Autonomous Risk-Behavior Change, and Non-Linear Incidence Rate, in Models of Sexually Transmitted Diseases.

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ABSTRACT

Changes in risk-behavior effecting the numbers of partners taken per unit time, and the probability of transmission per infected partnership, due to direct response to disease incidence or prevalence, are incorporated in simple models of sexually transmitted diseases. Single-group homosexual models where risk behavior is encapsulated in a single parameter product are considered. An S-I-R-S model is studied, with arbitrary risk-behavior response, and results presented on global stability of the disease free equilibrium, and uniqueness and global stability of the endemic equilibrium. A model of S-I-loss type, more suitable for HIV/AIDS dynamics, is also presented. Given reasonable biological assumptions about the nature of risk-behavior change, global results are presented for the case where risk-behavior change depends only on infection prevalence and number of susceptibles, and uniqueness and local stability are proved for the more general case where the effect of AIDS incidence is incorporated. The effects of seasonality in risk-behavior are shown to be minimal. A delayed response in risk-behavior change is shown to destabilize the endemic equilibrium in some circumstances, causing the system to oscillate.

KEYWORDS: **risk behavior; sexually-transmitted disease; autonomous response; non-linear incidence, HIV/AIDS.**

1. INTRODUCTION

Changes in sexual behavior in response to information about the risks of acquiring sexually transmitted diseases (STDs) may play an integral role in determining the level of incidence or rate of spread of STDs particularly in this era of HIV/AIDS. Dramatic changes of behavior have been experienced in some homosexual communities in San Francisco (McKusick *et al.* 1985*a*; Winkelstein Jr. *et al.* 1987; CDC MMWR 1985; Shilts 1987) including the reduction in the number of steady partners from a median of 16 to a median of 1 (Third International Conference on AIDS, Washington D. C. 1987), a 40% reduction on the number of male partners per month and a 50% reduction on the frequency of anal intercourse without a condom (see the results of the AIDS Behavioral Research Project as reported in McKusick *et al.* 1985*b*). Reports on substantial behavioral changes among homosexually active men in cities such as New York (see Martin 1986, 1987) and Boston (see McCusker *et al.* 1988, Saltzman *et al.* 1987, McCusker *et al.* 1989) have also been documented. Yet, most mathematical models for the transmission of STDs fail to incorporate a method for modeling behavioral change. If such change is addressed at all, it is usually by computing the effect of a presumed change of a certain percentage in a parameter such as the contact rate.

Changes in behavior may affect *recruitment* of new susceptibles (see Fineberg 1988 and references therein), the level of sexual *activity*, as measured for example by the mean number of partnerships per unit time (see McKusick *et al.* 1985*b*), and the nature of sexual practices (see McCusker *et al.* 1988, 1990; Baldwin and Baldwin 1988; Evans *et al.* 1989; Shechter *et al.* 1988). It may also affect the *mixing* of individuals, for example, the systematic avoidance of individuals who are perceived to have a high probability of being infected (see Fox 1987, McCusker *et al.* 1988; van Griensven *et al.* 1989*b*; Wiktor *et al.* 1990; Curran *et al.* 1988 and Fineberg 1988). In each case, such changes might be incorporated in a model by specific (*i.e.* autonomous or state- but not time-dependent) functions, by

explicit functions of time, or by using differential equations to model the processes controlling the evolving rate or quantity.

Changes in recruitment reflect potential members of a population choosing not to enter it, and may therefore implicitly include behavioral changes that are external to the population under consideration. In this study we assume no change in recruitment. The effects of state dependent recruitment are analyzed, also in the context of STDs, in Brauer *et al.* (1991).

The work of Kinsey *et al.* (1948, 1953) opened the door for a series of behavioral studies on a variety of populations including pre-adolescents (Jackson 1975), college students (Dignan *et al.* 1985; Earle and Perricone 1986), male college students (Finger 1975), female college students (see DeBuono *et al.* 1990), Christian oriented individuals (Peretti 1976), registered prostitutes (Papaevangelou 1988), male prostitutes (Tirelli *et al.* 1988), intravenous drug users (Belongia *et al.* 1988; Coutinho 1990), men and women of color (D'Aquila *et al.* 1989), teenage women (Hofferth *et al.* 1987), swingers (Jenks 1985), hospital outpatient populations (Marmor *et al.* 1990), heterosexual women (Perlman *et al.* 1990), homosexuals (Reiche and Dannecker 1977; McCusker *et al.* 1990; Fay *et al.* 1989; Shilts 1987), and many others. The purpose of this article is to illustrate the possible effects on the probability of transmission and the number of partners taken per unit time on a population that is responding to the prevalence of a sexually-transmitted disease. A reduction on these parameters has already taken place in HIV transmission among homosexually active individuals in San Francisco. Unfortunately, as Finberg (1988) puts it: this reduction "brings scant comfort when half or more are already infected."

Given the objectives of this article, we concentrate on the study of the effects of STD prevalence in sexual activity and in sexual practices. We hope that our results will point out some of the potential differences between the dynamics predicted by classical models of STDs and those that incorporate

state-dependent effects. Obviously, other effects such as those due to the age-structure of the population need to be considered. Here, however, we keep things mathematically tractable by studying exclusively the effects of risk-behavior changes in activity alone on the simplest homosexually active population. The single group population under consideration allows for the simultaneous study of both activity and practices, since the two relevant parameters appear in a common product. In this manuscript, we study the homosexual one-group model, with constant recruitment, for the S-I-R-S type STD and present some results for models of the S-I-loss type, which may be more appropriate for AIDS. We model the behavioral responses autonomously using explicit functions of the "state variables" S, I, and R because homosexually-active males do change their behavior in response to their perceptions of the incidence or prevalence of infection in their community (see for example Wiktor *et al.* 1990). We note that the effect of this choice is to introduce a non-linear incidence function into the model, and therefore our results bear relation to previous work on epidemic models with non-linear incidence (albeit not motivated by considerations of behavioral change rates). Explicitly, the incidence assumed in our model has the form

$$\beta c F(S,I,R) \frac{I}{S+I+R} S,$$

where $S(t)$, $I(t)$ and $R(t)$ are, respectively, the numbers of susceptible individuals, infected/infectious individuals, and removed or temporarily immune individuals; c represents the mean maximal contact rate before infection has entered the population; and β is the fraction of contacts with infected partners that result in transmission of the infection. The function $F(S,I,R)$ takes values between 0 and 1, and represents the fractional reduction in the *effective* maximal contact rate βc when the state variables have value S, I, and R. The reduction modeled by $F(S,I,R)$ could be due to a decrease in contacts (activity c) or in transmission (practices β). In subsequent sections, we present our assumptions about the function F and the consequences from such assumed functional relationships. Ascertaining the precise form of F within a given population at a given time may be very difficult and, consequently,

we assume a general form that can be fitted to parameters. In this paper, we concentrate on determining what qualitative conclusions can be drawn when very general and plausible assumptions are made about the F. A different approach is explored in Palmer *et al.* (1991).

In classical epidemic models, the incidence is generally taken to be proportional to SI which corresponds to $F = 1$ (F is dimensionless), and a constant total population $T = \mathrm{S} + I$ which is absorbed into the constant of proportionality. For populations that are not constant (and this includes any cases where there is disease-induced mortality) the form $\beta cSI/T$ is used by many authors, specially in models for AIDS (see Anderson *et al.* 1986; Blythe and Anderson 1988*a,b*; Jacquez *et al.* 1988; Castillo-Chavez 1989*a*; Castillo-Chavez *et al.* 1989*a,b,c,d,e*; Castillo-Chavez 1989*b* and references therein; Brauer 1990*a,b*; Pugliese 1990*a,b*; Sattenspiel and Castillo-Chavez 1990; Thieme and Castillo-Chavez 1989, 1990). General incidence terms of the form $I\,H(I,S)$, where H is a non-negative function that is non-decreasing with respect to S, were introduced by Liu *et al.* (1986, 1987); in these papers, $H(I,S) = \beta\, I^{p-1} S^{q}$, where $p \geq 1$, $q > 0$. Hethcote *et al.* (1989) treated a model with delay and non-linear incidence of this type, and Hethcote and van den Driessche (1991) dealt with $H(I,S) = \beta\, g(I)\, S/I$, where g is a saturating function. This type of incidence rates is capable of generating sustained oscillations, for a review of mechanisms capable of generating sustained oscillations; in epidemiological models, see Hethcote and Levin (1989).

Among other work related to the subject of this paper we may mention Anderson *et al.* (1989) and Gupta *et al.* (1989). These authors study behavioral change by considering a set of behavioral rules that require changes both in levels of sexual activity, and in mixing structures. These behavioral changes, in their work, are imposed by the fact that the population sizes of higher activity classes are reduced by mortality more rapidly than the population sizes of lower activity classes. thus, intentional changes in behavior in response to education or perceived threat are not included. Palmer *et al.* (1991)

study the effects of state-dependent mixing in single group models through the general formulation of Busenberg and Castillo-Chavez (1989, 1991), in single group models. Our approach here is more empirical in nature (the definition of F) than that of Palmer *et al.* (1991) and the dynamical behavior exhibited by both type of models can be different under similar mixing assumptions.

2. AN S-I-R-S MODEL

The model used in this section has the form

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - B - \mu S + \delta R \\ \frac{dI}{dt} &= B - (\gamma+\mu) I \\ \frac{dR}{dt} &= \gamma I - (\delta+\mu) R, \end{aligned} \tag{2.1}$$

with $S(0) = S_o \geq 0$, $I(0) = I_o > 0$, $R(0) = R_o \geq 0$. The incidence B is given by

$$B = \beta c \, F(S, I, R) \, S \frac{I}{T}, \tag{2.2}$$

where

$$T \equiv S + I + R. \tag{2,3}$$

Here $\Lambda > 0$ is the rate of recruitment of new susceptibles; $\mu > 0$ is the rate of removal of of individuals

from the population (for example by death); $\gamma > 0$ is the recovery rate of infecteds into the (removed or) immune class; $\delta > 0$ is the rate of loss of immunity (or return to the susceptible class); $c > 0$ is the maximal average sexual contact rate of the population; and $\beta > 0$ is the proportion of effective sexual contacts which result in the transfer of infection from an infected to a susceptible individual. All these parameters are assumed to be constant. As discussed in Section 1, $F(S,I,R)$ is a dimensionless (because of the definition of β and c) factor that modifies βc mostly in response to disease prevalence in the population.

In Eq (2.2), the factor I/T is the proportion of a susceptible's contacts which are infected, and hence infectious in this model. We do not assume that T is a constant. Systems with F equal to a constant, of the same or similar form as (2.1) but with distributions of durations of the infected and removed classes, have been studied by Hethcote and van den Driessche (1991). Following the approach used by Adler *et al.* (1989) in host-parasitoid models, we make the following hypothesis on F:

$$0 \leq \mathrm{F(S,I,R)} \leq \mathrm{F}\left(\frac{\Lambda}{\mu},0,0\right) \leq \mathrm{F}(\infty,0,0) = 1 \tag{2.4}$$

for $S \geq 0$, $I \geq 0$, $R \geq 0$. From (2.1) we note that

$$\frac{\mathrm{dT(t)}}{\mathrm{dt}} = \Lambda - \mu \mathrm{T}, \qquad \mathrm{T}(0) > 0, \tag{2.5}$$

and consequently $T(t)$ tends to Λ/μ as $t \to \infty$. In the absence of infection, $S(t)$ will therefore be close to Λ/μ. Realistic versions of this model will assume that $F(\Lambda/\mu,0,0)$ is very close to its limiting value $F(\infty,0,0) = 1$ when the population Λ/μ is sufficiently large that we may ignore demographic stochastic effects.

Our mathematical results involve hypotheses on the signs of the partial derivatives $\partial F/\partial S$, $\partial F/\partial I$ and $\partial F/\partial R$. There are several possibilities and we choose the following:

$$\frac{\partial \mathrm{F}}{\partial \mathrm{S}} \geq 0, \quad \frac{\partial \mathrm{F}}{\partial \mathrm{I}} \leq 0,$$

i.e. an increasing number of infectives in the population is likely to induce a reduction in activity whereas an increasing number of susceptibles is not. Note however that there is not an obvious way of modeling the effect of an increasing number of recovered individuals. This may result in an increasing awareness of the risks and lead to a reduced contact rate or it may through the presence of a large number of individuals known to be non-infectious, inspire confidence and lead to an increase rate of contact. As we present our results, we shall make clear exactly what hypotheses are being considered. The following inequality

$$\frac{\partial \mathrm{F}}{\partial \mathrm{S}} \geq \frac{\partial \mathrm{F}}{\partial \mathrm{R}} > \frac{\partial \mathrm{F}}{\partial \mathrm{I}}, \tag{2.5}$$

will often be required.

Lemma 1 The system (2.1) is well-posed. That is, given initial conditions $S(0) \geq 0$, $I(0) \geq 0$, $R(0) \geq 0$, there is a unique solution $S(t)$, $I(t)$, $R(t)$ that exists for all $t \geq 0$, and satisfies $S(t) \geq 0$, $I(t) \geq 0$, $R(t) \geq 0$. Moreover,

$$\lim_{t \to \infty} \mathrm{T(t)} = \frac{\Lambda}{\mu}, \tag{2.6}$$

where $T(t) = S(t) + I(t) + R(t)$. To prove this, we show that the region $S \geq 0$, $I \geq 0$, $R \geq 0$ is an invariant region for (2.1). Indeed, when $S = 0$ we have $dS/dt > 0$, hence a solution trajectory must satisfy $S \geq 0$, and when $R = 0$ we have $dR/dt > 0$, so that $R \geq 0$. When $I = 0$ we have $dI/dt = 0$, and

further argument is needed. Integrating $dI/dt + (\gamma+\mu)\, I = B(t)$, we obtain

$$\mathrm{I(t)} = \mathrm{I(0)}\, \mathrm{e}^{-(\gamma+\mu)\mathrm{t}} + \mathrm{e}^{-(\gamma+\mu)\mathrm{t}} \int_0^{\mathrm{t}} \mathrm{B(t\text{-}s)}\, \mathrm{e}^{(\gamma+\mu)\mathrm{s}}\, \mathrm{ds},$$

and this shows that $I(t) \geq 0$. From (2.5),

$$\mathrm{T(t)} = \mathrm{T(0)}\, \mathrm{e}^{-\mu \mathrm{t}} + \frac{\Lambda}{\mu}\left(1 - \mathrm{e}^{-\mu \mathrm{t}}\right),$$

and (2.6) follows. Since $S(t) \leq T(t)$, $I(t) \leq T(t)$, $R(t) \leq T(t)$, the variables S, I, R are bounded, the flow occurs in a compact region of $\mathfrak{R}^3$, and $S(t), I(t), R(t)$ exist for all $t \geq 0$.

We next look for equilibria of (2.1) which are defined by solutions to the equations

$$\Lambda - \mathrm{B} - \mu \mathrm{S} + \delta \mathrm{R} = 0$$

$$\mathrm{B} - (\gamma+\mu)\, \mathrm{I} = 0 \tag{2.7}$$

$$\gamma \mathrm{I} - (\delta+\mu)\, \mathrm{R} = 0.$$

One equilibrium is $(S, I, R) = (\Lambda/\mu, 0, 0)$, the disease-free or trivial equilibrium. Other equilibria are possible, their number depending primarily on the assumptions on F, and on parameter values.

Theorem 1 Assume that (2.4) holds and that

$$R_0 \equiv \frac{\beta \mathrm{c}}{\gamma+\mu}\, \mathrm{F}\left(\frac{\Lambda}{\mu}, 0, 0\right) < 1, \tag{2.8}$$

then the disease-free equilibrium attracts all solutions, that is,

$$\lim_{t\to\infty} \big(S(t), I(t), R(t)\big) = \big(\tfrac{\Lambda}{\mu}, 0, 0\big).$$

If $R_0 > 1$, then the disease-free equilibrium is (locally) unstable.

Proof The proof is given in Appendix A.

The number R_0 in (2.8), the basic reproductive number, is a dimensionless quantity representing the expected number of secondary cases caused by one infective during the mean infectious period $1/(\gamma+\mu)$ in a population of susceptibles, here fixed at the equilibrium size Λ/μ.

We next consider the number and stability of endemic equilibria, which may exist when $R_0 > 1$. Suppose that (S^*, I^*, R^*) is an equilibrium of (2.7) with $I^* > 0$, and let $F^* = F^*(S^*, I^*, R^*)$, and $T^* = S^* + I^* + R^*$. Then the equilibrium satisfies the following system of equations:

$$\begin{aligned} \Lambda - \mu S^* &= \beta c\, F^*\, S^*\, \frac{I^*}{T^*} - \delta R^*, \\ \beta c\, F^*\, S^*\, \frac{I^*}{T^*} &= (\gamma+\mu) I^*, \\ \gamma I^* &= (\delta+\mu) R^*. \end{aligned} \tag{2.9}$$

By (2.6), $T^* = \Lambda/\mu$. For $I^* \neq 0$ we have from (2.9) that

$$\frac{S^*}{T^*} = \frac{\gamma+\mu}{\beta c F^*},$$

and

$$\mu - \mu S^* = \frac{\Lambda - \mu S^*}{T^*} = \frac{(\delta+\mu) I^* - \delta R^*}{T^*} = \left(\delta+\mu-\frac{\gamma\delta}{\delta+\mu}\right)\frac{I^*}{T^*}.$$

Therefore if we let $q \equiv \gamma/(\delta+\mu)$ and

$$\eta \equiv \frac{\beta c F^*}{\gamma+\mu}, \tag{2.10}$$

then we obtain

$$\frac{S^*}{T^*} = \frac{1}{\eta}, \quad \frac{I^*}{T^*} = \frac{1}{1+q}\left(1-\frac{1}{\eta}\right), \quad \frac{R^*}{T^*} = \frac{q}{1+q}\left(1-\frac{1}{\eta}\right). \tag{2.11}$$

The equation (2.10) may now be written in the form $R_o F^* = F_o \eta$, where $F_o \equiv F(\Lambda/\mu,0,0)$. That is, by (2.11)

$$R_o F\left(\frac{T^*}{\eta}, \frac{T^*}{1+q}\left(1-\frac{1}{\eta}\right), \frac{q T^*}{1+q}\left(1-\frac{1}{\eta}\right)\right) - F_o \eta = 0, \tag{2.12}$$

where $T^* = \Lambda/\mu$.

Any solution of (2.12) for which $S^* < T^* = \Lambda/\mu$ (*i.e.* $\eta > 1$) yields an endemic equilibrium for (2.1). In discussing the existence and number of such equilibria we note that

$$T(t) = T(0)\, e^{-\mu t} + \frac{\Lambda}{\mu}\left(1 - e^{-\mu t}\right),$$

and $R(t) = T(t) - S(t) - I(t)$. Therefore we can replace (2.1) by the non-autonomous system

$$\frac{dS}{dt} = \Lambda - \beta c\, F\left(S, I, T(t)-S-I\right) S \frac{I}{T(t)} - \mu S + \delta\,(T(t)-S-I) \tag{2.13}$$

$$\frac{dI}{dt} = \beta c\, F\Big(S, I, T(t)-S-I\Big)\, S\, \frac{I}{T(t)} - (\gamma+\mu) I.$$

Since $T(t)$ tends to Λ/μ as $t \to \infty$, it may be reasonable to expect that the asymptotic behavior of solutions of (2.13) in a compact region will be the same as the asymptotic behavior of the limiting autonomous system

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta c\, F\Big(S, I, \tfrac{\Lambda}{\mu}-S-I\Big)\, S\, \frac{I}{\Lambda/\mu} - \mu S + \delta\big(\tfrac{\Lambda}{\mu}-S-I\big), \\ \frac{dI}{dt} &= \beta c\, F\Big(S, I, \tfrac{\Lambda}{\mu}-S-I\Big)\, S\, \frac{I}{\Lambda/\mu} - (\gamma+\mu) I. \end{aligned} \tag{2.14}$$

We have ran a large number of simulations in epidemiological situations of interest which suggest that the dynamic behavior of (2.13) and (2.14) is qualitatively the same. We conjecture that whenever the number of endemic equilibria is finite (the only situation of epidemiological relevance) then the local behavior of both models is qualitatively the same. A formal proof of this result is in preparation. In the rest of this section we make no differentiation between the original and the two-dimensional limiting model to facilitate the discussion through the elimination of these technical considerations.

Theorem 2 If $\frac{\partial F}{\partial S} > \frac{\partial F}{\partial I}$ for $S > 0$, $I > 0$, Eq (2.14) has no limit cycles in the region $S > 0$, $I > 0$. Therefore we conjecture that solutions of (2.13) cannot asymptotically approach a limit cycle.

Proof By an application of the Dulac Criterion, which we show in Appendix B.

We next study the curves in the (S, I) plane determined from setting $dS/dt = 0$ and $dI/dt = 0$. The equation $dI/dt = 0$ yields $I = 0$ and

$$F\left(S, I, \frac{\Lambda}{\mu}-S-I\right)S = \frac{(\gamma+\mu)\Lambda}{\beta c\mu}.$$

Implicit differentiation gives

$$\frac{dS}{dI} = \frac{S(F_R - F_I)}{F + S(F_S - F_R)},$$

where

$$F_S \equiv \frac{\partial F}{\partial S}, \quad F_I \equiv \frac{\partial F}{\partial I}, \quad F_R \equiv \frac{\partial F}{\partial R}.$$

Under the assumption

$$\frac{\partial F}{\partial S} \geq \frac{\partial F}{\partial R} > \frac{\partial F}{\partial I}, \tag{2.15}$$

we see that $dS/dI > 0$, that is, S is an increasing function of I (S is also positive when $I = 0$). The equation $dS/dt = 0$ gives

$$\Lambda - \frac{\beta c\mu}{\Lambda} SIF\left(S, I, \frac{\Lambda}{\mu}-S-I\right) + \frac{\delta\Lambda}{\mu} - (\delta+\mu)S - \delta I = 0.$$

Implicit differentiation gives

$$\frac{dS}{dI} = \frac{-\beta c\mu SF + \beta cSI(F_R - F_I) - \delta\Lambda}{\beta cIF + \beta c\mu SI(F_S - F_R) + (\delta+\mu)\Lambda}.$$

The denominator is positive when (2.15) holds but the sign of the numerator is not uniquely determined. When $I = 0$, we have $S = \Lambda/\mu$, and the slope is

$$\frac{dS}{dI} = \frac{-\beta c F_o - \delta}{\delta+\mu} = -\left[\frac{(\gamma+\mu)R_o + \delta}{\delta+\mu}\right],$$

so that $R_o > 1$ implies $dS/dI > -1$ and, consequently, the curve penetrates into the allowable region $S \geq 0$, $I \geq 0$, $S + I \leq \Lambda/\mu$. The existence and number of endemic equilibria depends on the numbers of crossings of this curve, if any, in this region. It does not seem to be possible on the basis of phase-plane analysis to determine the number of crossings or to determine their stability. However, examination of (2.12) suggests sufficient conditions for uniqueness. If we define

$$G(\eta) \equiv R_o F\Big(h_1(\eta), h_2(\eta), h_3(\eta)\Big) - \eta,$$

where the $\{h_i\}$ are the argument functions of Eq (2.12), then endemic equilibria are each characterized by a η such that $G(\eta) = 0$. From (2.11) and (2.12) we note that as $\eta \to 1$, $F \to F_o$, and $G(\eta) \to (R_o - 1) > 0$. Further, as $0 < F \leq 1$ by (2.4), for $\eta > R_o$ we have $G(\eta) < 0$. Hence for continuous F there is at least one $\eta \in (1, R_o)$ for which $G(\eta) = 0$, and thus at least one endemic equilibrium of (2.1). Note that

$$\begin{aligned}
\frac{dG}{d\eta} &= R_o\left\{\frac{\partial F}{\partial h_1}\frac{dh_1}{d\eta} + \frac{\partial F}{\partial h_2}\frac{dh_2}{d\eta} + \frac{\partial F}{\partial h_3}\frac{dh_3}{d\eta}\right\} - 1, \\
&= R_o\frac{T^*}{\eta^2}\left\{-\frac{\partial F}{\partial h_1} + \frac{1}{1+q}\frac{\partial F}{\partial h_2} + \frac{q}{1+q}\frac{\partial F}{\partial h_1}\right\} - 1 \\
&= R_o\frac{T^*}{\eta^2}\left\{-\left(\frac{\partial F}{\partial S}\right)^* + \frac{1}{1+q}\left(\frac{\partial F}{\partial I}\right)^* + \frac{q}{1+q}\left(\frac{\partial F}{\partial R}\right)^*\right\} - 1,
\end{aligned}$$

and, consequently, if $dG/d\eta < 0$ there is only one feasible $\eta \in (1, R_o)$ such that $G(\eta) = 0$. Thus the inequality

$$\frac{\partial F}{\partial S} \geq 0, \quad \frac{\partial F}{\partial I} \leq 0, \quad \frac{\partial F}{\partial R} \leq 0, \tag{2.16}$$

in combination with the assumption $R_o > 1$ provide sufficient conditions for the existence of a unique

endemic equilibrium of (2.1). This restricts us to cases where the behavioral response to increasing number of recovereds R cannot involve an increase in risk activities.

In numerical simulations with several "reasonable" functions that satisfy (2.15), but not necessarily (2.16), we have found a unique locally stable positive equilibrium when $R_0 > 1$. However, it should be possible to construct situations for which multiple endemic equilibria are possible. Indeed in Palmer *et al.* (1991) this has been accomplished but through a different mechanism.

3. AN HIV/AIDS MODEL

Models for HIV/AIDS infection differ from the SIRS models of the previous sections, in that there is no known recovery, and people with AIDS do not mix sexually to any significant extent. Writing $S(t)$ and $I(t)$ for the susceptibles and HIV-infecteds, and $A(t)$ for people with AIDS, a simple one-group model for a homosexually-active mixing population, is given by

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta c\, F(S,I,A)\frac{SI}{S+I} - \mu S, \\
\frac{dI}{dt} &= \beta c\, F(S,I,A)\frac{SI}{S+I} - (\gamma+\mu) I, \qquad (3.1) \\
\frac{dA}{dt} &= \gamma I - (\alpha+\mu) A,
\end{aligned}$$

where Λ, β, c, μ and γ are as in the SIRS model, and α is the *per capita* death rate from AIDS. In

general and without loss of generality we require $S+I+A \leq \Lambda/\mu$ for all t, $S(0) > 0$, $I(0) > 0$, and $A(0) \geq 0$. We study (3.1) for various hypotheses concerning F: as a function of any or all of S, I and A (Section 3.1); as a seasonally varying parameter (Section 3.2); as a delayed function of I (Section 3.3).

3.1 State dependent F

When F is a function of S and I only, the A equation drops out, and we are left with an SI model with non-linear incidence. Simon and Jacquez (1991) have analyzed a broad class of such models of this type with incidence of the form $\beta c H(S,I)$ for some H. We may recast their results using our notation as follows: there is a globally stable disease-free equilibrium for $R_0 < 1$, and a globally stable endemic equilibrium for $R_0 > 1$, for $F(S,I)$ such that

$$F(S,0) = 0 ,$$

$$\frac{1}{F}\left(\frac{\partial F}{\partial I}\right) \leq -\frac{S}{I(S+I)} < \frac{1}{F}\left(\frac{\partial F}{\partial S}\right), \tag{3.2}$$

with

$$R_0 \equiv \frac{\beta c}{\gamma+\mu} F\left(\frac{\Lambda}{\mu},0\right).$$

The system of equations (3.1) has also been studied in the context of host-parasitoid interactions, with $F(S,I,A)$, by Adler *et al.* (1989). These authors studied the case

$$F(\infty,0,0) = 1, \quad F(0,I,A) = 0,$$

$$\frac{1}{F}\left(\frac{\partial F}{\partial S}\right) > -\frac{I}{S(S+I)} , \tag{3.3}$$

$$\frac{1}{F}\left(\frac{\partial F}{\partial I}\right) \leq -\frac{S}{I(S+I)}$$

$$\frac{1}{F}\left(\frac{\partial F}{\partial A}\right) \leq \frac{1}{A}.$$

Adler *et al.* (1989) found a transition between a stable unique disease-free equilibrium and a unique endemic equilibrium when $R_0 = \beta c/(\gamma+\mu)$ increased through unity (that is, a transcritical bifurcation). Their analysis did not require that $S+I+A \leq \Lambda/\mu$ (it is not a disease-transmission model) and included general distributions for the duration of the I stage, rather than just an exponential distribution. In all the cases studied in Adler *et al.* (1989) the existence of a unique endemic equilibrium implied its local stability.

We may extend some of these results slightly, using the methods of Section 2, and $F(\infty,0,0) = 1$ (with the understanding that $F(\Lambda/\mu,0,0)$ is close to this limiting value for any reasonably large Λ/μ). Using the method of proof of Theorem 1, for the case $F(S,I,A)$ one shows that the disease-free equilibrium $(\Lambda/\mu,0,0)$ is unstable for $R_0 = \beta c/(\gamma+\mu) > 1$. Following the arguments in Lemma 1 and Theorem 1 one shows that for $R_0 < 1$, $I(t) \to 0$ and $A(t) \to 0$ as $t \to \infty$. If $T = S+I$, that is, if A-individuals are not sexually-active then

$$\frac{dT}{dt} + \mu T = \Lambda - \alpha A \leq \Lambda, \tag{3.4}$$

$\lim \sup S(t) = \Lambda/\mu$, and the disease-free equilibrium attracts all solutions, regardless of the form of F. We cannot perform a global analysis of (3.1) using the method of Section 2, because Eq (3.4) does not permit the use of the limiting argument employed before, but we note that for $F = F(S,I)$, Dulac's test (performed as in the proof of Theorem 2 in Appendix B) rules out oscillations provided that

$$\frac{\partial F}{\partial S} \geq \frac{\partial F}{\partial I}. \tag{3.5}$$

Condition (3.5) is slightly less restrictive than (3.2), but adds no practical insight, as one would naturally expect on biological grounds that $\partial F/\partial S > 0$ and $\partial F/\partial I \leq 0$.

For $F = F(S,I,A)$, the endemic equilibrium may be calculated much as in Section 2, with

$$\eta \equiv \left[\frac{S^*}{T^*}\right]^{-1}, \tag{3.6}$$

and

$$T^*(\eta) = \frac{\Lambda/\mu}{\left[1+\frac{\gamma}{\mu}\frac{\eta-1}{\eta}\right]}. \tag{3.7}$$

Hence η is given (and hence the steady-state population values) by any solution of the equation

$$R_0 F\left(\frac{1}{\eta}T^*(\eta), \frac{(\eta-1)}{\eta}T^*(\eta), \gamma\frac{(\eta-1)}{\eta}T^*(\eta)\right) - \eta = 0, \text{ for } \eta \geq 1. \tag{3.8}$$

By definition, $R_0 > 1$ at endemic equilibrium, so as $\eta \to 1$, the LHS of Eq (3.8) becomes $R_0 - 1 > 0$; further, as $0 \leq F \leq 1$, for $\eta \geq R_0$ the LHS of (3.8) is negative. Therefore for continuous F we are guaranteed at least one solution (endemic equilibrium) in the interval $1 < \eta < R_0$. For uniqueness, it is sufficient to assume that $dF/d\eta \leq 0$ for $1 < \eta < R_0$, which we may write as the requirement

$$\left(\frac{\partial F}{\partial S}\right)^* - \frac{\mu}{\gamma+\mu}\left(\frac{\partial F}{\partial I}\right)^* - \frac{\mu\gamma}{(\gamma+\mu)(\alpha+\mu)}\left(\frac{\partial F}{\partial A}\right)^* > 0. \tag{3.9}$$

Since we may reasonably expect

$$\left(\frac{\partial F}{\partial S}\right) \geq 0, \quad \left(\frac{\partial F}{\partial I}\right) \leq 0, \quad \left(\frac{\partial F}{\partial A}\right) \leq 0 \tag{3.10}$$

under reasonable biological assumptions (there is no question of risk activity increasing with increasing numbers of AIDS cases), inequality (3.9) is always satisfied, and there is a unique endemic equilibrium.

Theorem 3 For $F = F(S,I,A) \in [0,1]$, constrained by the inequalities (3.10), and $R_o > 1$, then the unique endemic equilibrium (S^*, I^*, A^*) of the system (3.1) is locally stable.

Proof The proof is by standard application of the Routh-Hurwitz criteria; an outline is given in Appendix C.

Numerical realizations of Eqs (3.1) strongly suggest global stability of the endemic equilibrium for $F = F(S,I,A)$, subject to constraints (3.10). The results of Adler *et al.* (1989) suggest that stability holds for the case of a distributed delay in the I-stage.

3.2 Seasonality in F

Although the stress in this study is on autonomous F, it is of interest to see how the system reacts to externally forced fluctuations in F, for example seasonality in the average activity level c, or random fluctuations in βc. Consider an F of the form $F(t) = \overline{F}[1 + af(t)]$, where $\overline{F}$ is the long-term average, and $2a$ the amplitude of the fluctuations $f(t)$ is sufficiently small to maintain the interpretation of model values; f may be periodic, aperiodic, or even a noise source. The analysis of response to F is straightforward (*e.g.* Nisbet and Gurney 1982), and hinges on two parameters,

$$\Gamma = q + \zeta - 1 \quad \text{and} \quad \omega_o^2 = \left(\frac{\zeta - 1}{\zeta}\right)(q + \zeta - 1),$$

where

$$\zeta \equiv \frac{\beta c \overline{F}}{\gamma+\mu} = R_o \overline{F} > 1 \quad \text{and} \quad q \equiv \frac{\mu}{\gamma+\mu} < 1.$$

If $\Gamma > 2\omega_o$ the system can be entrained by fluctuations with low enough frequency, but no resonant quasi-cyclic behavior can occur; for $\Gamma < \omega_o$, quasi-cycles can arise, even if the driver F is aperiodic. We find that for $\zeta > 4$, Γ always exceeds $2\omega_o$, and only fluctuations with period greater than $1/(\beta c\overline{F}-\gamma)$ will capture the system dynamics. For example with $\beta c = 0.5$ year^{-1}, $\overline{F} = 0.6$, and $\gamma = 0.1$ year^{-1}, periods longer than 5 years are needed to capture the system. For $\zeta < 4$, if $0 < q < (\zeta-1)(4-\zeta)$ there is a small region of parameter space where (3.1) exhibits damped oscillations in its approach to equilibrium; however, these are generally almost un-noticeable, and the coherence number (number of cycles during which system memory persists, Nisbet and Gurney (1982)) never exceeds $9/(16\pi) \simeq 0.179$, so that no visible quasi-cyclic behavior can be expected. For $\zeta < 4$ and $q > (\zeta-1)(4-\zeta)$, the behavior is the same as for $\zeta > 4$. We may conclude that periodic external fluctuations will have a visible effect only for such low frequencies that the transient dynamics will rarely be altered, and that there will be no discernible resonance effect due to random fluctuations.

3.3 Delayed response

If changes in F occur in response to the system variables after some delay (*e.g.* a reporting delay), then there exists the possibility that the endemic equilibrium can destabilize, and oscillations result. If we write Eqs (3.1) in the scaled form $S' = S \Big/ (\Lambda/\mu)$, $I' = I \Big/ (\Lambda/\mu)$, $t' = (\gamma+\mu)t$ (and hereafter drop the primes), and look at the particular case where F varies in response to delayed prevalence I only, we then arrive at the following set of equations:

$$\frac{dS}{dt} = q(1-S) - R_o F(I(t-\tau)) \frac{SI}{S+I},$$

(3.11)

$$\frac{dI}{dt} = R_o F(I(t-\tau)) \frac{SI}{S+I} - I,$$

with

$$F(0) = 1,$$

$$\frac{\partial F}{\partial I} \leq 0, \quad I \geq 0. \tag{3.12}$$

Note that this form of control could reflect either a response to the total number of infecteds or to the rate of incidence of new AIDS cases ($\gamma I(t)$ in the unscaled model, with exponentially distributed infectious period). The endemic equilibrium analysis proceeds exactly as before, with η given as the (unique given Eq (3.12)) solution to

$$R_o F\left(\frac{q(\eta-1)}{q+\eta-1} \right) - \eta = 0, \quad 1 \leq \eta < R_o. \tag{3.13}$$

This system can produce oscillations for suitable parameter values. For example, say $F(I) = e^{-I/\theta}$; an example of the stability boundary on the (θ,τ) plane is shown in Fig (1), and Fig (2) shows a realization of the model for particular parameter values producing oscillations. If the quantity

$$\psi \equiv -\frac{1}{F^*} \left(\frac{\partial F}{\partial I} \right)^* \tag{3.14}$$

is sufficiently large, then there exists a critical value of the delay τ above which the system Eq (3.11) is unstable (see Appendix D). In *unscaled* units of time (t not t') the minimum value of this critical delay is

$$\tau_{min} = \frac{\pi}{2(\gamma+\mu) ln(R_o)} = \frac{\pi}{\left[2(\gamma+\mu) ln\left(\frac{\beta c}{\gamma+\mu} \right) \right]} \tag{3.15}$$

for $R_o > 1$. Furthermore

$$\frac{\partial \tau_{\min}}{\partial \beta c} < 0$$

and

$$\frac{\partial \tau_{\min}}{\partial (\gamma+\mu)} \begin{cases} > 0, & R_o < e \\ < 0, & R_o > e \end{cases},$$

where $e \simeq 2.71828$. From the point of view of control of the infection, the value of $\tau_{\min}$ is a direct measure of stability: the larger this value is, the better. As most control measures will aim at decreasing βc, it is advantageous to know that this also increases stability in the presence of a reporting/response delay. Changes in the mean time spent active and infectious, $1/(\gamma+\mu)$, are ambiguous with respect to this measure of stability. In Appendix D we outline the local stability analysis of the unique endemic equilibrium of Eq (3.11).

It is clear that a delayed response in the change in risk-behavior adds considerable complication to the process of control of STDs. If the response is strong (meaning a small characteristic value θ in the example studied here) then the potential for destabilizing the dynamics exists, for long enough delay; further, the minimum length of delay for destabilization changes with the key parameters βc and $\gamma+\mu$, and not always in an simple manner. For parameter values that seem plausible for AIDS ($0.1 < \beta c < 1.0$ year^{-1}, $0.1 < \gamma+\mu < 0.15$ year^{-1}), the delay would have to be of the order of several years before destabilization could occur.

4. DISCUSSION

The study of the role of human sexuality and behavior has been significantly advanced but in the context of the HIV/AIDS epidemic. As J. H. Gagnon (1988) puts it "A strong consequence of this disease

is that *sex itself can become confused with disease and being sexual in various ways becomes treated as an illness or as an evidence of an illness.*" The HIV/AIDS epidemic has had strong impact in almost all aspects of human discourse because in the words of Grmek (1990) "AIDS, both unpredicted and unpredictable within the framework of the old nosology, is the first of the postmodern plagues." Over the last few years a tacit agreement has been reached that the most effective way of preventing the spread of HIV/AIDS, and consequently of other STDs, is through substantial albeit not clearly quantified behavioral changes.

Fineberg (1988) claims that "Long-term protection of an individual from infection requires extreme changes in risk-taking behavior" while at the population level "Partial shifts toward safer practices may be epidemiologically important in retarding the rate and extent of spread of infection." He adds that "Short of stifling the epidemic altogether, prolonging the course of its development can provide time for other strategies of control and treatment to come into being." We have written this manuscript in order to explore qualitatively the consequences of population-level behavioral changes that result from perceived global (rather than individual) risks using mathematically tractable models.

The evaluation of control measures that may reduce or slow down the spread of the HIV/AIDS epidemic can be quantitatively evaluated using mathematical models of the type discussed here. Unfortunately, one of the first results obtained from dynamic models of this type is discouraging: it indicates that the effects of control measures may not been seen within a politically reasonable time-scale due to the long period of infectiousness and the fact that most infected individuals are essentially new infectees (see Castillo-Chavez *et al.* 1989*a,b,c,d*). However, there is strong evidence that preventive measures do work. Finberg (1988) provides an example that took place during World War II (taken from Brandt, 1985): the Army warned the troops about the dangers of STDs and promoted condoms in a variety of ways, even while critics forced the withdrawal of several films on the basis that they

promoted promiscuity. The Army sold or distributed freely 50,000,000 condoms every month from 1940 to 1943 (penicillin was not yet available), reducing by 45% the rate per 1000 individuals of STDs. Preventive measures can work and currently provide the most promising avenue for the reduction of HIV/AIDS. Unfortunately, the success of preventive measures depend strongly on the nature of perceived risks. Substantial changes in behavior took place in San Francisco in response to HIV/AIDS prevalence but they came very late. Less conclusive experimental evidence is provided below.

DeBuono *et al.* (1990) studied the sexual behavior of women in 1975, 1986, and 1989. They found little change in sexual practices, except for an increase in the percentage of women that use condoms (less than 50%). Furthermore, they found no change in the number of sexual partners or the frequency of fellatio, cunnilingus, or anal intercourse. Baldwin and Baldwin (1988) surveyed college students and found that "The most consistent predictors of cautious sexual behaviors were age at first intercourse, average number of [sexual] partners per year, being female and using seatbelts while driving. Sexual practices were not influenced by religiosity or having had a course in human sexuality or religiosity." Their results showed that students appear to be "engaging in few activities that would protect them from contracting the human immunodeficiency virus (HIV)." We note that the reported incidence of AIDS and the estimated incidence of HIV among college students is very low. McCusker *et al.* (1988) studied a population of 270 homosexually-active (more than one sexual partner per month) healthy (no AIDS symptoms) individuals in Boston, of which 21% were aware of their HIV status. They found that "Except for the number of steady partners, the levels of all sexual activities of all groups of study participants declined over time. No effects of test awareness of antibody status were found on protective behavior for receptive anogenital contact. Elimination of unprotected insertive anogenital contacts (by elimination of the practice or by condom use) was reported somewhat more often among seropositive men who became aware of their test result." Knowledge of HIV status had some effect in reducing risky behavior among the individuals in this population. Wiktor *et al.* (1990) studied two

different populations of healthy homosexually-active individuals. Group 1 (G1) consisting of 134 homosexuals residing in New York and Washington D.C. and Group 2 (G2) consisting of 139 homosexuals residing in Copenhagen and Aarhus, Denmark. Their sexual practices and status were monitored over a period of 12 months. 63% of G1- and 70% of G2-individuals participated in anal intercourse during this period. Wiktor *et al.* (1990) report that "Knowledge of one's own HIV status by itself did not have a significant effect on participation in anal intercourse, partner number, or condom use." They note, however, those 23% of G1 and 24% of G2 men who always asked potential partners about their HIV status were very unlikely to engage with a partner of opposite HIV status. G2 were more likely to practice anal intercourse and more likely to be in monogamous relationships. 14% of G1 and 21% of G2 were not aware of their HIV status, and 52% of G1 and 31% of G2 individuals had anal intercourse with men of unknown status. Although these results can be interpreted in a variety of ways, it appears that knowledge of HIV/AIDS prevalence in one's community may, above a certain threshold, have a significant effect in sexual practices and behaviors. Given the fact that most HIV infected individuals are unaware of their own status, as well as the levels of HIV infection in their communities, the current evaluation of one's risk may be seriously underestimated.

In this paper we have incorporated the effects of levels of STD prevalence in disease transmission. In Section 2 a model of the SIRS type was analyzed. We found that the "typical" behavior of classical epidemic models of this type survives under restricted behavioral assumptions. For example, we found that if condition (2.5) is satisfied and if the basic reproductive number exceeds one, then there is a unique endemic equilibrium and the qualitative behavior of our model is in line with that of classical models for STDs. If we only assume that the incidence rate increases with the number of susceptibles, and decreases with the number of infected, then multiple endemic equilibria are possible, that is, abrupt changes in behavior or on initial conditions (such as the sudden influx of infected individuals) may significantly affect the levels of prevalence, but we are able to rule out oscillations. Similar results

were established for a model of HIV/AIDS transmission in Section 3.1. In Section 3.2, we show that small random fluctuations cannot produce sustained oscillations in those situations approximated by our HIV/AIDS model. Finally, in Section 3.3 we look at the potential effects that reporting delays may have on the efficacy of preventive measures. We found that long delays may cause persistent fluctuations in AIDS incidence. This result has different possible interpretations, given the fact that most members of sub-population have no knowledge of their HIV status, but only of their AIDS status. Furthermore, we note that even if the levels of estimated HIV prevalence (from reported number of AIDS cases) were known and absorbed by most population members, the level of under-reporting in developing countries could amount to a long delay in the population knowledge of true levels of prevalence. One may conclude in both cases that persistent fluctuations are quite possible.

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APPENDIX A: PROOF OF THEOREM 1

We assume that (2.4) and 92.8) hold. If $I(0) = 0$, the solution has $I(t) = 0$ for all $t \geq 0$, as we see from (2.1), and therefore $R(t) \to 0$, $T(t) \to \Lambda/\mu$, and $S(t) \to \Lambda/\mu$. If $I(0) > 0$ then since $S(t) \leq T(t)$,

$$\frac{1}{\gamma+\mu}\frac{dI}{dt} = \left[\frac{\beta c}{\gamma+\mu}F(S,I,R)\frac{S}{T} - 1\right]I$$

$$\leq \left[\frac{\beta c}{\gamma+\mu}F(S,I,R) - 1\right]I \quad \leq \quad \left[\frac{\beta c}{\gamma+\mu}F\left(\frac{\Lambda}{\mu},0,0\right) - 1\right]I$$

$$= \left(R_o - 1\right)I \; < 0.$$

Since $I(t)$ is decreasing, $\lim_{t\to\infty} I(t) = 0$. Then the variation of parametric formula gives

$$R(t) = R(0)\, e^{-(\delta+\mu)t} + \gamma \int_0^t I(s)\, e^{-(\delta+\mu)(t-s)}\, ds,$$

and it follows that $R(t) \to 0$ as $t \to \infty$. And since $T(t)$ tends to Λ/μ, we deduce that $S(t) \to \Lambda/\mu$.

The Jacobian matrix of (2.1), with derivatives evaluated at $I = R = 0$, $S = T = \Lambda/\mu$, is

$$\begin{bmatrix} -\mu & -\beta c F_o & \delta \\ 0 & \beta c F_o - (\gamma+\mu) & 0 \\ 0 & \gamma & -(\delta+\mu) \end{bmatrix}.$$

The eigenvalues are the diagonal entries. hence the disease-free equilibrium is unstable if $\beta c F_o > (\gamma+\mu)$ or equivalently when $R_o > 1$. this completes the proof of Theorem 1.

APPENDIX B: PROOF OF THEOREM 2.

Let $g_1(S,I)$ and $g_2(S,I)$ be the functions in the right members of (2.14), that is

$$\frac{dS}{dt} = g_1(S,I), \quad \frac{dI}{dt} = g_2(S,I).$$

Then

$$\frac{\partial}{\partial S}\left\{\frac{g_1(S,I)}{SI}\right\} + \frac{\partial}{\partial I}\left\{\frac{g_2(S,I)}{SI}\right\} = -\frac{\Lambda(\delta+\mu)}{\mu S^2 I} - \frac{\beta c\mu}{\Lambda}\left(F_S - F_R\right) + \frac{\beta c\mu}{\Lambda}\left(F_I - F_R\right) + \frac{\delta}{S^2}$$

$$= \frac{(\delta+\mu)}{S^2}\left(\frac{\delta}{\delta+\mu} - \frac{\Lambda}{\mu I}\right) + \frac{\beta c\mu}{\Lambda}\left(F_I - F_S\right).$$

Since $0 < I \leq \Lambda/\mu$, the first term is negative. The second term is negative, by hypothesis. thus the expression is of fixed sign in the region $S > 0$, $I > 0$, $S + I \leq \Lambda/\mu$, and it follows from Dulac's Criterion test that (2.14) has no limit cycles in the region.

APPENDIX C: PROOF OF THEOREM 3

Define the dimensionless quantities $S' \equiv S \Big/ \Lambda/\mu$, $I' \equiv I \Big/ \Lambda/\mu$, $A' \equiv A \Big/ \Lambda/\mu$, and $t' \equiv (\gamma+\mu)t$, and recast (3.1) in terms of these quantities. Dropping the primes, and defining the quantities

$$\Psi_S \equiv \frac{1}{F^*}\left(\frac{\partial F}{\partial S}\right)^*, \quad \Psi_I \equiv -\frac{1}{F^*}\left(\frac{\partial F}{\partial I}\right)^*, \quad \Psi_A \equiv -\frac{1}{F^*}\left(\frac{\partial F}{\partial A}\right)^*,$$

$$q \equiv \frac{\mu}{\gamma+\mu}, \quad p \equiv \frac{\alpha+\mu}{\gamma+\mu}, \quad R_o \equiv \frac{\beta c}{\gamma+\mu}, \quad K(\eta) \equiv \frac{q(\eta-1)}{q+\eta-1},$$

then the Jacobian at the endemic equilibrium is

$$J = \begin{bmatrix} -\left(q + \frac{(\eta-1)^2}{\eta} + K(\eta)\Psi_S\right) & -\frac{1}{\eta} + K(\eta)\Psi_I & K(\eta)\Psi_A \\ \frac{(\eta-1)^2}{\eta} + K(\eta)\Psi_S & -\left(\frac{\eta-1}{\eta} + K(\eta)\Psi_I\right) & -K(\eta)\Psi_A \\ 0 & 1-q & -p \end{bmatrix} \tag{C1}$$

The characteristic polynomial is then

$$\lambda^3 + \kappa_2\lambda^2 + \kappa_1\lambda + \kappa_0 = 0, \tag{C2}$$

where

$$\kappa_2 \equiv p+q+\eta-1 + K(\eta)\left(\Psi_S+\Psi_I\right)$$

$$\kappa_1 \equiv \frac{\eta-1}{\eta}\left(q+\eta(1+p)-1\right) + pq + K(\eta)\left\{(1+p)\,\Psi_S + (p+q)\,\Psi_I + (1-q)\,\Psi_A\right\}$$

$$\kappa_0 \equiv p\frac{\eta-1}{\eta}\left(q+\eta-1\right) + q\,K(\eta)\left\{p\,\Psi_S + p\,\Psi_I + (1-q)\,\Psi_A\right\}.$$

As $\eta > 1$, and all the Ψ_i terms are non-negative by (3.10), then all the κ_i are positive, satisfying two of the three Routh-Hurwitz criteria. It only remains to show that the final Routh-Hurwitz criterion $\kappa_1\kappa_2 - \kappa_0 > 0$ is satisfied. Expanding the κ_i, and evaluating $\kappa_1\kappa_2 - \kappa_0$ term by term (cross-products $\Psi_i\Psi_j$, linear in Ψ_i, constant term), we find that all the terms are positive. Then the third criterion is satisfied, and the unique endemic equilibrium is always locally stable, completing the proof.

APPENDIX D: LOCAL STABILITY OF (3.11)

The unique endemic equilibrium is given by $S^* = q/(q+\eta-1)$ and $I^* = q(\eta-1)/(q+\eta-1)$, for $1 \le \eta < R_o$ given by Eq (3.13). Note that with $R_o > 1$, for delay $\tau = 0$ the endemic equilibrium is stable, by the results of section 4; for $\tau > 0$, the linearized system for $S = S^* + x$, $I = I^* + y$ is

$$\begin{aligned} \frac{dx}{dt} &= -\left[q + \frac{(\eta-1)^2}{\eta}\right]x(t) - \frac{1}{\eta}y(t) + \psi\frac{q(\eta-1)}{(q+\eta-1)}y(t-\tau) \\ \frac{dy}{dt} &= \frac{(\eta-1)^2}{\eta}x(t) - \frac{(\eta-1)}{\eta}y(t) - \psi\frac{q(\eta-1)}{(q+\eta-1)}y(t-\tau) \end{aligned} \tag{D1}$$

where

$$\psi \equiv -\frac{1}{F^*}\left(\frac{\partial F}{\partial I}\right)^*. \tag{D2}$$

The characteristic equation for Eqs (D1) is

$$\lambda^2 + \lambda\left[q+\eta-1 + \psi\frac{q(\eta-1)}{(q+\eta-1)}e^{-\lambda\tau}\right] + \frac{(\eta-1)}{\eta}(q+\eta-1) + q\psi\frac{q(\eta-1)}{(q+\eta-1)}e^{-\lambda\tau} = 0, \tag{D3}$$

which is of a form studied by Cooke and Grossman (1982) and Blythe *et al.* (1985); in particular, where switches in stability occur with increasing delay τ, they are one-way, with no re-stabilization possible for larger τ. We may generate locii of $\lambda = i\omega$ in the (ψ,τ) plane with $R_o > 1$ and $0 < q < 1$ fixed, by *(i)* taking a series of values of $\eta > 1$, *(ii)* using Eq(•) to get the equivalent ψ, *(iii)* finding the largest positive root Z_+ of the quadratic (in $Z = \omega^2$)

$$Z^2 + BZ + C = 0, \tag{D4}$$

where

$$B = (q+\eta-1)\left(q + \frac{(\eta-1)(\eta-2)}{\eta}\right) - \psi^2\left(\frac{q(\eta-1)}{(q+\eta-1)}\right)^2$$

and

$$C = \left(\frac{\eta-1}{\eta}\right)^2 (q+\eta-1)^2 - q^2\psi^2\left(\frac{q(\eta-1)}{(q+\eta-1)}\right)^2,$$

and *(iv)* obtaining τ on the boundary from

$$\tau_c = \frac{1}{\sqrt{Z_+}}\ cos^{-1}\left[-\frac{(\eta-1)(q+\eta-1)\left(Z_+ + \frac{q}{\eta}(q+\eta-1)\right)}{\psi q\left(Z_+ + q^2\right)}\right]. \tag{D5}$$

For small enough ψ, when $\tau < \tau_c$ the linear system is stable, while for $\tau > \tau_c$ it is unstable, and the non-linear system exhibits oscillations. The critical value of ψ corresponds to the case when $B^2 - 4C = 0$. For ψ smaller than this critical value, the system is always stable. The results in Fig (•), for $F(I) = e^{-I/\theta}$, were obtained in this manner, using $\psi = 1/\theta$, with θ on the boundary given parametrically by

$$\theta_c = \frac{q(\eta-1)}{(q+\eta-1)\,ln(R_o/\eta)}. \tag{D6}$$

As $\eta \to 1$, $\theta_c \to 0$, but

$$\frac{1}{\theta_c}\frac{q(\eta-1)}{(q+\eta-1)} = \ln(R_o/\eta) \to \ln(R_o), \tag{D7}$$

so that (D4) becomes

$$Z^2 + \left\{q^2 - \left[\ln(R_o)\right]^2\right\} Z - \left[q \ln(R_o)\right]^2 = 0, \tag{D8}$$

and $Z_+ \rightarrow [\ln(R_o)]^2$. Then

$$\tau_c \rightarrow \frac{1}{ln(R_o)} \cos^{-1}(0) = \frac{\pi}{2\, ln(R_o)}. \tag{D9}$$

This represents the minimum delay τ for which destabilization is possible.

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FIGURE CAPTIONS

Figure 1:

Local stability boundary for Eqs (3.13) with $F(I) = \exp\{-I/\theta\}$, for the case $q = 0.2308$, $R_o = 3.846$. θ is scaled by Λ/μ, and τ by $1/(\gamma+\mu)$.

Figure 2:

Oscillations produced by delayed response in risk-behavior change in the dimensionless Eqs (3.13). Parameter values used were $q = 0.2308$, $R_o = 3.846$, $\theta = 0.1$, $\tau = 2.0$. Initial values: $S(0) = 1.0$, $I(0) = 0.001$, $I(t) = 0.0$ for $-\tau \leq t < 0$.

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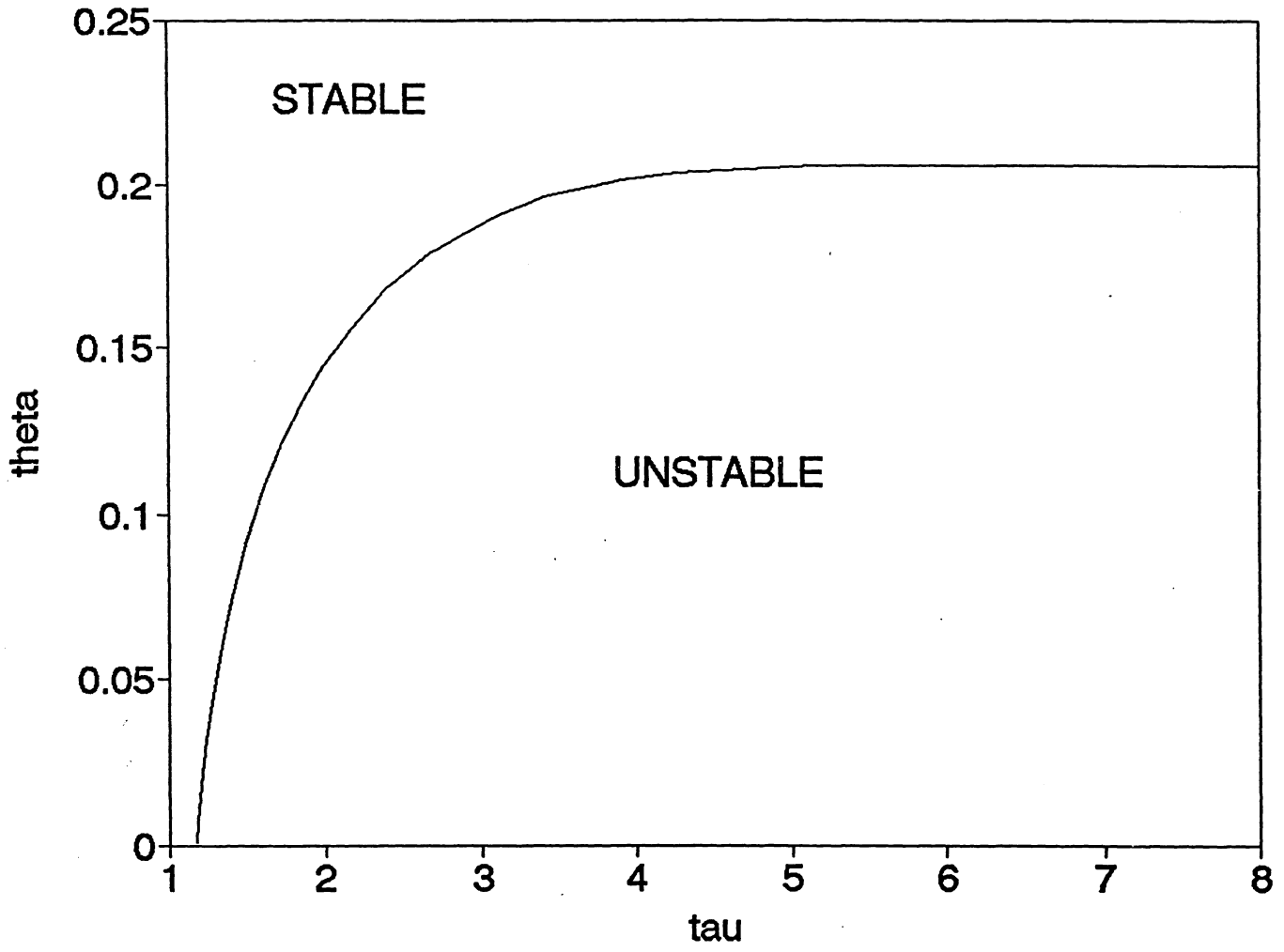
FIG 1



Blythe / Cooke / Castillo-Chavez

Fig 2