SIAM J. APPL. MATH.
Vol. 56. No. 1. pp. 143-166. February 1996
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A DIFFUSION MODEL FOR AIDS IN A CLOSED, HETEROSEXUAL POPULATION: EXAMINING RATES OF INFECTION*

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Abstract. This paper considers a model for the spread of acquired immunodeficiency syndrome (AIDS) in a closed, purely heterosexual population. Using asymptotic expansions, we derive a set of governing partial differential equations to approximate the population of proportion infected. By assuming a very narrow distribution of partners and a closed population, we examine both the initial spread of the AIDS epidemic and specific subculture populations which lend themselves well to this scenario. A main issue explored in this paper is determining a way to estimate an individual's infection rate—the probability of becoming infected with HIV given a fixed individual risk. In particular, as an individual's risk increases, which we define to be the number of different sexual partners per year, we observe, through traveling wave solutions, the increase of an individual's chance of becoming infected.

Key words. AIDS heterosexual epidemic model, partial differential equation, asymptotics

AMS subject classifications. 92D30, 35J65, 45M05

1. Introduction.

A number of models have looked at the acquired immunodeficiency syndrome (AIDS) epidemic in different venues. This paper's contribution lies in the estimation of the chance of becoming infected with human immunodeficiency virus (HIV) given a fixed risk. Comprehensive reviews of existing models can be found in [1], [25], for example. To examine the AIDS epidemic, it is important to try and quantify some of the contributing factors. One major problem concerning individuals is their personal risk of becoming infected with HIV. Quantifiers have been suggested to estimate this risk. They range over age, number of people with whom needles are shared, population density of the city in which one lives, ethnic group, socioeconomic status, sexual preference, behavior, and number of new sexual partners per year. These are just a few of the many possible groupings that put one in a higher or lower risk group of becoming infected. Given an individual has a fixed risk, we explore a way of quantifying an individual's chance of becoming infected with HIV. We choose as the risk factor the number of different sexual partners per year per individual and denote this number r. From data, this appears to be the main counter [30]. This does not take into account how many contacts take place with each different partner, just the total number of different partners. We can account for this through multiplication by an appropriate function to deal with multiple contacts. We must also distinguish between the actual number of partners an individual has per year from the desired number of partners per year. A person is placed in the category of risk r if their desired number of partners is r (whether male or female). We balance this with a term $r_i(t, r)$, which is the actual number of different partners one has given one is of risk r (i represents either male or female). Using asymptotic expansions, we derive expressions for this rate of infection for both males and females and explore the results through numerical simulations. Other important factors relate to how people choose their partners. Only now with

*Received by the editors October 19, 1992; accepted for publication (in revised form) November 18, 1994.

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the onset of AIDS has the great importance of this type of information been recognized. This type of behavior has recently begun being studied at the sociological, epidemiological, and mathematical modeling level [7].

Much work has been done on modeling the effects of HIV in the homosexual community [2],[5],[10],[13]–[16]. According to the Centers for Disease Control, the five states with the highest incidence of HIV infection in the heterosexual population are New York, California, Florida, Texas, and New Jersey. These states contain the largest cities, and inner cities, in the United States. Therefore, given the increasing numbers of heterosexual AIDS victims in Africa and in these inner cities of the United States, we are motivated to consider only a closed, purely heterosexual community with a strong preference toward partners of similar risk. The reasons for this are many. For example, in the inner-city groups, intravenous drug users share needles and exchange sex for drugs on a regular basis. Inner-city gangs usually have fixed "groupies," individuals of the opposite sex of the gang members who socialize only with gang members. Individuals in these groups share similar risk behaviors; hence, considering a similar risk-based heterosexual population is plausible. This risk behavior is commonly referred to as the like-vs.-like phenomenon and has been studied extensively (e.g., [4]).

2. The model. For AIDS, since there is no recovery, we divide the population into three groups: the susceptibles (S), the infecteds (I), and those diagnosed with AIDS (A). The susceptible population consists of those individuals who are sexually active and are not yet infected. The infected population are those who have been infected but are not showing signs of clinical AIDS as of yet (i.e., cancer, loss of weight, respiratory or nerve disorders, etc.). And, last, the AIDS grouping is for those who have progressed to AIDS and/or are removed from the sexually active population. We always assume that once one moves from susceptibles to infecteds or infecteds to clinical AIDS, one cannot flow back. With the inclusion of vaccine and chemotherapy treatments, this model can be extended to have movement in all directions.

Time t is calendar time, and once an individual moves from group S to group I, we begin marking τ, the age of infection. Then, upon moving to group A, we mark time as $\hat{\tau}$, the age since diagnosis. For the structure variable, we will define the risk of the individual, r, which governs the probability of moving from one population category to the next.

We begin with a simple interaction to describe the events which take place during the epidemic. If a single heterosexual population is considered according to the three mutually exclusive groups—susceptibles, infecteds, and diagnosed AIDS cases—we observe compartmental changes in the usual way, in that the susceptible population can become infected (at rate λ) and become infecteds. These infecteds can convert to AIDS at rate $\gamma(\tau)$ (dependent on time since infection).

We delineate a two-population model simply by using the subscript i, where $i \in \{M, F\}$. Note that an i or j subscript will *always* refer to sex (i.e., male or female). Thus, S_i refers to the variable S for the sex i. There are six equations with six auxiliary conditions, which are

$$\partial S_i(t,r)/\partial t = So_i(t,r) - \mu S_i(t,r) - \lambda_i(t,r)S_i(t,r), \tag{1}$$

$$I_i(t,r,0) = \lambda_i(t,r)S_i(t,r) \tag{1'}$$

$$\partial I_i(t,r,\tau)/\partial t + \partial I_i(t,r,\tau)/\partial \tau = -(\gamma(\tau)+\mu)I_i(t,r,\tau), \tag{2}$$

$$A_i(t,r,0) = \int_0^\infty \gamma(\tau) I_i(t,r,\tau)\,d\tau, \tag{2'}$$

$$\partial A_i(t,r,\hat{\tau})/\partial t + \partial A_i(t,r,\hat{\tau})/\partial \hat{\tau} = -(\delta(\hat{\tau}) + \mu) A_i(t,r,\hat{\tau}), \tag{3}$$

$$\lambda_i(t,r) = r_i(t,r) \int_0^\infty \rho_i(t,r,s) K_j(t,r,s)\,ds, \qquad i \neq j \in \{M,F\}. \tag{4}$$

A list of variables and parameters is given in Table 1, but we present a brief description here as to the meaning of each:

$r_i(t,r)$ = actual number of different partners per year (to simplify notation, we will write r_i only);
μ = natural death rate of persons in sexually active population;
So_i = density of people with r new partners per year before AIDS was introduced into the population;
$\gamma(\tau)$ = per-person rate of developing AIDS for those infected τ units of time ago;
$\delta(\hat{\tau})$ = per-person death rate due to AIDS for those diagnosed $\hat{\tau}$ units of time ago.

In the system (1)–(6), $\lambda_i(t,r)$, the per person rate of infection per susceptible, is needed to fully specify the system. This is one of the main issues explored in this paper: a way to estimate the infection rate, λ_i. If we examine the definition of $\lambda(t,r)$ given in (4), we see it depends on three functions: $K_j(t,r,s)$, the probability that a person of sex j and risk r will be infected by a person of sex i with risk s; $r_i(t,r)$, the actual number of different partners per year; and $\rho_i(t,r,s)$, the mixing function, which is discussed in the next section.

2.1. Mixing functions: $\rho_F(t,r,s)$ and $\rho_M(t,r,s)$. The functions $\rho_i(t,r,s)$ are, more precisely, the density functions for population i of risk r of their partners of risk s of sex j. Many have explored partner selection and pair formation in regards to disease modeling—for example, [6],[8],[9],[11],[16], [18]. In 1991, Busenberg and Castillo-Chavez [5] gave nine general cases for mixing functions, each arising from different assumptions. Here we present a few to motivate our choice.

For any one- or two-sex model with heterogeneous activity Blythe and Castillo-Chavez [4] described mixing functions of the form $\rho_i(t,r,s), i \in \{M,F\}$. The idea is that $\int_s^{s+\Delta s} \rho_i(r,x)dx$ represents the fraction of partners that a person with activity r has among individuals with activities in the range $[s, s+\Delta s]$.

For these functions $\rho_i(t,r,s)$, there are some natural conditions which arise, namely,

$$\int_0^\infty \rho_i(t,r,s)\,ds = 1, \tag{5a}$$

$$\rho_i(t,r,s) \geq 0, \tag{5b}$$

and

$$\rho_M(t,r,s) r_M(t,r) N_M(t,r) = \rho_F(t,s,r) r_F(t,s) N_F(t,s), \tag{6}$$

where $N_i(t,r) = S_i(t,r) + \int_0^\infty I_i(t,r,\tau)d\tau$ represents the total sexually active population.

Equation (5a) guarantees that the probability density function integrates to one so that the people of risk r actually have an average of $r_i(t,r)$ different partners per

TABLE 1

Independent variables

t = calendar time,
τ = age of infection,
$\hat{\tau}$ = age since AIDS diagnosis,
r = risk factor of individuals (i.e., number of desired different sexual partners per year),
s = risk factor of partners ($r, s \geq 0$ and $r, s \in \mathbf{Z}^+$).

Dependent variables

To model risk-based behavior, we suppose that the population can be distributed according to the number of its new sexual partners per year. Distribution refers to the total numbers per risk at time t.

$S_i(t,r)$ = distribution of uninfected individuals having sex with partners of risk r,
$I_i(t,r,\tau)$ = distribution of non-AIDS infecteds with risk r at time t, and duration of infection τ at time t,
$A_i(t,r,\hat{\tau})$ = distribution of AIDS cases with risk r and duration of AIDS $\hat{\tau}$,
$N_i(t,r)$ = total number of sexually active individuals with risk r in the population at time t,
$N_i(t,r) = S_i(t,r) + \int_0^\infty I_i(t,r,\tau)d\tau$,
$r_i(t,r)$ = actual number of different partners per year (to simplify notation, we will write r_i only).

Parameters

μ = natural death rate of persons in sexually active population,
So_i = density of people with r new partners per year before AIDS was introduced into the population,
$\gamma(\tau)$ = per person rate of developing AIDS for those infected τ units of time ago,
$\delta(\hat{\tau})$ = per person death rate due to AIDS for those diagnosed $\hat{\tau}$ units of time ago,
$\beta(\tau,r)$ = probability, for an given individual of risk r, of an infection per contact with an infected person who has been infected for τ time units (usually, we assume $\beta(\tau,r) = \beta$).

Functions

$\lambda_i(t,r)$ = per person rate of infection per susceptible (incidence at a fixed time),
$\rho_i(t,r,s)$ = the fraction of partners of people with risk r who have risk s (i.e., the fraction of their sexual partners chosen at time t that have risk between s and $s+ds$ is $\rho_i(t,r,s)ds$),
$K_j(t,r,s)$ = probability of a person of sex j with risk r being infected by a partner of risk s at time t,
$K_j(t,r,s) = \int_0^\infty \beta(\tau,r)\frac{I_j(t,\tau,s)}{N_j(t,s)}d\tau$.

year, and (5b) ensures nonnegative densities. Equation (6) is a balancing equation such that the number of partners with risk s with whom people of risk r have sex is the same as the number of partners with risk r with whom people of risk s have sex. The functions $\rho_i(t,r,s)$ are discussed below.

In a single-sex scenario, proportionate mixing is represented as (cf. [4],[11])

$$\rho(s,r) = \frac{rN(r)}{\int_0^\infty xN(x)dx}.$$

In [9], Castillo-Chavez and Busenberg presented a scheme and discussed solutions for a two-sex mixing problem. The equations are

$$\rho_M(s) = \frac{r_F(s)N_F(s)}{\int_0^\infty r_M(x)N_M(x)dx} \quad \text{and} \quad \rho_F(r) = \frac{r_M(r)N_M(r)}{\int_0^\infty r_F(x)N_F(x)dx},$$

where an alternative balance law to (6) is assumed, i.e., $\int r_M N_M = \int r_F N_M$. They proved that this is the only separable two-sex mixing function satisfying conditions (5) and (6).

Hyman and Stanley gave a more general alternative to proportionate mixing [16], which is expressed as

$$\rho(s,r) = \left\{ \begin{array}{ll} \rho(r,s)\dfrac{rN(r)}{sN(s)}, & r < s \\ \dfrac{f(s,r)rN(r)}{\int_s^\infty f(s,x)xN(x)dx}\left(1 - \int_0^s \rho(s,x)dx\right), & r > s \end{array} \right\}.$$

A main difference of these equations of Castillo-Chavez and Busenberg from that of Hyman and Stanley is the function $f_F(r,s)$. This function $f(s,r)$ is arbitrary and can aid in fine tuning the behavior of the mixing. This ρ can also be shown to satisfy constraint (5).

Expanding the notion of proportionate mixing, we refer to the self-selection rules from Stanley in [17],[27], [28], namely, the *asymmetric rule*, where only one of the sexes in the pairing does the choosing:

$$\rho_i(t,r,s) = \frac{[1 - \int_0^r \rho_j(t,s,x)dx]f_i(r,s)r_j(t,s)N_j(t,s)}{\int_0^\infty (1 - \int_0^r \rho_j(t,y,u)du)f_i(r,y)r_j(t,y)N_j(t,y)dy},$$

$$\rho_j(t,r,s) = \frac{\rho_i(t,s,r)r_i(t,s)N_i(t,s)}{r_j(t,r)N_j(t,r)}.$$

For our purposes here, we assume two major factors: first, that the females, in this purely heterosexual population, do the choosing—in other words, if a pairing takes place, then it was the female who chose the partner, and second, that people choose partners of similar risks. This means that someone who has a risk of 15 most likely pairs with partners of risk 15 in this like-vs.-like scenario. These assumptions are dependent on the particular environment of the heterosexual population as mentioned above. Hence, we choose the asymmetric rule.

This yields the equation for the density of male partners for women as

$$\rho_F(t,r,s) = \frac{[1 - \int_0^r \rho_M(t,s,x)dx]f_F(r,s)r_M(t,s)N_M(t,s)}{\int_0^\infty (1 - \int_0^r \rho_M(t,y,u)du)f_F(r,y)r_M(t,y)N_M(t,y)dy}. \tag{7a}$$

The idea is as follows. The numerator comes from multiplying the distribution of females and their male partners, times the acceptance function f_F of male partners of risk s for the females of risk r, times the actual number of male partners those females have, times the number of available males. This is normalized by integrating over all the possible male partners of risk s.

The equation for the density of female partners for men is

$$\rho_M(t,r,s) = \frac{\rho_F(t,s,r)r_F(t,s)N_F(t,s)}{r_M(t,r)N_M(t,r)}, \tag{7b}$$

which follows from (6).

Here, the function $f_F(r,s)$ represents the "acceptance" of females with risk r by partners with risk s. (It is chosen the same for males here as well, i.e., $f_F = f_M$.) In choosing this partner's acceptance function, we refer to [15], [16]. They considered intuition as well as data and suppose that partners are chosen according to a

"Gaussian-like" distribution with density function with mean $\mu = r$ and variance $\sigma^2 = \varepsilon^2(r+a)^2$:

$$f_i(r,s) = c \cdot \exp\left\{-\frac{1}{2\varepsilon^2}\left(\frac{r-s}{r+a}\right)^2\right\}.$$

Here the variable is s, which represents the risk of the partner, and r is the fixed risk of the person. Since we assume $s, r \geq 0$, the values of the density function for $s < 0$ are taken to be zero. These assumptions allow for a widely spread distribution for high-risk individuals and a very narrow distribution for low-risk individuals. The $(r+a)$ prevents the low-risk distribution from degenerating (i.e., that there is some minimum variance). The parameter a is going to be of order one. So this choice of f allows the distinction between high- and low-risk groups (i.e., risk factors of $r = 3$ or $r = 4$ give basically the same distribution but one very different from that of $r = 15$) which is independent of sex (i.e., the same function for both males and females). The c is a constant which cancels in (7a). For any of the following discussions, we must impose the restriction that $f(r,s)$, when left in general (as there are mant appropriate choices for $f(r,s)$), is strictly positive. Otherwise, the mixing functions, $\rho_i(t,r,s)$ do not satisfy the conditions (5) and (6).

To simplify the formuation and to combine the two properties (7a) and (7b), examine the expression for the female infectivity, (7a), and use condition (5) to replace the $1 - \int_0^r \rho_M(t,r,x)dx$ term by $\int_r^\infty \rho_M(t,r,x)dx$. Then, substitute equation (7b) into (7a). Cancel like terms, suppress the time notation (as this equation should hold for all time), and define $\varphi_i(t,x) = r_i(t,x)N_i(t,x)$. These simplifications yield an integral equation for $\rho_F(t,r,s)$, namely,

$$\rho_F(r,s) = \frac{\int_r^\infty \rho_F(x,s)\varphi_F(x)dx f_F(r,s)}{\int_0^\infty \left[\int_r^\infty \rho_F(x,y)\varphi_F(x)dx\right] f_F(r,y)dy}.$$

2.2. Properties of the asymmetric mixing function. It is important to understand the properties of these mixing functions (7a) and (7b) as formulated in § 2.1. The two main questions are: (i) Do they satisfy constraints (5) and (6) ? (ii) Does a solution exist to the integral equation (21) defining them? These problems are nontrivial and have been formulated as a purely mathematical exercise. Namely, the existence and uniqueness of a solution to (8) have been proven in [21]. We also present a way to actually construct a formula for the exact solution. This solution clearly satisfies constraints (5) and (6). So we assume this is given and examine further. For a numerical discussion of this asymmetric mixing function $\rho_i(t,r,s)$, see [17].

3. Estimation of an individual's incidence $\lambda_i(t,r)$. Reducing incidence of infection for each individual is the single most important issue in disease control. The need for a more understandable and workable expression for describing the per-person rate of infection is the focus of this section. The term $\lambda_i(t,r)$ is represented in terms of general functions for which we have no good quantitative or qualitative estimates. To estimate this infectivity function $\lambda_i(t,r)$, we will use the method of asymptotic integral expansions. The complete asymptotic analysis appears in [19]; however, a detailed overview is presented in Appendix A.

We begin by examining the densities for males and females (i.e., the partnership pairing functions) as they appear in (4), the definition of $\lambda_i(t,r)$. We first calculate a workable expression for the female infectivity function, $\lambda_F(t,r)$ and then proceed

to calculate it for the males, namely, $\lambda_M(t,r)$ The final expression, derived in Appendix A and [19], is

$$(8a)\lambda_F(t,r) \sim \left[K_M(t,r,x) + \frac{\varepsilon^2(r+a)^2}{2}\left[K''_M(t,r,x) - 2\frac{\varphi'_M(t,x)}{\varphi_M(t,r)}K'_M(t,r,x)\right]\right]\Bigg|_{x=r},$$

where $'$ denotes derivative with respect to the structure variable, x and where $K_M(t,r,s) = \int_0^\infty \beta(\tau,r)\frac{I_M(t,\tau,s)}{N_M(t,s)}d\tau$. It now remains to find an expression for the infectivity of males, namely, $\lambda_M(t,r)$. Intuition tells us it should be similar to that of $\lambda_F(t,r)$, but in actuality, because of the nonsymmetric partner-choosing functions, it is different. However, if we were to let the males do the choosing in this scenario, we would derive equal but opposite expressions for the λ_i's, respectively. We omit here the details of this derivation, since the asymptotics used are similar to that in the case of deriving $\lambda_F(t,r)$. The expression for $\lambda_M(t,r)$ is

$$\begin{aligned}(8b)\qquad \lambda_M(t,r) \sim{}& \frac{r_M(t)K_F(t,r,x)}{\quad\quad}\left[\frac{\varphi_F^2(t,r)}{(2\varphi_M(t,r)-\varphi_F(t,r))}\right] \\ &+ K'_F(t,r,x)\varepsilon(r+a)r_M(t)\left[3\varphi_F(t,r) + 2\varepsilon(r+a)\varphi'_F(t,x)\right] \\ &+ K''_F(t,r,x)r_M(t)\varepsilon^2(r+a)^2\varphi_F(t,r)\Big|_{x=r}.\end{aligned}$$

We add the assumption that $\varphi_M(t,r) \neq 2\varphi_F(t,r)$. This is reasonable since the sum over all risks of total numbers of male and female partners should be equal, *i.e.*, $\int \varphi_M(t,r)dr = \int \varphi_F(t,s)ds$. This holds from equation (6), which can alternatively be viewed as

$$\int_0^\infty r_M(t,r)N_M(t,r)dr = \int_0^\infty r_F(t,s)N_F(t,s)ds.$$

This would imply that the functions r_F and r_M should depend on N_F and N_M, respectively.

4. Discussion of the differential equations. We now would like to use the information gained in the derivation of workable expressions for $\lambda_M(t,r)$ and $\lambda_F(t,r)$ in the most useful way. If we consider the epidemic in its early stages, or in a subculture scenario, we can ignore birth and death influences (i.e., $So_i = \gamma = \mu = 0$). Examine equations (1), (1′), and (2) for the time derivatives of $S_i(t,r)$ and $I_i(t,r)$ and integrate over all τ. Under these assumptions, these three equations now reduce to $\partial_t S_i(t,r) = -\lambda_i(t,r)S_i(t,r)$ and $\partial_t I_i(t,r) = \lambda_i(t,r)S_i(t,r)$. Since we have integrated over all τ, we will now use the notation $I_i(t,r)$ for $\int_0^\infty I_i(t,r,\tau)d\tau$. If we let $N_i(t,r) = S_i(t,r) + \int_0^\infty I_i(t,r,\tau)d\tau$, which is the total population available, then for small time this implies $\partial_t N_i(t,r) \equiv 0$ (i.e., $N \sim$ constant), yielding a closed system. Define $V_i(t,r) = \frac{I_i(t,r)}{N_i(t,r)}$ to represent the fraction infected. This implies that the dependent variables will satisfy $0 \leq V_i \leq 1$. This, together with the equation $\partial_t I_i(t,r) = \lambda_i(t,r)S_i(t,r) = \lambda_i\big(N_i(t,r) - I_i(t,r)\big)$, while suppressing the (t,r) notation for ease, yields

$$(9)\qquad \partial_t V_i = \lambda_i(1 - V_i).$$

Examine equation (8a). From the definition for $K_M(t,r,s)$, together with neglecting changes in the probability of infection per contact with an infected partner since we are considering the like-vs.-like scenario (i.e., $\beta(\tau,r)=\beta$), for a fixed time and at $\tau=0$ we have $K_M(t,r,r)=\frac{\beta I(t,r)}{N(t,r)}=\beta V_M(t,r)$; hence, the expression for (9) becomes a nonlinear partial differential equation in V_M and V_F:

$$\partial_t V_F=\beta(1-V_F)r_F\left[V_M+\left(\frac{\varepsilon^2(r+a)^2}{2}\right)\left(\frac{-\varphi'_M(r)}{\varphi_M(r)}\partial_r V_M+\partial_r^2 V_M\right)\right]$$

In a manner similar to that of the equation for females, we have $\partial_t V_M=\lambda_M(1-V_M)$, with $K_F(r,r)=\beta V_F(r)$, and substituting in our now asymptotically expanded version of λ_M, (8b), implies that our coupled set of nonlinear partial differential equations is

$$\partial_t V_F=\beta(1-V_F)r_F\left[V_M+\frac{\varepsilon^2(r+a)^2}{2}\left(\frac{-\varphi'_M(r)}{\varphi_M(r)}\partial_r V_M+\partial_r^2 V_M\right)\right]$$

$$\partial_t V_M=\beta(1-V_M)r_M\left[\frac{\varphi_F^2(r)}{\varphi_M(r)(2\varphi_M(r)-\varphi_F(r))}V_F+\right.$$

$$\left.\left(\frac{3\varepsilon^2(r+a)\varphi_F(r)}{\varphi_M(r)}+\frac{2\varepsilon^2(r+a)^2\varphi'_F(r)}{\varphi_M(r)}\right)\partial_r V_F+\frac{\varphi_F(r)}{\varphi_M(r)}\partial_r^2 V_F\right]$$

To simplify this for analysis, let the coefficients of V_M, $\partial_r V_M$ and $\partial_r^2 V_M$ be $C_0(r)$, $C_1(r,\varepsilon)$, and $C_2(r,\varepsilon)$ and those of V_F, $\partial_r V_F$ and $\partial_r^2 V_F$ be equal to $C_3(r)$, $C_4(r,\varepsilon)$, and $C_5(r,\varepsilon)$, respectively, i.e.,

(13a) $$C_0(r)=\beta r_F,$$

$$C_1(r,\varepsilon)=\frac{-\varphi'_M(r)}{\varphi_M(r)}\frac{\beta r_F\varepsilon^2(r+a)^2}{2}.$$

(13c) $$C_2(r,\varepsilon)=\frac{\beta r_F\varepsilon^2(r+a)^2}{2}.$$

$$C_3(r)\sim\frac{\beta r_M\varphi_F^2(r)}{\varphi_M(r)(2\varphi_M(r)-\varphi_F(r))},$$

$$C_4(r,\varepsilon)=\frac{3\beta r_M\varepsilon^2(r+a)\varphi_F(r)}{\varphi_M(r)}+\frac{2r_M\beta\varepsilon^2(r+a)^2}{\varphi_M(r)}\varphi'_F(r),$$

and $$C_5(r,\varepsilon)=\frac{\beta r_M\varepsilon^2(r+a)^2\varphi_F(r)}{\varphi_M(r)}.$$

This yields the system

$$\partial_t V_F = (1 - V_F)[C_0 V_M + C_1 \partial_r V_M + C_2 \partial_r^2 V_M], \tag{14}$$

$$\partial_t V_M = (1 - V_M)[C_3 V_F + C_4 \partial_r V_F + C_5 \partial_r^2 V_F].$$

When one has no partners, one's probability of becoming infected is zero, and when one has an "infinite" number of partners, one's probability of becoming infected is one. (In actuality, of course, we assume the right boundary condition is less than ∞, and we replace the ∞ with a large number R_∞.) Therefore Dirichlet boundary conditions of the form $V_F(t,0) = V_M(t,0) = 0$ and $V_F(t,R_\infty) = V_M(t,R_\infty) = 1$ are chosen. These are the simplest form of boundary conditions for the epidemiological problem. Another correct boundary condition would be to impose a zero flux of infection. This could be achieved by using an approximation to this condition, such as $V_{F_r}(t,R_\infty) = V_{M_r}(t,R_\infty) = 0$.

The first derivative term in the first equation of (14) is interpreted as the female's partners of higher-risk men. The following diffusion term represents the fact that the females have male partners of both higher and lower risk than their own risk. However, due to the ε^2 coefficients, convection and diffusion occur slowly in that they are centered around partners of similar risk. The second equation of (14) for the males is interpreted similarly. Models involving both convection and diffusion have been used to explain spatial distributions of animal populations that are principally controlled by the interference between individuals and other environmental conditions [12]. If convective effects are large compared with diffusion, then the population dynamics are, except near the boundaries, dominated by the convection and interaction terms [22].

A first question that arises focuses on the type of system this is (e.g., parabolic, hyperbolic, etc.). Examining the system (26) we see it "looks parabolic."

In matrix form it becomes

$$\begin{pmatrix} U \\ V \end{pmatrix}_t = \begin{pmatrix} 0 & A(U) \\ B(V) & 0 \end{pmatrix} \begin{pmatrix} U \\ V \end{pmatrix}_{xx} + \begin{pmatrix} (C(U,V,V_x) \\ D(U,V,U_x) \end{pmatrix}.$$

The eigenvalues of the highest order coefficient matrix are $\lambda = \pm\sqrt{A(U)B(V)}$. This says that the system cannot be parabolic since the matrix is not positive definite. The system is not well posed as an initial value problem unless the matrix has real nonnegative eigenvalues. It is the negative eigenvalue which corresponds to the solution to the backward diffusion equation (see below). Systems of a similar form, however, are referred to as *cross-diffusion*. They occur when the diffusion matrix is not strictly diagonal. Cross-diffusion is not common, and little work has been done in this area. The general form of a cross-diffusion system is

$$\begin{pmatrix} U \\ V \end{pmatrix}_t = \begin{pmatrix} 0 & A(U,V) \\ -B(V,U) & 0 \end{pmatrix} \begin{pmatrix} U \\ V \end{pmatrix}_{xx} + \begin{pmatrix} C(U,V,V_x) \\ D(U,V,U_x) \end{pmatrix}.$$

Since our equations do not satisfy the above criteria, we refer to them as *pseudo-cross-diffusion* equations and examine further.

Let $V_F - V_M = W$ and subtract the second equation of (14) from the first. Suppressing the coefficients, this yields the equation

$$W_t = -W_{rr} - W_r - W + \text{ other terms.}$$

This is parabolic in form; however, the $\partial_t W = -W_{rr}$ part implies there is a backwards diffusion equation embedded in the system. This does not appear all the time, as the coefficients must be of a certain form to derive this exact scenario; however, the irregularity can be seen numerically, as the solutions can wildly oscillate for small time and certain parameters and coefficients, especially focused near and on the boundary [20].

This irregularity is a result of the asymptotics carried out to determine the expressions for $\lambda_F(t,r)$ and $\lambda_M(t,r)$ (see Appendix A). The methodology carried out was a straightforward one, and this sometimes is insufficient for the needs of the individual problem. This was the case for these equations. When dropping terms of $O(\varepsilon^2)$, which was thought justified, we lost terms that would have accounted for this irregularity. These all need to be reexamined to uncover the irregularity. Stanley [27] has reexamined this work presented here and discovered a second method in deriving similar equations. We only mention this here and cite a paper where one can see this different procedure in full detail. (Her procedure also faces problems with asymptotic breakdown.) Stavros Busenberg (personal communication) hypothesized that the reason for the breakdown in both the asymptotic approaches mentioned is that the higher-order terms were dropped in the expansions and hence the balancing equation constraint (6) is no longer satisfied. We plan to explore this possibility as further study. We now explore this irregularity further.

4.1. Analytical results.

4.1.1. Solving for steady-state solutions. To begin an analytical discussion, we first point out that the full system (14) has two constant solutions: $V_F = V_M = 0$ and $V_F = V_M = 1$. However, because the boundary conditions are not satisfied, these are not valid solutions. To solve the equations for steady-state solutions, the left-hand side of (14) is set equal to zero. This is solved if $V_F = V_M = 1$ (case 1) or if $C_0V_M + C_1\partial_r V_M + C_2\partial_r^2 V_M = 0$ and $C_3V_F + C_4\partial_r V_F + C_5\partial_r^2 V_F = 0$ are both satisfied (case 2). (The other combination yields the first case again.) Since the constant solution does not satisfy the boundary condition, we look to case 2 for a solution.

Since the two equations of case 2 are of similar form, we will examine the first, $C_0V_M + C_1\partial_r V_M + C_2\partial_r^2 V_M = 0$. For notation ease, we will write $V = V_M$ for this discussion. Using equations (13a)–(13c), the coefficients, we can rewrite this system as $V_{rr} - \frac{\varphi_M'(r)}{\varphi_M(r)}V_r + \frac{2}{\varepsilon^2(r+a)^2}V = 0$. As discussed below in § 4.1.3, we can approximate $\varphi_M(r)$ by $\frac{1}{r^3}$; therefore, the coefficient of V_r is $\frac{-3}{r}$. For simplicity, we choose $a = 0$. The results discussed below are similar to the case $a \neq 0$, so we omit the details. Therefore, the equation we are solving is $\varepsilon^2 r^2 V_{rr} - 3\varepsilon^2 r V_r + 2V = 0$. A Cauchy–Euler transformation, namely, $r = e^z$, yields the constant coefficient equation $\varepsilon^2 V_{zz} - 4\varepsilon^2 V_z + 2V = 0$. Since $\varepsilon < 1$ (and here we must require $\varepsilon < \sqrt{.5}$=.71 to have the imaginary eigenvalues), the solution is:

$$V(r) = r^3\left[c_1\cos(\omega ln|r|) + c_2\sin(\omega ln|r|)\right],$$

where $\omega = \frac{1}{\varepsilon}\sqrt{2-4\varepsilon^2}$. The boundary conditions $V(0) = 0, V(R_\infty) = 1$ must be satisfied. The first is satisfied for any choice of c_1, c_2. The second gives an explicit formula for one in terms of the other. For a particular value of R_∞, we can find the exact solution. For example, with $R_\infty = 50$, and $c_1 = c_2$, we get the graph in Fig. 1.

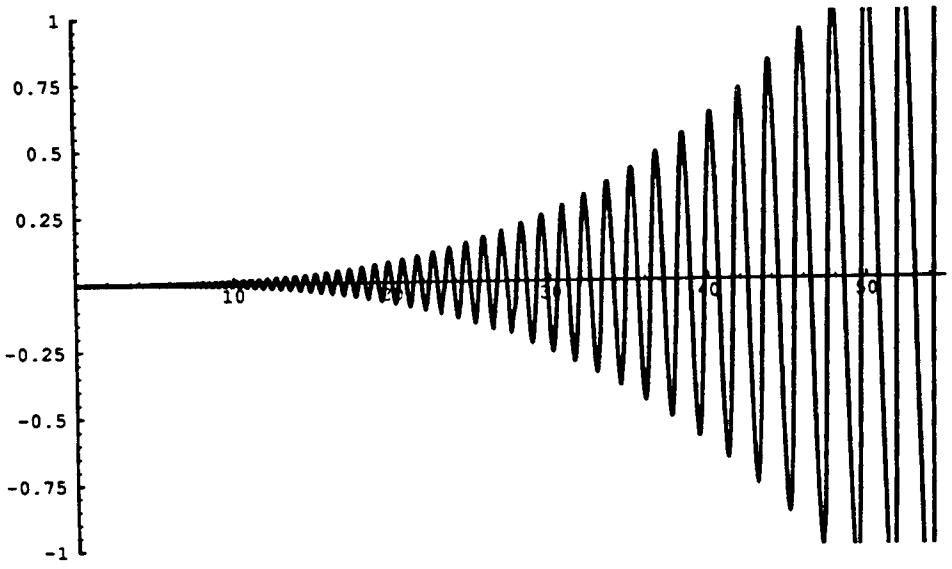


FIG. 1.

The second equation yields a similar result. Again, we see the underlying irregularity in the derived equations. We attempt to remedy this in the next section.

4.1.2. Solving the partial differential equations. Here we attempt to regularize equations (14). This is possible because the trouble arises in the $\mathcal{O}(\varepsilon^2)$ term (a high-order term), and we assume that the next-order term in the expansion would regularize the equations. Consider the full system (14). We will use an idea similar to that in regularizing the Korteweg–de Vries equation by the Benjamin–Bona–Mahony equation [3]. We replace only the cross-diffusion terms as they are the irregular terms (as seen in the backward diffusion equation discussion) and get the new system

(15)
$$V_{F_t} = (1 - V_F)\left[C_0 V_M + C_1 V_{M_r} + C_2\left(\frac{V_{F_{trr}}}{C_0} + \frac{(C_0 V_F V_M)_{rr}}{C_0} - \frac{2C_0' V_{M_r} + C_0'' V_M}{C_0}\right)\right],$$
$$V_{M_t} = (1 - V_M)\left[C_3 V_F + C_4 V_{F_r} + C_5\left(\frac{V_{M_{trr}}}{C_3} + \frac{(C_3 V_M V_F)_{rr}}{C_3} - \frac{2C_3' V_{M_r} + C_3'' V_M}{C_3}\right)\right].$$

This approximation to (14), which is valid up to order ε^2, can be verified as follows. First consider (14) up to order one:

(16)
$$V_{F_t} = (1 - V_F) C_0 V_M \quad \text{and} \quad V_{M_t} = (1 - V_M) C_3 V_F.$$

If we take the Laplacian, we have

$$V_{F_{trr}} = (C_0 V_M)_{rr} - (C_0 V_F V_M)_{rr} \text{ and } V_{M_{trr}} = (C_3 V_F)_{rr} - (C_3 V_F V_M)_{rr}.$$

Rearranging the equations and solving for the irregular terms we have

(17)
$$V_{M_{rr}} = \frac{V_{F_{trr}}}{C_0} + \frac{(C_0 V_F V_M)_{rr}}{C_0} - \frac{2C_0' V_{M_r} + C_0'' V_M}{C_0} \quad \text{and}$$

(18)
$$V_{F_{rr}} = \frac{V_{M_{trr}}}{C_3} + \frac{(C_3 V_F V_M)_{rr}}{C_3} - \frac{2C_3' V_{M_r} + C_3'' V_M}{C_3}.$$

If one wishes to verify the above approximation, examine (15) with (17) and (18) substituted in and retain terms only up to order ε^2. This reestablishes (14) with a cubic error term:

$$V_{F_t} = (1 - V_F)[C_0 V_M + C_1 V_{M_r} + C_2 V_{M_{rr}}] + \mathcal{O}(\varepsilon^3), \tag{19}$$

$$V_{M_t} = (1 - V_M)[C_3 V_F + C_4 V_{F_r} + C_5 V_{F_{rr}}] + \mathcal{O}(\varepsilon^3).$$

Therefore, up to $\mathcal{O}(\varepsilon^3)$, (15) is a good approximation to (14). Hence, we will do our analysis on (15), which we rewrite, for notation purposes, as

$$U_t = (1 - U)[K_0 V + K_1 V_r + K_2 U_{trr} + K_2 (C_0 UV)_{rr}], \tag{20}$$

$$V_t = (1 - V)[K_3 U + K_4 U_r + K_5 V_{trr} + K_5 (C_3 UV)_{rr}],$$

with $0 \leq U,\ V \leq 1$, $V(0) = U(0) = 0$, $V(R_\infty) = U(R_\infty) = 1$, $K_0 = C_0 - \frac{C_0''}{C_0}$, $K_1 = C_1 - \frac{2C_2 C_0'}{C_0}$, $K_2 = \frac{C_2}{C_0}$, $K_3 = C_3 - \frac{C_3''}{C_3}$, $K_4 = C_4 - \frac{2C_5 C_3'}{C_3}$, and $K_5 = \frac{C_5}{C_3}$.

A discussion of the existence of a solution to the regularized system is presented in detail in [19]. We omit the details here for brevity.

4.1.3. Numerically solving the regularized system. We numerically analyze the regularized system (20). Define the structure and time grid as follows. Let h represent the change in the structure (i.e., Δx) and k represent the change in time (i.e., Δt). The superscripts will refer to the discretization in time, and subscripts will refer to discretization in structure. If we discretize with respect to both time and structure, we can write our equations in matrix form as $\mathbf{A}X = \mathbf{B}$, where $X(i)$ are the $U^{n+1}(i)$ terms (respectively, $V^{n+1}(i)$), $B(I)$ are the functions of $U^n(i)$ (respectively, $V^n(i)$), and the matrix elements are

$$A_1(i) = 1 + \frac{2K2(i)}{h^2}(1 - U_i^n),\ A_2(i) = \frac{-K2(i)}{h^2}(1 - U_i^n),\ \text{and}\ A_3(i) = \frac{-K2(i)}{h^2}(1 - U_i^n)$$

for $i = 1, \ldots, n-1$ for the first equation and

$$A_1(i) = 1 + \frac{2K5(i)}{h^2}(1 - V_i^n),\ A_2(i) = \frac{-K5(i)}{h^2}(1 - V_i^n),\ \text{and}\ A_3(i) = \frac{-K5(i)}{h^2}(1 - V_i^n)$$

for the second equation. Since both of these coefficient matrices are diagonally dominant (i.e., $| A1 | > | A2 | + | A3 |$), we solve these two systems using a simple Gaussian elimination explicit method [25]. The boundary conditions, since they are known quantities, are included in the $B(1)$ and $B(n-1)$ terms, respectively.

To carry out the numerics we make the simplification that the total number of partners per time for each sex is equal, and in similar risk populations, this behavior is not unexpected. Therefore, we assume $\varphi_M(t, r) = \varphi_F(t, r)$, implying $r_M(t, r) N_M(t, r) = r_F(t, r) N_F(t, r)$. For the simulations, choose the population sizes, N_i, to be identical for each of the respective populations (i.e., $N_F = N_M$) and distributed as $\frac{1}{r^4}$ [16], [17]. This means that the size of each population with respect to risk decreases as an inverse quartic as the risk grows. Of possible interest would be to investigate the effects of different distributions such as inverse cubics or squares.

The actual number of different partners per year, $r_i(t, r)$, will be taken to be the same as the desired number of partners per year, r, i.e., $r_i(r) = r$. This follows

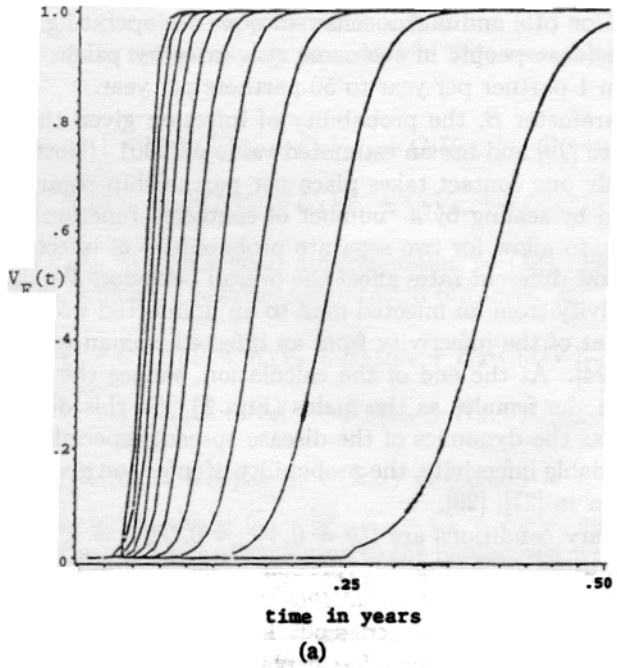
from the definition of r and makes sense since we are operating under the scenario of like vs. like (that people in the same risk category pair). We range possible values of r from 1 partner per year to 50 partners per year.

For the parameter β, the probability of infection given that a contact takes place, we refer to [29] and use an estimated value of 0.001. (Note that here we considered that only one contact takes place per partnership regardless of risk. This can be modified by scaling by a "number of contacts" function.) It is also appropriate, however, to allow for two separate probabilities of infection, i.e., β_{FM} and β_{MF}, and see how different rates affect the overall behavior. We did this by considering the infectivity from an infected man to an uninfected woman (β_{MF}=0.01) to be ten times that of the infectivity from an infected woman to an uninfected man (β_{FM}=0.001) [24]. At the end of the calculation, we see the percentage infected as twice that in the females as the males (Fig. 2). So this difference in infectivity greatly affects the dynamics of the disease spread, especially in a heterosexual population. Variable infectivity, the probability of infection given contact, has been examined further in [21], [29].

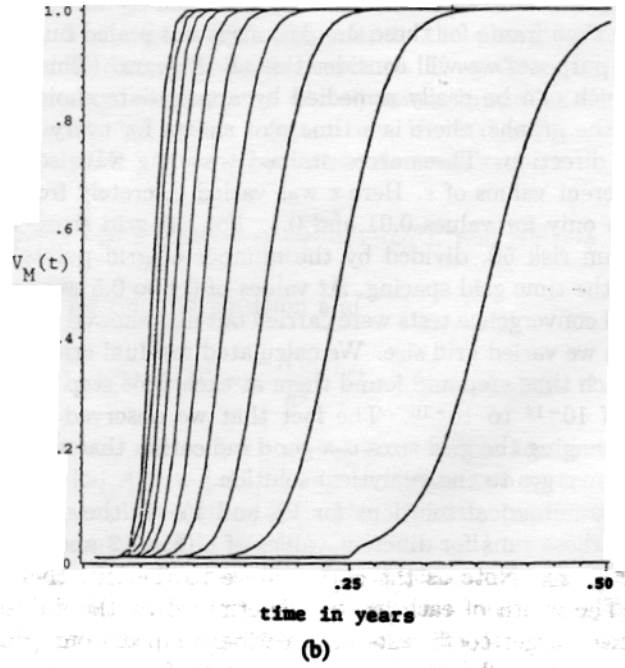
The boundary conditions are $U_0^n = 0, V_0^n = 0, U_{R_\infty}^n = 1, V_{R_\infty}^n = 1$. The runs were calculated using an S-shaped, hyperbolic tangent initial condition of the form $.5(\text{Tanh}(4/25(t,r)-25)+1)$, which satisfies the boundary conditions. (Calculations with different initial curves were carried out, but we do not present them for brevity.)

We centered this S-curve at different places to see the effects of only high-risk populations infected and watch the curves diffuse slowly into the low-risk groups over time. The time frame for these simulations is not scaled but presented in 1800 units. For our purposes we will consider this as 18 years. (This is a direct scaling calculation, which can be easily remedied by appropriate choices of parameters.) If we examine the graphs, there is a time plot shown for every 2 years, moving in the negative x direction. These are sustained traveling wave solutions. We begin by varying different values of ε. Here ε was varied discretely from 0.01 to 0.1, but runs are shown only for values 0.01 and 0.1. For the grid sizes, we choose Δx to be the maximum risk 50, divided by the number of grid points. In most cases, $\Delta x = 0.1$. For the time grid spacing, Δt values of 0.1 to 0.5 were considered. Both consistency and convergence tests were carried out and showed little or no change in the solutions as we varied grid size. We calculated residual errors for the difference equations on each time step and found them at each time step to be approximately on the order of 10^{-14} to 10^{-16}. The fact that we observed little change in the output when changing the grid sizes is a good indication that the numerical system is stable and converges to the analytical solution.

We find the numerical solutions for V_F and V_M as they appear in Figs. 2–4. We carried out these runs for different values of ε (Figs. 3 and 4) and for different values of β (Fig. 2). Note as the curves move to the left, they become steeper and steeper. The width of each front is determined by the diffusion term in the equations; hence, larger coefficients give a wider-shaped front (this would be the case for large r) and small coefficients give a steeper front (the case for small r) (cf. 13a–13f). This follows from the fact that the distribution of partners for higher-risk individuals is wider than that of individuals at lower risk. (Think of the individuals at lower risk as being "choosier" in the like-vs.-like scenario.) It is the partners of the upper-middle-risk to middle-risk individuals who "diffuse" over risk and time into higher risk categories. However, for long enough time, every risk category can be infected. The advection is the dominant term in the equations; this is what retains the S-shape of the curve.

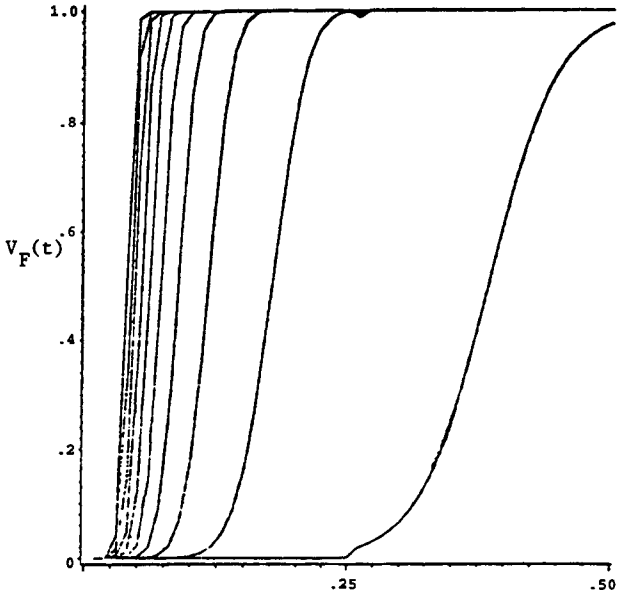


(a)

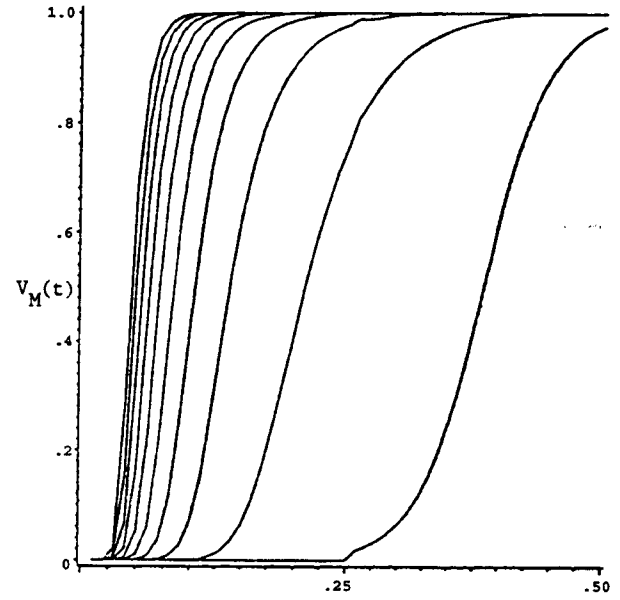


(b)

FIG. 2. (a) *Graph of infected females, V_F vs. τ for* (15). *Time is in years. The curve on the extreme right is the initial condition, and consequent years, by two, move to the left. Here $\varepsilon = 0.01, \beta_{MF} = \beta_{FM} = 0.001, h = 0.1$, and $k = 0.5$. Even with ε smaller, for long time we still see that the percentage infected reaches a high proportion.* (b) *Graph of infected males, V_M vs. τ for* (15). *Time is in years. The curve on the extreme right is the initial condition, and consequent years, by two, move to the left. Here $\varepsilon = 0.01, \beta_{MF} = \beta_{FM} = 0.001, h = 0.1$, and $k = 0.5$. Even with ε smaller, for long time we still see that the percentage infected reaches a high proportion.*

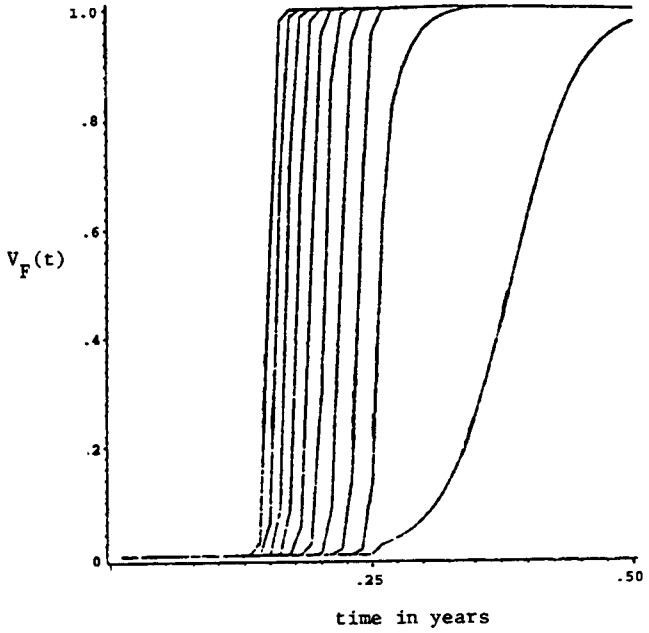


(a)

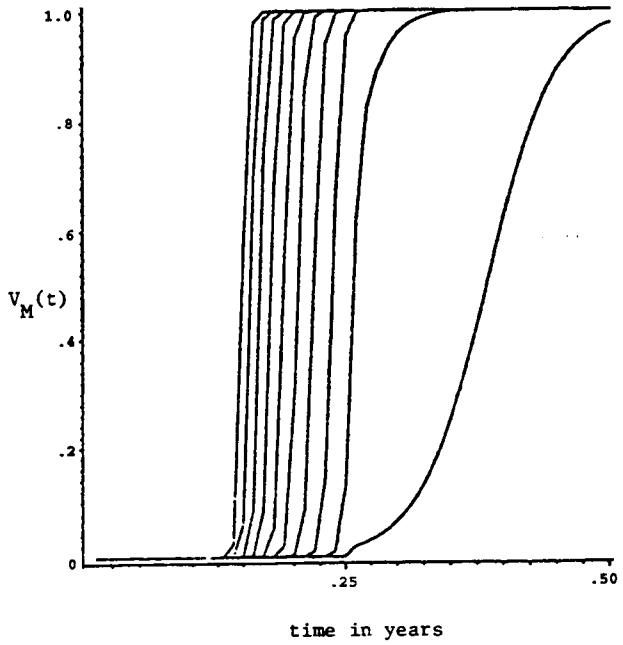


(b)

FIG. 3. (a) *Graph of infected females, V_F vs. r for* (15). *Time is in years. The curve on the extreme right is the initial condition, and consequent years, by two, move to the left. Here* $\varepsilon = 0.1, \beta_{MF} = \beta_{FM} = 0.001, h = 0.1$, *and* $k = 0.5$. *Now, for much longer time, we see that the percentage infected reaches a high proportion.* (b) *Graph of infected males, V_M vs. r for* (15). *Time is in years. The curve on the extreme right is the initial condition, and consequent years, by two, move to the left. Here* $\varepsilon = 0.1, \beta_{MF} = \beta_{FM} = 0.001, h = 0.1$, *and* $k = 0.5$. *With the higher diffusion rates, due to larger* ε, *the populations saturate faster as compared to Fig.* 3.



(a)



(b)

FIG. 4. (a) *Graph of infected females, V_F vs. r for* (15). *Time is in years. The curve on the extreme right is the initial condition, and consequent years, by two, move to the left. Here* $\varepsilon = 0.1, \beta_{FM} = 0.001, h = 0.1$, *and* $k = 0.5$. *Here, we considered different β's. We see the fraction of females infected twice that of the males for time equivalent to that in* (b). (b) *Graph of infected males, V_M vs. r for* (15). *Time is in years. The curve on the extreme right is the initial condition, and consequent years, by two, move to the left. Here* $\varepsilon = 0.1, \beta_{MF} = 0.01, h = 0.1$, *and* $k = 0.5$. *However, for the males, the distribution remains wide, even for low risk.*

The epidemiological implications are explained as follows. Following from our assumptions, we are examining this system at the early stages of the epidemic or in certain subculture scenarios where a closed, similar-risk population can be found. The results show that there are higher concentrations of individuals at low risk than at high (in either males or females). However, a higher concentration of the high-risk individuals is infected. We see an enormous difference between the high-risk individual's outcome and the low-risk individual's. And those individuals at high risk have significant increases in their numbers of infecteds as time increases. Because we are examining the populations with the birth and death processes removed, eventually, for long enough time, everyone will become infected.

We observe, finally, that the equations have different signs in the convection terms. The first equation of (20) has $K_1 > 0$, while, the second has $K_4 < 0$. This arises from the nonsymmetry in the mixing functions and the biasing in the choosing of partners (that women get to do the choosing). Because there are more lower-risk individuals in a population, more partners are chosen from lower risk categories. And since the women do the choosing, they most likely choose from risks which are the same or lower.

5. Conclusions. Here we have presented a simple model to describe the interactions of a purely heterosexual population. By defining risk as the number of different sexual partners per year, we have seen, clearly, that the higher the risk of the individual, the greater the chance of becoming infected and that this risk increases over time. This is true specifically in the like-vs.-like scenario where a pairing would take place between partners of similar risk. This result is more restrictive than that uncovered for the homosexual population by Hyman and Stanley [15] because we impose two things: female choosing and equivalent population distributions. However, here we are addressing certain communities as mentioned above.

Estimating rates of infection now lies in the correlation between risk category and sex (male or female). It is clear that if the infectivity (probability of infection per contact) is higher for male-to-female transmission than female-to-male and that the number of different sexual partners per year puts one at a higher risk of becoming infected; thus, educating these high-risk individuals can clearly be a powerful deterrent in the spread of AIDS.

Appendix A. As $\lambda_F(t,r)$ is composed of functions, we begin first by approximating those in order to get an expression for this infection rate.

Substituting equation (7b) into (7a) gives an integral equation for $\rho_F(t,r,s)$, namely,

$$\rho_F(t,r,s) = \frac{\left(1 - \int_0^r \frac{\rho_F(t,x,s) r_F(t,x) N_F(t,x) dx}{r_M(t,s) N_M(t,s)}\right) \cdot f_F(r,s) r_M(t,s) N_M(t,s)}{\int_0^\infty \left(1 - \int_0^r \frac{\rho_F(t,u,y) r_F(t,u) N_F(t,u) du}{r_M(t,y) N_M(t,y)}\right) f_F(r,y) r_M(t,y) N_M(t,y) dy}. \tag{21}$$

Substituting an expression for the total number of partners available per unit time, $\varphi_i(t,x) = r_i(t,x) N_i(t,x)$, and using definition (22a) below yield (22b) (with time fixed):

$$g_F(r,s) = \int_0^r \rho_F(x,s) \cdot \frac{\varphi_F(x)}{\varphi_M(s)} dx, \tag{22a}$$

$$\rho_F(r,s) = \varphi_M(s)\frac{(1-g_F(r,s))f_F(r,s)}{\int_0^\infty \varphi_M(x)(1-g_F(r,x))f_F(r,x)dx}$$

evaluated at a fixed time t.

Now we would like to compute $\lambda_F(r) = r_F \int_0^\infty \rho_F(r,s)K_M(r,s)\,ds$.

We will use the methods of asymptotic expansions to estimate this. Substituting for $\rho_F(r,s)$ from (22b) gives

$$\lambda_F(r) = r_F \int_0^\infty K_M(r,s)\cdot \frac{\varphi_M(s)\big(1-g_F(r,s)\big)f_F(r,s)}{\int_0^\infty \varphi_M(x)\big(1-g_F(r,x)\big)f_F(r,x)dx}ds.$$

Substituting in for the acceptance function $f_F(r,s)$ gives

$$\lambda_F(r) = \frac{r_F \int_0^\infty \varphi_M(s)K_M(r,s)\big(1-g_F(r,s)\big)e^{-\frac{1}{2}\left((r-s)/\varepsilon(r+a)\right)^2}}{\int_0^\infty \varphi_M(x)\big(1-g_F(r,x)\big)e^{-\frac{1}{2}\left((r-x)/\varepsilon(r+a)\right)^2}dx}ds.$$

Let N (resp., D) be the numerator (resp., denominator) so that $\lambda_F(r) = N/D$. Now define

$$\begin{cases} p_1(r,s) = r_F K_M(r,s)\big(1-g_F(r,s),\big)\varphi_M(s), \\ p_2(r,x) = \big(1-g_F(r,x)\big)\varphi_M(x). \end{cases}$$

We will make the assumptions p_1 and $p_2 \in C^3(\mathbf{R}^+)$, where $\mathbf{R}^+ = [0,\infty)$. This is not unreasonable since the functions which p_1 and p_2 are composed of are expected to be well behaved.

This falls in line with the epidemiology. We are considering a special case of mixing, namely, like vs. like, and under this scenario these assumptions are not unreasonable. Assuming the derivatives of p_1 are of exponential order (bounded) is also reasonable. For example, if $r_i = r$ and $N_i = \frac{1}{r^4}$, $r > 0$ (see § 4.1.3), The functions $\rho_i(t,r,s)$ and $K_i(t,r,s)$ are also expected to be well behaved. (For a discussion of ρ_i see § 2.1, mixing functions.) $K_i(t,r,s)$ behaves like a scaled version of $V_i(t,r,s)$, which is the proportion of infecteds, which is certainly bounded (by definition) and most likely smooth.

Then the expressions which make up p_1 and p_2 are composed of functions which are expected to be well behaved, and hence assuming they are in $C^3(\mathbf{R}^+)$ is valid.

We can now write (23) as

$$\lambda_F(r) = \frac{\int_0^\infty e^{-\frac{1}{2}((r-s)/\varepsilon(r+a))^2}p_1(r,s)\,ds}{\int_0^\infty e^{-\frac{1}{2}((r-x)/\varepsilon(r+a))^2}p_2(r,x)\,dx} = \frac{N}{D}.$$

Since N and D have the same form, we can expand

$$N(r) = \int_0^\infty e^{-\frac{1}{2}((r-s)/(\varepsilon(r+a)))^2}p_1(r,s)\,ds$$

and then use the result to approximate D. Make the change of variables $t = \frac{s-r}{\varepsilon(r+a)}$, which implies $s = r+\varepsilon(r+a)t$ and $ds = \varepsilon(r+a)\,dt$. For the limits of integration, $s = 0$ implies $t = -r/\varepsilon(r+a)$ and $s \to \infty$ implies $t \to \infty$. Hence,

$$N = \int_{-r/\varepsilon(r+a)}^{\infty} e^{-t^2/2}p_1(r,\varepsilon(r+a)t+r)\cdot\varepsilon(r+a)\,dt.$$

Break N into two integrals as follows:

$$N = N_1 + N_2 = \int_{-r/\varepsilon(r+a)}^{0} e^{-t^2/2} p_1(r, \varepsilon(r+a)t + r) \cdot \varepsilon(r+a)\, dt$$

$$+ \int_0^{\infty} e^{-t^2/2} p_1(r, \varepsilon(r+a)t + r) \cdot \varepsilon(r+a)\, dt.$$

Examine N_2 first. The idea used here is that we are examining the integral $\varepsilon(r+a)$ $\int_0^{\infty} e^{-t^2/2} \tilde{p}_1(\varepsilon(r+a)t)\, dt$. Expand $\tilde{p}_1(t)$ in a Maclaurin series about $t = 0$:

$$\tilde{p}_1(t) = \tilde{p}_1(0) + \varepsilon(r+a)t\tilde{p}_1'(0) + \varepsilon^2(r+a)^2 \frac{t^2}{2} \tilde{p}_1''(0) + R_3(t),$$

where $R_3(t)$ is the remainder term obtained by approximating $\tilde{p}_1(t)$ by a second-degree Taylor polynomial. We make the assumption (justified above) that the third derivative of $\tilde{p}_1$ is bounded. Call this bound C. (We can also assume it is of exponential order as well, and the proofs below are similar, but constants arise differently.) Examine the integrals

$$\varepsilon(r+a) \int_0^{\infty} e^{-t^2/2} \left(\tilde{p}_1(0) + \varepsilon(r+a)t\tilde{p}_1'(0) + \varepsilon^2(r+a)^2 \frac{t^2}{2} \tilde{p}_1''(0) \right)$$

$$+ \varepsilon^4(r+a)^4 \int_0^{\infty} e^{-t^2/2} \tilde{p}_1'''\xi(t) \frac{t^3}{6} dt,$$

for some $\xi(t)$ between 0 and t by Taylor's theorem. We will first examine the last integral above. Since we assume the third derivative is bounded by C, then we bound this integral by

$$<< \frac{\varepsilon^4}{6}(r+a)^4 C \int_0^{\infty} e^{-t^2/2} t^3 dt. \tag{25}$$

Clearly, $Ce^{-t^2/2}t^3 < e^{-t^2/4}$ for t, sufficiently large. Now, choose α based on this t, and hence equation (25) is bounded by

$$<< \varepsilon^4(r+a)^4 C \left(\int_0^{\alpha} e^{-t^2/2} t^3 dt + \int_{\alpha}^{\infty} e^{-t^2/4} dt \right).$$

The first integral is bounded, as is the second. Call their sum M. Therefore, equation (25) is bounded by $\varepsilon^4(r+a)^4 CM$. This term is bounded by δ if $\varepsilon(r+a) < \left(\frac{\delta}{MC}\right)^{1/4}$, and hence is small. Therefore, we will restrict our analysis to

$$N_2 \sim \varepsilon(r+a) \int_0^{\infty} e^{-t^2/2} \left[p_1(r,r) + \varepsilon(r+a)\partial_2 p_1(r,x) \cdot \right.$$

$$\left. t + \frac{(\varepsilon(r+a))^2}{2} \partial_2^2 p_1(r,x) \cdot \frac{t^2}{2} \right] dt \Big|_{x=r}.$$

We have three integrals to evaluate with the restriction that $\varepsilon(r+a) << 1$, i.e., $r << \frac{1}{\varepsilon}$. We state equivalently that $a < 1$. But, as is standard,

$$\text{(i)} \qquad \varepsilon(r+a)\int_0^\infty e^{-t^2/2}p_1(r,r)\,dt = \sqrt{\frac{\pi}{2}}\varepsilon(r+a)p_1(r,r),$$

$$\text{(ii)} \qquad (\varepsilon(r+a))^2\int_0^\infty \partial_2 p_1(r,r)\cdot t\,e^{-t^2/2}\,dt = (\varepsilon(r+a))^2\partial_2 p_1(r,r),$$

$$\text{(iii)} \qquad \frac{\varepsilon^3(r+a)^3}{2}\int_0^\infty \partial_2^2 p_1(r,r)\cdot t^2/2\,e^{-t^2/2}\,dt = \frac{\varepsilon^3(r+a)^3}{2}\sqrt{\frac{\pi}{2}}\partial_2^2 p_1(r,r).$$

Thus,

$$N_2 \sim \varepsilon(r+a)\left[\sqrt{\frac{\pi}{2}}p_1(r,r) + \varepsilon(r+a)\partial_2 p_1(r,x) + \sqrt{\frac{\pi}{2}}\frac{\varepsilon^2(r+a)^2}{2}\partial_2^2 p_1(r,x)\right]\Bigg|_{x=r}.$$

We carry out similar analysis on N_1, and combining both N_1 and N_2 gives

$$N \sim 2\varepsilon(r+a)\left[p_1(r,r)\sqrt{\frac{\pi}{2}} + \frac{\varepsilon^2(r+a)^2}{2}\partial_2^2 p_1(r,x)\sqrt{\frac{\pi}{2}}\right]\Bigg|_{x=r}$$

with a similar expression for D in terms of p_2. This implies

$$\lambda_F(r) \sim \frac{p_1(r,r) + \frac{\varepsilon^2(r+a)^2}{2}\partial_2^2 p_1(r,x)}{p_2(r,r) + \frac{\varepsilon^2(r+a)^2}{2}\partial_2^2 p_2(r,x)} = \frac{p_1(r,r)}{p_2(r,r)}\left[\frac{1 + \frac{\varepsilon^2(r+a)^2}{2}\frac{\partial_2^2 p_1(r,x)}{p_1(r,r)}}{1 + \frac{\varepsilon^2(r+a)^2}{2}\frac{\partial_2^2 p_2(r,x)}{p_2(r,r)}}\right]\Bigg|_{x=r}.$$

Now, expanding the denominator and neglecting the $\mathcal{O}(\varepsilon^4)$ terms we have

$$\lambda_F(r) \sim \frac{p_1(r,r)}{p_2(r,r)} + \frac{\varepsilon^2(r+a)^2}{2}\left[\frac{\partial_2^2 p_1(r,x)}{p_2(r,r)} - \frac{p_1(r,r)}{(p_2(r,r))^2}\cdot\partial_2^2 p_2(r,x)\right]\Bigg|_{x=r}.$$

From (23), $p_1(r,s) = r_F K_M(r,s)p_2(r,s)$; thus substituting this and its appropriate derivatives in (26) with $\varphi_M(x)$, we have (with $'$ denoting derivative with respect to the structure variable)

$$\lambda_F(r) = r_F\left[K_M(r,x) + \frac{\varepsilon^2(r+a)^2}{2}\left[K_M''(r,x) + \frac{2K_M'(r,x)\cdot\partial_2[1-g_F(r,x)]\varphi_M(r)}{[1-g_F(r,r)]\varphi_M(x)}\right]\right]\Bigg|_{x=r}.$$

From the expression for $\lambda_F(t,r)$ in (27), we see the only expressions left to evaluate are $g_F(r,r)$ and $\partial_2 g_F(r,x)|_{x=r}$. If the expansions are valid, our assumption is that we need $\partial_2 g_F(r,x)$ to first order. This is because $K_M(t,r,s)$ is a specified function. Namely, $K_M(t,r,s)$ is the probability of a male of risk s being infected by a female partner of risk r. From equation (22a),

$$g_F(t,r,s) = \int_0^r \rho_F(t,u,s)\frac{\varphi_F(t,u)}{\varphi_M(t,s)}\,du.$$

Plugging in for $\rho_F(r,s)$ from equation (22b) gives

$$g_F(r,s) = \int_0^r \frac{\varphi_F(u)(1-g_F(u,s))f_F(u,s)}{\int_0^\infty \varphi_M(x)f_F(u,x)(1-g_F(u,x))\,dx}du.$$

Now define

$$q(u,s) = \frac{(1-g_F(u,s))\varphi_F(u)}{\int_0^\infty \varphi_M(x)f_F(u,x)(1-g_F(u,x))\,dx}. \tag{28}$$

This yields

$$g_F(r,s) = \int_0^r ce^{-\frac{1}{2}((x-s/\varepsilon(x+a))^2}q(x,s)\,dx.$$

If we examine (15), it is evident that the denominator also depends on u, the variable of integration for the outside integral. Thus we cannot expand this first. The expansions are valid for $r > 0$. Let $t = \frac{s-x}{\varepsilon(x+a)}$. This implies $x = \frac{s-\varepsilon at}{1+\varepsilon t}$ and $dx = \frac{-\varepsilon(a+s)}{(1+\varepsilon t)^2}\,dt$. This change of variables is justified because $t \to x$ is a strictly monotone decreasing function. For the limits of integration, $x = 0$ implies $t = s/\varepsilon a$ and $x = r$ implies $t = (s-r)/\varepsilon(r+a)$. Replacing the $s/\varepsilon a$ by ∞ (this implies that $s >> \varepsilon$) because $\varepsilon > 0$ is small, together with the change of variables and if we expand the $\frac{1}{(1+\varepsilon t)^2}$ term and keep only terms of order $\dot{\varepsilon}$, yields

$$g_F(r,s) \sim \varepsilon(a+s)c\int_{(s-r)/\varepsilon(r+a)}^\infty e^{-t^2/2}q\left(\frac{s-\varepsilon at}{1+\varepsilon t}, s\right)dt.$$

Evaluating at $s = r$, assuming $q \in C^2$, and expanding q in a Taylor series about the first component, we get (we also assume that the same assumptions are present here on q as on p_1 above)

$$g_F(r,r) \sim \varepsilon(a+r)c\int_0^\infty e^{-t^2/2}\left[q(r,r) + \left(\frac{r-\varepsilon at}{1+\varepsilon t} - r\right)\partial_1 q(x,r)t\right]\Bigg|_{x=r} + \mathcal{O}(\varepsilon^2)\,dt.$$

Here we assume that $\mathcal{O}(q(r,r)) = \mathcal{O}(\partial_1 q(x,r))$. Since the coefficient of $\partial_1 q(x,r)$ is $\mathcal{O}(\varepsilon^2)$, and we retain only up to order ε, we can drop this term from the expansion. This yields

$$g_F(r,r) \sim c\cdot\varepsilon(a+r)\sqrt{\frac{\pi}{2}}q(r,r) + \mathcal{O}(\varepsilon^2). \tag{29}$$

Now we need an expression for $q(r,r)$. From equation (28)

$$q(r,r) = \frac{(1-g_F(r,r))\varphi_F(r)}{\int_0^\infty \varphi_M(x)f_F(r,x)(1-g_F(r,x))\,dx}. \tag{30}$$

From the same arguments applied to (25), we approximate this denominator in an asymptotic expansion and neglect the $\mathcal{O}(\varepsilon^3)$ terms, and plugging into (30) yields $q(r,r) \sim \frac{\varphi_F(t,r)}{2\varphi_M(r)\varepsilon(r+a)\sqrt{\frac{\pi}{2}}}$. If we use this expression for $q(r,r)$ in (29)

$$g_F(r,r) \sim \varepsilon(a+r)\sqrt{\frac{\pi}{2}}\left[\frac{\varphi_F(r)}{\varphi_M(r)2\varepsilon(r+a)\sqrt{\frac{\pi}{2}}}\right],$$

so we have

$$g_F(r,r) \sim \frac{\varphi_F(r)}{2\varphi_M(r)}.$$

Since we are considering the scenario of like vs. like, we are concerned only with the limiting case of $s = r$; hence we can now use this asymptotic expansion for $g_F(r,r)$ in (27), the expression for $\lambda_F(r)$,

$$\lambda_F(r) \sim r_F \left[K_M(r,x) + \frac{\varepsilon^2(r+a)^2}{2} \left[K''_M(r,x) - \frac{2K'_M(r,x) \cdot \partial_2(1 - g_F(r,x))\varphi_M(x)}{\left[\varphi_M(r) - \frac{\varphi_F(r)}{2}\right]} \right] \right] \Bigg|_{x=r} + \mathcal{O}(\varepsilon^3).$$

We neglect the $\mathcal{O}(\varepsilon^3)$ term. Now the only term left to approximate is

$$\frac{\partial}{\partial x} g_F(r,x) \Big|_{x=r}.$$

Begin by examining (28), the expression for $g_F(r,s)$. We wish to calculate its first partial derivative and evaluate at $x = r$:

$$g_F(r,s) = c \int_0^r q(x,s) e^{-\frac{1}{2}((x-s)/\varepsilon(x+a))^2} \, dx,$$

$$\frac{\partial g_F(r,x)}{\partial x} = \frac{\partial}{\partial x} \left(c \int_0^r q(y,x) e^{-\frac{1}{2}((y-x)/\varepsilon(y+a))^2} \right) dy \Bigg|_{x=r}.$$

Using similar asymptotic expansion arguments, we can show

$$\frac{\partial g_F(r,x)}{\partial x} \Big|_{x=r} = 0.$$

The complete details for deriving this result (31) are found in [19].

Therefore, our final expression for $\lambda_F(r)$ is

$$\lambda_F(t,r) \sim r_F \left[K_M(r,x) + \frac{\varepsilon^2(r+a)^2}{2} \left[K''_M(r,x) - 2\frac{\varphi'_M(x)}{\varphi_M(r)} K'_M(r,x) \right] \right] \Bigg|_{x=r}.$$

A complete, detailed discussion of what has been presented here can be found in [19].

We mention here that the equation for $q(r,r)$ is possibly problematic. The ε in the denominator is an indication that there may be problems using this type of asymptotic expansion. We discuss this in § 4.

Acknowledgments. As the majority of the work presented here is direct results from my thesis, I would like to thank my advisors, Jerry Goldstein and J. Mac Hyman, for all their help and support. I also would like to thank Carlos Castillo-Chavez for critical comments and suggestions in the writing of this manuscript. Although she was not a member of my Ph.D. committee, E. Ann Stanley aided

significantly with the asymptotic results presented here. I would like to thank her for her generosity of time, ideas, and assistance. Special thanks also go to the referees, whose helpful comments improved the quality of this paper.

REFERENCES

[1] R. M. ANDERSON (1989), *Editorial review on mathematical and statistical studies of the epidemiology of HIV*, AIDS, 3, pp. 333–346.

[2] R. M. ANDERSON AND R. M. MAY (1987), *Transmission dynamics of HIV*, Nature 326, pp. 137–142.

[3] T. B. BENJAMIN, J. L. BONA, AND J. L. MAHONY (1972), *Model equations for nonlinear dispersive systems*, Philos. Trans. Roy. Soc. London Ser. A, 272, pp. 47–78.

[4] S. BLYTHE AND C. CASTILLO-CHAVEZ (1989), *Like with like preference and sexual mixing models*, Math. Biosci., 96, pp. 221–238.

[5] S. BUSENBERG AND C. CASTILLO-CHAVEZ (1991), *A general solution of the problem of mixing and its application to risk- and age-structured epidemic models for the spread of AIDS*, IMA J. Math. Appl. Med. Biol., 2, pp. 1–29.

[6] ———(1991), *Interaction, pair formation, and force of infection terms in sexually transmitted diseases*, in Mathematical and Statistical Approaches to AIDS Epidemiology, C. Castillo-Chavez, ed., Lecture Notes in Biomathematics, vol. 83, Springer-Verlag, Berlin, 1989.

[7] C. CASTILLO-CHAVEZ, S. SHYU, G. RUBIN, AND D. UMBACH (1992), *On the estimation problem of mixing/pair formation matrices with applications to models for sexually-transmitted diseases*, in AIDS Epidemiology: Methodological Issues, K. Dietz, V. Farewell, N. P. Jewel, eds., Birkhäuser, Boston, pp. 384–402.

[8] C. CASTILLO-CHAVEZ, S. BUSENBERG, AND K. GEROW (1991), *Pair formation in structured populations*, in Differential Equations with Applications in Biology, Physics, and Engineering, Lecture Notes in Pure and Appl. Math. 133, Marcel Dekker, New York.

[9] C. CASTILLO-CHAVEZ AND S. BUSENBERG (1991), *On the solution of the two-sex mixing problem* in Proc. International Conference on Differential Equations and Applications to Biology in Population Dynamics, S. Busenberg and M. Martelli, eds., Springer-Verlag, New York, Lecture Notes in Biomathematics, Vol. 92, pp. 80–98.

[10] C. CASTILLO-CHAVEZ, K. COOKE, W. Z. HUANG, AND S. LEVIN (1989), *Results on the dynamics for models for the sexual transmission of HIV*, Appl. Math. Lett., 4, pp. 327–331.

[11] C. CASTILLO-CHAVEZ AND S. BLYTHE (1989), *Mixing framework for social/sexual behavior*, in Mathematical and Statistical Approaches to AIDS Epidemiology, C. Castillo-Chavez, ed., Lecture Notes in Biomathematics, Vol. 83, Springer-Verlag, Berlin.

[12] J. E. COHEN AND J. D. MURRAY (1983), *On non-linear convective dispersal effects in an interacting population model*, SIAM J. Appl. Math., 43, pp. 66–78.

[13] K. DIETZ (1988), *On the transmission of HIV*, Math. Biosci., 90, pp. 397–414.

[14] H. HETHCOTE (1989), *A dynamic model of HIV transmission and AIDS in San Francisco*, Abstract presented at the V International Conference on AIDS, p. 149.

[15] J. M. HYMAN AND E. A. STANLEY (1988), *Using mathematical models to understand the AIDS epidemic*, Math. Biosci. 90, pp. 415–473.

[16] ——— (1989), *The effects of social mixing patterns on the spread of AIDS*, in Mathematical Approaches to Problems in Resource Management and Epidemiology, C. Castillo-Chavez, S. A. Levin, and C. A. Shoemaker, eds., Lecture Notes in Biomathematics, Vol. 81, Springer-Verlag, Berlin, pp. 190–219.

[17] ——— (1993), *A risk-based heterosexual model for the AIDS epidemic with biased sexual partner selection*, in Modelling the AIDS Epidemic, Kaplan and Brandeau, eds.

[18] J. JACQUEZ, C. SIMON, AND J. KOOPMAN (1989), *Structured mixing: Heterogeneous mixing by the definition of activity groups*, in Mathematical and Statistical Approaches to AIDS Epidemiology, C. Castillo-Chavez, ed., Lecture Notes in Biomathematics, vol. 83, pp. 301–315.

[19] D. E. KIRSCHNER (1991), *Mathematical Modeling of the AIDS Virus in Epidemiology and Immunology*, Doctoral Thesis, Department of Mathematics, Tulane University, August 1991.

[20] D. E. KIRSCHNER (1992), *Examining a system of pseudo-cross diffusion equations*, International Journal on Scientific Computing and Modeling, 1, pp. 174–179.

[21] J. COOK, D. KIRSCHNER, AND J. MORGAN (1994), *Solution of the asymmetric mixing problem*, SIAM J. Appl. Math., submitted.

[22] S. LAYNE, J. SPOUGE, AND M. DEMBO (1989), *Measuring HIV infectivity*, in Mathematical and Statistical Approaches to AIDS Epidemiology, C. Castillo-Chavez, ed., Lecture Notes in Biomathematics, Vol. 83, Springer-Verlag, Berlin.

[23] J. D. MURRAY (1989) *Mathematical Biology*, Springer-Verlag, New York, pp. 400–450.

[24] N. PADIAN AND J. WILEY (1987), *Male to female transmission of HIV: Current results, infectivity rates and San Francisco population seroprevalence estimates*, Presented at III International Conference on AIDS, Washington, D.C.

[25] L. SATTENSPIEL (1990), *Modeling the spread of infectious disease in human populations*, Yearbook of Physical Anthropology, 33.

[26] G. A. SOD (1985), *Numerical Methods in Fluid Dynamics*, Cambridge University Press, New York.

[27] E. A. STANLEY (1995), *Partner selection in heterosexual populations*, in Mathematical Population Dynamics: Analysis of Heterogeneity, Vol. 1: Theory of Epidemics, O. Arino, M. Kimmel, M. Langlais, and D. Axelrod, eds., Wuerz, pp. 311–330.

[28] ———(1990), *Interagency Working Group Model Equations for the Transmission Dynamics of HIV/AIDS*, Los Alamos Technical Report LA-UR-90-2807.

[29] H. THIEME, AND C. CASTILLO-CHAVEZ (1989), *On the role of variable infectivity in the dynamics of the HIV epidemic*, in Mathematical and Statistical Approaches to AIDS Epidemiology, C. Castillo-Chavez, ed., Lecture Notes in Biomathematics, Vol. 83, Springer-Verlag, Berlin.

[30] J. WILEY (1990). *AIDS: The Second Decade*, National Academy of Science Publication for National Research Council, Chapter 6.