depends on individuals' behavior. For examples, safe sex practices such as the use of condom could dramatically reduce the chance of infection. Also, the way of HIV/AIDS transmission for one to another is various, depending on the interactions between people, and thus the probability of getting infected is determined in part by transmission routes and can be quite different between infected-male/susceptible-female and susceptible-male/infected-female interactions. Under this assumption, the models that are confined to a single high-risk human group are not suitable in overall population cases. New models are needed to explicitly consider the complexity. Therefore, we make an extension to the traditional CA model by introducing the extended definition of neighborhood and attaching some attributes to each agent such as infectivity and resistibility. We also define four types of agents that are characterized by different infectivity (and susceptibility) and various forms of neighborhood to represent four types of people in real life. In doing so, we will be able to investigate the dynamics of HIV/AIDS with heterogeneous groups in a realistic way.

Thirdly, classical CA models assume that agents in the grid are spatially fixed, that is, once an agent is placed in a cell, it does not move into another cell. This assumption is problematical because people in the real world are migratory. For instance, in China, millions of rural people leave their hometowns and seek jobs in the cities. The migration of population is a driving force for the spread of HIV/AIDS. Ignoring the mobility of agents in epidemic models would jeopardize the creditability of the results obtained. Considering this point, we incorporate agents' mobility into their behaviors. In our model, each agent is allowed to move randomly into one of its adjoined and unoccupied cells at random time intervals.

Recently, Agent-based modeling is used in various fields to solve plenty of problems (see, e.g. Zhang and Bhattacharyya 2007; Luo et al. 2007). Some reader might notice that our improved CA model have features that usually found in Agent-based methodology. As a matter of fact, our method borrows much from agent-based simulation modeling. To make things simple, we prefer to view this model as being a CA models.

This paper is organized as follows. In the next section, we present our extended CA simulation model. Section 3 gives a detailed description of simulation results and analyzes some influential factors that affect the dynamical behavior of the model. Section 4 concludes and points out some possible extensions and directions for future research.

2 CA model for HIV/AIDS transmission

2.1 Cellular automata

Cellular automata have been extensively used as tools for modeling complex adaptive systems such as traffic flow, financial markets, chemical systems, biological groups, and other social systems (see e.g. Gerhard and Schuster 1989; Gerhardt et al. 1990; Weimar et al. 1992; Karafyllidis and Thanailakis 1997; Karafyllidis 1998). Usually, a typical CA model consists of a regular two-dimension grid with a certain boundary condition and a swarm of agents living in the grid.[1] The *neighborhood* of an agent is defined to some (or all) of the immediately adjacent cells and the agents who inhabit in the neighborhood are called *neighbors*. Agents are restricted to local neighborhood interaction and hence are unable to communicate globally. There are several states agents can be in at each time and an agent's

[1] Note that in the conventional view, the cells in the grid and the agents who occupy them are equivalent. Here we take another view and see them as being different to facilitate the modeling.

state at time $t+1$ is determined based on its neighbors' states at time t. The rules used in the determination of next-time states can be written as a mapping:

$$f : (S, t) \to S, \tag{1}$$

where S is the set of states and t denotes simulation time. The mathematical properties of cellular automata have been studied in Martin et al. (1984).

In our Model, we consider a population of size $N(t)$ at time t randomly distributed in a two-dimension $w \times w$ lattice. Population growth rate r is fixed throughout the simulation. At each time, new agents are added to the model, and the dead removed. Simulation time advances in a discrete way. The time interval $(t, t+1)$ is specified to represent one week in real life. This assumption makes the simulations run reasonably fast (with respect to the whole progress of epidemics) without losing any time-specific clinical properties associated with HIV/AIDS.

2.2 Epidemic stages and state transitions

Explicitly modeling the post-infection progression to AIDS is one feature of our model compared with conventional CA models. Classical epidemic models divide the closed population into three subgroups: susceptible, infective, and recovered (removed). This simplified classification is not consistent with the epidemics in real life. Particularly, it is well established that an individual, once infected with HIV, undergoes roughly three clinical phrases towards the full-blown AIDS: (1) infected, not yet infectious, (2) infectious, not yet asymptomatical, and, (3) symptomatical (May and Anderson 1987; May et al. 1988). The lifetime of an individual should cover not only the process from health to infection, but also the sub-stages after infection. Thus, we assume that each agent can go through the following states:

- S1: Healthy state,
- S2: Dangerous (or critical) state,
- S3: Infected state,
- S4: Infectious state,
- S5: Symptomatic state,
- S6: Deceased state.

Figure 1 shows the possible state transitions throughout the lifetime of an agent.

Initially, each agent is set to be in S1 state. Healthy agents have no risk of being infected. When a healthy agent moves into the neighborhood of an infectious one or an infectious agent approaches him, the healthy agent's state will change from S1 to S2 because contacts with infectives incur the danger of infection.

As for an agent in S2 state, it can transit in two directions: One direction is to change from S2 back to S1, after all its infectious neighbors move away (or its dead neighbors are removed from the grid) or he leaves the neighborhoods of its infectious neighbors; the other direction is to change from S2 to S3 if he unluckily get infected. Note that we assume infection is instantaneous, i.e., instantaneous transmission from an infected individual to a susceptible.

A newly infected agent is unable to transmit HIV virus until seroconversion. The S3 state corresponds to the early stages of HIV infection. Let T_1 denote the duration of this period. Empirical works have been done to estimate the parameter. Anderson et al. (1988), Anderson and Medley (1988) report T_1 to lie between 40 and 60 days in transfusion-induced AIDS cases. In our model we assume that T_1 is a random variable following a normal distribution with the mean μ_1 and the variance σ_1^2.

After T_1, the infected agent enters S4 state: infectious state. Medically, the duration during which an infected is infectious but not yet symptomatic is called incubation period. We let T_2 donate the period. Empirical work suggests an average incubation period of around 4 to around 15 years (Medley et al. 1987, 1988). A Weibull distribution are commonly used to describe this incubation period (see, e.g., Anderson 1988; Anderson and Medley 1988). Furthermore, Anderson et al. (1988), Anderson and Medley (1988) estimated T_2 with a Weibull distribution (with a mean of 7.7 years and a median of 7.4 year) based on 545 transfusion-induced AIDS cases. For simplicity, we take T_2 as a real number drawn from a normal distribution with the mean μ_2 and the variance σ_2^2 rather than a Weibull distribution. It should be pointed out that the simulation results generated with the normal distribution here are proved to have little, if any, difference compared with those generated when a Weibull distribution is employed.

During the T_2 period, HIV viruses in the victim's body are constantly cloning themselves and eventually the immunity system collapses. At this point, the victim starts to show some symptoms and thus transit to S5 state: symptomatic stage. As usually, let T_3 denote the duration of this period. Rothenberg et al. (1987) report 5–7 year survival rates among 1660 IDUs (Intravenous Drug User) in New York City and find a median time of survival of 282 days. Chang et al. (1993) report a median survival time of 10.5 months. Empirical work shows that almost all HIV infectives, excluding those who die from other causes, will inevitably develop AIDS and die of it (May and Anderson 1987). Similarly, we assume T_3 follows a normal distribution with the mean μ_3 and the variance σ_3^2.

Eventually, the ill agent enters S6 state after T_3 passes by Agents in S6 state will be removed from the population at the beginning of the next time and all of their uninfected neighbors will be released from S2 state, back to S1 state.

Generally, these state transitions take place in the order of S1, S2, S3, S4, S5, and S6. It is impossible for an agent to return from S3 state to S1 or S2 state. This backward transition S2 to S1, demonstrated by a dashed line in Fig. 1, is due to the disappearance of threats posed by infectious agents. Moreover, although the transitions among S3, S4, and S5 state are not relevant to the propagation process, this process is closely related to HIV/AIDS transmission. Taking account of this procession is essential for a better understanding of HIV/AIDS transmission.

In the model, all the events triggering transitions could be divided into two categories. One category is a rule-based, such as healthy-to-dangerous, dangerous-to-infected, and dangerous-to-healthy state-transition events. These events occur according to the CA transition rules: An agent's state at time $t + 1$ is based not only on its own state but also on the states of its neighbors at time t. The other category is time-based, meaning that these events are scheduled at pre-specified times. For instance, an agent entering S3 state will be assigned a time indicating when to change to S4 state. After that amount of time elapse, the transition occurs spontaneously. Despite the distinction between these two categories, the subtlety of implementing the two event-triggering mechanisms is very trivial and leaves no further elaboration necessary.

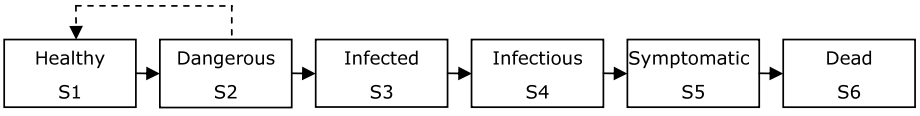

Fig. 1 Transitions among six states

Actually, these six states can be divided into three "super" classes in terms of the taxonomy used in the classical mathematical models: S1 and S2 states correspond to the susceptible state; S3, S4, and S5 states belongs to the infection state, and S6 state is the removed state. Obviously, S1 and S2 state could be treated as one single state without changing any results. The reason why we divide this into two sub-states is that this makes our model easily implemented and our logic decent and legible.

2.3 Heterogeneity in agents' behavior

HIV/AIDS epidemic differs from other epidemics in that its dynamics is heavily affected by individual's behavioral patterns and the interactions between them. For example, careful sex practices and sanitization measures in drug taking will make individuals less likely to be infected. Behavioral patterns and interactions are mostly determined by individual's life styles, personalities, social networks, etc. However, the majority of models fail to take account of the heterogeneity in agents' behaviors. To capture this, we extend classical CA models by allowing each agent to have its own attributes such as mobility, infectivity, resistibility (susceptibility)[2] and different extent of neighborhood.

2.3.1 Mobility

Assume that each cell in the grid can be occupied by at most one agent at a time. At time t, agent i can move from one cell into one of its adjacent cells with probability p_i^m. Here, p_i^m is a measurement of agent i's activity level. It is a fixed real number, drawn from a uniform distribution $(p_{\min}^m, p_{\max}^m)$ $(0 \le p_{\min}^m \le p_{\max}^m \le 1)$. When $p_{\min}^m = p_{\max}^m$, the activity level across agents is equal and therefore agents have the same inclination to move around. One extreme case is $p_{\min}^m = p_{\max}^m = 0$, which corresponds to the situation in which agents stay in their initial places during simulation, whilst $p_{\min}^m = p_{\max}^m = 1$ means that each agent will move into one empty neighboring cell at almost each time (he could get stocked and not move anywhere if it's neighborhood is occupied). It is easy to induce that the average time per move is calculated as $1/p_i^m$. Intuitively, high level of activity leads to speedy spreading. Our simulation results verify this.

2.3.2 Various infectivity and susceptibility

Besides the heterogeneity in agents' activity, another kind of heterogeneity is introduced when we assign various levels of infectivity and susceptibility to agents. Let f_i denote the infectivity level of agent i. f_i is a real number drawn uniformly from the interval $(0, 1)$. It measures the possibility that agent i transmits HIV viruses to others when they meets. Evidently, greater values of f_i indicate higher infectiousness of agent i.

Suppose also that each agent has resistance to being infected. We denote this resistibility as R_i for agent i. Similarly, R_i is also a real number drawn uniformly from the interval $(0, 1)$ and has the property that the greater the resistibility, the less is the chance of getting infected.

Note that the infectivity of an agent need not be a constant. An agent can have different level of infectivity, depending both on its state as well as on its behavior. It is widely believed that infectives experience two periods of high infectivity (see e.g. May and Anderson 1987;

[2]Notice in our model that resistibility and resistibility have the following relationship: resistibility $= 1 -$ susceptibility.

May et al. 1988), one shortly after being infected and the other at the late stage of his illness. Another example is that a patient might have high infectivity during the incubation period and low infectivity owing to good health care during the symptomatic period. Although our model allow for various infectivity at different stages for a single agent, we adopt the fixed infectivity for each agent. In doing so, we can focus our attention on some significant issues. We leave various infectivity scenarios for future work.

2.3.3 Extended neighborhood

Conventional CA models define two types of neighborhoods: Moore neighborhood and von Neumann neighborhood. In this paper, we extend the concept of CA neighborhood in order to better describe various situations encountered in agent-based modeling. Figure 2 illustrates the definition.

As we can see in Fig. 2b shows the classical Moore neighborhood, and Fig. 2d classical von Neumann neighborhood. Figure 2c represents an extended Moore neighborhood with the order of 2×2, and Fig. 2e an extended 2×2 von Neumann neighborhood. Specially, Fig. 2a can be simply viewed as an extended 0×0 Moore (or von Neumann) neighborhood. Note that Fig. 2b, f–i have the neighborhoods with one direction. This directional structure is able to capture the biases or preferences embedded in individuals' behavioral patterns and we can use different directions to represent variable ways of interactions. It is easy to see that the greater is the neighborhood, the larger extent to which an agent can exert its influence to its neighbors.

Given the above neighborhood definition, a concept of distance is naturally induced. Let a pair of integer numbers (x, y) represent an agent's coordinates in the grid. The distance between agent i and j is thus given as

$$d_{i,j} = \sqrt{(x_i - x_j)^2 + (y_i - y_j)^2}. \tag{2}$$

Next, we specify that the influence indicator $M_{i,j}$ of agent i and j satisfies the following condition:

$$M_{i,j} = \begin{cases} \frac{1}{d_{i,j}^2} & j \text{ in the neighborhood of } i \\ 0 & j \text{ not in the neighborhood of } i. \end{cases} \tag{3}$$

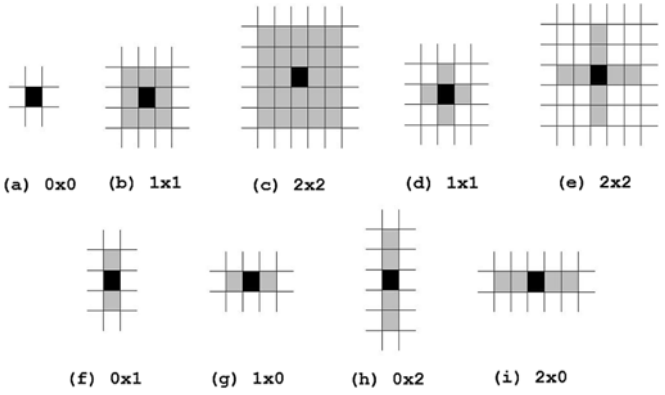

Fig. 2 The definition of neighborhood

That is to say, the influence intensity is inversely proportional to the distance between them if the influence can be exerted, and zero otherwise. Therefore, the infective impact $I_{i,j}$ of agent i on agent j can be expressed as

$$I_{i,j} = M_{i,j} f_i. \tag{4}$$

And the probability of an agent infected by one of its neighbors is defined as:

$$p_{i,j} = p(I_{i,j}, R_i), \tag{5}$$

where $p(\cdot)$ is a function satisfying the conditions: (1) $p(\cdot)$ is a real-valued function with the value between zero and one; (2) $p(\cdot)$ is increased in $I_{i,j}$ and decreased in R_i. In this model, we assume $p(\cdot)$ takes the form of the following equation:

$$p(I_{i,j}, R_i) = \sqrt{I_{i,j}(1 - R_i)}. \tag{6}$$

Here, $p(I_{i,j}, R_i)$ is interpreted as the probability of agent i being infected by its neighbor j. Denoted by B_i the set of all its neighbors, agent i's overall probability of infection is thus given by

$$p_i = \max_{j \in Bi} \{p_{i,j}\} \tag{7}$$

It indicates that this overall probability is determined by the most influential neighbor. Such specification makes sense in most cases.

3 Simulation and results

The model developed in Sect. 2 is implemented using Java programming language with the REPAST software package.[3] Detailedly commented source code is available from the authors upon request. Next, we begin our analysis by considering first a typical simulation run as a benchmark case.

3.1 Benchmark case

Table 1 lists the input parameters chosen for the benchmark case. In this case, the grid consists of 100×100 sites ($w = 100$). The population size n is set to 2000 with the initial infected ratio $\alpha = 0.005$. All agents are homogeneous in terms of having the same infectivity $f_i = 0.2$, resistibility $R_i = 0.5$, and 1×1 Moore neighborhood. They are uniformly distributed in the grid. The total simulation time T for each run is set to 2000. Figure 3 shows a snapshot of the spatial distribution of the population at some time in a typical simulation.

It is commonly believed that as the epidemic develops, its spread ends up with two typical situations: extinction or prevalence. Figure 4 depicts these two situations. In Fig. 4a, the number of infections[4] climbs early in the process. After about time $t = 470$, the infection level starts to drop slowly until it reaches zero at time $t = 2000$, while in Fig. 4b, the number of infections increases slowly and reaches an equilibrium level after $t = 1600$. The intuition

[3] See North et al. (2006) for more details.

[4] In our model, we only count the agent in S4 state as infectives due to its extraordinarily long incubation period.

Table 1 Parameters for the benchmark case

Parameters	Symbol	Value
Grid size	w	100
Initial infected ratio	α	0.005
Initial total number of agent	N_0	2000
Min activity level	$p^m_{\min}$	0.0
Max activity level	$p^m_{\max}$	0.05
Infectivity	f_i	0.2
Resistibility	R_i	0.5
Population growth rate	r	0.0001
Mean of S3 stage (T_1)	μ_1	10
Variance of S3 stage (T_1)	σ_i^2	1
Mean of S4 stage (T_2)	μ_2	500
Variance of S4 stage (T_2)	σ_2^2	100
Mean of S5 stage (T_3)	μ_3	400
Variance of S5 stage (T_3)	σ_3^2	50
Total simulation time	T	2000

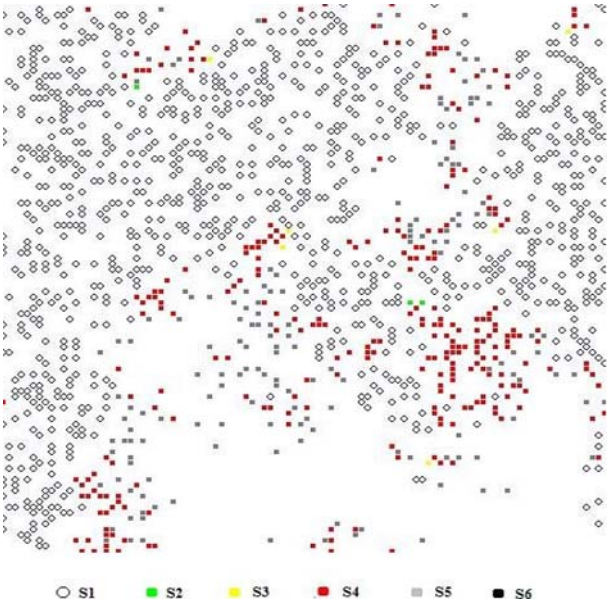

Fig. 3 The spatial distribution of the population at time $t = 1275$

behind is that in the first case, newly infected continuously enter the pool of infectives at a fairly low rate in early stages. After the lengthy incubation period, these infectives begin to develop AIDS and eventually die. The total number of them drops when the infection rate is very low with regard to the rate at which infectives leave the pool for some reason (dead in the model). While in the second case, healthy agents get infected at a relatively high rate in early stages. The infection level continues to increases because the number of removed agents is relatively small in later stages. Thus high infection rate often leads to prevalence as demonstrated in Fig. 4b. These two results can be found in real-world situations. Notice that

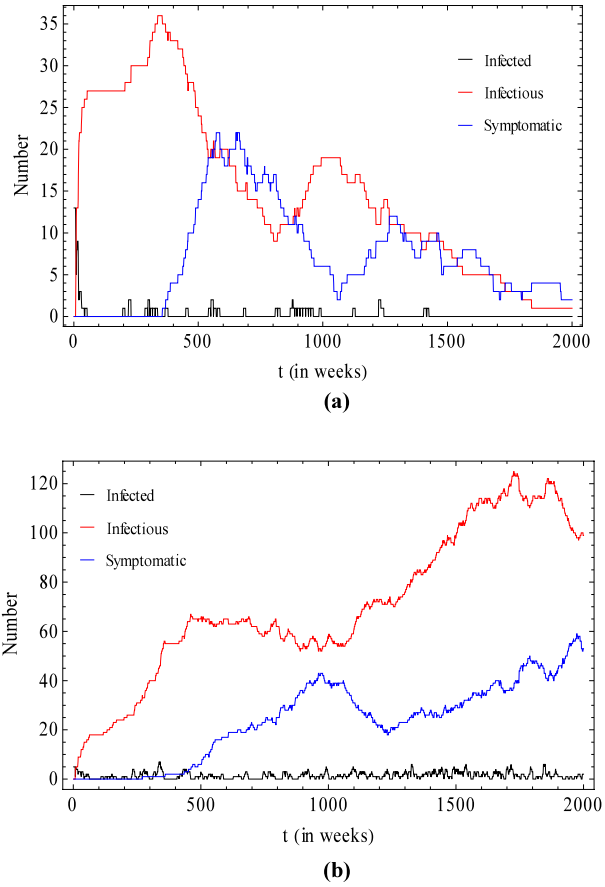

Fig. 4 The infections vs. time result in two simulations

in the parameters setting, the growth rate r is almost zero ($r = 0.0001$). In next subsection, we will explore the effects of various factors, such as population density, initial infection ratio, and infectivity, on the epidemic.

3.2 Effect of population density β

Now we keep other parameters constant as before and let the population density β vary to see how β ($\beta = n/w^2$) affects the dynamics of HIV/AIDS transmission. Tarwater and Martin (2001) investigate this issue when studying the outbreaks of measles or measles-like infectious diseases. As one would expect, many common infectious diseases spread more rapidly at a high population density than at a low population density. Figure 5 illustrates the time series of the mean numbers[5] of infectives for different population sizes $n = 1500$, 2000, 2500, 3000, and 3500. We can see that when population density is relatively low ($n = 1500$, 2000), the infection levels are relatively low during the entire simulation and decline slowly in the later stages. This suggests that the epidemic died out eventually. In the

[5]The mean number of infection is obtained by average the results of 50 simulation runs for each parameters combination. This is also applied to the subsequent simulation experiments, unless otherwise stated.

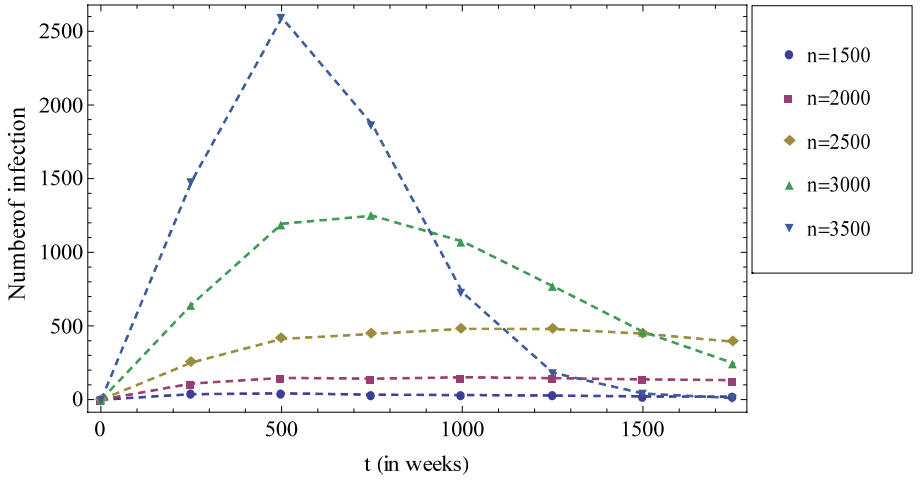

Fig. 5 Mean number of infections vs. time for various population sizes

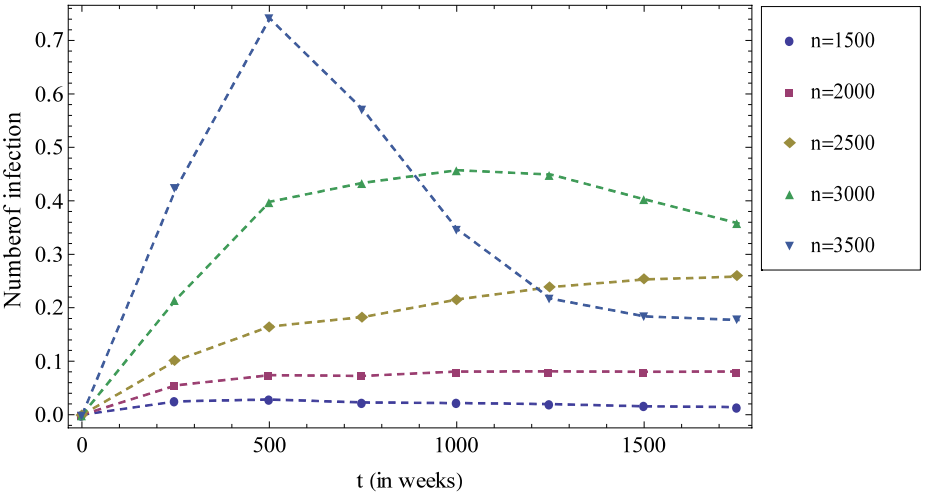

Fig. 6 Mean fractions of infections in total population vs. time for various population sizes

case of $n = 2500$, the infection level goes up at first and fluctuates in the range between 300 and 400 at last. For $n = 3000$ and 3500, the infection numbers reach to a very high level and then drop rapidly. The collapse is because that so many infectives are removed from the model that the pool of infectives shrinks. For clarity, we also plot the fractions of infectives in the population vs. time in Fig. 6. Clearly, in late stages of the epidemic, the fractions are greater when β is great than when β is small.

In summary, HIV/AIDS infection is more likely to persist at higher population densities. This is due to that with the population density increasing, the population contact rate rise, leading to increases in the probability of infection. Early work (see, e.g., Rhodes and Anderson 1996) suggests that there is a threshold below which the epidemic would eventually dies out and above which it would persist. Due to the limitation of CA methods, it is diffi-

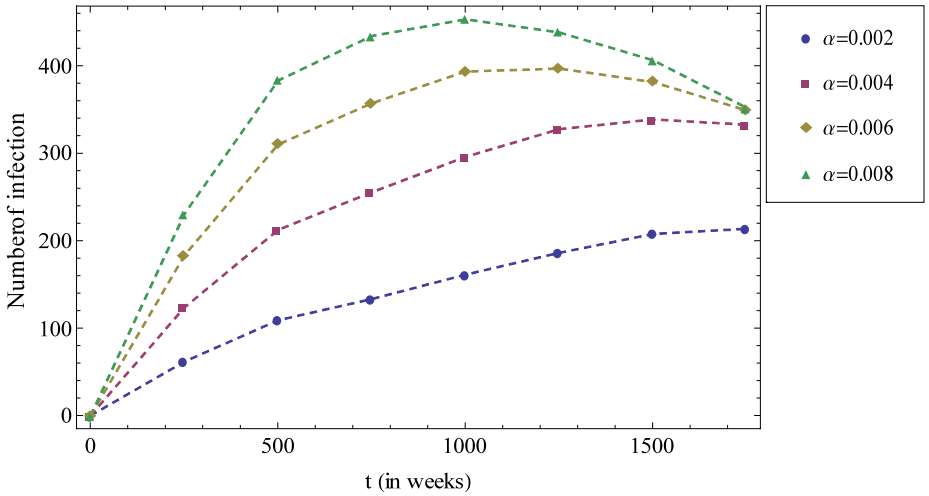

Fig. 7 Mean number of infections vs. time for different initial infected ratios

cult to pin down its exact value. However, with many simulation runs, we still can give an approximate interval in which the threshold lies.

3.3 Effect of initial infected ratio α

An interesting question one may ask is how epidemic spreading is affected by initial configurations of susceptibles and infectives, or whether the multiple infection sources will make the disease more likely to become endemic. With other parameters fixed as before, we run the simulations with $\alpha = 0.002$, 0.004, 0.006, and 0.008, respectively. Figure 7 presents the result.

Clearly, as α increase, the infection level shifts upwards. In the case of $\alpha = 0.004$, the infection level reach 300 at time $t = 2000$, higher than 200 in the case of $\alpha = 0.002$. By contrast, the level in the case of $\alpha = 0.006$ climb to around 380 at time $t = 1000$ and drops slightly to 340 at time $t = 2000$. The maximum infection is reached in the case of $\alpha = 0.008$ at time $t = 1000$, which is more than 440. Spatially, more sources of infection imply greater chance of being infected within a certain area, letting HIV/AIDS epidemics to be more likely to spread out and persist. Statistically speaking, an individual's probability of infection is generally proportional to the number of infectious sources.

3.4 Effect of mobility

Intuitively, the more migratory the population, the more likely that an epidemic is to spread. Suppose an agents' activity can be measured by the number of contacts it makes with others within a unit of period of time. As a result, our model assumes that individuals' activity is measured by mobility.

Figure 8 shows that the time evolutions of infection under the conditions of $p^m_{\min} = 0$ and $p^m_{\max} = 0.001$, 0.003, 0.005, 0.007, and 0.009. As we can see in the figure, when $p^m_{\max} = 0.001$ and 0.003, the infections increase and then decline afterwards. The infection level in former case is greater than in latter case at most of time. While in the cases of $p^m_{\max} = 0.005$ and 0.007, the two infection levels stay at two different equilibriums in

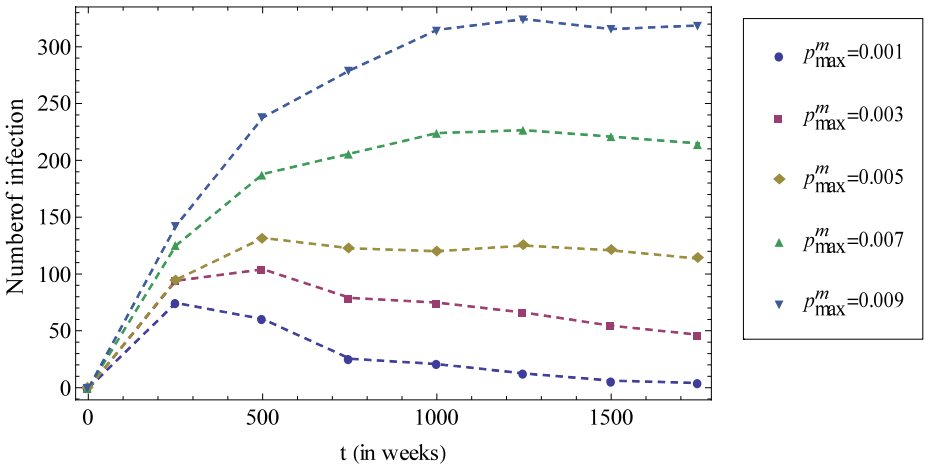

Fig. 8 Mean number of infections vs. time for various mobilities

later stages. This is, in the case of $p^m_{\max} = 0.005$, the level is above 120 and in the case $p^m_{\max} = 0.007$, the level is in the range $(180, 210)$. In the last case of $p^m_{\max} = 0.009$, the infection level fluctuates above 300, higher than those of other cases. So we conclude that mobility plays a significant role in the dynamics. It could explain why Chinese government took rather strong measures to control the migratory people and quarantine the infectives or the suspects during the outbreak of SARS in the spring of 2003. As to our model, if agents are configured with higher mobilities, it is more likely that the HIV/AIDS infection can persist in the population, whilst if configured with lower mobilities, the infection would gradually diminish and eventually die out.

3.5 Effect of forms of neighborhood

Next, we are to examine how neighborhood forms affect HIV/AIDS epidemic dynamics. The parameter sets are kept the same as in benchmark case, except for the adoption of different neighborhood forms. Figure 9 illustrates the simulation results generated in two cases: one with 1×1 von Neumann neighborhood and the other with 1×1 Moore neighborhood. In the case where the von Neumann neighborhood is used, the level of infection goes up to about 140. It is clearly greater than that of the case with 1×1 Moore neighborhood in which the level only reach about 100. Such result suggests that with wider neighborhood, an agent is more likely to get influenced by its neighbors and therefore the likelihood of getting infected increases accordingly. It is easy to induce that the infection level rises with the order of neighborhood. This result also suggests that HIV/AIDS epidemic dynamics is significantly affected by strong interactions between agents.

3.6 Four-class agents scenario

We now turn to examine heterogeneous mixing, i.e., different at-risk groups coexist. Usually, heterogeneous mixing will make the dynamics more complicated and unpredictable. In the following, we assume the whole population is divided into four different groups as shown in Table 2.

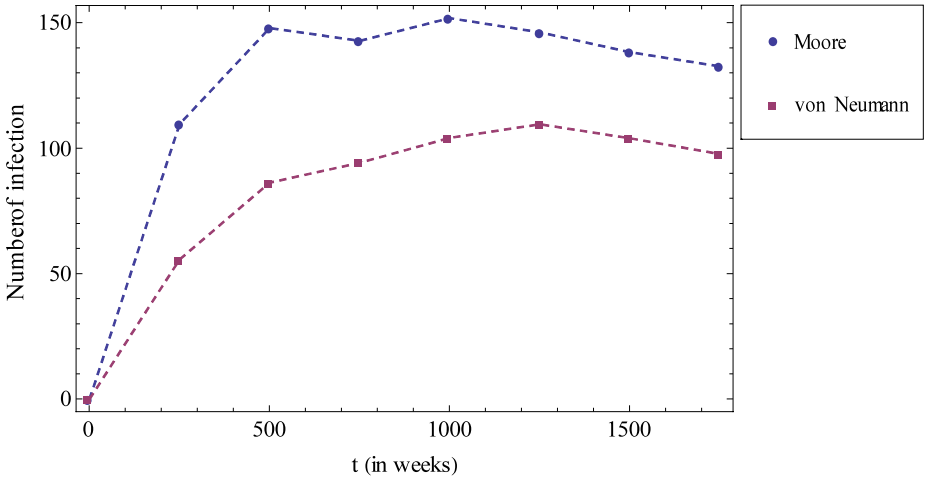

Fig. 9 Mean number of infections vs. time for two neighborhood forms

Table 2 Four classes of agents

Classes	Number	Infectivity[1]	Susceptibility	Neighborhood form
P0	A few	Very low	Low	1×1 von Neumann
PL	Many	Low	Low	1×1 Moore
PH	A few	High	High	0×1 (or 1×0) von Neumann
PH+	Very few	Very high	Very high	2×2 Moore

As shown in Table 2, Class P0 has very low infectivity, low susceptibility, and 1×1 von Neumann neighborhood. It can represents children and (or) elders in the population who hardly infect others and are easy to be infected. Class PL refers to ordinary people who have relatively low infectivity and high resistibility (therefore low susceptibility). In our model, this class amounts to a large fraction of the whole population. Class PH and Class PH+ can represent the two high-risk groups observed in real life. Agents of Class PH have high infectivity and low resistibility duo to their high-risk behaviors like incautious sex without protection, needle sharing, unhygienic blood transfusion and so on. The biased 0×1 (or 1×0) von Neumann neighborhood captures their potential oriented or biased behaviors. In contrast, Agents of Class PH+ with both higher infectivity and higher susceptibility represent those who are, although being in the minority, the most dangerous and malevolent group. Such group does exist in real life. For instance, some crimes were reported in China in recent years that a few AIDS infectives intentionally have sex with innocent people or shot people with contaminated syringes in public places. They blame their infection on the society and the government for not being able to provide necessary health service and compensating too little. The 2×2 Moore neighborhood indicates their intensive influence on others. We distinguish Class PH+ from Class PH in order to see whether such malevolent behaviors have significant impacts on the spread of HIV/AIDS and to what extent. While such rough classification may be incorrect or even erroneous, it surely is reasonable and well supported by our extended CA model. Table 3 gives the values of f_i and R_i used in the

Table 3 The resistibility and infectivity assigned to four classes

Classes	Infectivity	Resistibility
P0	$f_{P0} = 0.05$	$R_{P0} = 0.95$
PL	$f_{PL} = 0.2$	$R_{PL} = 0.50$
PH	$f_{PH} = 0.7$	$R_{PH} = 0.25$
PH+	$f_{PH+} = 0.9$	$R_{PH+} = 0.15$

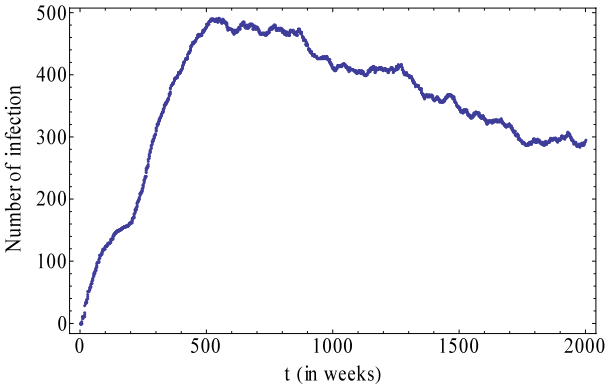

Fig. 10 Infection vs. time under four-class agent scenario

following simulations. As we will see later, the results generated with this classification are fairly consistent with those obtained through empirical work.

Figure 10 gives a typical simulation result in the four-class scenario. The fractions of four classes here are: $n_{P0} = 0.1$, $n_{PL} = 0.7$, $n_{PH} = 0.1$, and $n_{PH+} = 0.1$. The infection curve in Fig. 10 is quite similar to that obtained in the single-class scenarios except that its level is fairly higher.

3.6.1 Effect of susceptibility (or resistibility)

In this section, we will investigate the effect of agents' susceptibility on the dynamics of HIV/AIDS. Given $n_{P0} = 0.05$, $n_{PL} = 0.8$, $n_{PH} = 0.1$, $n_{PH+} = 0.05$ and others as before, Let R_{PH+} vary. Figure 11 gives the infection levels when R_{PH+} is set to be 0.6, 0.7, 0.8, and 0.9, respectively. As we can see, these equilibrium infections are almost at the same level, which is inconsistent with our expectation. The differences are so small that we cannot assure with confidence whether changes in susceptibility have impact on the epidemic dynamics. The possible reasons, we believe, are twofold: First, the role played by susceptibility may be not as decisive as the above factors. Second, we may describe susceptibility in the wrong way and make it an inessential factor in our model. Future work will reconsider this issue and find the better way to describe susceptibility.

3.6.2 Effect of changes in the fractions of four classes

At last, we will examine whether changes in the fractions of some classes can affect epidemic behaviors. Given $n_{P0} = 0.1$, $n_{PL} = 0.7$ and other parameters as before, we take $n_{PH} = 0.12$ ($n_{PH+} = 0.08$), 0.14 (0.06), 0.16 (0.04), 0.18 (0.02), 0.20 (0.0), respectively. Figure 12 shows the infection levels in these five combinations.

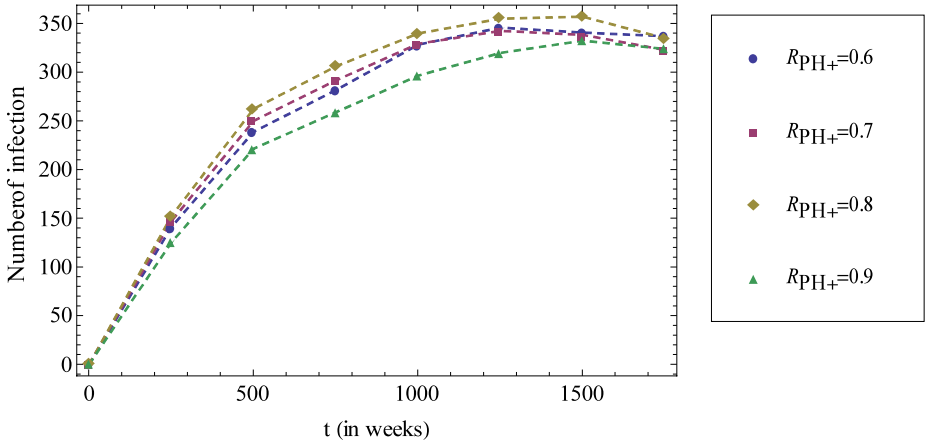

Fig. 11 Mean number of infection with different individual susceptibility

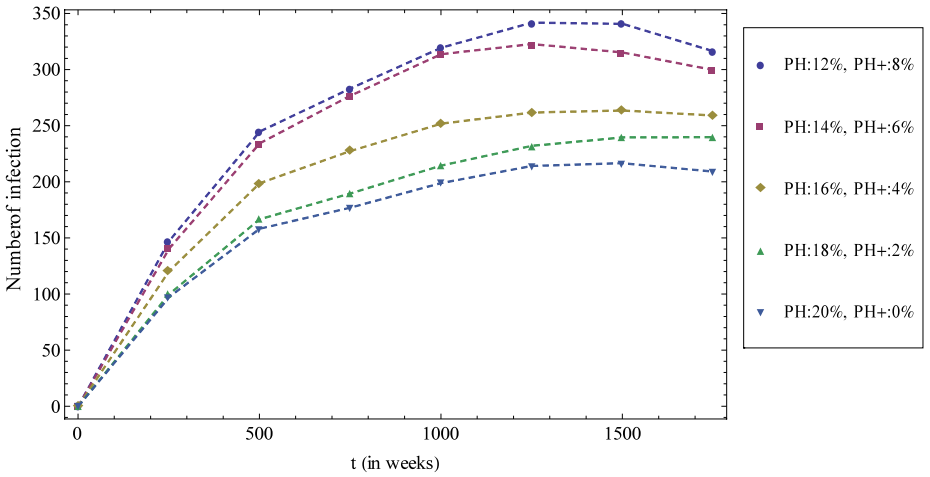

Fig. 12 Mean number of infection vs. time for various fraction of Class PH+

As observed from Fig. 12, we obtained very similar results, compared with those in the foregoing analysis. With n_{PH+} decreasing, infection level declines. Recall that PH+ has more influence on its neighbors than PH, thus leading to greater transmission to others and larger infection rate. This finding suggests the government should pay more attention to those who have high-risk life styles and revengeful behaviors. This makes it the essential issue of how highly infectious and malevolent individuals are restricted and controlled.

4 Conclusions

The focus of the paper is on the modeling of the entire course of HIV/ACID epidemics and heterogeneity in agents' behaviors. Even though classical CA models are capable of describing the spread of common epidemics but fail to represent the complicated epidemics

like HIV/AIDS disease. The ignorance of heterogeneity gives rise to unacceptable errors in the prediction of the development trends. In addition, components of a conventional CA system such as topological forms of grid, the definition of neighborhood, and state transition rules are simple and unchanged over time. This makes the modeling of complicated dynamics such as HIV/AIDS transmission difficult and uncontrollable. In this paper, we have developed an extended CA model to capture key epidemiological and clinical features of HIV/AIDS epidemic. First, We explicitly models and simulates the whole progression of HIV/AIDS disease (i.e., infected but not infectious, infectious but asymptomatic, symptomatic, and deceased). Such improvement can give us a better understanding of the dynamics during the entire HIV/AIDS epidemics. In order to examine various degrees of influence between agents, we have introduced an extended definition of neighborhood to represent the intensity and bias of influence. This lets us gain insight into how various degrees of interactions affects the HIV/AIDS epidemics. Another type of heterogeneity in disease-related attributes such as susceptibility, infectivity and durations of epidemic phrases is also taken into consideration. Moreover, we also consider the effect of agents' mobility on epidemics dynamics.

Given all the improvements, we have obtained richer simulation results similar to those usually found in the mathematical models or other classical simulation models. We have identified some influential factors that greatly affect the HIV/AIDS epidemic dynamics. The main findings are that 1) HIV/AIDS epidemic can end up in the two regimes: extinction and persistence; 2) with these factors such as agents' mobility, population density, initial infection ratio, and the extent of neighborhood increasing, the infection level get higher. After crossing some critical point, the regime generated could change from dying-out to persistence at some point. This result is robust across many of the tested parameter combinations; 3) in four-class scenarios, the great fraction of 'super' infectives (the PH+ class in our model) can also produce higher level of infection.

However, our simulation study above is still preliminary. There are some issues needed to be addressed. First, as said before, we should redefine susceptibility in a better way to check its role in the dynamics of HIV/AIDS epidemic. Second, most models posit that a virus carrier's infectivity is constant during the progress of a disease. However, this is not the case for HIV/AIDS epidemic. Various infectivity at different stages could have substantial impact on the dynamics of HIV/AIDS transmission. This problem needs special attention. Additional, Further developments of our model, e.g. by adding age-related structure (Griffiths et al. 2000), different subgroup classification, and other heterogeneity, would greatly add to the appeal of this model. With these additions, a better understanding of HIV/AIDS and thorough empirical work are required. Finally, a natural extension of the model is to include the assessment of various control policies and managerial strategies, and this will be a firm support for the decision-making in prevention programs against HIV/AIDS.

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