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Environmental context, social interactions, and the spread of HIV

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ABSTRACT

The spread of HIV through a population is influenced by many factors. These include the frequency and type of sexual activity, age distribution of the population, use of intravenous drugs and behaviors associated with their use, the pattern of infectivity of the virus during the several stages of the infection, geographic location, and the patterns of interaction among individuals and the context in which those interactions occur.

A number of questions about the natural history and epidemiology of AIDS remain unanswered. Since the disease has only recently been recognized, historical data are unavailable to aid in answering these questions. Mathematical models provide one approach that can be of use in understanding the spread of the disease.

Because of the complexity of the biology and epidemiology of AIDS, useful models must focus on specific aspects of the disease. In addition to a brief review of existing models for the spread of AIDS, a new model which incorporates the effects of social context operating at the time of a sexual encounter is described. The model considers the spread of the disease in a population of homosexual men divided into groups based on relative risk for the disease. Individuals are assigned to a specific group on the basis of their normal sexual activities, but they may take on the characteristics of a different risk group temporarily given the conditions operating when they engage in a sexual encounter. Results from preliminary analyses of the new model indicate that the major effects of incorporating social context include a decrease in the number of cases of the disease, sometimes by an order of magnitude or more, delayed spread of the disease, and a decreased impact of the disease on low and medium risk groups.

The AIDS epidemic is one of the major public health emergencies of this century. This has stimulated highly productive research efforts in a number of fields, including epidemiology, immunology, clinical medicine, sociology, and applied mathematics. This paper will review the contributions made by applied mathematicians and mathematical or theoretical epidemiologists to understanding the spread of AIDS in modern populations. In addition, a new model which incorporates factors operating at the time of contact between individuals will be described.

A number of mathematical models have been developed to describe the transmission system of HIV, the virus responsible for AIDS. These models help to provide a framework with which to explore the nature of disease spread through a population, the effects of behavior change on the rates of spread, the effectiveness of control strategies, the consequences of different patterns of contact among individuals in a population, and other similar questions. Analysis of such models provides valuable knowledge and understanding of biological and social factors influencing disease transmission — understanding that may not be available from statistical analyses.

NATURAL HISTORY AND EPIDEMIOLOGY OF AIDS

AIDS is a clinical syndrome associated with severe damage to the immune system as a consequence of infection with a retrovirus, human immunodeficiency virus (HIV). Because of the immune suppression a number of opportunistic infections, rare cancers, and other unusual infections may occur. These are the immediate causes of death among AIDS patients. The earliest marker to indicate infection is usually seroconversion, which occurs when antibodies to HIV begin to be detected in the serum of the individual. The average time to seroconversion is estimated to be about 2-6 months. At some point after infection more than 90% of individuals will start to show an initial immune suppression. AIDS itself develops in most, if not all infected individuals who do not die of other causes.

There are three primary modes of transmission of the virus; a) sexual activity, b) blood and blood products, and c) perinatal transmission. As a consequence of these modes of transmission, the biology of the virus, and the history of the virus in the U.S. population, there are seven recognized high risk groups in the United States: 1) homosexual males, 2) intravenous drug users, 3) sexual contacts of intravenous drug users (both male and female), 4) recipients of blood transfusions prior to 1985, 5) hemophiliacs, 6) sexual contacts of transfusion recipients and hemophiliacs, and 7) children of infected individuals.

The proportions of reported AIDS cases in different risk groups remained relatively constant during the mid-1980s, with about 65% of all cases in the U.S. occurring among homosexual males, another 20% occurring among intravenous drug users (both heterosexual and homosexual), and the remaining 15% from other risk groups or with unknown risk factors (Centers for Disease Control, 1987). Recently there has been a decrease in the proportion of men whose only apparent risk factor was homosexual activities (from 70% of cases reported in 1987 to 63% in 1988) and an increase in the proportion of cases among intravenous drug users. This change is likely due to a combination of the effects of the 1987 revision of the case definition for AIDS and changing incidence rates in the homosexual and intravenous drug-using populations (Centers for Disease Control, 1989).

Realistic models for the spread of HIV in the U.S. population should consider all of the risk groups mentioned, but because of limited data on the type and frequency of human interactions (i.e. the social/sexual mixing) most existing models have concentrated on one or two risk groups. In addition to the risk groups themselves there are a number of other factors commonly considered in models of HIV transmission. These include frequency of sexual behavior (which has been estimated to range from 0 to over to over 50 partners per year for homosexual males (data from BMRB, 1987, cited in May and Anderson, 1988), the nature of the sexual behaviors and preventive measures taken, geographic location, the age distribution of the population (since sexual behavior and intravenous drug use are clearly age-dependent), behaviors associated with intravenous drug use, such as sharing needles or using rented equipment, and the patterns of

interaction among individuals within and between risk groups. Models for HIV transmission also generally include two main factors associated with the natural history of the disease: 1) the pattern of infectivity, which probably is not constant throughout the infectious period, and 2) the progression of the disease within an individual. This last factor is important because the individual behaviors and possibly the infectivity of the virus are likely to vary in different stages of the disease.

MODELS FOR THE SPREAD OF HIV

Because there are such well-defined risk groups for HIV transmission and because there clearly is nonrandom mixing among risk groups, reasonable models for HIV transmission must consider structured populations. Rather than considering one large randomly mixing population, an obviously unrealistic assumption, these models consider the population to be divided into a number of subpopulations within which mixing is usually assumed to be random but among which mixing is nonrandom.

Although a few structured epidemiological models were developed earlier, the biggest push toward the development of such models began in the mid-1970s with the work on sexually transmitted diseases of Cooke and Yorke (1972, 1973), Lajmanovich and Yorke (1976), Yorke et al. (1978), Nold (1980), Hethcote et al. (1982) and Hethcote and Yorke (1984). These models have proven very useful in several areas, including policy planning. A drawback of them, however, is that most assume that the population under consideration is stratified into subpopulations of constant size. This assumption implies that the mixing between groups (i.e., the social structure) does not change over time, which is unrealistic, because even in the case of proportionate (or generalized random) mixing, the social structure depends dynamically on the size of the interacting subpopulations. An extensive mathematical study of the dynamic effects of

variable population size in the dynamics of HIV in structured populations can be found in the work of Castillo-Chavez, Cooke et al. (1989b,c), Huang (1989), and Huang et al. (1990).

Models should not include every detail unless their purpose is simply to describe a process. Rather, to be useful they should be developed with the idea of addressing very specific questions such as the role of the long periods of infectiousness in the dynamics of HIV, the effects of different vaccination strategies on the future dynamics of a disease, etc. Often, in order to begin to answer many of these kinds of questions it is necessary to use structured models. These models have arisen in response to the recognition that virtually all human populations are structured in some way, so that the assumption that the population consists of one large group of randomly mixing individuals is rarely appropriate for any disease. Structured models have been developed for a number of diseases in addition to sexually transmitted diseases. These other diseases include, for example, measles (Haggett, 1972, 1976; Cliff et al., 1975) influenza (Baroyan, Rvachev, and Ivannikov, 1977; Rvachev and Longini, 1985; Longini, 1988; Castillo-Chavez, Hethcote et al., 1988, 1989), smallpox (Travis and Lenhart, 1987), and hepatitis A (Sattenspiel, 1987; Sattenspiel and Simon, 1988).

Most of the existing models for the transmission of HIV consider only the sexual transmission of the disease, largely because, for historical reasons, there are better data available for this problem. A few recent papers, however, address the transmission of the disease within intravenous drug-using populations (for example, Kaplan, 1989; Blower and Medley, 1990). Although there are several aspects of the natural history of the disease which models can address, the bulk of the modeling efforts are concerned with the consequences of one or more of four factors: 1) the effect that variability in infectivity throughout the course of the disease in an individual has on the spread of the infection through the population, 2) the role of the incubation period on the dynamics of HIV models, 3) the effect of level of sexual activity on an individual's risk for disease, and 4) the effect that assumptions about mixing between groups have on both individual risk and transmission throughout a population.

Most early models and a few more recent models of the transmission of HIV are concerned primarily with projecting the spread and impact of the disease given the limited data available. Pickering et al. (1986) developed a discrete-time model where the probability of transmission depended upon length of the incubation period, variable infectivity, level of infection within a population, and changes in sexual behavior. Knox (1986) considered variations in the removal rate, the new partner acquisition rate, and the infectivity on equilibrium levels for prevalence and incidence. DeGruttola and Mayer (1988) developed a model for heterosexual spread of HIV and fit the results to surveillance data on prevalence of the infection. Wilkie (1988) developed an actuarial model for AIDS that could be used to aid in the development of insurance policies. Such a goal requires the best possible estimates of long-term trends of the disease. Aron and Sarma (1989) analyze demographically data on sexual behavior in the United States to project the potential impact of AIDS on the heterosexual population. Hethcote (1989) developed a model for the transmission of HIV that incorporates homosexual transmission, heterosexual transmission, and intravenous drug use. His model was intended to aid in an assessment of the effects of all the major modes of transmission on the spread of the virus through a population. Bailey (1988) advocated the development of relatively simplified models that could be used by decision makers to act more effectively immediately. Mode et al. (1988) developed a stochastic computer model of an AIDS epidemic to explore the consequences of ignoring random effects in transmission on projections of the epidemic. Results of the analysis of their model showed that predictions from deterministic models may be overly pessimistic.

One consequence of these models was a recognition of the sensitivity of predictions to the parameter values chosen. A variety of outcomes are possible depending on particular choices of critical parameters. Because of this sensitivity to parameters and the lack of sufficient epidemiological and behavioral data, many recent models are more concerned with the identification of key epidemiological and behavioral parameters that need to be measured, the determination of key mechanisms for transmission, the study of the possible role of models for planning, and in the study of HIV dynamics at a variety of scales (immune system models,

population dynamic models, geographic models, etc.) rather than with specific projections of the epidemic.

Several modeling groups have developed structured models for HIV transmission which address these issues. Anderson et al. (1986) introduced simple models in order to estimate and identify parameters and begin to address specific questions such as the effects of the incubation period distribution on the dynamics of HIV. This model provided a starting point for a variety of the most recent models. For further details see Anderson (1988) or Schwager, Hethcote, and Castillo-Chavez (1989).

The Michigan HIV group developed a similar model to that of Anderson et al. (1986). Because the simulated dynamics of the new model presented later in this paper will be compared explicitly with the simulated dynamics of the Michigan model (Jacquez et al. 1988; Koopman et al. 1988; Sattenspiel et al. 1990), the Michigan model will be discussed in some detail before reviewing additional models that have been developed by other groups.

The Michigan model considers the spread of HIV in a population of homosexual males divided into five groups on the basis of sexual activity levels. Transmission can occur when a susceptible individual engages in sexual activity with an infective individual, who may be a member of any one of the five subgroups. The likelihood of an average individual from subgroup i coming into contact with an individual from another subgroup j is given by ρ_{ij}, the ij th element of the contact matrix, ρ.

The most common type of contact matrix in epidemic models is built upon the assumption that individuals from different subgroups mix randomly. This leads to a mixing model called proportionate mixing, which was first introduced by Barbour (1978). In this type of generalized random mixing the probability of contact between individuals from different groups is proportional to the size or amount of activity of the groups involved (Hethcote and Yorke 1984). This is not generally a very realistic assumption, especially for sexually transmitted diseases, although the use of proportionate mixing in age-structured models has generated some useful practical and

theoretical results (see, for example, Castillo-Chavez, Hethcote et al., 1988, 1989; Dietz, 1975; Hethcote and Van Ark, 1987; Hoppensteadt, 1974; May, 1986; Schenzle, 1985; and Webb, 1985).

The Michigan model relaxes some of the constraints on mixing patterns imposed by proportionate mixing, although the model still considers a simplified pattern called preferred mixing. Althought preferred mixing and other, more general forms of mixing have been used extensively in the populations genetics literature, this type of mixing was first introduced into disease modeling by Nold (1980) and Hethcote and Yorke (1984) Their ideas were formalized and named preferred mixing by the Michigan HIV group. In preferred mixing a specified proportion of contacts are made between individuals within the same group, while the remaining contacts occur between individuals from different groups and are made in proportion to the amount of sexual activity of the groups. Preferred mixing is thus a combination of restricted, endogamous mating and random, exogamous mating. Generalizations to like-with-like (or assortative) mixing have been carried out by Hyman and Stanley (1988, 1989), Blythe and Castillo-Chavez (1989), Castillo-Chavez and Blythe (1989), and Anderson et al. (1989). Recently, an axiomatic framework has been developed by Busenberg and Castillo-Chavez (based on the work of Blythe and Castillo-Chavez) to describe one- and two-sex mixing interactions of age- and risk-structured populations. Further, formulas describing all solutions to these frameworks (i.e. formulas describing all forms of mixing) have been computed, and a simple recipe for superimposing mixing structures (such as in the case of preferred mixing) has been provided (see later in this paper; Busenberg and Castillo-Chavez, 1989, 1990; and Castillo-Chavez et al. 1990).

In the Michigan model, a model for homosexually active populations, the disease progresses through four stages within an individual. An individual is in stage 1 of the disease from the time of infection to the time of seroconversion. Stage 2 includes the time from seroconversion to the time of initial immune suppression. Stage 3 lasts from initial immune suppression to the development of clinical AIDS (stage 4). In addition, there is constant recruitment of susceptibles into the population and death from both AIDS and other causes.

The model resulting from this framework is:

$$\frac{dX_i}{dt} = - c_i X_i \sum_{j=1}^{n} \rho_{ij} \sum_{r=1}^{4} \beta_{ijr} \frac{Y_{jr}}{X_j+Y_j} - \mu X_i + U_i$$

$$\frac{dY_{i1}}{dt} = c_i X_i \sum_{j=1}^{n} \rho_{ij} \sum_{r=1}^{4} \beta_{ijr} \frac{Y_{jr}}{X_j+Y_j} - (k_1+\mu) Y_{i1}$$

$$\frac{dY_{ir}}{dt} = k_{r-1} Y_{i,r-1} - (k_r+\mu) Y_{i,r} \qquad r=2,3,4$$

$$\frac{dZ_i}{dt} = k_4 Y_{i4} - \partial Z_i$$

where:

X_i = the number of susceptibles in sexual activity level *i*,

Y_{ir} = the number of infecteds in sexual activity level *i* who are at stage *r* of the disease,

Z_i = the number of AIDS patients who are removed from transmission activity,

U_i = the number of individuals recruited into activity level *i* per unit time. Note that the model assumes that all recruits are susceptible.

c_i = the number of sexual contacts per person in group *i* per unit time,

ρ_{ij} = the proportion of the contacts of an individual in subgroup *i* made with persons in subgroup *j*,

β_{ijr} = the transmission fraction; the fraction of contacts between a susceptible in subgroup *i* and an infective in subgroup *j* at disease stage *r* that result in transmission of the virus,

k_r = the fractional transfer rate of infectives from stage *r* to stage *r+1*,

μ = the fractional rate at which members transfer out of the groups for all reasons other than the development of AIDS. μ is called the competing mortality rate, and is assumed to be constant for all X_i and Y_i and small in relation to k,

∂ = the mortality rate for those with AIDS.

In the above model the parameters, U_i, c_i, β_{ijr}, k_r, μ, and ∂, are assumed to be constant. This assumption restricts the model in a variety of ways. For example, the fact that the removal rates from the infective classes are constant constrains the incubation period distribution to flexible but specific parmetric distributions (see Blythe and Anderson, 1988a,b; Castillo-Chavez 1989a, Castillo-Chavez et al. 1989c,d). Although, these restrictions on the parameters may not have practical implications they may restrict our theoretical understanding of this type of epidemiological model (see Thieme and Castillo-Chavez 1989, 1990).

The assumption of preferred mixing used in the model leads to a mixing matrix with elements of the following form:

$$\rho_{ii} = \rho_i + (1 - \rho_i) \frac{c_i (1 - \rho_i)(X_i + Y_i)}{\sum_k c_k (1 - \rho_k)(X_k + Y_k)}$$

$$\rho_{ij} = (1 - \rho_i) \frac{c_j (1 - \rho_j)(X_j + Y_j)}{\sum_k c_k (1 - \rho_k)(X_k + Y_k)}, \qquad j \neq i$$

In these equations ρ_i is the fraction of group *i*'s contacts reserved for individuals within the group, usually assumed to be constant. As indicated earlier this type of mixing is a mixture of solutions to the framework of Blythe, Busenberg, and Castillo-Chavez (Blythe and Castillo-Chavez, 1989; Castillo-Chavez and Blythe, 1989; Busenberg and Castillo-Chavez, 1989, 1990). In general any positive convex linear combination of mixing matrices is a mixing matrix. In other words, if ρ_{ij}^l ($l = 1,...,n$) are *n* mixing matrices and if $\alpha_1,...,\alpha_n$ are positive constants with $\sum_{l=1}^{n} \alpha_l = 1$, then $\sum_{l=1}^{n} \alpha_l \rho_{ij}^l$ is also a mixing function.

Although, this procedure provides us with a simple recipe for generating mixing matrices, there is no clear procedure for choosing the coefficients α_l. Further, if we assume that these coefficients

are constant, as in the case of preferred mixing, then we introduce behavioral constraints arbitrarily. These constraints are incompatible with populations of variable size. Despite these shortcomings, preferred and proportionate mixing are still very useful as they provide us with reference models (mixing matrices) against which we can test more realistic forms of mixing.

Results from numerical analyses of the Michigan model and other similar models show that 1) the pattern of infectivity in an individual can markedly influence the outcome of the epidemic, 2) the level of sexual activity is an important individual risk factor, and 3) the pattern of mixing among individuals profoundly affects the rate and amount of spread of the infection through the population (Castillo-Chavez, Cooke et al. 1989b; Hyman and Stanley 1988, 1989; Jacquez et al. 1988; Koopman et al. 1988; Sattenspiel et al. 1990).

Jacquez et al. (1989) and Koopman et al. (1989) extend the original Michigan HIV model and introduce a new type of mixing. They divide the population into both structural and mixing groups, which overlap but are not equivalent. The mixing matrix, f, has elements f_{ij} which give the fraction of structural group *i*'s contacts allocated to mixing group *j*. A mixing group may contain contributions from any number of structural groups and a structural group may appear in any number of mixing groups. Jacquez et al. (1989) consider the nonrandom mixing associated with the definition of mixing groups while Koopman et al. (1989) use numerical simulations to explore nonrandomness associated with selection of partners within mixing groups. The large number of parameters involved in these models makes this type of mixing useful mostly for theoretical considerations. The model that will be introduced in the next section has the same shortcomings. However, even with the shortcomings we will be able to address our specific question — the role of environmental context in the dynamics of HIV.

A number of other models for the transmission of HIV have been developed. The Michigan model presented above is a special case of models developed by Hyman and Stanley (1988, 1989), May et al. (1988a,b), Castillo-Chavez, Hethcote et al. (1989), and Castillo-Chavez, Cooke et al (1989b,c). These more general models have continuously distributed characteristics and incorporate arbitrary incubation period distributions. In addition, the extensions of May et al.

(1988a,b) incorporate age-structure, while the recent extensions of Busenberg and Castillo-Chavez (1989, 1990) include age- and risk structure and arbitrary mixing structures.

Morris (1989) has extended a basic HIV transmission model to incorporate the effects of selective mixing in both stable and non-stable populations. She found that in non-stable populations behavioral responses to changes in relative group sizes must be explicitly modeled. This is done by making the elements of the contact matrix a function of mixing preferences, which may be stable over time, and the population structure, which changes over time. In addition to these structural considerations, log-linear models were proposed as a method to estimate statistically the underlying parameters that regulate selective mixing. Simulations based on behavioral, sociological, demographic, and epidemiological data from a number of sources were used to demonstrate the usefulness of this approach.

The effect of level of sexual activity has been studied by Anderson et al. (1986), Blythe and Anderson (1988a), Blythe and Castillo-Chavez (1989), Busenberg and Castillo-Chavez (1989, 1990), Castillo-Chavez and Blythe (1989), Castillo-Chavez et al. (1990), Jacquez et al. (1988), Kießling et al. (1986), Koopman et al. (1988), and May and Anderson (1987). These studies clearly show that the level of sexual activity is a critical factor influencing the rate of spread and intensity of the epidemic in each subgroup.

Blythe and Anderson (1988b), Castillo-Chavez, Cooke et al. (1989), Hyman and Stanley (1988, 1989) and Jacquez et al. (1988) have studied the effects of variation in the shape of the infectivity curve over the course of infection. These models use an infectivity curve that has a sharp peak early in the infectious period, followed by a low level of infectivity that rises gradually with the time since infection. Blythe and Anderson (1988b) and Hyman and Stanley (1988, 1989) use a continuous distribution of infectivity in their simulations, while simulations presented in Jacquez et al. (1989) or Castillo-Chavez, Cooke et al. (1989b) are more restrictive. Results from these models show that in a model in which all individuals have the same risk behavior, the rate at which the susceptible population is infected changes dramatically when the infectivity curve is altered. These effects are most marked when the alterations change the early peak in infectivity.

Theoretical analyses of HIV models with variable infectivity have been conducted by Thieme and Castillo-Chavez (1989, 1990). They have shown that variable infectivity may cause the incidence of the disease to oscillate over time, even in a single group model. Their results have shown that this potential oscillatory behavior would not be observed in compartmental models like those used by Jacquez et al. (1988) or Castillo-Chavez, Cooke et al. (1989b) that use only differential equations with constant parameters. It has not yet been determined whether oscillations are possible within realistic parameter ranges for models with homogeneously mixing populations and variable infectivity .

Single group models have also been developed to explore the effects of variability in the length of the incubation period (Blythe and Anderson, 1988b; Castillo-Chavez, 1989a; Castillo-Chavez, Cooke et al., 1989a,d). Results from the analysis of these models show that the qualitative outcome of the epidemic does not change much (i.e. there is no oscillatory behavior, but rather a steady approach to an endemic equilibrium), but the path taken to get to that end can vary significantly. However, the interplay between variable and long periods of incubation and variable mean partnership change rate can potentially change the qualitative outcome of the epidemic (Thieme and Castillo-Chavez 1989, 1990).

May et al. (1988a, b) and Anderson et al. (1988) have developed a series of models to look at the demographic consequences of the AIDS epidemic. They take a simple epidemic model and combine it with standard demographic models to explore the effects of horizontally- and vertically-transmitted HIV on overall growth rates and age profiles of a population. Because their models are focused on understanding basic ways that AIDS deaths might affect demographic patterns rather than predictions of prevalence and incidence rates, they simplify the epidemiology by assuming constant infectiousness, constant transmission rates, and random mixing of individuals within the population.

A few models have attempted to incorporate more long-term behavioral changes in AIDS transmission models. Hethcote et al. (1990) developed a model for homosexual transmission of the virus and attempted to determine the model structure necessary to reproduce the San Francisco

AIDS epidemic. They found that in order to get satisfactory fits to the data changes in sexual behavior must be included.

Anderson et al. (1989) have also looked at the effects of changes in sexual behavior on the dynamics of HIV transmission in the male homosexual population in the United Kingdom. Numerical simulations show that the manner in which behavioral changes occur and who in the population is influenced by such changes have a major impact on the future time course of the epidemic. The strongest effects will occur when the behavioral changes occur disproportionately among the most active individuals in the population.

Marriage models that follow pairs of individuals and consider the dynamics of pairs have been developed by Kendall (1949) and Fredrickson (1971) to aid in demographic projections of populations. Similar types of models for transmission of sexually-transmitted diseases have been developed by Dietz (1988), Dietz and Hadeler (1988), Waldstätter (1989), and Castillo-Chavez et al. (1990). These models explicitly consider how individuals form partnerships and allow an examination of the effect of variations in the duration of partnerships as well as variability in the number of contacts with the same partner. This kind of model allows one to incorporate the effects of changing sexual activity rates with length of partnership or the consequences of infection of one partner after a variable amount of time within a partnership. These factors cannot be explored effectively with most other existing models. However, we note that the mixing framework developed by Busenberg and Castillo-Chavez (1989, 1990) allows for the unification of both modeling approaches (for a detailed example, see Castillo-Chavez et al., 1990). Finally, we note that Blythe and Castillo-Chavez (1990) have begun to explore the processes of pair formation and dissolution in the context of stochastically interacting populations. Their main focus has been to explain the "power law" of Anderson and May (1988) regarding the levels and variability of sexual activity in human populations.

A MODEL FOR HIV TRANSMISSION WHICH INCORPORATES SOCIAL CONTEXT

One question that is not addressed in any of the models described above is the effect of the social context of the sexual interactions on the likelihood of transmission of the infection. These models assume that the only factors influencing the transmission are characteristics associated with the assigned groups *i* and *j* of the individuals involved in an interaction. However, in some situations this is not a reasonable assumption to make. Behaviors are dynamic and plastic and tend to change when the environmental conditions change. Thus, although an individual may normally engage in certain types of sexual activities, the actual behaviors present under the conditions of a specific sexual interaction may be of higher or lower risk than normal for that individual. The risks for transmission of a disease depend on the behaviors that are occurring at the time of contact, though, not on the behaviors that normally occur and therefore it is important to have some mechanism for behavioral plasticity in the model.

One way to approach modeling the effects of social context and the resulting behavioral plasticity on the spread of HIV is to define the groups on the basis of a set of risk behaviors rather than using a simple variable like number of new sexual contacts. Individuals are classified into a particular group on the basis of their usual activities, but at the time of a sexual contact they may take on behaviors characteristic of other risk groups. Since the probability of transmission will be most closely related to behaviors occurring at the time of contact a mechanism is required to model the probability that an individual from group *i* takes on behaviors characteristic of group *j* when that individual enters a sexual encounter. This temporary behavioral change can be described by a "movement" matrix, b, the elements of which give the probability that a typical individual whose normal behaviors are characteristic of risk level *k* engages in sexual activities characteristic of risk level *l*.

A model incorporating this behavioral "mobility" can be developed by combining the movement matrix approach described in Sattenspiel (1987) and Sattenspiel and Simon (1988) with generalized compartmental models like the model developed by the Michigan HIV modeling group (Jacquez et

al., 1988; Koopman et al., 1988; Sattenspiel et al., 1990). Castillo-Chavez (1989b) indicates further that the movement matrix approach can be combined with any classical modeling approach. He and Busenberg addressed the possiblity of developing such models, but because of the improbability of collecting reasonable data to validate their models, the models were not fully developed (Busenberg and Castillo-Chavez, 1989).

To describe our model that incorporates the effects of social context, we begin by considering a population of homosexually-active males divided into five risk groups. Rather than defining these groups on the basis of sexual activity levels alone, consider a definition based on a number of different risk factors, such as sexual activity level, type of sexual behavior, presence of other sexually transmitted diseases, etc. Assuming that estimates could be made for the relative risks of any type of behavior, the total risk of an individual could be determined as some combination of their degrees of risk from each individual factor. The population is divided into five behavioral classes with the assignment of a given individual reflecting where they fall on a multivariate scale determined by consideration of all possible combinations of risk factors.

Classical epidemiological models, including the Michigan model on which this new model is based, describe how an average individual from group *i* and an average individual from group *j* come together, but they cannot address the problem of what happens to their behavior after they make contact. In addition to the contact, the two individuals must be doing the same thing, and the behaviors they take on may be characteristic of the normal behaviors of one or both of the individuals or they may be different from the normal behaviors of either. For example, consider the contact between an average individual from risk group 2 and one from risk group 4. If this contact occurs at the home of individual 2 the behaviors they engage in may be relatively more sedate than if the contact occurred in a bathhouse in San Francisco (which may result in the highest risk behaviors (by definition, characteristic of group 5)). For a successful contact at 2's home both 2 and 4 would need to engage in behaviors characteristic of group 2. The probability of this happening (assuming independence) is $b_{22}b_{42}$. For a successful contact at the bathhouse both 2 and 4 would take on behaviors characteristic of group 5. The probability of this occurring is

$b_{25}b_{45}$ (assuming independence and that both individuals are at the bathhouse at the same time). Theoretically the two individuals could take on behaviors characteristic of any of the five groups. The only requirement is that they must simultaneously take on characteristics of the same group. The total probability that this happens is $\sum_k b_{2k}b_{4k}$ which gives the probability that two average individuals from groups 2 and 4 meet and take on any of the types of risk behaviors. Note however, that they may meet more than once at location k (i.e. take on the behavior k) per unit time, and hence the frequency and distribution of the types of behaviors taken by different individuals needs to be considered. The probability that an average individual from group *i* and an average individual from group *j* simultaneously take on the same behaviors is $\sum_k b_{ik}b_{jk}$. If we let m_{ik} denote the average number of "visits" that a typical individual from risk-group *i* takes to risk-group *k* per unit time (i.e., the average number of times that he takes on the *k*th type behavior), then, assuming independence, we have that $\sum_k m_{ik}m_{jk}b_{ik}b_{jk}$ denotes the rate at which the *i* and *j* individuals meet at neighborhood *k* and hence the rate at which they behave like *k*th type individuals.

This framework allows one to look at the effects of social context on the spread of HIV. Since transmission risk is likely to be most closely related to the sexual behaviors operating at the time of contact rather than the normal sexual behaviors of the individuals involved the probability of transmission is a function of the temporary risk group *k* rather than the groups *i* or *j*.

Combining this formulation with the Michigan HIV model and its notation, we arrive at the following compartmental model for the spread of HIV within an environmental context:

$$\frac{dX_i}{dt} = -c_i^* X_i \sum_{j=1}^{n} \rho_{ij} \sum_{r=1}^{4} \sum_{k=1}^{5} m_{ik}m_{jk}b_{ik}b_{jk} \cdot \frac{Y_{jr}}{X_j+Y} \beta_{rk} - \mu X_i + U_i$$

$$\frac{dY_{i1}}{dt} = c_i^* X_i \sum_{j=1}^{n} \rho_{ij} \sum_{r=1}^{4} \sum_{k=1}^{5} m_{ik}m_{jk}b_{ik}b_{jk} \cdot \frac{Y_{jr}}{X_j+Y_j} \cdot \beta_{rk} - (k_1+\mu)Y_{i1}$$

$$\frac{dY_{ir}}{dt} = k_{r-1}Y_{i,r-1} - (k_r+\mu)Y_{i,r} \qquad r=2,3,4$$

$$\frac{dZ_i}{dt} = k_4Y_{i4} - \partial Z_i$$

In this model, since not all individuals within a risk group have the same number of sexual partners, c_i^* must represent the average number of sexual contacts per unit time (one month) of an individual from group *i* rather than the actual number of sexual contacts as in the Michigan model, ρ_{ij} gives the probability that an individual from group *i* comes into contact with an individual from group *j*, $\sum m_{ik}m_{jk}b_{ik}b_{jk}$ gives the rate that both individuals take on the same level of risk behaviors, $X_i \cdot \frac{Y_{jr}}{X_j+Y_j}$ gives the proportion of contacts a susceptible from group *i* has with an infective from group *j* in stage *r* of the disease, and β_{rk} gives the probability of transmission given that the infective individual is at stage *r* of the disease and that the behaviors operating at the time of contact are characteristic of risk group *k*. The Michigan HIV model can be generated from this model by assuming that $\beta_{rk} = \beta_r$ for all k and $\sum m_{ik}m_{jk}b_{ik}b_{jk} = 1$. The latter condition will hold, for example, if $b_{ik} = b_{jk} = 1/5$ and $m_{ik} = m_{jk} = \sqrt{5}$ for all i, j, and k.

Although other values will also guarantee that the condition holds, these particular values of b_{ik} and m_{ik} for the Michigan model make intuitive sense. Setting all b's equal to 1/5 is equivalent to saying that there is no differentiation among individuals with regard to taking on particular behaviors, a critical feature of models without context. However, because the context model considers the simultaneous behavioral change of two individuals it will always lead to fewer transmissions than the non-context Michigan model. This is due to the fact that there will be an effective contact between two individuals only if they both change their behavior to the same type, and so the probability of effective contact will be less than one (since it does not include the situations where they change to different types of behaviors). Consequently, in order to have the model equivalent to the Michigan model, individuals must come into contact more often to offset the decrease in effective contacts. The m_{ik} are the weights that compensate for this. What then becomes interesting is to see if there are qualitative differences between risk groups in the context model over and above a simple lowering of the number of cases compared to models without context.

NUMERICAL EXPLORATIONS OF THE MODEL

The Michigan model is used as a control in order to assess the importance of social context as incorporated into the new model. We therefore use in our simulations the same basic parameters as in Jacquez et al. (1988), Koopman et al. (1988), and Sattenspiel et al. (1990). These are as follows:

1) There is an underlying death rate from all causes other than AIDS of $\mu = 0.012$ per year, assumed to be constant over time and to occur among individuals from all disease classes in all subgroups.
2) The model assumes a constant recruitment rate of 100 individuals per month distributed among activity groups as follows: 24 per month into each of groups 1 and 2, 36 per month into group 3, 14 per month in group 4, and 6 per month in group 5. These numbers correspond to 0.1% of the equilibrium group sizes in an uninfected population of 100,000.
3) The rates at which the individuals proceed through the different stages of the disease are $k_1 = 0.5$ (for the transition from stage 1 infection to stage 2 infection), $k_2 = 3$, $k_3 = 1$, $k_4 = 1$ (for the transition from stage 4 infection to removal from sexual activity).
4) The probability of transmission per partnership at different stages is set at 0.03 for individuals in stages 1,3, and 4 while individuals in stage 2 of the disease are unable to transmit the infection. These values reflect the belief that infectivity is relatively high both early and late in the course of the disease and is much lower in the middle stages. These values are also per partner rather than per sexual contact, which does not allow the model to deal with the situation of one person having 100 contacts with the same partner, a limitation which this model shares with the Michigan model.
5) The starting population is set at 50,000 individuals in risk group 1, 33,000 individuals in risk group 2, 9,000 in risk group 3, 2,000 in risk group 4, and 1,000 in risk group 5. This initial distribution was chosen to be consistent with the simulations of the Michigan HIV model.

6) The average number of sexual contacts per person per month is 1 in risk group 1, 2 in risk group 2, 4 in risk group 3, 8 in risk group 4, and 16 in risk group 5. These were also chosen to be consistent with the Michigan simulations.

7) The probabilities of contact between individuals from different risk groups are assumed to follow the model of preferred mixing proposed by the Michigan HIV modeling group and described above. Runs were made with the degree of restriction of mixing, ρ, set at 0 (or random mixing), 0.3, and 0.9. The degree of restriction is the same for all risk groups.

In addition to these features in common with the classical compartmental models, there are two new features of the model presented here. First, the probability of transmission per partnership can be varied with the type of risk behaviors taken on at the time of contact as well as with the disease stage of the infected partner. The probability is constant for a given context, but can vary within a particular type of intergroup partnership. In other words, the probability of transmission per partnership is not constant for all partnerships between an individual from group *i* and one from group *j*. Rather, the probability depends on both the disease stage of the infected partner and the temporary assignment to risk group of the individuals involved. The latter factor reflects the behaviors taken on at the time of contact. Although this is a very useful aspect of the context model, initial simulations were designed to facilitate comparisons with the Michigan model and were run with no change in transmission rates across temporary behavior groups. Future studies will be addressed to the consequences of variable transmission rates associated with behaviors operating at the time of sexual contact.

The second new feature is the matrix, b, giving the probability of behavioral change (weighted by the frequency, m_{ik}, with which individuals take on specific behaviors per unit time). This matrix allows for the direct incorporation of context effects. Three different forms of the behavioral mobility matrix, b, were analyzed. Model I assumes that most individuals are consistent in the types of risk behaviors they engage in, and when they do deviate it is only rarely and only to the neighboring groups (Figure 1a). Models II and III assume that individuals usually take on a small range of behaviors in the neighborhood of their own behaviors but on occasion they may deviate

more from normal behaviors and engage in the highest risk behaviors. In model II 1% of individuals in groups 1-4 take on type 5 behaviors, while in model III 5% of individuals in groups 1-4 take on these behaviors. The behavior of group 5 individuals does not vary from that of model II (Figures 1b, c). In all of these models $m_{ik} = m_{jk} = 1$. In addition, simulations were run with a matrix in which all elements were equal to 1/5 and in which $m_{ik} = m_{jk} = \sqrt{5}$ for all i, j, and k. This matrix leads to a model in which context is not included and duplicates the results of the Michigan model.

As in the Michigan HIV models the simulations begin with one infective individual at the first stage of disease in each of the five subgroups. The simulations were run for 150 years in monthly steps.

[FIGURE 1 ABOUT HERE]

RESULTS

Effect of adding context, with equal transmission rates for each type of risk group

The first explorations of the model are concerned with the effect of incorporating the context structure into the model. Comparisons were made between the Michigan model, which incorporates preferred mixing but no context and models I-III which also use preferred mixing but have incorporated various formulations to take into account the context of sexual interactions. Three levels of intergroup contact (or degrees of restricted mixing) were used: 1) random contact among individuals from different risk groups ($\rho_i = 0$), 2) 30% restriction to own risk group ($\rho_i = 0.3$), and 3) 90% restriction to own group ($\rho_i = 0.9$). In all of these comparisons the risk of transmission did not vary by context of the interaction, i.e. all types of sexual behaviors resulted in the same risk of transmission.

Results from these analyses are presented in Table 1 and Figures 2 and 3. Table 1 gives the peak number and proportion of cases, the time at which the peak occurred, and the final number

and proportion of cases for each run of the simulation. A comparison of the values for runs without context with those for all context models shows that, as expected, the size of the epidemic is significantly reduced in all risk groups when context effects are included. The peak number of cases of the disease in a model without context varies from 1.05 to approximately 2500 times the number of cases in a context model, depending upon the model and risk group chosen for comparison. The effects are much stronger for low risk groups than for high risk groups.

[TABLE 1 ABOUT HERE]

The context model is expected to result in a decrease in the number of cases of the disease because it requires that individuals not only come into contact but simultaneously change to the same types of behaviors. Since a proportion of all contacts will involve individuals who change to different types of behaviors, and since these types of contacts will no longer result in transmission, fewer transmissions will occur than in models without context, which consider all contacts between individuals potentially capable of resulting in transmission.

This inherent restriction on the number of effective contacts does not appear to explain all of the differences between the context and no context models, though, since different risk groups seem to be differentially affected by the incorporation of context. This result shows up clearly in graphic comparisons of the prevalence of the disease (Figures 2a and 2b give examples of some of the simulation results). There are qualitative differences in the results as well as numerical changes. Figure 2a shows that under conditions without context risk group 3 has the largest number of cases in the population at the peak of the epidemic (this is true for most of the simulations without context). Because of the relatively larger decrease in cases in group 3, when context is added the peak number of cases is largest for either group 4 or group 5 depending upon the context model used (Figure 2b).

[FIGURE 2 ABOUT HERE]

The peak prevalence is an important variable to look at, because it gives an idea of the actual number of cases to be expected at any given time. However, when the initial group sizes vary, as they do in these simulations, the peak prevalence is strongly influenced by the group size chosen. This is a major reason why there are more cases in group 3 than in other groups in the models without context effects.

Comparisons of the fraction infected in each risk group for different models are not subject to these effects of variable group size. These comparisons indicate the relative importance of the disease for different risk groups. A sample from the simulations is given in Figures 3a-d. In all simulations, both with and without context, and including simulations other than those presented in Figures 3a-d, a larger fraction of individuals from high risk groups were infected than from lower risk groups. The relative ordering of the fractions infected was risk group 5 > risk group 4 > risk group 3 > risk group 2 > risk group 1 for all simulations and at almost all times. Rarely the peak prevalence for risk group 4 lagged long enough behind the peak prevalence for risk group 5 that the proportion infected in group 4 was equal to or slightly larger than the proportion infected in group 5. This occurred in some of the simulations with 90% restriction of mixing and was only present for short periods of time (for example, see Figure 3d).

Comparisons of Figure 3a with Figure 3b and Figure 3c with Figure 3d also indicate that the fraction infected in each risk group is markedly reduced when context is included in the model. In addition, the reduction in the fraction infected is not equal for all risk groups. There is a much greater reduction in low risk groups than in high risk groups (Table 1), which indicates that the reduction is probably not solely due to a restriction in the proportion of effective contacts. This is true for all groups in all simulations with random mixing, for all but the highest risk group in simulations with 30% restriction, and for the lower three risk groups in simulations with 90% restriction. These results clearly indicate that there are interactions between the effects of restricted mixing and the effects of context as well as a differential effect due to the incorporation of context itself.

[FIGURE 3 ABOUT HERE]

The results from Figures 2 and 3 and Table 1 show, in addition, that under many conditions the decrease in prevalence as a consequence of context effects is enough that the two lowest risk groups are barely touched by the epidemic. This is because of the increased restrictions on sexual contact imposed by context (in that individuals must both come into contact and be in the same temporary behavioral class).

In addition to the number and proportion of cases, the timing of the epidemic peaks is significantly influenced by context. Figures 2 and 3 and Table 1 show that the epidemics are delayed when context is included relative to simulations without context. Thus the major effects of context are to decrease the number and proportion of cases in each risk group, with differential decrease in lower risk groups, and to significantly slow the epidemic process. In addition, numerical results indicate that when context is present in the models there is low amplitude cycling of the values of the variables. This cycling does not appear to be present in models without context, but it may be that the models have not been run long enough for it to become apparent.

Comparison of different behavioral mobility models

Comparison of the nearest neighbor mobility model (Model I), the model with extreme behavioral mobility of 1% of the population (Model II), and the model with extreme behavioral mobility of 5% of the population (Model III) shows that when the transmission probabilities do not vary with the behavioral risk group, Model I and Model II have very similar disease prevalences, while the prevalences in Model III are somewhat different (although the differences are not as marked as the effects of context in general) (Table 1). The patterns are less clear for fraction infected, especially in the lower risk groups with variable transmission probabilities. These results

indicate that the frequency with which individuals deviate markedly from their normal behaviors often must be relatively high in order to affect significantly the outcome of the epidemic.

DISCUSSION

Most previous non-age-structured HIV models have considered behavior to be a constant for a given individual. However, actual behavior is extremely plastic. People are constantly adjusting their behavior to the conditions or context present at any particular time. This plasticity of behavior often includes variability in the risk of transmission experienced by an individual. Truly realistic models must be able to take account of individual flexibility in behavior. The model presented above attempts to incorporate the effects of changes in behavior occurring in specific contexts.

Results from the model clearly show that context can significantly affect the course of an epidemic. These effects can be quite marked — in the simulations presented here the decrease in the prevalence of the disease was as high as three orders of magnitude for some risk groups and the fraction infected in a risk group in a context model was sometimes 2% to 5% that of a similarly-structured model that did not include context. In addition, context tends to slow the epidemic down.

These results are similar to those of Dietz and Hadeler (1987) and Dietz (1988) who use models which explicitly consider pair formation at the individual level. In these models temporary periods of immunity occur because individuals are immune when involved in partnerships where both partners are uninfected. Introducing social context has a similar effect — two individuals must be in the same place at the same time and be engaging in the same types of behaviors for effective contact to occur. If these conditions are not met then the individuals are immune from transmission. Both of these mechanisms reduce the overall incidence and hence generate less serious epidemics.

However, the added realism of these kinds of models presents costs as well. In order to use complex models for forecasting and other practical considerations one needs to be able to estimate

the values of all parameters. Additional complexity often adds parameters for which it is difficult or impossible to find relevant data. There have been several studies in biology where simple models better predict the output generated by complex models than the complex models do themselves (Ludwig, 1989). However, this level of detail is critical in order to answer theoretical questions such as the one posed in this paper.

A major question that needs to be answered given these problems is whether the incorporation of context is necessary in models for HIV transmission. The answer to this depends on the focus of the study. If the focus is accurate projections of epidemics then clearly context is necessary. Projections require as realistic a model as possible.

On the other hand, the qualitative results of this model are not too different from models without context. Generally the addition of context has a much stronger influence on the lower risk groups than on the high risk groups. This results in a shift in the group with the highest prevalence of disease from risk group 3 to risk group 4 or 5. However, although the relative prevalence of infection changes, the ordering of the groups in terms of fraction infected does not change. The higher the risk group the larger the fraction of that group infected with the virus. Thus, whether context is included or not, a larger proportion of high risk individuals become infected than lower risk individuals.

In general, the effects of context are significant enough that if the goal is to realistically model the course of the epidemic contextual factors should certainly be considered. If the goal is to increase basic understanding of various factors influencing the disease (such as variability of infectivity, patterns of mixing, etc.) then a simplifying assumption that context is not important is probably justified. It is important to realize, though, that in investigations of factors which are highly dependent upon contextual influences, such as variability in transmission rates, a model ignoring context is likely to lead to serious misconceptions.

No model is totally realistic and the model presented above is no exception. In addition to simplifying assumptions common to most other HIV models, this model contains a few assumptions that deserve comment. First, the structure of the model is such that once two

individuals meet each other they need only be classed into the same behavioral group for a sexual contact to occur. Although this is more realistic than most other models, which assume that meeting is synonymous with sexual contact, it is not what happens in the real world. There are complex, mostly poorly understood patterns of behavior that determine whether a social interaction will lead to a sexual partnership. In addition, assignment to a particular risk group does not imply a particular set of risk behaviors. One individual may fall into a low risk group because of engaging in moderately risky sex but with low frequency, while a second individual may fall into the same risk group because of a moderate frequency of low risk sex. Clearly, two individuals must take on the same level of risk behavior in order to have a successful interaction and they must be willing to engage in the same sorts of behaviors, but there are many other factors involved and willingness does not mean the behaviors actually occur. This is one aspect in the modeling of sexual partnerships that deserves further research.

A second limitation of the model is the problem associated with definition of the risk groups. What risk behaviors must be included? How are these behaviors to be quantified in such a way that the population variability can be collapsed into a few discrete groups? These are problems inherent in any discrete model, and although the use of discrete models presents obvious limitations they have resulted in great increases in our understanding of a number of problems. (Some work has been done comparing the results from continuous and corresponding discrete HIV models (Blythe and Anderson, 1988a). Results show that for the model studied a discrete version with six behavioral classes gives a reasonable approximation to a continuous model.)

Attempts were made to study the effects of variability in the behavioral mobility matrix. Although the results from these simulations showed that there was not much difference in the outcome of the epidemic for the models chosen (Models I-III), the choice of parameters was quite limited. Further study of other possible models is necessary to evaluate the importance of behavioral mobility for transmission of HIV.

CONCLUSIONS

The use of models to understand the transmission system of HIV has led to a significant increase in our understanding of the important factors driving the epidemic. Previous studies have shown that sexual activity level, variability in infectivity of the virus, and patterns of mixing among subgroups are important in determining the impact of the disease on the population. Results from this study indicate that the social context operating at the time of a potential sexual interaction can also affect the course of the epidemic. These effects are most marked for the prevalence of the disease in different risk groups and the timing of the epidemic peaks. Incorporation of the effects of context is most important for models whose goal is prediction of the future course of the epidemic or allocation of resources towards control. When the goal of the modeling is to increase understanding of the transmission system of HIV then the decision to incorporate context effects must be evaluated with reference to the focus of the particular model used. Models that focus on variability in parameters that would be strongly affected by the conditions operating at the time of contact, such as the probability of transmission for a sex act, must consider the effects of context. Models which focus on other factors may be justified in assuming that context is unimportant.

The development and analysis of mathematical models for the transmission of HIV has led to much greater understanding of the factors most important in explaining the observed spread of the disease. Because the spread of HIV is an epidemic that is happening at the present and not one that has already happened, and because there has never before been such an epidemic, long-term forecasting must be done with the use of models that are based on a solid understanding of the biology of the virus and the biology and behavior of the host. Without this biological understanding and without the knowledge that is derived from models based on this understanding, predictions of the effects of the epidemic can only be justified for a very short time into the future. The model presented above adds to the base of scientific knowledge necessary for long-term predictions.

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Table 1. Peak and final prevalence and proportion infected by risk group.

RISK GROUP 1

run of simulation[1]	peak prevalence	time	final prevalence	peak proportion	time	final proportion
rm,nc	2367.68	16	924.46	0.3123	18	0.2426
rm,mI	1.00	0	0.19	0.000104	0	0.0000195
rm,mII	3.09	37	1.53	0.000322	37	0.00016
rm,mIII	16.14	36	7.46	0.00169	36	0.000781
30,nc	1967.12	15	853.68	0.2508	18	0.2007
30,mI	2.24	50	1.52	0.000233	52	0.000158
30,mII	4.88	17	4.07	0.000509	17	0.000425
30,mIII	24.37	17	14.32	0.00254	17	0.00151
90,nc	433.79	20	345.15	0.0488	22	0.0463
90,mI	1.11	2	0.687	0.000116	2	0.0000716
90,mII	1.58	47	1.43	0.000165	47	0.000149
90,mIII	4.92	11	4.39	0.000513	11	0.000459

RISK GROUP 2

rm,nc	3563.95	14	1088.04	0.5136	18	0.3904
rm,mI	10.29	54	5.46	0.00107	55	0.000571
rm,mII	14.17	49	8.63	0.00148	50	0.000904
rm,mIII	41.72	38	22.19	0.00437	38	0.00235
30,nc	3235.95	13	1058.46	0.4623	17	0.3562
30,mI	66.57	48	44.28	0.00705	48	0.00475
30,mII	67.65	47	48.06	0.00718	48	0.00517
30,mIII	76.91	44	64.39	0.00823	44	0.00700
90,nc	1515.18	21	848.08	0.2275	31	0.1977
90,mI	66.02	49	55.13	0.00698	49	0.00595
90,mII	64.19	49	53.24	0.00679	49	0.00574
90,mIII	58.48	50	49.68	0.00620	51	0.00535

RISK GROUP 3

run of simulation[1]	peak prevalence	time	final prevalence	peak proportion	time	final proportion
rm,nc	6512.39	12	1591.52	0.7264	16	0.5616
rm,mI	268.13	51	140.56	0.0212	56	0.0118
rm,mII	283.05	49	150.44	0.0232	50	0.0127
rm,mIII	357.21	44	189.29	0.0294	45	0.0163
30,nc	6389.14	11	1585.85	0.7147	15	0.5527
30,mI	1352.65	40	791.84	0.1434	47	0.1006
30,mII	1286.65	39	766.43	0.1341	46	0.0956
30,mIII	1092.48	35	683.52	0.1072	41	0.0802
90,nc	5403.57	14	1576.07	0.6876	19	0.5378
90,mI	3516.00	34	1457.73	0.5109	41	0.3970
90,mII	3388.23	34	1433.79	0.4930	41	0.3841
90,mIII	2854.69	36	1377.78	0.4175	44	0.3302

RISK GROUP 4

rm,nc	3611.05	11	731.81	0.8738	14	0.7193
rm,mI	673.03	41	288.53	0.1524	44	0.0761
rm,mII	677.26	40	288.60	0.1529	43	0.0761
rm,mIII	698.99	38	291.66	0.1567	41	0.0774
30,nc	3725.03	9	733.77	0.8843	13	0.7300
30,mI	1935.37	20	624.50	0.5480	25	0.3695
30,mII	1902.39	20	619.69	0.5367	25	0.3602
30,mIII	1780.79	20	600.05	0.4929	25	0.3257
90,nc	3976.57	8	739.46	0.9079	12	0.7627
90,mI	3328.31	13	727.22	0.8562	16	0.6951
90,mII	3298.12	13	725.97	0.8506	17	0.6888
90,mIII	3082.33	13	720.55	0.8257	18	0.6623

RISK GROUP 5

run of simulation[1]	peak prevalence	time	final prevalence	peak proportion	time	final proportion
rm,nc	1797.14	10	321.84	0.9452	12	0.8367
rm,mI	550.96	32	196.39	0.3421	35	0.1680
rm,mII	557.41	32	197.84	0.3492	36	0.1706
rm,mIII	583.32	31	204.05	0.3699	35	0.1821
30,nc	1922.14	8	322.73	0.9538	10	0.8514
30,mI	1413.35	13	306.00	0.8369	16	0.6323
30,mII	1413.35	13	306.02	0.8373	16	0.6326
30,mIII	1415.34	12	306.11	0.8391	16	0.6336
90,nc	2179.10	5	324.41	0.9704	7	0.8830
90,mI	2066.29	6	322.50	0.9584	8	0.8475
90,mII	2066.27	6	322.50	0.9584	8	0.8476
90,mIII	2066.22	6	322.51	0.9585	8	0.8477

[1]rm = random mixing, 30 = 30% restriction, 90 = 90% restriction; mI-mIII refers to the context model of the run, nc refers to the Michigan model (no context).

FIGURE LEGENDS

Figure 1. Forms of the behavioral mobility matrix, b, used in the simulations. a) Model I — neighboring behaviors only, b) Model II — neighboring behaviors plus 1% taking on extreme behaviors, c) Model III — neighboring behaviors plus 5% taking on extreme behaviors. These models are explained in more detail in the text.

Figure 2. Disease prevalence by group for models with and without context. a) random mixing , no context (Michigan model), b) random mixing, context Model I (neighboring behaviors only). The numbers above the curves represent the risk groups, with 1 the lowest risk group and 5 the highest risk group. In the Michigan model these groups are defined on the basis of level of sexual activity only, in the context models they are defined on the basis of a number of risk characteristics.

Figure 3. Proportion infected by group for models with and without context. a) random mixing, no context effects (Michigan model), b) random mixing, context Model III (5% extreme behaviors), c) 90% restricted mixing, no context (Michigan model), d) 90% restricted mixing, context Model I (neighboring behaviors only). The numbers above the curves represent the risk groups, with 1 the lowest risk group and 5 the highest risk group. In the Michigan model these groups are defined on the basis of level of sexual activity only, in the context models they are defined on the basis of a number of risk characteristics.

$$\begin{pmatrix} 0.89 & 0.1 & 0 & 0 & 0.01 \\ 0.05 & 0.89 & 0.05 & 0 & 0.01 \\ 0 & 0.05 & 0.89 & 0.05 & 0.01 \\ 0 & 0 & 0.05 & 0.89 & 0.06 \\ 0 & 0 & 0 & 0.1 & 0.9 \end{pmatrix}$$

(a) Model I

$$\begin{pmatrix} 0.9 & 0.1 & 0 & 0 & 0 \\ 0.05 & 0.9 & 0.05 & 0 & 0 \\ 0 & 0.05 & 0.9 & 0.05 & 0 \\ 0 & 0 & 0.05 & 0.9 & 0.05 \\ 0 & 0 & 0 & 0.1 & 0.9 \end{pmatrix}$$

(b) Model II

$$\begin{pmatrix} 0.85 & 0.1 & 0 & 0 & 0.05 \\ 0.05 & 0.85 & 0.05 & 0 & 0.05 \\ 0 & 0.05 & 0.85 & 0.05 & 0.05 \\ 0 & 0 & 0.05 & 0.85 & 0.1 \\ 0 & 0 & 0 & 0.1 & 0.9 \end{pmatrix}$$

(c) Model III

Figure 1

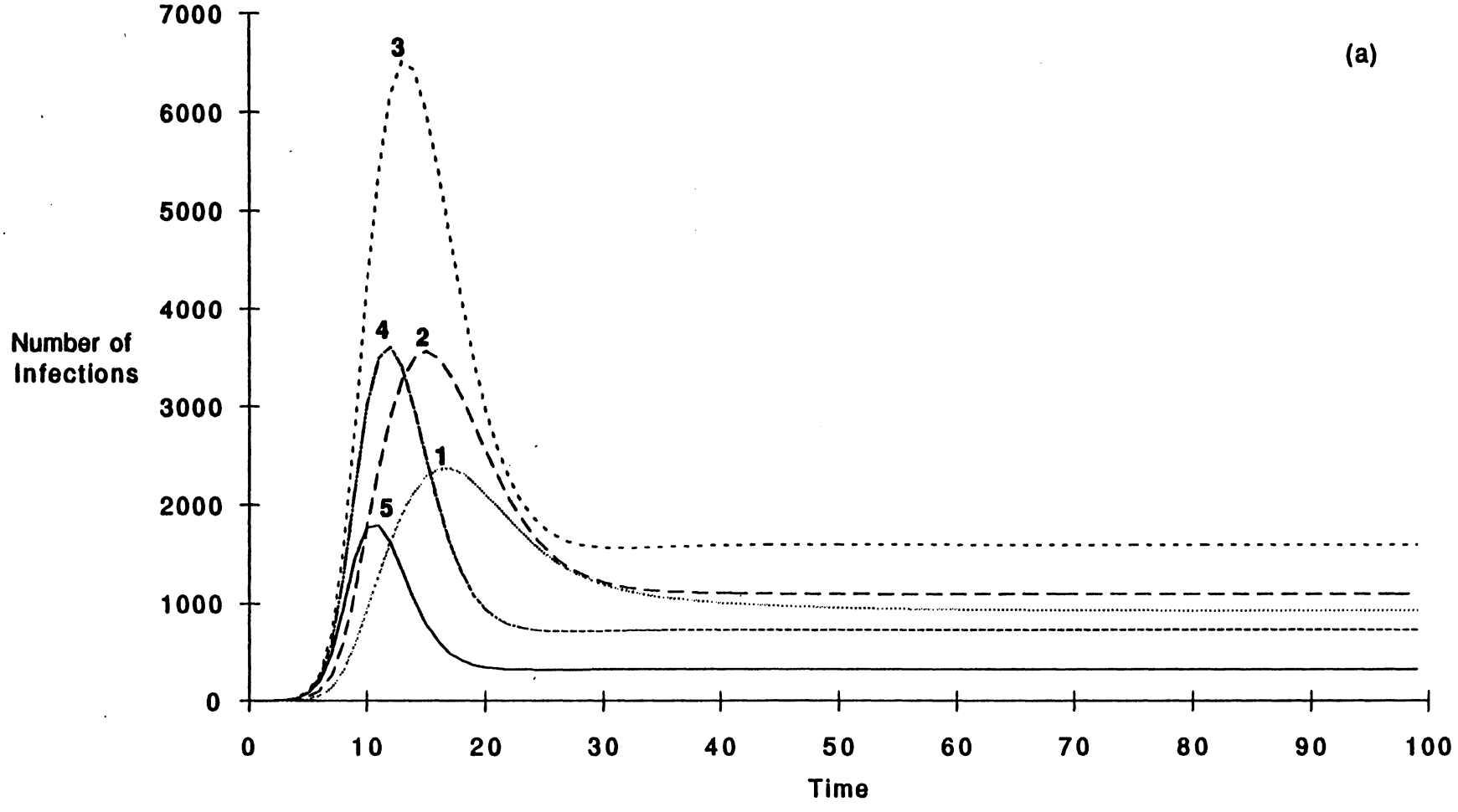
(a)
Number of Infections
7000
6000
5000
4000
3000
2000
1000
0
0
10
20
30
40
50
60
70
80
90
100
Time
1
2
3
4
5

Figure 2a

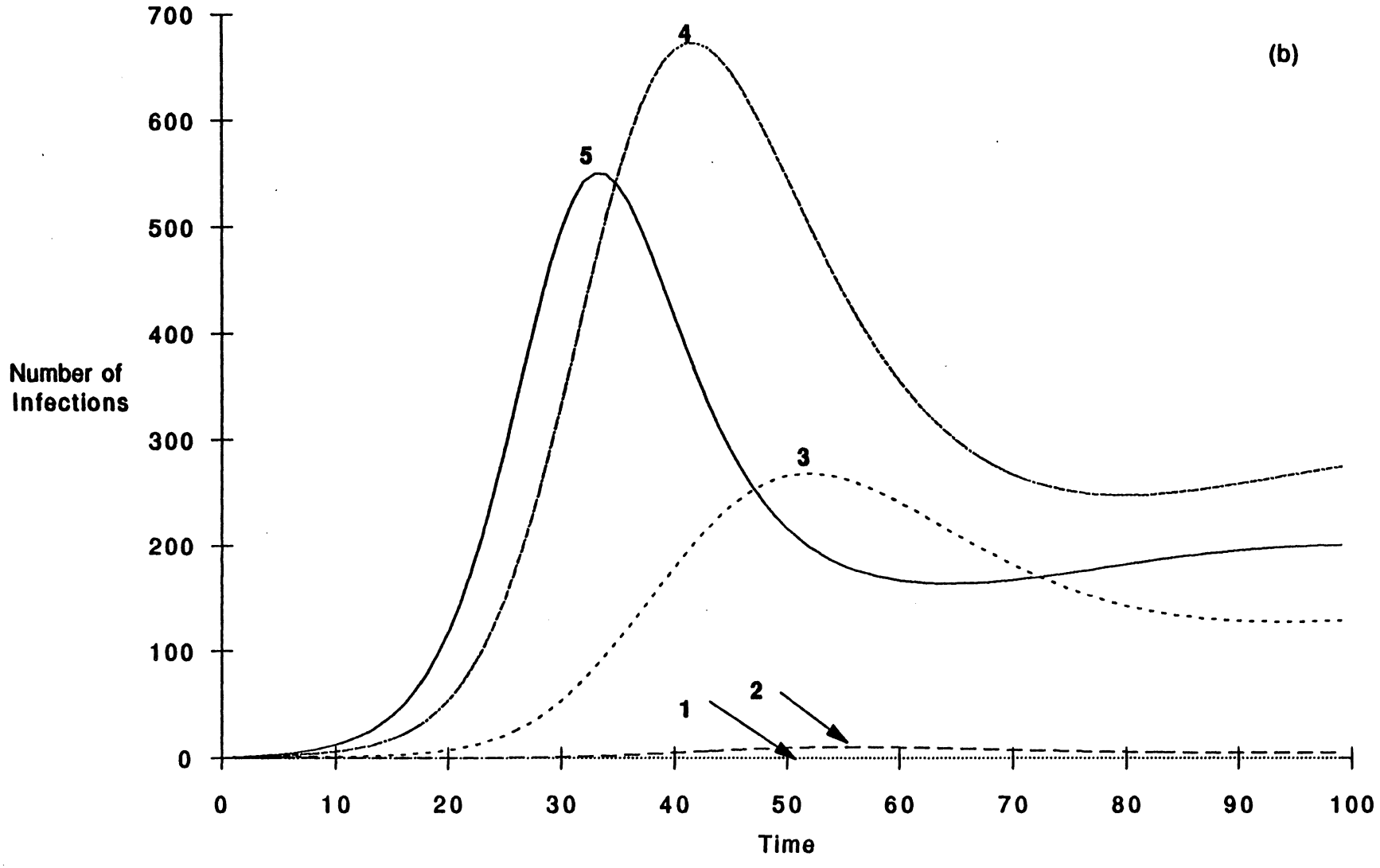
(b)
700
600
500
400
300
200
100
0
Number of Infections
0
10
20
30
40
50
60
70
80
90
100
Time
1
2
3
4
5

Figure 2b

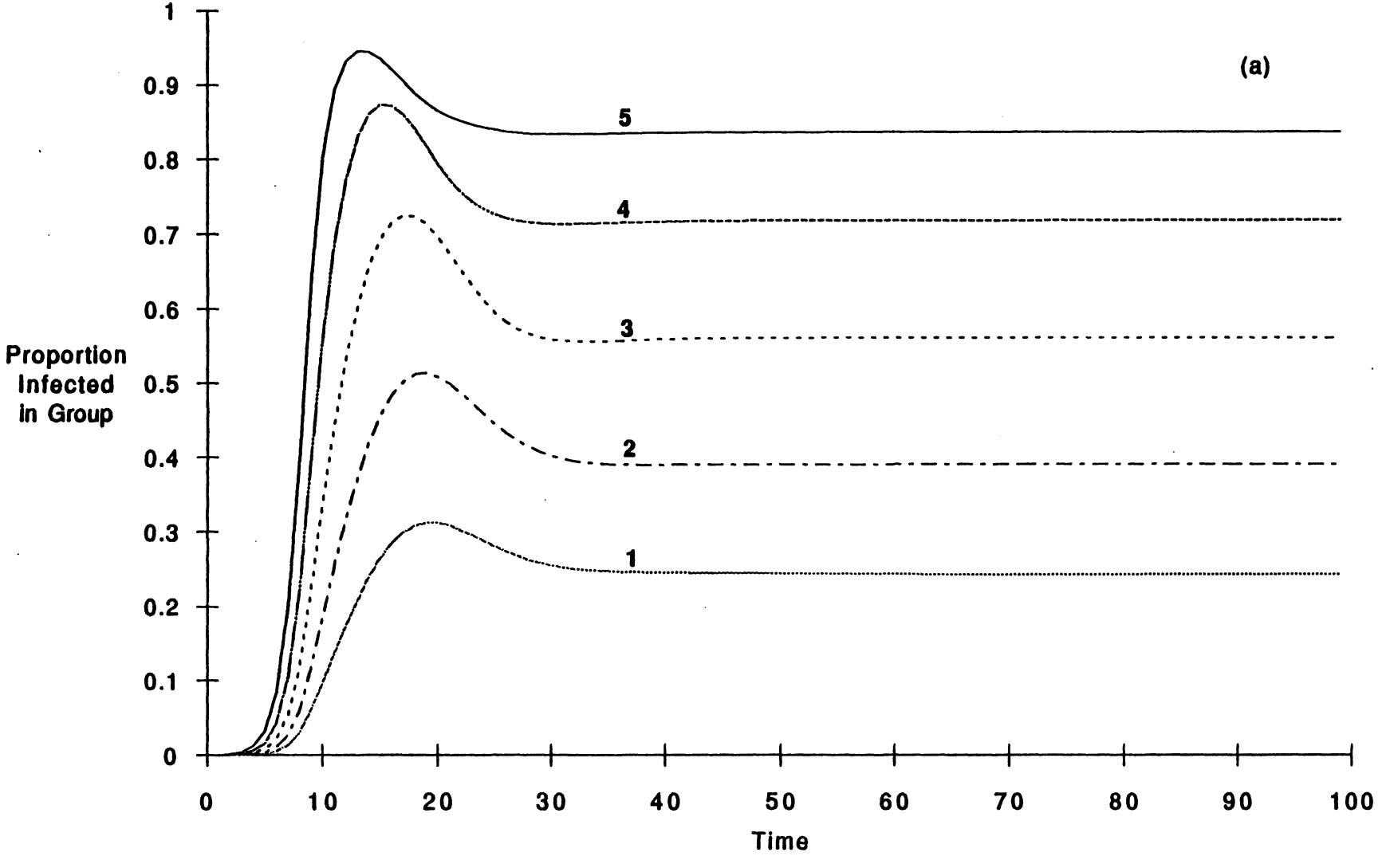
(a)
Proportion Infected in Group
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
5
4
3
2
1
0
10
20
30
40
50
60
70
80
90
100
Time

Figure 3a

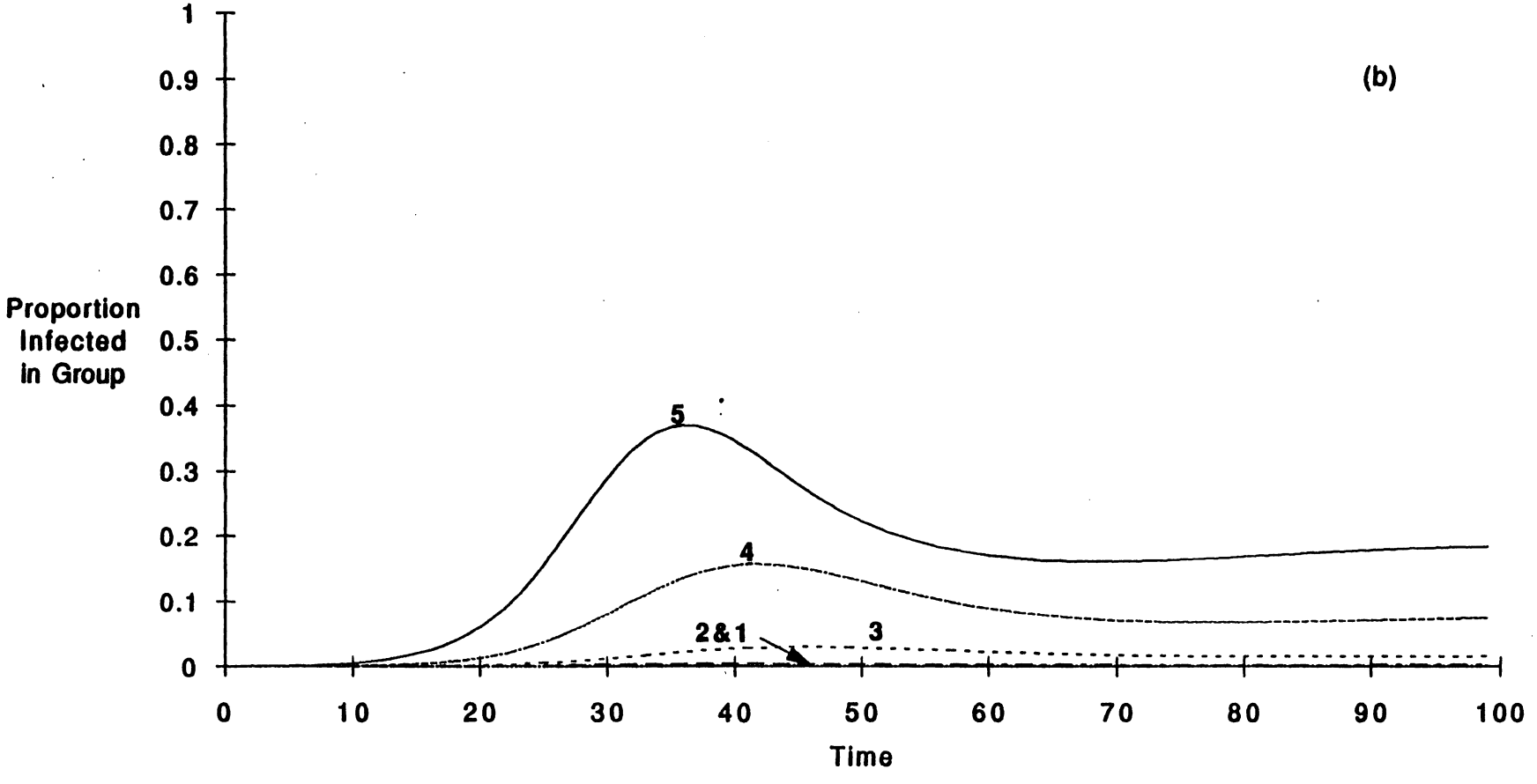
(b)
Proportion Infected in Group
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
5
4
2&1
3
0
10
20
30
40
50
60
70
80
90
100
Time

Figure 3b

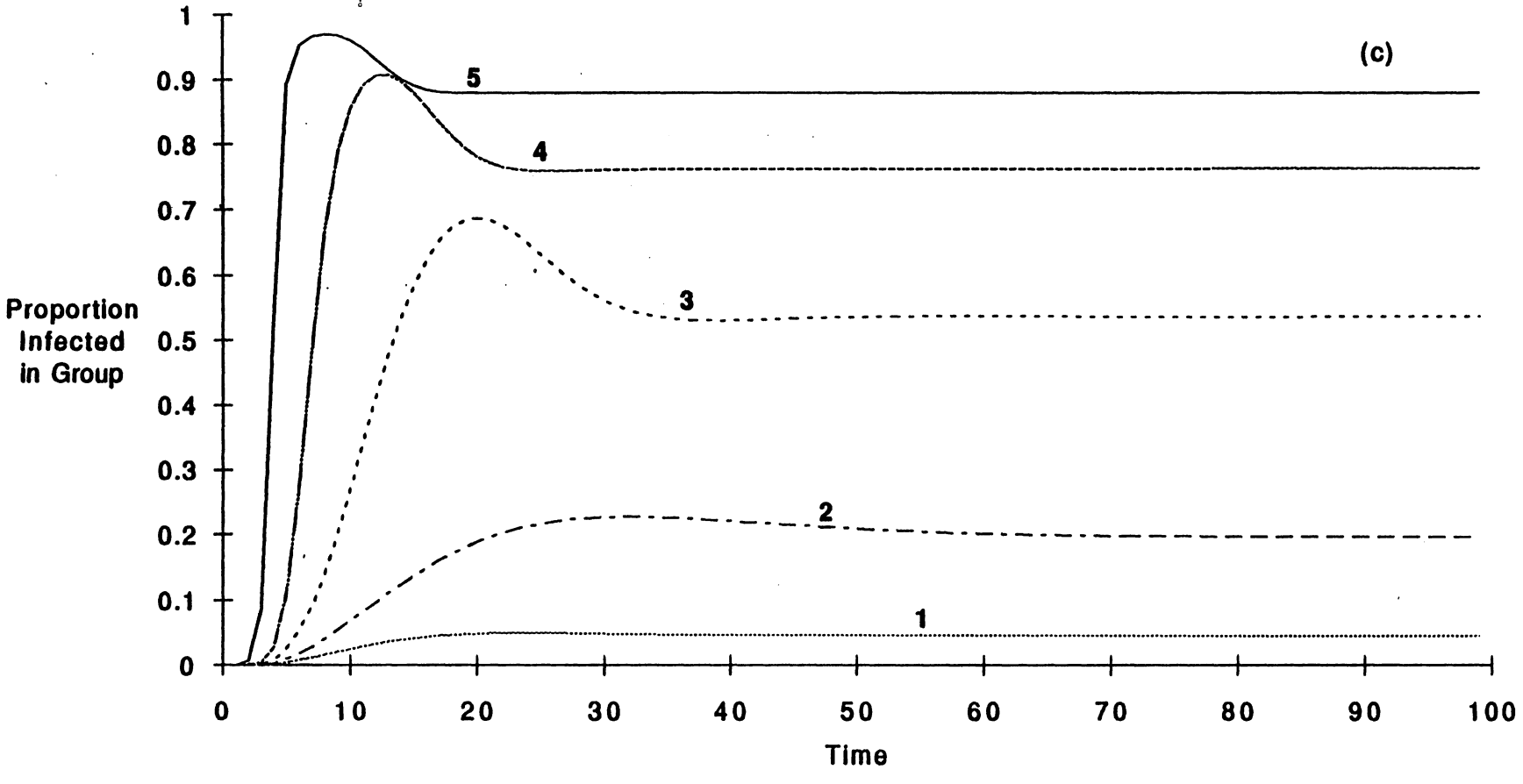
(c)
Proportion Infected in Group
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
5
4
3
2
1
0
10
20
30
40
50
60
70
80
90
100
Time

Figure 3c

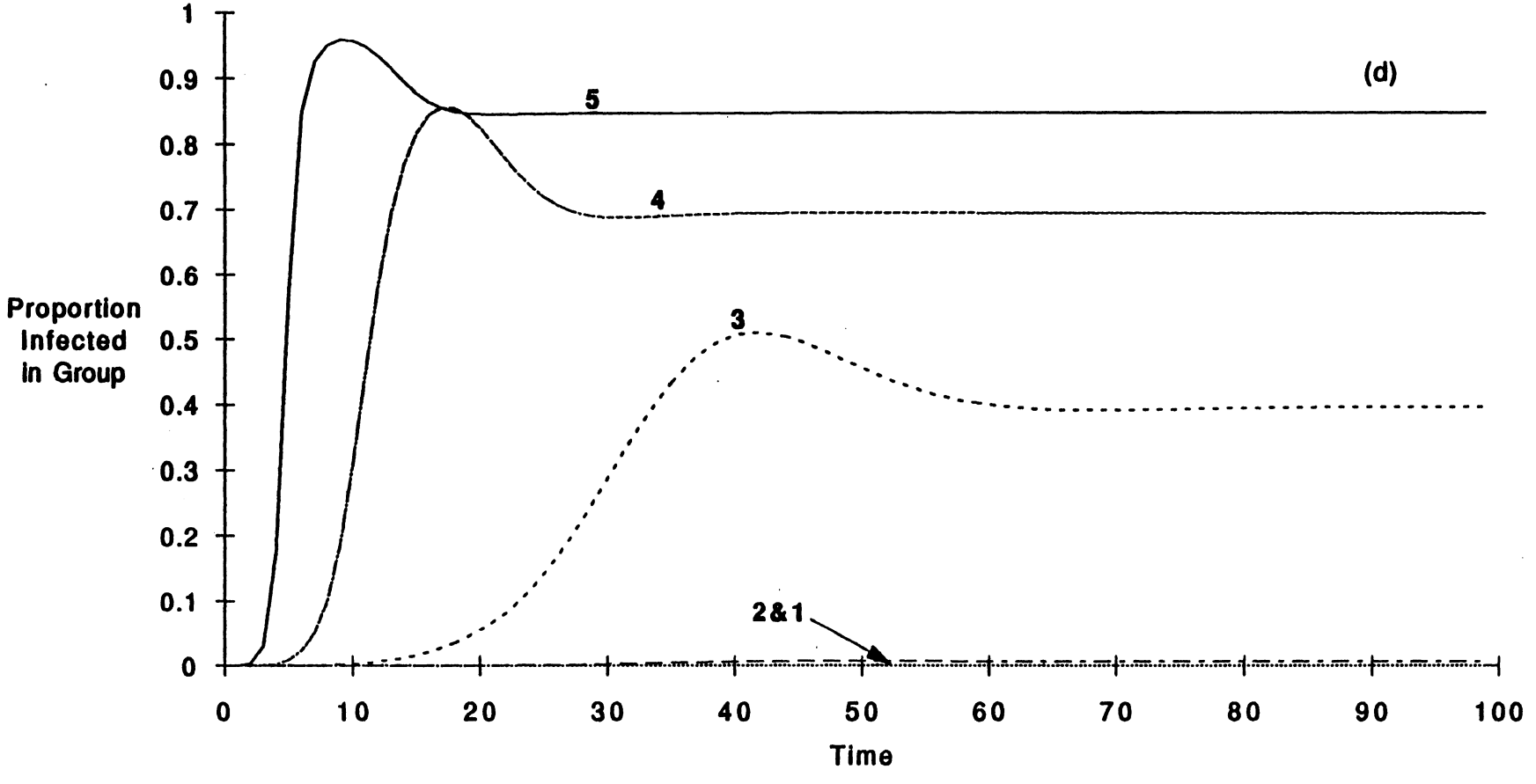
(d)
5
4
3
2&1
Proportion Infected in Group
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
10
20
30
40
50
60
70
80
90
100
Time

Figure 3d