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THE SPREAD AND QUARANTINE OF HIV INFECTION IN A PRISON SYSTEM*

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Abstract. This paper is concerned with models for the spread of HIV in prisons. A simple deterministic model for the spread of HIV in a single prison is considered, a discrete-time stochastic analogue of it is then discussed, and some simulations of epidemics are provided. The model is generalized to a two-prison system in both the deterministic and stochastic cases. Finally the possibilities for intervention in a prison are outlined and a simple cost-effectiveness analysis in the context of screening and quarantine is undertaken. The computations for the stochastic models make use of a new technique for bounding variability in a Markov chain with a large state space.

Key words. prison, HIV infection, deterministic and stochastic epidemic models, quarantine, error bounding for Markov chains

AMS subject classifications. 92D30, 60J20, 62C12

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1. Introduction.

One of the serious problems of prison life is the spread of HIV by both sexual contacts and needle sharing activities among prisoners (see Brewer et al. (1988) and Horsburgh et al. (1990)). Decker and Rosenfield (1992) point out that in 1990, 14 million people passed through U.S. jails, of which a sizable fraction indulged in drug use and sexual practices placing them at high risk for AIDS. A similar situation is known to exist in many European countries and Australia. Anonymous (1993) reports that about 70% of jailed detainees test positive for injected drug use, and New York state reports an HIV infection rate of 17.4% among its prisoners. The publication cited as U.S. Department of Justice (1993, p. 15) lists that between November 1992 and March 1993, 11,500 AIDS cases were reported in Federal prisons, and the number of new cases has been growing at approximately 50% a year for the past several years. The book edited by Thomas and Moerings (1994) gives reports from a number of other countries, and from this literature, one sees that HIV/AIDS in prison systems is of worldwide concern. A special report in the *New York Times* (Sims (1996)) notes that a recent study by the World Health Organization estimates the number of HIV infectives in Argentine federal prisons to be of the order of 30% of all inmates and suggests that local and rural prisons have similar prevalences, as do prisons in other Latin American countries, including Brazil.

Education is an important weapon in combating the AIDS epidemic, and nowhere more so than in prisons. Magura, Rosenblum, and Herman (1991) and Stevens (1993) found that intravenous drug users were receptive to counseling in jails. But counseling needs to be based on facts, and these are not readily available, particularly as HIV screening of inmates is not widely or uniformly practiced. Blumberg and Langston (1991) have discussed various proposals for mandatory HIV testing, while Hammett (1989) suggested that voluntary testing and AIDS education would prove acceptable to the majority of inmates. If HIV+ individuals report their status, medical care is

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available, but some harassment may ensue; if they do not, they will not receive medical help (Siegal et al. (1993)). Hsieh (1992) has investigated a differential equation model which incorporates screening and quarantine of infectives. An objective in his study is to find the screening level needed to make the reproductive rate of HIV infection fall below 1.

We need to understand the situation better and to direct the attention of medical and prison authorities to those data which must be collected in order to map the spread of HIV in prisons accurately. In order to do this, we present some models, both deterministic (section 2) and stochastic (section 3), which attempt to describe the HIV epidemic in a prison or a set of prisons (section 4). In section 5, we extend the model to allow for mandatory HIV screening and the possibility of quarantine of those prisoners thereby found to be seropositive. A decision problem arises through these considerations: what level of screening is appropriate? In an economic sense, the answer depends on the length of decision horizon. Screening and quarantine cost money, and the expense is valid only in terms of preventing further spread of infection. If the time horizon is short, the expense may be difficult to justify. On the other hand, in the long run, quarantine may be worthwhile. At the close of section 5, hypothetical costs are posed, and a simple decision analysis is undertaken as a guide to techniques involved in determining the cost-effectiveness of screening and quarantine policies.

Stochastic processes have not been widely embraced by the AIDS modeling community, although epidemics are patently random. This reluctance stems in part from the fact that it is difficult to get analytic results of interest; further, if the population is at all sizable, aside from brute force and sometimes inconclusive simulation, stochastic models and their associated large Markov transition matrices are troublesome or even intractable. Methodologically, we proceed by solving deterministic counterparts of the stochastic processes and then use concepts recently developed elsewhere (Gani and Yakowitz (1995)) to assess the effects of the random variability.

2. A deterministic model. Let us assume that a prison consisting of N inmates is subject at time t $(t = 0, 1, 2, \ldots)$ to a simultaneous inflow and outflow of $n < N$ prisoners, after which there are at this time $y(t)$ prisoners who are HIV+ and $N - y(t)$ susceptibles. During the interval $(t, t+1)$, let homogeneous mixing occur in the prison with an infection rate $\beta = \frac{\gamma}{N-1}$, so that $\beta y(t)\,(N - y(t))$ new infectives are produced, and at $t+1$ there are

$$z(t+1) = y(t) + \beta y(t)\,(N - y(t)) \tag{2.1}$$

infectives.

Let an inflow of n new prisoners join the prison at time $t+1$ from the outside world, where the proportion of HIV+ is $0 \le \mu \le 1$, so that

$$u(t+1) = n\mu$$

infectives are added. Simultaneously, an outflow of n prisoners leaves the prison of which

$$v(t+1) = \frac{n}{N} z(t+1) = \frac{n}{N}\left(y(t)(1 + \beta N) - \beta y^2(t)\right) \tag{2.2}$$

are infectives. Thus

$$\begin{aligned} y(t+1) &= u(t+1) + z(t+1) - v(t+1) \\ &= n\mu + y(t)\,(1 + \beta N - \beta y(t))\left(1 - \frac{n}{N}\right) \end{aligned} \tag{2.3}$$

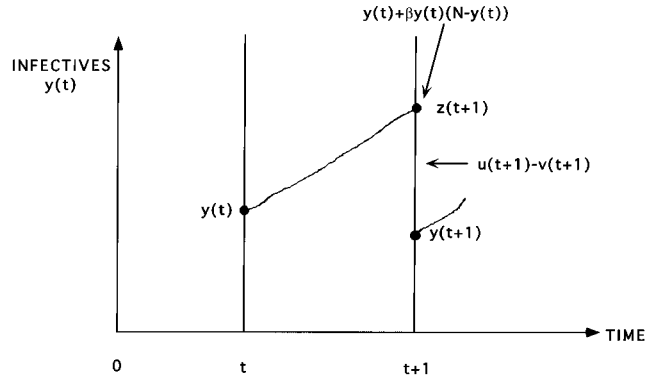


FIG. 1. *Infectives at times $t, t+1$ in prison.*

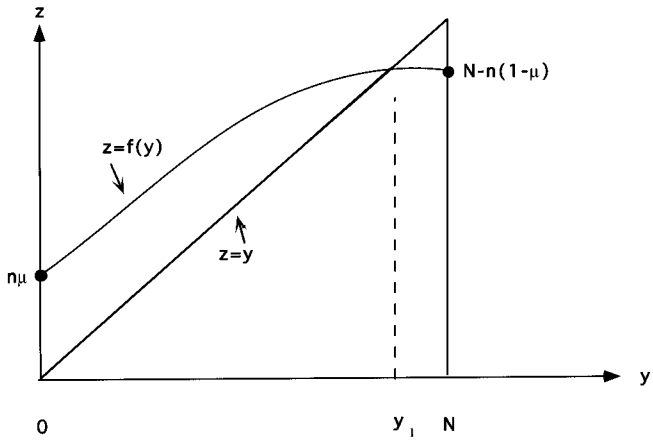


FIG. 2. *Graphs $z = f(y)$ and $z = y$.*

expresses the number $y(t+1)$ of HIV+ prisoners at time $t+1$ in terms of $y(t)$, those at time t. Figure 1 illustrates these events and variables in an epoch.

A stable point exists at y_1, where this is the positive solution of

$$n\mu + y\left(\beta(N-n) - \frac{n}{N}\right) - \beta\left(1 - \frac{n}{N}\right)y^2 = 0;$$

namely,

$$y_1 = \frac{1}{2}\left\{\frac{N\beta(N-n)-n}{\beta(N-n)} + \sqrt{\left[\frac{N\beta(N-n)-n}{\beta(N-n)}\right]^2 + \frac{4Nn\mu}{\beta(N-n)}}\right\}$$

$$= \frac{1}{2}\left\{N - \frac{n}{\beta N\left(1-\frac{n}{N}\right)} + \sqrt{\left(N - \frac{n}{\beta N\left(1-\frac{n}{N}\right)}\right)^2 + \frac{4n\mu}{\beta\left(1-\frac{n}{N}\right)}}\right\} \tag{2.4}$$

with $0 < y_1 < N$ if $\mu < 1$. This can be readily seen from Figure 2 showing the parabola $z = f(y) = n\mu + y\left(1-\frac{n}{N}\right)(1+\beta N) - \left(1-\frac{n}{N}\right)\beta y^2$, where $f(0) = n\mu$ and $f(N) = N - n(1-\mu) < N$ if $\mu < 1$.

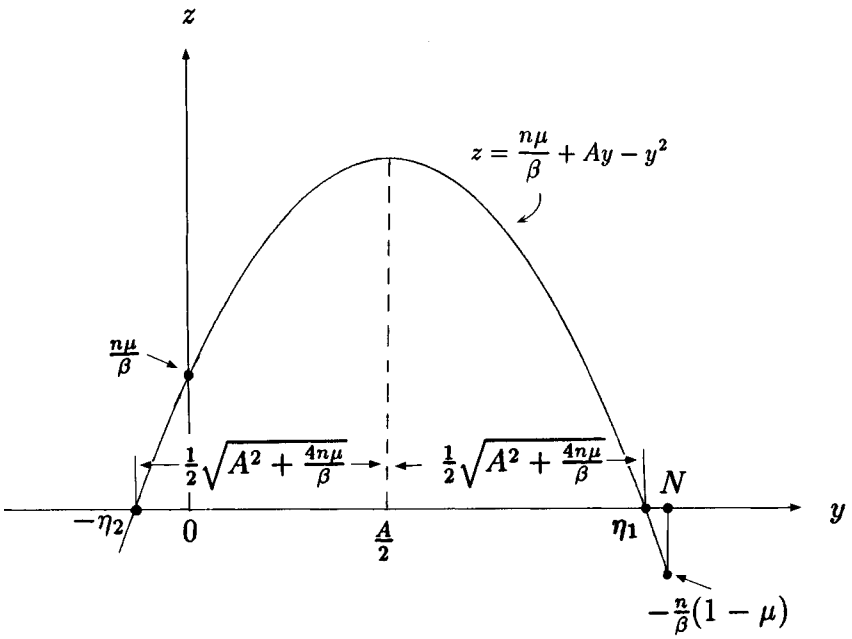


FIG. 3. *Roots* η_1, η_2.

While the discrete version (2.3) of the model lends itself readily to computation, explicit solutions can be found more easily for its continuous time analogue

$$\begin{aligned}\frac{dy}{dt} &= n\mu - n\left(\frac{y(t)}{N}\right) + \beta y(t)\,(N - y(t)) \\ &= n\mu + \beta y(t)\left(N - \frac{n}{\beta N} - y(t)\right) \\ &= \beta\left(\frac{n\mu}{\beta} + Ay(t) - y^2(t)\right) \\ &= \beta\,(\eta_1 - y(t))\,(\eta_2 + y(t))\,. \qquad (2.5)\end{aligned}$$

with $A = N - \frac{n}{\beta N}$. Here,

$$\begin{aligned}\eta_1 &= \frac{1}{2}\left\{A + \sqrt{A^2 + \frac{4n\mu}{\beta}}\right\}, \\ -\eta_2 &= \frac{1}{2}\left\{A - \sqrt{A^2 + \frac{4n\mu}{\beta}}\right\}, \qquad (2.6)\end{aligned}$$

where η_1 plays the same role as y_1 of (2.4). Figure 3 illustrates the values (2.6) when $\mu < 1$; we have $-\eta_2 < 0 < \eta_1 < N$. We now need to solve (2.5) or

$$\frac{dy}{\eta_1 + \eta_2}\left(\frac{1}{\eta_1 - y} + \frac{1}{\eta_2 + y}\right) = \beta dt,$$

which yields

$$y(t) = \frac{\eta_1 K e^{\beta(\eta_1+\eta_2)t} - \eta_2}{1 + K e^{\beta(\eta_1+\eta_2)t}}. \qquad (2.7)$$

If the initial value at time $t = 0$ of the number of infectives is $y_0 > 0$, then

$$K = \frac{\eta_2 + y_0}{\eta_1 - y_0} \gtreqless 0 \text{ for } y_0 \lesseqgtr \eta_1,$$

and $y(t)$ in (2.7) is fully defined. Note that as $t \to \infty$, $\lim_{t\to\infty} y(t) = \eta_1$, a stable value similar to (2.4) for the discrete-time case with $\beta\left(1 - \frac{n}{N}\right)$ replaced by β. We illustrate the behavior of $y(t)$ by some examples.

Example 1. Case of $A = N - \frac{n}{\beta N} > 0$. Let $N = 500$, $n = 10$, $\mu = 0.1$, $\beta = \frac{\gamma}{N-1} = 0.000300$. Then

$$A = 500 - \frac{10}{0.15} = 433.3333,$$
$$\eta_1 = 440.8937 \eta_2 = 7.5604.$$

If $y_0 = 100 < \eta_1$, say, then

$$K = 0.3155 > 0$$

and

$$y(t) = \frac{139.1129 e^{0.1345t} - 7.5604}{1 + 0.3155 e^{0.1345t}}.$$

If, however, $y_0 = 450 > \eta_1$, then

$$K = -50.2468 < 0$$

and

$$y(t) = \frac{-22153.4838 e^{0.1345t} - 7.5604}{1 - 50.2468 e^{0.1345t}}.$$

In both cases of $K \gtreqless 0$, $\lim_{t\to\infty} y(t) = 440.9$.

In Figure 4, we have plotted the susceptibles $N - y(t)$ as a function of time for the two initial conditions in this example. Both solutions are evidently asymptotically approaching the stable point. Toward investigating the issue of the discrepancy between the differential equation (2.5) and the original difference equation model (2.3), which will serve as the basis of analysis in the remaining sections, we compare in Figure 5 the trajectories, with the same parameters, and the initial condition $y_0 = 100$. This gives us confidence that, at least in this case, not much is lost by our abandonment of the conventional continuous-time AIDS model for a more computer-handy discrete-time model.

Example 2. Case of $A = N - \frac{n}{\beta N} < 0$. Let $N = 500$, $n = 10$, $\mu = 0.1$, $\beta = 0.000020$. Then

$$A = 500 - \frac{10}{0.01} = -500,$$
$$\eta_1 = 85.4102 \eta_2 = 585.4102.$$

If $y_0 = 50 < \eta_1$, then

$$K = 17.9443 > 0$$

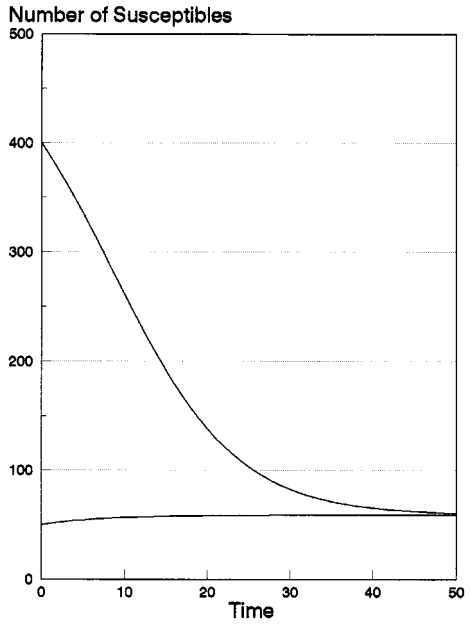


FIG. 4. *Two solution trajectories for equation* (2.5).

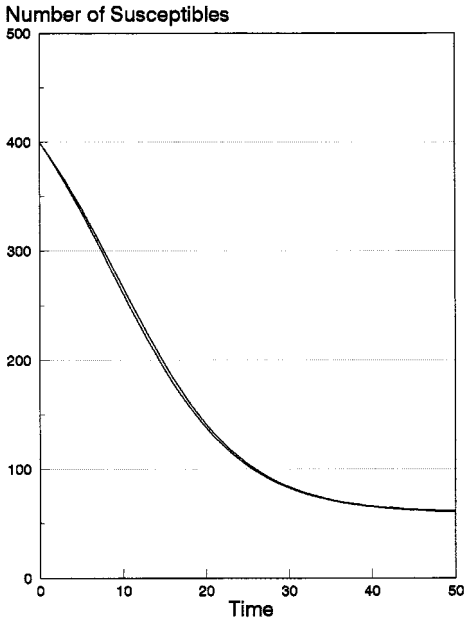


FIG. 5. *Comparison of differential and difference equation solutions.*

and

$$y(t) = \frac{1532.6238e^{0.0134t} - 585.4102}{1 + 17.9443e^{0.0134t}}.$$

If, however, $y_0 = 100 > \eta_1$, then

$$K = -46.9787$$

and

$$y(t) = \frac{\left(-4012.4612e^{0.0134t} - 585.4102\right)}{(1 - 46.9787e^{0.0134t})}.$$

Again, for both $K \gtrless 0$, we have the same limiting number of infectives, i.e., $\lim_{t\to\infty} y(t) = 85.4$.

3. The stochastic analogue. We now consider the discrete-time stochastic analogue of model (2.1), where in a prison of size N there are inflows and outflows of $n < N$ prisoners at the times $t = 0, 1, 2, \ldots$. We shall assume that at time t, just after such an inflow and outflow, the number of HIV+ prisoners is $Y(t)$, and that there is homogeneous mixing of these with the $N - Y(t)$ susceptibles during the period $(t, t+1)$. An approximation of the Reed–Frost-type model for homogeneous mixing when β is small results in the creation of a new number $M(t)$ of infectives having the binomial distribution

$$M(t) \sim \text{Bin}(N - Y(t), \beta Y(t)). \tag{3.1}$$

A rationale for (3.1) is that during each epoch, each susceptible chooses one individual at random from the entire population for contact. Contact can be intercourse, needle sharing, or just transmission of virus. Hence the chance of an infective being thus selected is $\frac{Y(t)}{N-1}$. Let γ be the probability that pairing with an infective results in HIV contagion. Then each individual has probability $\beta Y(t)$ of becoming HIV+, where $\beta = \frac{\gamma}{N-1}$. Viewing the chance of a susceptible becoming infected as a Bernoulli trial with parameter $\beta Y(t)$, independently of the outcomes of the other susceptibles, we arrive at the binomial model (3.1).

From this binomial model, we have that

$$E\left(M(t)\right) = \beta Y(t)\left(N - Y(t)\right). \tag{3.2}$$

Let us now consider the composition of $Y(t+1) = U(t+1) - V(t+1) + M(t) + Y(t)$, where $U(t+1)$ is the inflow from the outside world of new HIV+ prisoners and $V(t+1)$ is the outflow of current HIV+ prisoners at time $t+1$. The distribution of $U(t+1)$ is binomial

$$U(t+1) \sim \text{ Bin}\left\{n, \mu\right\}. \tag{3.3}$$

The outflow $V(t+1)$ will depend on $Y(t) + M(t) = Z(t+1)$, where from (3.1) the transition probability matrix from $Y(t)$ to $Z(t+1)$ for the model (2.1) is given by $P = \{p_{ij}\}$ with

$$\begin{aligned} p_{ij} &= \binom{N-i}{j-i} (i\beta)^{j-i}(1 - i\beta)^{N-j}, \quad && j \geq i, i = 0, \ldots, N, \\ p_{ij} &= 0, && i \geq j. \end{aligned} \tag{3.4}$$

The distribution of $V(t+1)$, conditioned on $Z(t+1) = z$, is hypergeometric:

$$V(t+1) \sim p(N, n, z, v) = \frac{\binom{z}{v}\binom{N-z}{n-v}}{\binom{N}{n}} \tag{3.5}$$

with $\max(0, n-(N-z)) \le v \le \min(z, n)$. For simplicity, let us write

$$p_v(z) = p(N, n, z, v)$$

and note that for $z = 0$,

$$p_0(0) = 1,$$

while the ranges of the distributions for the other values of z are

$$\begin{aligned} &p_0(z), \ldots, p_z(z) \quad \text{for } 1 \le z < n, \\ &p_0(z), \ldots, p_n(z) \quad \text{for } n \le z \le N-n, \\ &p_{N-z}(z), \ldots, p_n(z) \quad \text{for } N-n < z \le N. \end{aligned} \tag{3.6}$$

It is readily seen that the transition probability matrix from $0 \le Z(t+1) \le N$ to $0 \le Z(t+1) - V(t+1) \le N-n$ is given by the $(N+1) \times (N-n+1)$ matrix $Q = \{q_{ij}\}$:

$$\begin{aligned} q_{ij} &= p_{j-i}(i) \text{ for } 0 \le i \le j,\ 0 \le j \le n, \\ q_{ij} &= p_{i-j}(i) \text{ for } n+1 \le i \le N-n,\ i-n \le j \le i, \\ q_{ij} &= p_{i-j}(i) \text{ for } N-n+1 \le i \le N,\ i-n \le j \le N-n, \\ q_{ij} &= \quad 0 \quad \text{otherwise.} \end{aligned} \tag{3.7}$$

Thus Q has $N+1$ rows and $N-n+1$ columns and a diagonal band of probabilities of height $n+1$.

The distribution (3.3) of $U(t+1)$ allows us to write the transition probability matrix from $Z(t+1)-V(t+1)$ to $Z(t+1)-V(t+1)+U(t+1)$ as the $(N-n+1)\times(N+1)$ array determined by $R = \{r_{ij}\}$

$$\begin{aligned} r_{ij} &= \binom{n}{n-j+i} \mu^{j-i}(1-\mu)^{n-j+i} \text{ for } i = 0, \ldots, N-n,\ 0 \le j-i \le n, \\ r_{ij} &= 0 \qquad\qquad \text{otherwise.} \end{aligned} \tag{3.8}$$

It follows that the transition probability matrix from $Y(t)$ to $Y(t+1) = Z(t+1) - V(t+1) + U(t+1)$ is

$$S = PQR = \{p_{ij}\}\{q_{jk}\}\{r_{kl}\} \tag{3.9}$$

with P, Q, and R as determined by (3.4), (3.7), and (3.8). We now derive an equation for $E(Y(t+1) \mid Y(t))$.

From (3.1) we see that

$$E(M(t) \mid Y(t)) = \beta(N - Y(t))\, Y(t). \tag{3.10}$$

It follows that

$$E(Z(t+1) \mid Y(t)) = Y(t) + \beta(N - Y(t))\, Y(t). \tag{3.11}$$

Now, from (3.3),

$$E(U(t+1)) = n\mu, \tag{3.12}$$

and from (3.5), $E(V(t+1) \mid Z(t+1)) = \frac{n}{N} Z(t+1)$, so that

$$E(V(t+1) \mid Y(t)) = \frac{n}{N}\left(Y(t) + \beta(N - Y(t))\, Y(t)\right). \tag{3.13}$$

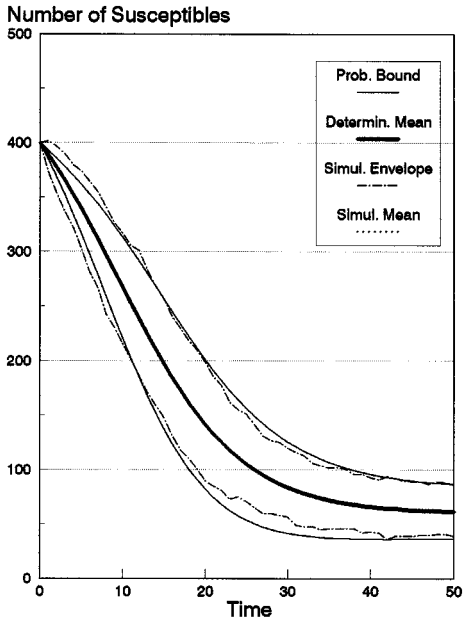


FIG. 6. *The deterministic epidemic and stagewise bounds for variability.*

Thus

$$E\left(Y(t+1) \mid Y(t)\right) = n\mu + \left(1 - \frac{n}{N}\right)\left(Y(t) + \beta\left(N - Y(t)\right)Y(t)\right). \tag{3.14}$$

The term on the right in (3.14) has precisely the form (2.3), so that the deterministic approximation of the type discussed by Gani and Yakowitz (1995) will apply in this case.

Example 3. Here we examine the stochastic analogue of the first case of Example 1. The parameters μ, β, N, and n in that model determine the transition matrices P, Q, and R as above. Toward validating the accuracy of the deterministic model as an approximator of the expectation trajectory of the stochastic analogue, a Monte Carlo study was undertaken. In Figure 6, we have plotted the solution (in terms of remaining susceptibles) of the difference equation, up to time 50, as the middle heavy line. Close to it is a dotted line which is the mean of 10,000 replications of the stochastic epidemic. The upper and lower continuous lines are the bounds as computed according to the "stagewise bound" technique of Gani and Yakowitz (1995) for showing the domain of variability in a stochastic population model. The broken lines are the envelope (i.e., the pointwise maxima and minima) of the 10,000 replications. We see that the stagewise bound method gives an accurate portrayal of the likely range of the epidemic through much of its evolution.

Toward filling in details of the construction of Figure 6 we present concepts behind the "stagewise bound" method for the interested reader. Let $\{y(t)\}$ denote the solution of the deterministic difference equation (2.3) which we express as

$$y(t+1) = G(y(t)).$$

Let $\{Y(t)\}$ be the stochastic model discussed in this section. Then assume $\delta(t)$ is a number which, with high probability, bounds $|Y(t) - y(t)|$. By linearizing the difference equation, we recall from Gani and Yakowitz (1995, equation (2.3)) that the stagewise

bound method entails propagating the error bound by

$$\delta(t+1) = \max_{z \in I_t} |G'(z)|\delta(t) + B(t). \tag{3.15}$$

In (3.15) we let I_t denote the interval $(y_t - \delta(t), y(t) + \delta(t))$ and B_t the standard deviation of $Y(t+1)|(Y(t) = y(t))$. From (2.3) of the preceding reference, one finds that

$$G'(z) = (1 - n/N)(1 + \beta * (N - 2z)).$$

In calculating $B(t)$, we observe that, conditional on $Y = y(t)$,

$$\begin{aligned}
Var(Z(t+1)) &= \beta y(t)(N - y(t))(1 - \beta(y(t))),\\
Var(U(t+1)) &= n\mu(1-\mu),\\
Var(V(t+1)) &= \frac{n(N-n)}{N^2(N-1)} E[(Z(t+1))(N - Z(t+1))]\\
&\quad + \frac{n^2}{N^2} Var(Z(t+1)),\\
Covar(Z(t+1), V(t+1)) &= \frac{n}{N} Var(Z(t+1)),
\end{aligned}$$

and so, with this notation and representation,

$$B(t) = \sqrt{Var(Z(t+1)) + Var(U(t+1)) + Var(V(t+1)) + 2Covar(Z(t+1), V(t+1))}.$$

A reviewer has suggested to us that it would be wise to incorporate into our explorations recognition that the typical prison population is composed of (i) people having shorter sentences who leave prison relatively quickly and (ii) others who are in for long terms. We generalized the model to investigate the effect of having such subpopulations. Thus, let the single-prison population be classified into those who are more likely to be released quickly, with the rest being longer-term residents. One can implement this by subdividing the one-prison population into four classes, according to HIV status and length of stay, which for our elementary study, will be either short or long. It is still presumed that mixing is homogeneous among all prisoners.

The question will be whether the effect of this compartmentalization is to change the evolution of the epidemic significantly. If so, the distribution of lengths of prison sentences must be accounted for. By the example to follow, it turns out that for the particular choices of parameters of Example 3, the net infection profiles are close. We will see, however, when we study quarantine strategy (section 5), that actually removing infectives from circulation can have a massive influence on the epidemic progression.

Example 4. We will choose parameters so that this compartmentalized population is comparable with the homogeneous prison of the previous example. The parameters are as in Examples 3 and 1, namely, $\mu = 0.1$ and $\gamma = 0.15$. However, the categories are distinguished by having different probabilities for imprisonment and release. Thus, as before, $n = 10$ people are exchanged at each epoch. But the probability is 0.8 that any one of those selected is a short-term prisoner. The population of Example 3 is split into $y_0^s = y_0^l = 50$ initial infectives among short- and long-term prisoners and $N_s = N_l = 250$ total prisoners in each category. Thus, as in Example 3, the initial prevalence of HIV is 20%.

The paths of the mean susceptible populations are shown in Figure 7. The broken line is the compartmentalized susceptible population as a function of time, and the

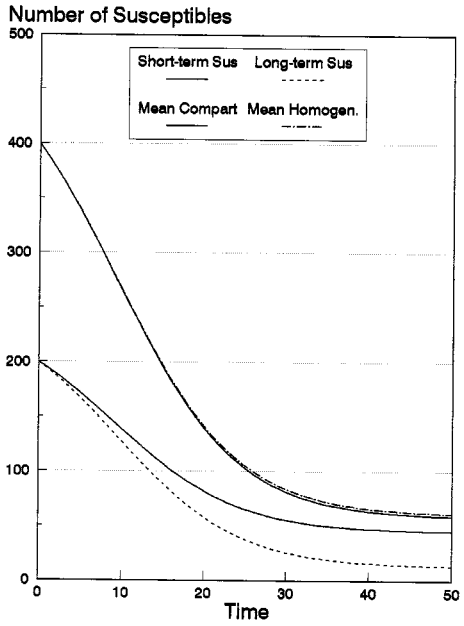


FIG. 7. *Compartmentalized versus homogeneous exchange.*

solid line is a repetition of the susceptible trajectory of Example 3. We see that there is virtually no net effect on the epidemic size. Also shown lower in this figure are two lines. The light solid line is the trajectory of the number of short-term susceptibles, and the dotted line is the long-term susceptibles. (Added together, they give the broken line.)

We experimented with other parameters. Changing the infectivity $\gamma = (N-1)\beta$ between 0.05 to 0.5 did not result in significant differences between the compartmentalized and noncompartmentalized trajectories. By increasing the inflow rate n from 10 to 50, we did get a relative difference of 20% in the number of susceptibles in the prison, but still the total number of new infections was about the same as the noncompartmentalized model. In Figure 8, we have compared the number of infections produced by the compartmentalized prison (the broken line) with the simple single prison (the solid line) as a function of time for the $n = 10$ case. Our conclusion is that in measuring new infections, in the range of our parameters the epidemic is not very sensitive to length of sentence, and so one does not need to segregate the population according to time in prison.

4. The two-prison case. The deterministic extension of our discrete-time model (2.3) to the case of two prisons Pr_1 and Pr_2 of sizes N_1, and N_2, respectively, is fairly straightforward. We will allow interaction between the prisons.

Consider the two prisons at time t, when the numbers of HIV+ prisoners in each are $y_1(t)$, $y_2(t)$. During the time interval $(t, t+1)$ there is homogeneous mixing within the prisons so that $\beta_1 y_1(t)\,(N_1 - y_1(t))$, $\beta_2 y_2(t)\,(N_2 - y_2(t))$ new infectives are produced in Pr_1 and Pr_2, respectively. Thus, at time $t+1$, there will be

$$\begin{aligned} z_1(t+1) &= y_1(t) + \beta_1 y_1(t)\,(N_1 - y_1(t))\,, \\ z_2(t+1) &= y_2(t) + \beta_2 y_2(t)\,(N_2 - y_2(t)) \end{aligned} \tag{4.1}$$

infectives in Pr_1, Pr_2.

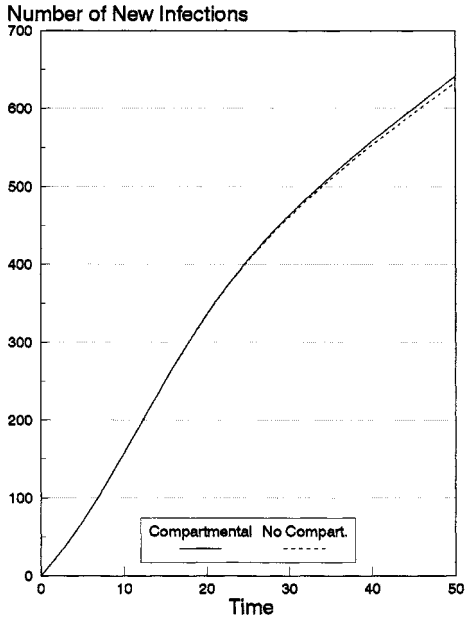


FIG. 8. *New infections, compartmentalized versus homogeneous exchange.*

Now, suppose that n_1, n_2 new prisoners join the prisons Pr_1, Pr_2 from the outside world, where the proportion of HIV+ is $0 \leq \mu \leq 1$ so that

$$u_1(t+1) = n_1\mu$$
$$u_2(t+1) = n_2\mu$$

infectives are added to Pr_1, Pr_2, respectively. Simultaneously, outflows of n_1, n_2 prisoners leave Pr_1, Pr_2 of which

$$\begin{aligned} v_1(t+1) &= \frac{n_1}{N_1} z_1(t+1) \\ &= \frac{n_1}{N_1}\left(y_1(t)\left(1+\beta_1 N_1\right) - \beta_1 y_1^2(t)\right) \\ v_2(t+1) &= \frac{n_2}{N_2} z_2(t+1) \\ &= \frac{n_2}{N_2}\left(y_2(t)\left(1+\beta_2 N_2\right) - \beta_2 y_2^2(t)\right) \end{aligned} \tag{4.2}$$

are infectives. Thus, at this stage, the numbers of infectives in Pr_1, Pr_2 are

$$\begin{aligned} y\prime_1(t+1) &= z_1(t+1) + u_1(t+1) - v_1(t+1) \\ &= n_1\mu + \left(1 - \frac{n_1}{N_1}\right)\left(y_1(t)(1+\beta_1 N_1) - \beta_1 y_1^2(t)\right), \\ y\prime_2(t+1) &= z_2(t+1) + u_2(t+1) - v_2(t+1) \\ &= n_2\mu + \left(1 - \frac{n_2}{N_2}\right)\left(y_2(t)(1+\beta_2 N_2) - \beta_2 y_2^2(t)\right). \end{aligned} \tag{4.3}$$

Next an exchange of $n < \min(N_1, N_2)$ prisoners occurs between the prisons Pr_1, Pr_2,

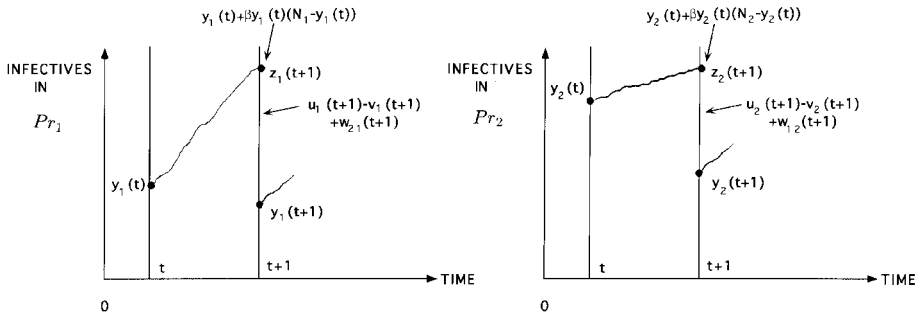


FIG. 9. *Infectives at times $t, t+1$ in two prisons Pr_1, Pr_2.*

so that

$$w_{12}(t+1) = \frac{n}{N_1} y\prime_1(t+1),\ w_{21}(t+1) = \frac{n}{N_2} y\prime_2(t+1)$$

infectives pass from Pr_1 to Pr_2 and Pr_2 to Pr_1, respectively. This results in

$$\begin{aligned} y_1(t+1) &= \left[1-\frac{n}{N_1}\right]\left[n_1\mu + \left(1-\frac{n_1}{N_1}\right)\left(y_1(t)(1+\beta_1 N_1) - \beta_1 y_1^2(t)\right)\right] \\ &\quad + \frac{n}{N_2}\left[n_2\mu + \left(1-\frac{n_2}{N_2}\right)\left(y_2(t)(1+\beta_2 N_2) - \beta_2 y_2^2(t)\right)\right], \\ y_2(t+1) &= \frac{n}{N_1}\left[n_1\mu + \left(1-\frac{n_1}{N_1}\right)\left(y_1(t)(1+\beta_1 N_1) - \beta_1 y_1^2(t)\right)\right] \\ &\quad + \left[1-\frac{n}{N_2}\right]\left[n_2\mu + \left(1-\frac{n_2}{N_2}\right)\left(y_2(t)(1+\beta_2 N_2) - \beta_2 y_2^2(t)\right)\right]. \end{aligned} \tag{4.4}$$

We have thus expressed $y_1(t+1)$, $y_2(t+1)$ in terms of the infectives $y_1(t)$, $y_2(t)$ at time t.

A stable point $\eta_1 > 0$, $\eta_2 > 0$ will exist, satisfying (4.4) in which $y_1(t+1) = y_1(t) = \eta_1$ and $y_2(t+1) = y_2(t) = \eta_2$. Figure 9 illustrates these events and variables for an epoch. Our attention will now turn to the stochastic analogue of this model.

Suppose that at time t there are $Y_1(t)$, $Y_2(t)$ HIV+ prisoners in Pr_1, Pr_2 and there is homogeneous mixing during $(t, t+1)$. Then, as in section 3, the new numbers of infectives in Pr_1, Pr_2 will be, respectively,

$$\begin{aligned} M_1(t) &\sim \text{ Bin}\left\{N_1 - y_1(t), \beta_1 Y_1(t)\right\}, \\ M_2(t) &\sim \text{ Bin}\left\{N_2 - y_2(t), \beta_2 Y_2(t)\right\}. \end{aligned} \tag{4.5}$$

Thus the numbers of infectives at $t+1$ are, respectively,

$$\begin{aligned} Z_1(t+1) &= Y_1(t) + M_1(t), \\ Z_2(t+1) &= Y_2(t) + M_2(t). \end{aligned} \tag{4.6}$$

In the fashion of (3.4), these relations (4.5), (4.6) determine transition matrices P_1 and P_2 relating $Z_1(t+1)$, $Z_2(t+1)$ to $Y_1(t)$, $Y_2(t)$, respectively.

Next we have outflows of n_1 prisoners from Pr_1 and n_2 from Pr_2, of which $V_1(t+1)$, $V_2(t+1)$ are HIV+; these will have the hypergeometric distributions

$$p(N_1, n_1, z_1, v_1) = \frac{\begin{pmatrix} z_1 \\ v_1 \end{pmatrix}\begin{pmatrix} N_1 - z_1 \\ n_1 - v_1 \end{pmatrix}}{\begin{pmatrix} N_1 \\ n_1 \end{pmatrix}},$$

$$p(N_2, n_2, z_2, v_2) = \frac{\begin{pmatrix} z_2 \\ v_2 \end{pmatrix}\begin{pmatrix} N_2 - z_2 \\ n_2 - v_2 \end{pmatrix}}{\begin{pmatrix} N_2 \\ n_2 \end{pmatrix}},$$

$$\begin{aligned} &\max\,(0, n_1 - (N_1 - z_1)) \le v_1 \le \ \min\,(z_1, n_1), \\ &\max\,(0, n_2 - (N_2 - z_2)) \le v_2 \le \ \min\,(z_2, n_2) \end{aligned} \tag{4.7}$$

when $Z_1(t+1) = z_1$, $Z_2(t+1) = z_2$. For simplicity we may write

$$p_{v_1}(z_1) = p(N_1, n_1, z_1, v_1),\ p_{v_2}(z_2) = p(N_2, n_2, z_2, v_2),$$

where $p_0(0) = 1$ for z_1 or $z_2 = 0$. The ranges of the distributions are

$$\begin{aligned} p_0(z_1), \ldots, p_{z_1}(z_1) &\quad \text{for } 1 \le z_1 < n_1, \\ p_0(z_2), \ldots, p_{z_2}(z_2) &\quad \text{for } 1 \le z_2 < n_2, \\ p_0(z_1), \ldots, p_{n_1}(z_1) &\quad \text{for } n_1 \le z_1 < N_1 - n_1, \\ p_0(z_2), \ldots, p_{n_2}(z_2) &\quad \text{for } n_2 \le z_2 < N_2 - n_2, \\ p_{N_1 - z_1}(z_1), \ldots, p_{n_1}(z_1) &\quad \text{for } N_1 - n_1 \le z_1 < N_1, \\ p_{N_1 - z_2}(z_2), \ldots, p_{n_2}(z_1) &\quad \text{for } N_2 - n_2 \le z_2 < N_2. \end{aligned}$$

The probability matrices Q_1, Q_2 for the transitions from

$$0 \le Z_1(t+1) \le N_1 \text{ to } 0 \le Z_1(t+1) - V_1(t+1) \le N_1 - n_1$$

and

$$0 \le Z_2(t+1) \le N_2 \text{ to } 0 \le Z_2(t+1) - V_2(t+1) \le N_2 - n_2,$$

respectively, have the same form as Q in (3.7), where now Q_1 has N_1 for N and n_1 for n and has order $(N_1 + 1) \times (N_1 - n_1 + 1)$. By the same token, Q_2 has N_2 for N and n_2 for n and order $(N_2 + 1) \times (N_2 - n_2 + 1)$. Since the inflows of infectives $U_1(t+1)$, $U_2(t+1)$ from the outside world are binomial and there is a probability μ of being HIV+, the probability matrices R_1 and R_2 for the transition from $Z_i(t+1) - V_i(t+1)$ to $Y_i(t+1) = Z_i(t+1) - V_i(t+1) + U_i(t+1)$, with $i = 1, 2$, will be of the form R in (3.8) where now R_1 has N_1 for N and n_1 for n and order $(N_1 - n_1 + 1) \times (N_1 + 1)$, and R_2 has N_2 for N and n_2 for n and order $(N_2 - n_2 + 1) \times (N_2 + 1)$. Thus the probability matrices S_1 and S_2 for the transition from

$$\begin{aligned} Y_1(t) \text{ to } {Y'}_1(t+1) &= Z_1(t+1) - V_1(t+1) + U_1(t+1), \\ Y_2(t) \text{ to } {Y'}_2(t+1) &= Z_2(t+1) - V_2(t+1) + U_2(t+1) \end{aligned}$$

will be of the type (3.9), with

$$S_1 = P_1 Q_1 R_1,\ S_2 = P_2 Q_2 R_2, \tag{4.8}$$

with the matrices P_i, Q_i, R_i, $i = 1, 2$ as determined as described above.

There is now the final step of exchanging n prisoners between Pr_1 and Pr_2, where $W_{12}(t+1)$ prisoners transferred from Pr_1 to Pr_2 are HIV+ and $W_{21}(t+1)$ prisoners transferred from Pr_2 to Pr_1 are HIV+. Thus

$$\begin{aligned} Y_1(t+1) &= Y_1{}'(t+1) + W_{21}(t+1) - W_{12}(t+1), \\ Y_2(t+1) &= Y_2{}'(t+1) + W_{12}(t+1) - W_{21}(t+1). \end{aligned} \tag{4.9}$$

We note that the distributions of $W_{12}(t+1)$, $W_{21}(t+1)$ are hypergeometric

$$p(N_1, n, y_1', w_{12}) = \frac{\begin{pmatrix} y_1' \\ w_{12} \end{pmatrix}\begin{pmatrix} N_1 - y_1' \\ n - w_{12} \end{pmatrix}}{\begin{pmatrix} N_1 \\ n \end{pmatrix}},$$

$$p(N_2, n, y_2', w_{21}) = \frac{\begin{pmatrix} y_2' \\ w_{21} \end{pmatrix}\begin{pmatrix} N_2 - y_2' \\ n - w_{21} \end{pmatrix}}{\begin{pmatrix} N_2 \\ n \end{pmatrix}},$$

$$\begin{aligned} \max\,(0, n - (N_1 - y_1')) &\le w_{12} \le \min\,(y_1', n), \\ \max\,(0, n - (N_2 - y_2')) &\le w_{21} \le \min\,(y_2', n), \end{aligned} \tag{4.10}$$

respectively with probability generating functions (PGFs) $\Psi_{1y_1'}(s)$, $\Psi_{2y_2'}(s)$, respectively. The PGF of $W_{21}(t+1) - W_{12}(t+1)$ is $\Psi_{2y_2'}(s)\Psi_{1y_1'}(s^{-1})$, while that of $W_{12}(t+1) - W_{21}(t+1)$ is $\Psi_{2y_2'}(s^{-1})\Psi_{1y_1'}(s)$, where we write

$$\begin{aligned} \Psi_{2y_2'}(s)\Psi_{1y_1'}(s^{-1}) &= \sum_k p_k(y_1', y_2')s^k, k \gtrless 0, \\ \Psi_{2y_2'}(s^{-1})\Psi_{1y_1'}(s) &= \sum_k p_k(y_1', y_2')s^{-k}, k \gtrless 0. \end{aligned} \tag{4.11}$$

It is clear that $(Y_1(t+1), Y_2(t+1))$ depends on the past only through $(Y_1'(t), Y_2'(t))$ and that the transition probability vector expressing this dependency is given by

$$P\{Y_1(t+1) = y_1' + k, Y_2(t+1) = y_2' - k \mid Y_1'(t) = y_1', Y_2'(t) = y_2'\} = p_k(y_1', y_2'), \tag{4.12}$$

which can be determined from (4.11). We now derive equations for $E(Y_1(t+1) \mid Y_1(t))$ and $E(Y_2(t+1) \mid Y_2(t))$. From (4.5), we see that

$$\begin{aligned} E(M_1(t)) &= \beta_1 Y_1(t)(N_1 - Y_1(t)), \\ E(M_2(t)) &= \beta_2 Y_2(t)(N_2 - Y_2(t)). \end{aligned} \tag{4.13}$$

Hence

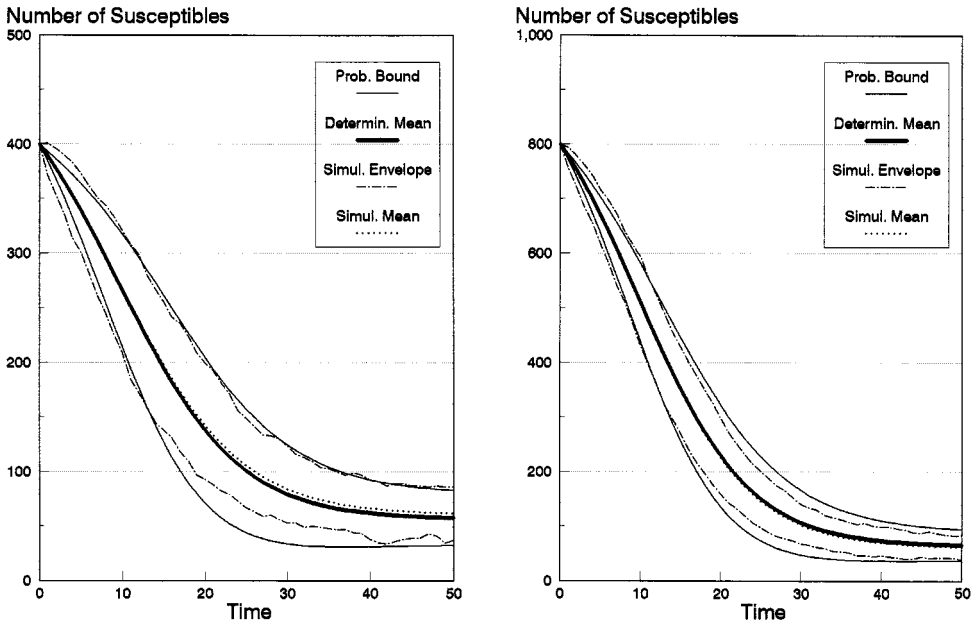
$$E(Z_i(t+1) \mid Y_i(t)) = Y_i(t)(1 - \beta_i N_i) - \beta_i Y_i^2(t), \quad i = 1, 2. \tag{4.14}$$

From (4.6), we see that $Z_i(t+1) - V_i(t+1)$ has the conditional expectation

$$E(Z_i(t+1) - V_i(t+1) | Y_i(t)) = \left(1 - \frac{n_i}{N_i}\right)\left(Y_i(t)(1 + \beta_i N_i) - \beta_i Y_i^2(t)\right), i = 1, 2,$$

and, given the binomial distribution of $U_i(t+1)$, we have

$$E(Y_i'(t+1)) = n_i\mu + \left(1 - \frac{n_i}{N_i}\right)\left(Y_i(t)(1 + \beta_i N_i) - \beta_i Y_i^2(t)\right), i = 1, 2. \tag{4.15}$$



(a) Prison 1, $N_1 = 500$ (b) Prison 2, $N_2 = 1000$

FIG. 10. *Solution of a two-prison model with stagewise bounds for variability.*

Finally, given the distribution of $W_{21}(t+1)$, $W_{12}(t+1)$ in (4.10), we have that

$$\begin{aligned}
E(Y_1(t+1) \mid Y_1(t), Y_2(t)) &= \left[1 - \frac{n}{N_1}\right]\left[n_1\mu + \left(1 - \frac{n_1}{N_1}\right)(Y_1(t)\,(1 + \beta_1 N_1 - \beta_1 Y_1(t)))\right] \\
&\quad + \frac{n}{N_2}\left[n_2\mu + \left(1 - \frac{n_2}{N_2}\right)(Y_2(t)(1 + \beta_2 N_2 - \beta_2 Y_2(t)))\right], \\
E(Y_2(t+1) \mid Y_1(t), Y_2(t)) &= \frac{n}{N_1}\left[n_1\mu + \left(1 - \frac{n_1}{N_1}\right)(Y_1(t)(1 + \beta_1 N_1 - \beta_1 Y_1(t)))\right] \\
&\quad + \left[1 - \frac{n}{N_2}\right]\left[n_2\mu + \left(1 - \frac{n_2}{N_2}\right)\right. \\
&\qquad \left. \times\,(Y_2(t) + (1 + \beta_2 N_2 - \beta_2 Y_2(t)))\right], \qquad (4.16)
\end{aligned}$$

where the results are of precisely the same form as (4.4) with

$$y_1(t+1) = E\left(Y_1(t+1) \mid Y_1(t), Y_2(y)\right) \text{ and } y_2(t+1) = E\left(Y_2(t+1) \mid Y_1(t), Y_2(y)\right).$$

Once again the approximations of Gani and Yakowitz (1995) will apply.

Example 5. In the spirit of Example 3, a computational study of the stochastic two-prison system was undertaken. The populations of the prisons were as follows. For prison 1, the values N_1, n_1, and $y_1(0)$ were taken to be exactly those of the prison in Example 1, case 1, namely $N_1 = 500$, $n_1 = 10$, and $y_1(0) = 100$. The populations for prison 2 were those of prison 1 multiplied by 2; i.e., $N_2 = 1000$, $n_2 = 20$, and $y_2(0) = 200$. For both prisons Pr_j, $j = 1, 2$, the model parameters were as in Example 1. That is, $\gamma_j = 0.15$ and $\mu_j = 0.1$ for $j = 1, 2$. The number n of prisoners exchanged at each epoch was taken to be 10. The Figure 10a and b represent trajectories of

prisons 1 and 2, respectively. We follow the format of the curves of Figure 6. Thus the heavy solid line is the solution of the deterministic version (4.4), and the nearby dotted line is the mean of 10,000 simulation replications. The thin solid lines give the stagewise probability bounds obtained by the method of Gani and Yakowitz (1995). (The procedure for computing the stagewise bounds was as described in Example 3, only the formulas become more messy because of the dimensionality. One replaces $|G'|$ in (3.15) with the analogous second-order matrix $\{|G_{i,j}|\}_{1\le i,j\le 2}$, where $G_{i,j}$ is the partial derivative with respect to $y_j(t)$ of the expression for $y_i(t+1)$ in (4.4). We spare the readers the details of this calculation and the formula for the two coefficients of B_t for 3.15.) The broken lines are the extrema (at each time point) of the 10,000 simulations of the stochastic version of the model.

The point of our experiment is that we have achieved some computational understanding of this stochastic model and are therefore in a position to explore enhancements such as prison-dependent mixing models, influence on the outside population pool of infectives, etc., or, as in the following section 5, the effect of screening and quarantine.

It is fairly obvious that these procedures could be extended to any number of prisons, and the computational burden would not be excessive if the number of prisons were moderate. We have explored different computational ideas for m-prison problems in a related work (Yakowitz, Blount, and Gani (1996)).

5. Intervention and quarantine. The prison setting, because incarceration entails forfeiture of many rights, presents an opportunity to explore educational and intervention programs aimed at reducing the severity of the HIV/AIDS epidemic, at least within the system. Educational possibilities are discussed, for example, by Stevens (1993), which contains a substantial reference list. The detailed publication (U.S. Department of Justice (1993)) mentioned that currently 14 states and the Federal prison system have mandatory HIV testing. Issues related to screening and segregation of infected prisoners are surveyed in a qualitative fashion in that work, which was prepared in collaboration with the Centers for Disease Control and Prevention.

The prison-system epidemic models introduced in the preceding sections, or modifications thereof, can give grounds for assessing the dynamics of the HIV+ population. In this section, we consider the cost effectiveness of screening and quarantine within that framework. The principles for postulating costs of such intervention have not been satisfactorily established, and so one must view the details in this section as somewhat hypothetical. Nevertheless, the principles we follow can, presumably, serve as guidelines, with the details to be filled in through experience.

We adopt the deterministic model of section 2 and augment it by presuming that at each decision time one can screen a portion $0 \le \tau \le 1$ of the prison population. Then, presumably, those detected as HIV+ are quarantined or sent to a special prison (in the case of California, Vacaville State Prison serves this function).

Let $x(t)$, $y(t)$, and $q(t)$ denote the number of susceptibles, infectives (not in quarantine), and quarantined prisoners, respectively, where $x(t) + y(t) + q(t) = N$ the prison capacity. Suppose that during the interval $(t, t+1)$, homogeneous mixing occurs in the prison between susceptibles and infectives with an infection rate β, so that $\beta x(t)y(t)$ new infectives are produced, and at $t+1$ there are

$$z(t+1) = y(t) + \beta x(t)y(t) = y(t)\,(1 + \beta x(t)) \tag{5.1}$$

infectives (not in quarantine).

Suppose an inflow of n new prisoners joins the prison with a proportion $0 \leq \mu \leq 1$ of HIV+ individuals so that

$$u(t+1) = n\mu \tag{5.2}$$

infectives are added. At the same time n prisoners leave the prison among them

$$v_1(t+1) = \frac{n}{N} z(t+1) \tag{5.3}$$

nonquarantined infectives and

$$v_2(t+1) = \frac{n}{N} q(t) \tag{5.4}$$

quarantined prisoners. Thus, before a further quarantine is imposed, we have a total of

$$z(t+1) + u(t+1) - v_1(t+1) = y(t)\left(1 + \beta x(t)\right)\left(1 - \frac{n}{N}\right) + n\mu \tag{5.5}$$

infectives, and

$$q(t) - v_2(t+1) = \left(1 - \frac{n}{N}\right) q(t) \tag{5.6}$$

quarantined prisoners.

If we now screen and quarantine a proportion $0 \leq \tau \leq 1$ of the nonquarantined prison population, then, from (5.5), at time $t+1$, the number of infectives will be

$$y(t+1) = (1-\tau)\left(n\mu + y(t)(1+\beta x(t))\left(1 - \frac{n}{N}\right)\right), \tag{5.7}$$

while the number of quarantined prisoners is

$$q(t+1) = q(t)\left(1 - \frac{n}{N}\right) + \tau\left(n\mu + y(t)\left(1 + \beta x(t)\right)\left(1 - \frac{n}{N}\right)\right). \tag{5.8}$$

Since the prison capacity is N, there are

$$x(t+1) = N - y(t+1) - q(t+1)$$

susceptibles.

We now consider the costs involved: let a be the cost of testing for HIV, b the unit-time cost of quarantining a prisoner, and c the unit-time cost of treating an infective, even if no admission of HIV+ status has been made. Then at any time t, the cost $C(t)$ will be

$$C(t) = a\tau(N - q(t)) + bq(t) + cy(t) = Na\tau + q(t)(b - a\tau) + cy(t). \tag{5.9}$$

The total cost over a fixed horizon of T time units is

$$J(\tau, T) = \sum_{t=1}^{T} C(t). \tag{5.10}$$

Presuming that $b > c > a$, one might expect the value of $\tau < 1$. For if T is short, there will be few new infections and their cost will not counterbalance the cost of screening the entire population. If, on the other hand, T is very large, the best strategy will be to quarantine at least some of the HIV+ prisoners and stop further infections, to the extent possible.

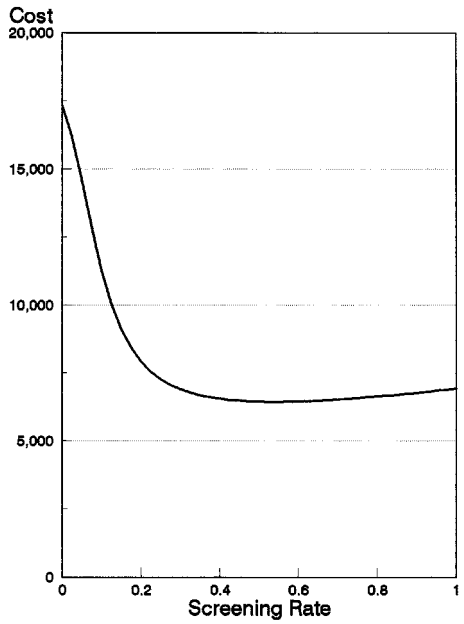


FIG. 11. *Epidemic cost, as a function of screening rate.*

Example 6. Purely for illustrative purposes, we adopt the cost parameters

$$a = 0.1, \quad b = 1.1, \quad c = 1.0$$

and employ the deterministic difference equation (2.3) with the parameters as in Example 3; that is, $\mu = 0.1$, $\beta = 0.0003$, $n = 10$, $N = 500$, and $y_0 = 100$. We then screen according to different values of τ and tabulate the costs over a 50-period epidemic. The resulting values are shown in Figure 11. The results suggest that screening and quarantine is worthwhile, to a certain point. The minimum occurs at $\tau = 0.525$, but beyond a level of about $\tau = 0.5$ further increase in screening does not yield savings.

In Figure 12, several susceptible trajectories associated with different screening rates are shown. There is little improvement in the disease prevention for τ larger than 0.4 or 0.5, and thus screening beyond this level is not cost effective according to our loss function choice.

One can use the stagewise bounds technique in the context of assessing the variability of epidemic cost. Thus by the procedure offered in (3.15), modified to take into account screening and quarantine as in (5.7, 5.8), after evaluations along the upper and lower quasiboundaries, we get that at $\tau = 0.5$ the costs fall into the interval (2900, 10250). For a horizon $T = 10$, the costs are in a smaller interval of (1100, 1800). The point of this section and Example 6, in particular, is that methodology for the decision problem of HIV screening in prison is at hand. Work remains to be done to reconcile the model with evidence, data, costs, and intuition of prison system decision makers.

Conclusions. From qualitative citations given, it is clear that AIDS in prison systems is a universal, serious, and expensive problem. The main contributions of the present study are (i) to generalize standard HIV epidemic models to cover the

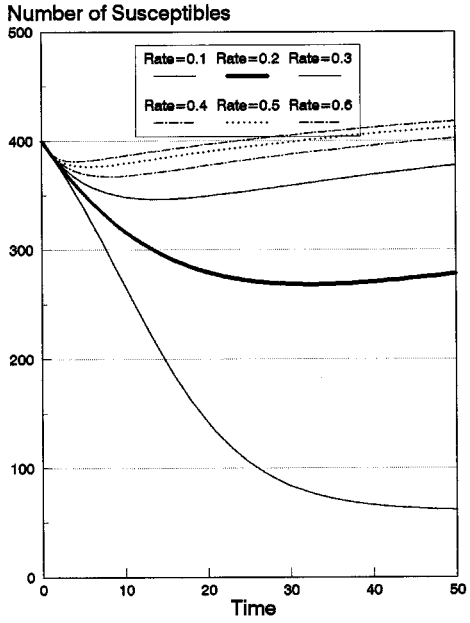


FIG. 12. *Paths for different screening rates.*

dynamics of a prison setting and (ii) within this framework, to explore a quantitative optimization problem of the cost effectiveness of screening and quarantine. To our knowledge, publications on HIV in prison are not quantitative enough for us to calibrate or verify the models and the parameters selected for this study. Hopefully the models and methodologies examined here are robust, and will accommodate the situation as data become available.

Among approximations made in this paper is that the proportion μ of infectives among detainees entering prison is constant in time. Were more informed estimates of the proportion available, they could, of course, be included in the computational analysis. One should account for the propensity of this prisoner inflow to contain former prisoners by taking μ to be higher than the infection rate of the general population. It would be very useful if prisons conducted HIV testing on all prisoners admitted, so that μ and its variation in time could be monitored.

On the methodological side, a probabilistic bounding technique developed in Gani and Yakowitz (1995) has been adopted to assess epidemic variability and the reliability of deterministic approximations. This continues efforts of the authors to understand the influence of stochasticity and the extent to which it can be ignored, as is the custom in much of the literature. Another aspect is our attempt to put epidemic problems into a decision-theoretic framework, as seen in the quarantine analysis.

Much remains to be done both in analysis of the AIDS epidemic within prison systems and, more generally, in the decision and policy analysis of all epidemics.

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