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A Computer Model for Predicting AIDS Among Intravenous Drug Abusers

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Abstract

Intravenous drug abuse (IVDA) is an important cause of HIV transmission. Computer simulation is one way to understand and predict the spread of HIV infection among IVDAs. We design and simulate HIV infection among IVDAs and the impact of AIDS on this community, and thereby predict future IVDA population, HIV levels, AIDS levels, and AIDS deaths in this group. The HIV to AIDS, and AIDS to Death latencies are described by probability density functions (PDFs) in this model. Factors such as the recruit, quit, and normal death rate of IVDAs, are considered, as well as the infection and removal rates for HIV and AIDS. All these PDFs and rates can be accessed by the user interactively. The impacts of these factors on the IVDA, HIV, and AIDS populations are demonstrated and compared. Discussion of the factors impacting the infection rate provides medical policy makers with useful information.

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Introduction

Intravenous drug abuse (IVDA) is an important source of HIV transmission. Computer simulation is one good way to analyze and predict the IVDA, HIV, and AIDS populations. There are deterministic models and stochastic ones. Some of them employ sophisticated mathematics and many variables and coefficients. However, due to the complicated human behaviors involved in IVDA it is difficult to accurately describe and predict HIV transmission in the IVDA population. Still, computer simulation provides an efficient way to predict trends, and by research on the factors that affect the disease transmission, can provide helpful information to policy makers in health services and education.

Model Description

Consider IVDA as a community consisting of HIV infected and uninfected populations. IVDA recruits are uninfected but susceptible immediately after they enter the community. Members who are infected with HIV will develop AIDS with a latency described by a certain probability density function (PDF). An AIDS patient will die after another PDF-described latency. Members leave the community because of death, quitting, showing AIDS symptoms, or being isolated for other reasons. We assume that no IVDA exists outside the community, no HIV transmission occurs outside the community, and all AIDS cases are removed from the community immediately. However, HIV infected individuals outside the com-

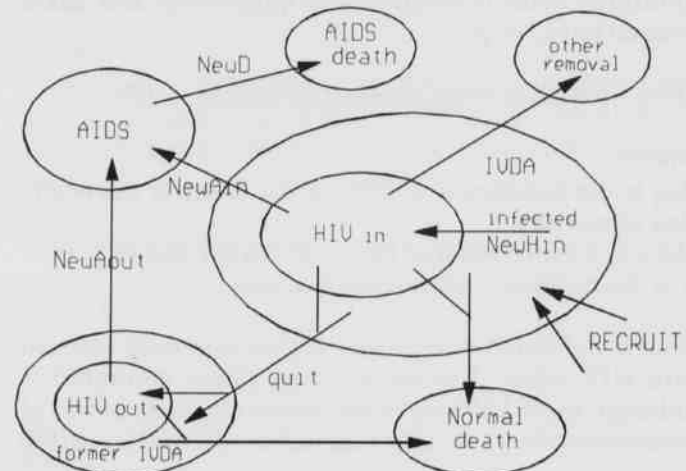munity will still develop AIDS eventually. The model in graphical form appears in Fig. 1.

Fig. 1.

Some important characteristics of this model are as follows:
*This is a dynamic model.
*Latencies are defined by PDFs.
*People remain infectious until removed due to AIDS or other reasons.
*No recovery from HIV and AIDS occurs.

Mathematical Model

A dynamic model of the IVDA population is described by Caulkins and Kaplan (1991). That model

considers the recruit and quit rates, giving:

$$dI/dt = c*I^v - \mu*I \quad ---(1)$$

where I is the IVDA population, c and v are the recruit coefficients and μ is the quit rate. We augment this formula by taking two additional factors into consideration: the normal death rate for the IVDA population and the removal of AIDS victims from this population. Then, we have:

$$\Delta I_j = c*I_j^v - (\mu+n)*I_j - NewAin_j \quad ---(2)$$

Where:

I_j is the IVDA population at the beginning of month j.
ΔI_j is the increment in I in month j.
$NewAin_j$ is the AIDS population that is removed from the IVDA population.
c, v are IVDA recruit coefficients.
μ is the IVDA quit rate.
n is the normal death rate.

An epidemiological model is described by Bailey (1957) which models infection rate as proportional to the product of infected and susceptible populations. With the additional consideration of the quit, death and AIDS removal rates, we get:

$$\Delta Hin_j = \alpha*(I_j - Hin_j)*Hin_j - (\mu+n)*Hin_j - NewAin_j \quad ---(3)$$

Where:

Hin_j is the population of HIV in the IVDA at the beginning of month j.
ΔHin_j is the increment of HIV in IVDA for month j.
α is the coefficient of the infection rate.

Hout is the number of former IVDAs who were infected with HIV while they were in the IVDA community. Although these HIVs no longer infect others by means of intravenous drug use, they themselves will develop AIDS:

$$\Delta Hout_j = \mu*Hin_j - n*Hout_j - NewAout_j \quad ---(4)$$

Where:

$Hout_j$ is the population of HIV outside IVDA for the month j.
$\Delta Hout_j$ is the increment in Hout for the month j.
$NewAout_j$ is the number of individuals in $Hout_{j-1}$ who developed AIDS in the past month.

The accumulated AIDS deaths:

$$AD_j = AD_{j-1} + NewD_j \quad ---(5)$$

Where:

AD_j is the accumulated AIDS deaths by the month j.
$NewD_j$ is the AIDS deaths in the month j.
NewAin, NewAout, NewD are associated with probability density functions (PDFs) describing latencies. Suppose the PDF of Fig. 2 describes how the HIVs who are infected in month j are going to develop AIDS over time.
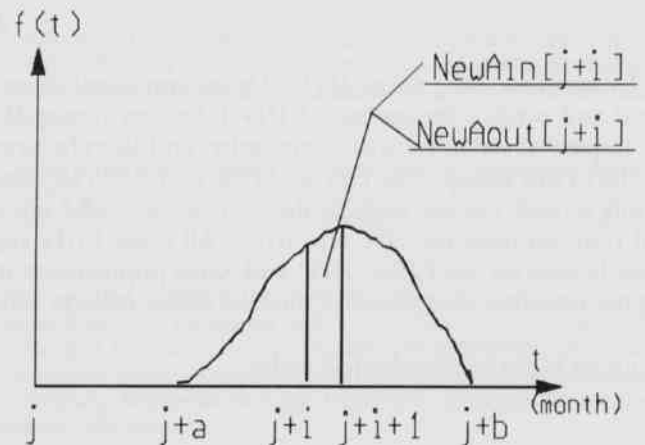

Fig. 2.

Let the number of new HIV cases in month j be called $NewHIV_j$. These HIVs will develop AIDS in the next a to b months according to above PDF. Then this group of HIV will contribute:

$$(\int_i^{i+1} f(t)dt)*NewHIV_j$$

to the newly developed AIDS cases for the month j+i, for a<i≤b.
The NewHIV is calculated in accordance with the first term of equation (3) as:

$$NewHIV_j = \alpha*(I_j - Hin_j)*Hin_j \quad ---(6)$$

However, as HIVs in the IVDA population keep quitting at rate μ, and dying at rate n, in month j+i:

$$\Delta NewAin_{j+i} = (1-\mu-n)^i*(\int_i^{i+1} f(t)dt)*NewHIV_j \quad ---(7)$$

and

$$\Delta NewAout_{j+i} = ((1-n)^i - (1-\mu-n)^i)*(\int_i^{i+1} f(t)dt)*NewHIV_j \quad --(8)$$

Of course, equations (7) and (8) only show those new AIDS cases in month j+i arising from those individuals who caught HIV in a previous month j. The total NewAin and NewAout in a month, say month p, are accumulated populations contributed by the $NewHIV_q$'s where p-b=<q<p-a, then:

$$NewAin_p = \sum_{q=p-b}^{p-a-1} ((1-\mu-n)^{p-q} * (\int_{p-q}^{p-q+1} * f(t)dt) * NewHIV_q) \quad ---(9)$$

$$NewAout_p = \sum_{q=p-b}^{p-a-1} (((1-n)^{p-q} - (1-\mu-n)^{p-q}) * (\int_{p-q}^{p-q+1} f(t)dt) * NewHIV_q)$$
$$---(10)$$

The sum of New Ain$_p$ and NewAout$_p$ gives the total AIDS developed in month p. Similarly, assuming an AIDS onset to AIDS death latency PDF g(t) and ignoring normal death rate because AIDS death latency is relatively short, we have:

$$\Delta NewD_{j+i} = (\int_i^{i+1} g(t)dt) * NewA_j \quad ---(11)$$

where a1=<i<b1, and
$NewA_j = NewAin_j + NewAout_j$
Then the total AIDS deaths in month p is:

$$NewD_p = \sum_{q=p-b1}^{p-a1-1} ((\int_{p-q}^{p-q+1} g(t)dt) * NewA_q) \quad ---(12)$$

From the above discussion, we have built a mathematical model using functions (2), (3), (4), (5), (6), (9), (10), and (12).

The Coefficients

v, μ, n AND c

We choose v=0.5 for the following reasons: v<0 is not meaningful; v>0 because the IVDA population does impact recruitment IVDAs; v<1 because the more the IVDAs, the more the IVDA recruits, but we can not expect that the recruits increases as fast or faster than the number of IVDAs. To compromise, we choose v=0.5. (Later we will see that v has a considerable affect on the model output, thus uncertainity in v should be handled in later work.)

Caulkins and Kaplan (1991) infer μ=0.12, from quit and IVDA population data in years for which such data are available. We therefore use this value.

Berleant et al. (1992) infer the AIDS death rate from the reciprocal of an AIDS survival interval. Similarly, we define the normal death rate as the reciprocal of life expectancy. Assuming a lifetime of 80 years, we get a normal death rate of 1/80. Although IVDAs die faster than average for many causes of death, this is not modeled here. This should probably be modeled in furture work, or at least a sensitivity analysis done to see if it should be modeled.

Assume that before the introduction of AIDS, the size of the IVDA population is approximately constant [Caulkins and Kaplan (1991) make the same assumption],

and the incidence of AIDS is zero. Equation (2) then reduces to:

$$\Delta Is = 0 = c * Is^v - (\mu+n) * Is \quad ---(13)$$

Then:

$$c = (\mu+n) * Is / Is^v = (\mu+n) * Is^{1-v} \quad ---(14)$$

ALPHA
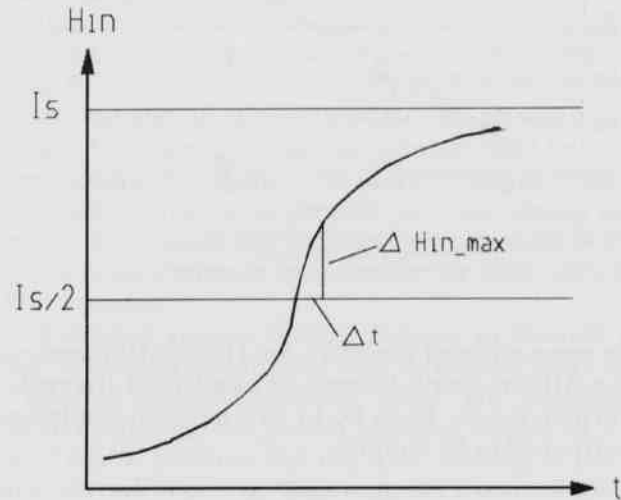

Fig. 3.

As shown in Fig. 3, Hin=α*(Is-H)*H is a logistic curve with the maximum value of (dHin/dt) occuring at Hin=Is/2. Let this dHin/dt equal δ. Then, from equation (6):

$$a * (Is - \frac{Is}{2}) * (\frac{Is}{2}) = \delta * Is$$

which gives:

$$\alpha = 4 * \frac{\delta}{Is}$$

or:

$$\delta = a * \frac{Is}{4}$$

δ is the fraction of Is which is infected with HIV in a unit time (1 month) when the highest infection rate occurs (when Hin=I/s./2). δ is more meaningful than α because δ is independent of Is while α does depend on Is. Therefore, the use of δ makes estimating the infection rate easier. Using δ instead of α in (3) gives:

$$\Delta Hin_j = (4 * \frac{\delta}{Is}) * (I_j - Hin_j) * Hin_j - (\mu+n) * Hin_j - NewAin_j \quad ---(3')$$

We can find the best value for δ by simulating equation (3') for various values of δ, and comparing the resulted trajectories with known historical data. The best value

of δ is that value used in the simulation whose results best match historical data. The best δ we found is 0.24. Results of simulation of this model are demonstrated in Fig. 4.
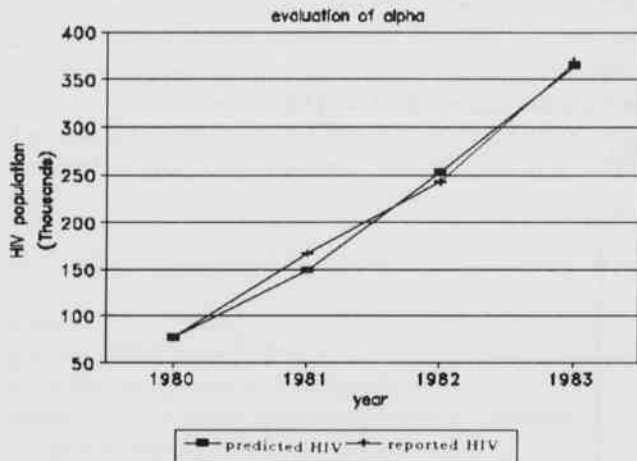

Fig. 4.

PDFS

We approximated the PDFs for HIV-to-AIDS latency, and for AIDS-to-death latency, as equilateral triangels. The former ranges from 60 to 140 months, the latter ranges from 12 to 36 months.
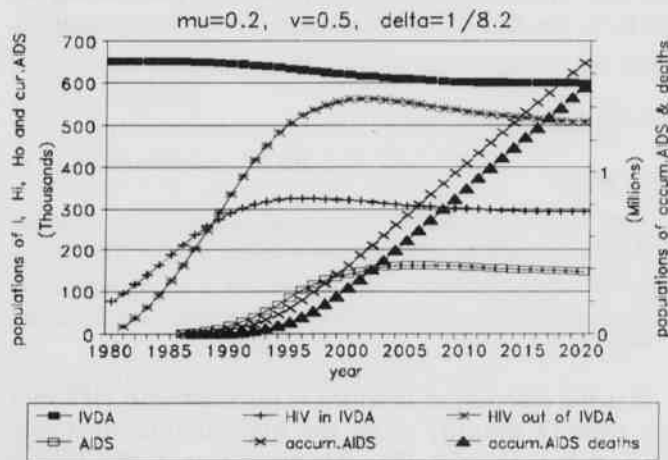
Results

IVDA, HIV, and AIDS PREDICTIONS

Fig. 5.

In Fig. 5 simulation starts in 1980. IVDA population remains essentially constant until 1988. The fastest HIV increase in IVDA happens when IVDA population is steady and at its highest value. As the susceptible population size decreases, HIV infection slows down. There would be more HIV infection if not for AIDS removal, which first occurs in large numbers in 1987, when HIV in IVDA reaches its estimated maximum of 0.24 million.

HIV among former IVDAs follows this trend but more slowly and mildle. AIDS population reaches its maximum of 0.2 million in 2005 and decreases thereafter. By the year 2005, IVDA, HIV and AIDS will be close to their asymptotic equilibria.
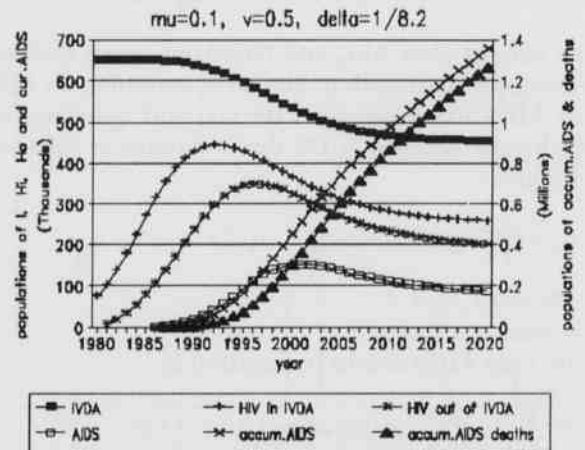
IMPACT OF μ, v AND δ

Fig. 6.

In Fig. 6 the quit rate μ is larger. Notice that the HIV outside the IVDA community (who were once inside and are out and inactive now because of quitting or removal by other reasons) has almost an equal population size (or even larger) compared to that of the HIV in IVDA. This is reasonable when the HIVs quit very quickly leading to large buildup of HIV positives among former IVDAs. Further, because relatively few HIV cases remain IVDAs long enough to be removed by developing AIDS, IVDA population is little changed.

In Fig. 7, v=1.0 which means the recruitment rate is more sensitive to the IVDA population. Therefore, as the IVDA decreases, the recruitment decreases faster (compared with the example in Fig. 5). Lower recruitment leads to fewer IVDAs which causes even lower recruitment and so on. By the year 2055, IVDA decreases to about 1/2 of its initial level and the HIV and AIDS
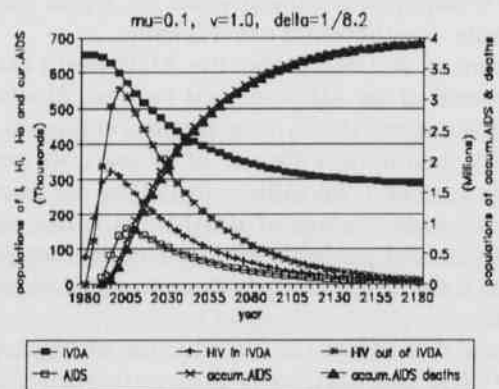

Fig. 7.

decrease asymptotically toward zero.

Figure 8 shows the impact of δ on the result. A higher δ means a higher infection rate. Compared with the corresponding curves in Fig. 5, while the steady state values of IVDA, HIV and AIDS are almost the same, the population of HIV in IVDA increases faster and has a higher maximum value. So does the AIDS population. The reason for the faster increases in HIV and AIDS population is straightforward, and the reason for higher maximum values is the HIV spreads faster and infects more IVDAs before they quit, thus populations of HIV both in and out of IVDA become larger causing higher maximum populations of HIV and AIDS.
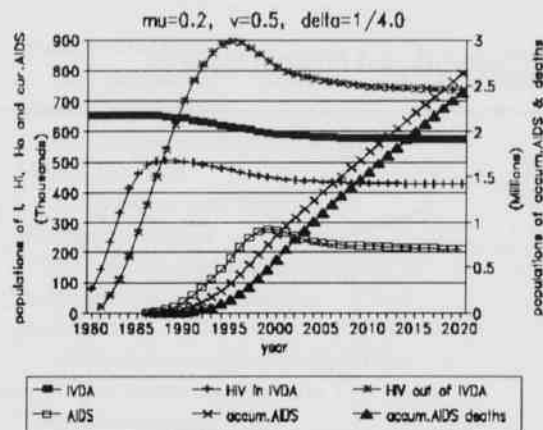

Fig. 8.

Conclusion

It is well known that in the intravenous drug abuse community, HIV spreads easily and quickly. Our results are consistent with that observation. Our results also bear out the conclusion of Caulkins and Kaplan (1991) that AIDS will significantly reduce the IVDA population. However, a critical observation is the apparent fact that the size of the IVDA population will not only remain substantial but will function as reservoir of infection: turnover in this population, and sexual interactions flow between population members and nonmembers, mean a continuous flow of HIV infection from this reservoir to the rest of society. Thus, understanding and dealing with the IVDA/HIV problem is of great social importance.

It is interesting to step back and look at the IVDA/HIV problem from a larger perspective. The HIV virus is known to mutate rapidly. Recent epidemiological work indicates that evolutionary pressures act on pathogens make them more virulent when opportunities to spread are plentiful Ewald (1993). Since needle sharing involves exchange of possible infected blood, it is a highly infective way of spreading HIV (Kaplan, 1989). This suggests that evolutionary pressures facing the HIV virus in the IVDA community will cause it to remain quite viru-

lent or become even more so. Virulence may be measured by the latency between initial infection and AIDS symptoms, with shorter latencies indicating greater virulence. Indeed, it has been found that the virulence of HIV among intravenous drug abusers has continued to be high, whereas among American homosexuals the virus has been becoming less virulent presumably as an adaptive strategy by the virus due to less risky practices among individuals in that group brought about by awareness concerning HIV (Ewald, 1993). Therefore, the IVDA community may serve not only as a reservior of HIV infection, but as a reservoir of particularly virulent HIV virus. In order to reduce not only HIV incidence among IVDAs but also to remove evolutionary pressure on the virus to be virulent, changes in behavior must be encouraged that will reduce the ease of spreading, analogous to the situation that has apparently occurred among homosexuals. Thus proposals like needle exchanging in which addicts could exchange used needles for clean ones merit close attention.

Ecological systems tend to increase in diversity over time. Ecological systems also tend to become more stable as diversity increases (Logofet, 1993). The mutability of the HIV virus and the known presence already of a variety of strains indicates that the virus' diversity is not only high but will increase. This suggests that humankind's relation with the HIV is, unfortunately, a "stable" one and therefore that the HIV problem will likely remain with us into the indefinite future.

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