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"Modeling Contact Structures in Biology"

Carlos Castillo-Chavez
Biometrics Unit
Cornell University
Ithaca, NY 14853-7801
U.S.A.

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MODELING CONTACT STRUCTURES IN BIOLOGY

CARLOS CASTILLO-CHAVEZ
Biometrics Unit, Warren Hall,
Cornell University, Ithaca, NY 14853.

JORGE X. VELASCO-HERNANDEZ †
Depto. de Matemáticas, División de Ciencias Básicas e Ingeniería,
UAM-Iztapalapa, Apdo. Postal 55-534, México D.F. 09340 MEXICO.

SAMUEL FRIDMAN
Biometrics Unit, Warren Hall,
Cornell University, Ithaca, NY 14853.

Abstract. Social dynamics have had a strong impact on the development of theoretical epidemiology over the last six years. Interactions or contacts among individuals have traditionally been modeled by the use of the mass-action law or proportionate mixing, giving limited understanding of effects that the environment, changing social structure, has on disease dynamics. Furthermore, while gender plays a central role in the dynamics of sexually-transmitted diseases, the use of two-sex models has been rare. In this article, we review briefly our mixing/pair formation framework and illustrate its application to population models of the type currently used in demography, epidemiology, and social dynamics. A new application to frequency dependent competitive interactions is discussed in more detail. Connections between deterministic and stochastic processes are presented. The results of the simulations of a demographic two-sex stochastic model that follows the dynamics of pairs are presented.

Keywords. Epidemiology, social dynamics, demography, food web dynamics, mixing and pair formation, contact rates, consumer-resource interactions, predator-prey systems, differential equation models, vector-transmitted diseases, AIDS, sexually-transmitted diseases, stochastic processes.

1. INTRODUCTION

The transmission of diseases, genetic characteristics, or cultural traits is influenced by many factors including the contact/social structure of the interacting subpopulation, that is, the social environment. Classical demography (see MacKendrick, 1926; Lotka, 1922; and Leslie, 1945) ignores social dynamics and usually concentrates on the birth and death processes of female populations under the assumption that they have reached a stable

† Jorge X. Velasco-Hernández dedicates his part of this work to the memory of Stavros Busenberg, who was his teacher, adviser and friend.

age distribution. They usually ignore the specific mating/contact structure of the population. The incorporation of mating structures or marriage functions, as they are commonly referred to in human demography, was pioneered by Kendall (1949) and Keyfitz (1949). However, despite the fact that their work was extended by Parlett (1972), Fredrickson (1971), McFarland (1972), and Pollard (1973) two decades ago, their impact on demography, epidemiology, and population biology has been minimal.

The grim scenario due to the HIV/AIDS epidemic has accelerated the pace at which social dynamics have been incorporated into epidemiological models. Researchers are developing new models and innovative modeling approaches to help us identify and/or improve our understanding of the mechanisms responsible for HIV transmission; for example, there has been intensive research activity on the effects of social dynamics, the immune system variability, etc., in HIV dynamics as well as on the development of methods for the evaluation of competing control measures.

Dietz (1988a) and Dietz and Hadeler (1988) have brought to the forefront, and for the first time in epidemiology, models that incorporate heterogeneity through the processes of pair formation and dissolution. Epidemic models can be very sensitive to changes in the sexual/social mixing structure of interacting subpopulations and may not only have different quantitative dynamics but also distinct global dynamics.

The work that we (this generic 'we' includes many collaborators who will be cited throughout the text) have conducted over the last few years has had as a major component the development of a mathematical framework for the systematic incorporation of very general contact structures. The modeling approach outlined in Section 2 of this manuscript has been used to develop models for the study of disease dynamics, the dynamics of frequency-dependent predation in heterogeneously mixing populations (food web dynamics), transmission dynamics of cultural traits, social dynamics, general demographic processes, and so forth. In addition, we have made serious efforts to connect these models to data (see Rubin *et al.*, 1992; Castillo-Chavez *et al.*, 1992; Hsu Schmitz and Castillo-Chavez, 1992) and have participated in the collection of appropriate data (see Crawford *et al.*, 1990).

This manuscript is organized as follows: Section 2 introduces the basic formalism and states the basic theoretical results; Section 3 uses it in the context of demography and social dynamics; Section 4 applies the same approach to model frequency dependent predation and food web dynamics; Section 5 discusses the uses of our approach in the study of models for vector-transmitted diseases. Section 6 illustrates the connections between deterministic and stochastic processes. The results of the simulations of a demographic two-sex stochastic model that follows the dynamics of pairs are presented.

2. BASIC FRAMEWORK FOR CONTACT STRUCTURES

The mass-action law has played a central role in the development of stochastic and deterministic epidemiological models (see Bailey, 1975; Anderson, 1982; Anderson and May,

1991; and references therein). The assumption that the rate of new infections (the incidence) is proportional to the product of susceptibles and infectives in the exposed population has no mathematical significance when one deals with interacting subpopulations that have a constant number of individuals (although it may have an important effect on the interpretation of relevant epidemiological parameters such as the transmission coefficient). However, the mass-action assumption seriously affects the qualitative and quantitative behavior of models with interacting subpopulations of varying size (that is, when the sizes of the interacting subpopulations vary according to deterministic or stochastic rules). Unfortunately, a thorough analysis of basic assumptions such as those implicit in the mass-action law was not carried out in a systematic fashion because mathematical epidemiology was growing almost independently of epidemiology (there are some exceptions, *e.g.*, see Hethcote and Yorke, 1984).

The HIV/AIDS epidemic revealed the deficiencies and inadequacies of the existing theory. Several questions relevant to the dynamics of heterogeneously mixing populations affected by fatal diseases could not be properly studied under the existing framework. The contact structure of the population must respond at least to the potential population changes due to a heterogeneously transmitted fatal disease (or more generally to frequency dependent predation). The importance of the contact process—well recognized by Ross (1911) in his work on malaria—in frequency-dependent systems has motivated the work that we present in the final volume of this series.

Our general approach for modeling contact processes describes who is mixing or pairing with whom. We let $M(a,t)$ denote the density of males of age a who are not in pairs at time t, and let $F(a,t)$ denote the density of females of age a who are not in pairs at time t. Pairing is defined through the mixing functions:

1. $p(a,a',t)$, proportion of partnerships of males of age a with females of age a' at time t, given that they formed a partnership;
2. $q(a,a',t)$, proportion of partnerships of females of age a with males of age a' at time t;
3. $C(a,t)$ expected or average number of partners of a male of age a at time t per unit time;
4. $D(a,t)$ expected or average number of partners of a female of age a at time t per unit time.

Definition 1. The following natural conditions characterize these mixing functions:

i) $p, q \geq 0$;
ii) $\int_0^\infty p\,(a,a',t)\,da' = \int_0^\infty q\,(a',a,t)\,da = 1$;
iii) $p\,(a,a',t)\,C\,(a,t)\,M\,(a,t) = q\,(a',a,t)\,D\,(a',t)\,F\,(a',t)$;
iv) $C\,(a,t)\,M\,(a,t)\,D\,(a',t)\,F\,(a',t) = 0 \Rightarrow p\,(a,a',t) = q\,(a',a,t) = 0$.

Condition (ii) is due to the fact that p and q are probabilities. Condition (iii) simply states that the total rate of pair formation between males of age a and females of age

a' equals the total rate of pair formation between females of age a' and males of age a (all per unit time and age). Condition (iv) says that there is no mixing in the age and activity levels where there are no active individuals; *i.e.*, on the set $\mathcal{L}(t) = \{(a, a', t) : C(r, a, t)M(a, t)D(a, t)F(a, t) = 0\}$.

Remarks on property (iii) :

Remark 1. This property states the obvious, that is, that the *effective* rate of outflow from the a-male compartment into the (a-male, a'-female) paired compartment must match the outflow from the a'-female compartment if we are to have only heterosexual pairs or contacts.

Remark 2. Property (iii) would be satisfied only for very special functions C and D if we insist on assuming that they are only functions of a and a' respectively. This can, and must, be relaxed in a variety of ways. We (Castillo-Chavez *et al.*, 1993c) have studied frameworks and models that assume that C and D are functions of M and F.

Remark 3. If we replace axiom (iii) by

$$\theta(t)C(a, t)M(a, t)p(a, a', t) = D(a', t)F(a', t)q(a', a, t) \qquad \text{for all } a, a'$$

where

$$\frac{1}{\theta(t)} = \frac{\int_0^\infty C(a, t)M(a, t)da}{\int_0^\infty D(a', t)F(a', t)da'} \qquad \text{(the sexual activity ratio)} \quad (*)$$

then property (iii) is automatically satisfied for "arbitrary" functions C and D. However, this solution, in some sense, implies that one sex calls all the shots. This can be modified so that the gender with the smaller number of total contacts per unit time has the upper hand. For further elaboration see Hsu-Schmitz (1994).

Remark 4. If we use property $(*)$ then all the results of this paper hold essentially without change.

The pair (p, q) is called a *two-sex mixing function* if and only if it satisfies axioms (i-iv). Further, a two-sex mixing function is *separable* if and only if

$$p(a, a', t) = p_1(a, t)\, p_2(a', t) \quad \textit{and} \quad q(a, a', t) = q_1(a, t)\, q_2(a', t).$$

If we let

$$h_p(a, t) = C(a, t)\, M(a, t) \tag{1}$$

and

$$h_q(a, t) = D(a, t)\, F(a, t) \tag{2}$$

then, omitting t to simplify the notation, one has the following results (see Castillo-Chavez and Busenberg, 1991):

Result 1. The only two-sex separable mixing function satisfying conditions (i-iv) is given by the Ross solution $(\hat{p}, \hat{q})$, where

$$\hat{p}(a') = \frac{h_q(a')}{\int_0^\infty h_q(u)du}, \tag{3}$$

$$\hat{q}(a) = \frac{h_p(a)}{\int_0^\infty h_p(u)du}. \tag{4}$$

We named this solution the *Ross solution* because Ross (1911) was aware of the importance and necessity of axiom (c) and used it in his model for malaria, although he only used one vector and one host type. This fact was clearly pointed out by Lotka (1923) in his review of Ross's work on malaria models (Ross also outlined the potential use of this work in models for STD's).

Result 2. Any solution of axioms (i)-(iv) can be written as a multiplicative perturbation of the Ross solution $(\hat{p}, \hat{q})$. These perturbations are a measure of the deviation from random or proportionate mixing among subpopulations (given by the Ross solutions) and can be parametrized by matrices that estimate the affinities/preferences of individuals.

In the next sections we discuss the applications of this framework in a variety of settings. We first outline its use in demographic and social contexts and then provide a new application of this approach in the context of frequency-dependent predation and food web dynamics. Finally, we discuss its use in epidemiology and its implementation in stochastic frameworks (Markov chain models).

3. DEMOGRAPHIC AND SOCIAL DYNAMIC MODELS

Classical demographic models that consider pairs and follow the dynamics of pairs have been studied by Kendall (1949), Keyfitz (1949), Parlett (1972), Fredrickson (1971), McFarland (1972), and Pollard (1973), and have been extended to epidemiology by Dietz and Hadeler (1988), Dietz (1988a), Hadeler (1989a,b), Hadeler and Nagoma (1990), and Waldstätter (1989). Their approach is based on the use of a nonlinear function to model the process (rate) of pair formation. This mixing/pair formation function is assumed to satisfy the Fredrickson/McFarland (1971, 1972) properties:

a) $\psi(0, F) = \psi(M, 0) = 0$. In the absence of either males or females there will be no heterosexual pair formation.
b) $\psi(\alpha M, \alpha F) = \psi(M, F)$ for all α, M, $F \geq 0$. If the sex ratio remains constant, then the increase in the rate of pair formation is assumed to be proportional to total population size.
c) $\psi(M+u, F+v) \geq \psi(M, F)$ for all u, v, F, $M \geq 0$. Increases in the number of males and/or females does not decrease the rate of pair formation.

Condition (b) implies that all mixing functions are of the form

$$\psi(M,F) = M\ g\left(\frac{F}{M}\right) = F\ h\left(\frac{M}{F}\right)$$

where h and g are functions of one variable.

Examples of mixing functions satisfying the above axioms include:

$$\begin{aligned}
&\psi(M,F) = k\min(M,F), \quad k \text{ a constant,}\\
&\psi(M,F) = k\sqrt{MF}, \quad \text{and}\\
&\psi(M,F) = 2k\frac{MF}{M+F}.
\end{aligned}$$

The simplest demographic model that takes into account pair formation is constructed by balacing the rates of flows between the different compartments/subpopulations; that is, by keeping track of the transition rates associated with the transfer of individuals and pairs of individuals (couples) as they form or dissolve pairings. To state the explicit equations: let μ denote the rate of pair dissolution, σ denote the natural mortality rate, Λ denote the 'recruitmen' rate, and W denote the number of (heterosexual) pairs. Then Kendall's demographic model is described by the following set of equations:

$$\begin{aligned}
\frac{dM}{dt} &= \Lambda - \mu M + (\sigma+\mu)W - \psi(M,F),\\
\frac{dF}{dt} &= \Lambda - \mu F + (\sigma+\mu)W - \psi(M,F),\\
\frac{dW}{dt} &= -(\sigma+2\mu)W + \psi(M,F).
\end{aligned} \tag{5}$$

If Λ, μ, and σ are constant, then there is always a globally stationary solution (M,F,W), where W is determined by non-trivial solutions to the equation

$$\psi\Big(\frac{\Lambda}{\mu} - W, \frac{\Lambda}{\mu} + W\Big) = (\sigma+2\mu)W$$

For references to this and related results see the work of Dietz and Hadeler (1988) and Waldstätter (1989). Extensions of this model that incorporate the age structure of a population have been carried out by Hadeler (1989a,b).

We now specify an age-structured demographic model equivalent to those studied by Hadeler (1989a,b) but using the framework of Section 2. Specifically, let $f(a',t)$ and $m(a,t)$ denote the age-specific densities for single males and single females respectively, and assume that C and D are defined in Section 2, and μ_m and μ_f are functions of age (the mortality rates for males and females). In addition, let $W(a,a',t)$ denote the age-specific density of heterosexual pairs (where a denotes the age of the male and a' the age of the

female). Then, using the two-sex mixing functions p and q of Section 2, we arrive at the following demographic model for heterosexual populations with pairing:

$$\begin{aligned}
\frac{\partial m}{\partial t}+\frac{\partial m}{\partial a} &= -C(a)\,m(a,t)-\mu_m(a)\,m(a,t)\\
&\quad+\int_0^\infty [\mu_f(a')+\sigma]\,W(a,a',t)\,da',\\
\frac{\partial f}{\partial t}+\frac{\partial f}{\partial a'} &= -D(a')\,f(a',t)-\mu_f(a')\,f(a',t)\\
&\quad+\int_0^\infty [\mu_m(a)+\sigma]\,W(a,a',t)\,da,\\
\frac{\partial W}{\partial t}+\frac{\partial W}{\partial a}+\frac{\partial W}{\partial a'} &= D(a')\,f(a',t)\,q(a,a',t)\\
&\quad-[\mu_f(a')+\mu_m(a)+\sigma]\,W(a,a',t).
\end{aligned} \tag{6}$$

To complete this model we must specify the initial and boundary conditions. To this effect we let λ_m and λ_f denote the female age-specific fertility rates, and let m_0, f_0, and W_0 denote the intial age densities. Hence, the initial and boundary conditions are given by

$$\begin{aligned}
m(0,t) &= \int_0^\infty \lambda_m(a')\,N_f(a',t)da',\\
f(0,t) &= \int_0^\infty \lambda_f(a')\,N_f(a',t)da',\\
W(0,0,t) &= 0,\\
f(a,0) &= f_0(a), \quad m(a,0)=m_0(a), \quad W(a,a',0)=W_0(a,a'),
\end{aligned} \tag{7}$$

where

$$N_f(a',t)=\int_0^\infty W(a,a',t)da.$$

We observe that N_f and $f+N_f$ satisfy the following set of equations:

$$\left(\frac{\partial}{\partial t}+\frac{\partial}{\partial a'}\right)(f+N_f)=-\mu_f(a')\,[f+N_f], \tag{8}$$

and

$$\begin{aligned}
\left(\frac{\partial}{\partial t}+\frac{\partial}{\partial a'}\right)N_f &= D(a')\,f(a',t)-[\mu_f(a')+\sigma]\,N_f\\
&\quad-\int \mu_m(a)W(a,a',t)\,da.
\end{aligned} \tag{9}$$

Remark 5. If we take into account *Remark* 4, then $C(a)m(a,t)$ in *Equation* (6.1) should be replaced by $\int_0^\infty D(a')f(a',t)q(a,a',t)da'$ (for more details, see Castillo-Chavez, 1993c, and Hsu-Schmitz, 1994).

If we let $\sigma \to \infty$ (while fixing a, t, and $\mu_m(a)$ constant) then $N_f(a,t) \to 0^+$ and formally equation (9) approaches the classical MacKendrick/Von Foerster model. However, since in the model given by equations (7)-(9) only pairs reproduce, we can not recover the classical boundary condition. This situation is easily corrected if one uses Dietz's (1988a) definition of a pair. For some preliminary analysis of this model see Castillo-Chavez *et al.* (1991).

Models like (7) are useful because of the importance of the mating system and the *average duration of partnerships* in the transmission dynamics of cultural traits. Superficially, the cultural transmission of traits appears similar to genetic transmission. However, there are further complications, as the inheritance of social traits such as language and religion is influenced by the level of heterogeneity of the population at large. Previous approaches to the modeling of cultural trait transmission have been either very specific, as in the bilingual competition model of Baggs and Freedman (1990), or quite general, albeit within very restricted or rigid frameworks. Cavalli-Sforza and Feldman (1981) and Boyd and Richerson (1985) assume that cultural transmission happens once per generation, *e.g.*, at birth. Hence they exclude many situations including religious conversion, otherwise their model has the usual inherent limitations of models with non-overlapping generations. In Lubkin *et al.* (1992), we have developed a flexible framework to study the transmission dynamics of cultural traits in age-structured populations with overlapping generations. The flexibility comes from the incorporation of pairings, partnership duration, and 'arbitrary' mating systems. Examples are provided that include the melting pot, biparental determination, and maternal determination models.

Finally, we note that the models of the *type* here constructed represent more than just an exercise in modeling, since their use has begun to have a serious impact in the area of sociology, epidemiology, immunology, and ecology. The current revisions of the theory have increased the number of interactions among a large number of scientists from biology, sociology, epidemiology, statistics, and mathematics. The large number of interdisciplinary conferences and workshops that have brought these groups together over the last five years has radically changed the fields of mathematical population dynamics and mathematical epidemiology.

4. FREQUENCY DEPENDENT PREDATION MODELS

Here we shift gears and discuss applications of the framework of Section 2 to situations in which frequency-dependent predation is important. Our emphasis is on the description of prey selection, competition for common resources (see Pimm, 1982, 1988), interaction strength (Pimm and Kitching, 1988), and their relation to food web dynamics. In this section, we define a food web as a network composed of *biological species* interacting through frequency-dependent predation and competition and illustrate our approach with

the use of a simple three-level food web (top predators, intermediate species, bottom species).

The mechanisms by which predators select prey for their diet has been the subject of intensive research (Akre *et al.*, 1979; Cock, 1978; Levin and Segel, 1982; Chesson, 1978, 1983; Gendron, 1987; Oaten and Murdoch, 1975; Teramoto *et al.*, 1979). A given predator's diet is, in principle, not necessarily related to the abundance of the different prey types available (Gendron, 1987). From a phenomenological point of view, this outcome can be seen as the product of a density-dependent risk of being captured and the density of other alternative prey (Gendron, 1987). To model these interactions we let c_{ij} denote the average *per capita* number of effective contacts (leading to a successful meal) between predators of type i and prey of type j per unit time; while r_{ji} denotes the average *per capita* number of prey of type j captured by predators of type i per unit time. If $T_i(t)$ denotes the number or density of predators of type i at time t and $N(t)$ denotes the number or density of prey of type j at time t, we must then have that

$$T_i c_{ij} = N_j r_{ji}.$$

By setting

$$c_{ij} = \sum_j^m c_{ij}, \quad r_j = \sum_i^n r_{ji},$$

one has

$$c_i T_i c_{ij} c_i^{-1} = r_j N_j r_{ji} r_j^{-1}.$$

Defining

$$p_{ij} = c_{ij} c_i^{-1} \quad and \quad q_{ji} = r_{ji} r_j^{-1},$$

permits the interpretation of p_{ij} as the proportion of prey of type j on the diet of the ith predator, and q_{ji} as the proportion of the jth prey type consumed by the ith predator, given thant it had a meal at time t. Hence the matrix (p_{ik}, q_{ki}) satisfies the discrete analog of properties (i)-(iv). Using these definitions we introduce the concept of a mixing matrix in the context of a finite number of interacting subpopulations:

Definition 2. The matrix (p_{ik}, q_{ki}) is called a mixing/contact matrix if and only if it satisfies the following properties:

di) $0 \leq p_{ik} \leq 1$, and $0 \leq q_{ki} \leq 1$;

dii) $\sum_{k=1}^m p_{ik} = 1 = \sum_{i=1}^n q_{ki}$, for $i = 1, \cdots, n$ and $k = 1, \cdots, m$;

diii) $c_i T_i p_{ik} = r_k N_k q_{ki}$,for $i = 1, \cdots, n$ and $k = 1, \cdots, m$; [1]

div) if for some i, $1 \leq i \leq n$ and/or some j, $1 \leq j \leq m$ we have that $c_i r_k T_i N_k = 0$, then we define $p_{ik} \equiv q_{ki} \equiv 0$.

[1] The assumption of constant contact rates is limiting, but facilitates the discussion. Modifications similar to those discussed in Sections 2 and 3 are easily made.

Condition (diii) is interpreted as a conservation of contacts law or group reversibility property. The total number of contacts per unit time of predators of type i and prey of type j has to be equal to the number of contacts between prey of type j with predators of type i, given that they had a contact at time t. The condition relates the rates at which k-prey are captured by i-predators, that is $c_i p_{ik}$ and $r_k q_{ki}$. The use of N_k and T_i as state variables introduces the abundance of prey and predators in a contact structure based on biological species interactions. Condition (div) asserts that the mixing of nonexistent subpopulations, either of prey or predators, cannot be arbitrarily defined. The symmetry involved in the total number of contacts per unit time required for predator-prey, consumer-resource or host-parasite interactions is an obvious fact that has not been fully explored until very recently (see Castillo-Chavez and Busenberg, 1991).

We re-derive expressions for the mixing probabilities that allow for the incorporation of handling times. Let τ_i be the fraction of the total time available to an average predator of type i spent foraging, and let σ_{ij} denote average fraction of time spent by a predator of species i handling prey in group j, $i = 1, \cdots, n$, $j = 1, \cdots, m$. Then

$$\tilde{\sigma} = \sum_{k=1}^{m} \sigma_{ik} c_{ik} \tag{10}$$

denotes the mean *per capita* handling time of predators of group i. The searching time (the available time that predators of species i have to find prey suitable for consumption) of predators of type i is $\tau_i - \tilde{\sigma}_i$. Hence, the number of contacts that an average predator of type i has with prey in group j during the searching time is

$$c_{ij} = (\tau_i - \tilde{\sigma}_i) b_{ij} N_j, \tag{11}$$

where b_{ij} denotes the proportion of contacts that result in a capture of prey from group j by predator species i. Substituting (10) in (11) gives

$$\tilde{\sigma} = (\tau_i - \tilde{\sigma}_i) \sum_{k=1}^{m} \sigma_{ik} b_{ik} N_k.$$

Solving for $\tilde{\sigma}$ leads to

$$\tilde{\sigma}_i = \frac{\tau_i \sum_{k=1}^{m} \sigma_{ik} b_{ik} N_k}{1 + \sum_{k=1}^{m} \sigma_{ik} b_{ik} N_k}, \tag{12}$$

while substitution of (12) into (11) leads, after some algebra, to

$$c_{ij} = \frac{\tau_i b_{ij} N_j}{1 + \sum_{k=1}^{m} \sigma_{ik} b_{ik} N_k}.$$

From the ratio c_{ij}/c_i, we conclude that the probability of an effective contact between a predator of species i and prey of group j is

$$p_{ij} = \frac{b_{ij} N_j}{\sum_{k=1}^{m} b_{ik} N_k}. \tag{13}$$

To derive the probability q_{ji} of an effective contact of predators of group i with prey of species j we solve the relation $T_i c_{ij} = N_j r_{ji}$ for r_{ji}. The formula $q_{ji} = r_{ji}/r_j$ leads to

$$q_{ji} = \frac{\tau_i b_{ij} T_j}{1 + \sum_{k=1}^{m} \sigma_{ik} b_{ik} N_k} \Bigg/ \sum_{u=1}^{n} \frac{\tau_u b_{uj} T_j}{1 + \sum_{k=1}^{m} \sigma_{uk} b_{uk} N_k}. \tag{14}$$

Equations (13) and (14) satisfy the mixing axioms (di)-(diii). Formula (14) may be interpreted in the following way: b_{ij} is the maximum capture proportion in the absence of frequency-dependent effects; $\tau_i b_{ij} T_i$ is the number of captures of prey of type j by predators of type i during the total foraging time characteristic of the predator species. The numerator of (14) gives the proportion of captures of all potential prey of predators of type i, while its denominator represents the total number of captures made by all types of predators per unit time; p_{ij} depends only on the weighted relative proportion of prey types, while q_{ji} depends also on the handling times of each predator species involved.

Definition 3. A predator-prey mixing probability is *separable* if and only if

$$p_{ij} = p_i \tilde{p}_j \quad \textit{and} \quad q_{ji} = q_j \tilde{q}_i.$$

To obtain separable solutions from formulae (12) and (13), one requires $b_{ij} = b$ for all indices (*i.e.*, the maximum capture proportion is the same for all predators regardless of the prey type they capture). This assumption leads to the following set of contact probabilities (Ross solutions):

$$\tilde{p}_j = \frac{N_j}{\sum_{u=1}^{m} N_u}, \tag{15a}$$

and

$$\tilde{q}_i = \frac{\tau_i T_i}{1 + b \sum_{k=1}^{m} \sigma_{ik} N_k} \Bigg/ \sum_{k=1}^{n} \frac{\tau_k T_k}{1 + b \sum_{u=1}^{m} \sigma_{ku} N_u}. \tag{15b}$$

Thus, the frequency of a prey type in the diet of a predator depends on the proportion of prey types available, while the presence of a given prey type in the diet of a predator depends on the relative foraging time invested in capturing it. This last factor is commonly associated with the functional response of the predator (see, *e.g.*, Price, 1990).

4.a The Components of Predation Risk

Gendron (1987) has shown that the components of risk (how likely it is for a prey of any given type to be captured by a predator) can be understood in terms of the following factors:

a) The efficiency of the search path.
b) The area searched by the predator per unit time.
c) The conditional probability of detecting prey.

d) The conditional probability of attacking and then capturing detected prey.

Models for predator switching behavior are defined in terms of the frequency of each prey type in the diet of the predator. Specifically, F_i, the frequency of prey type i is defined as

$$F_i = \frac{\beta_i N_i}{\sum_{j=1}^{m} \beta_j N_j}, \tag{16}$$

where β_i is a measure of the relative risk of prey i. Usually β_i is computed by the formula

$$\beta_i = \frac{r_i}{\sum_{j=1}^{m} r_j},$$

where r_i denotes the risk index of species i. Frequency-dependent predation requires risk indices which are functions of the relative density of the prey species and give rise to the switching behavior of predators. Generalizations of this switching behavior model useful for statistical analyses are of the form

$$F_i = \frac{f(X_i)}{\sum_j f(X_j)}, \tag{17}$$

where f is a nonlinear (usually a polynomial) function of X, the density of prey species j (*e.g.*, Gendron, 1987).

To account for several predator species competing for a collection of prey species, we reformulate equation (17) in the following way

$$F_{ij} = \frac{\beta_{ij} N_i}{\sum_{k=1}^{m} \beta_{ik} N_k}. \tag{18}$$

The model is complete after the postulation of appropriate functional forms for the relative risks of predation β_{ij}. These functional forms usually weigh each prey type according to the risk of being captured (see Gendron, 1987). The connection with the mixing theory described before is made by reinterpreting the matrix (p_{ij}, q_{ji}) in (13)-(14) to model F_{ij} above. It has the added advantage that it can be incorporated into dynamic models (see Sections 4b and 4c).

Predation in nature is a selective process and has also been explored in the context of food webs (Pimm, 1982, 1988; Fretwell, 1987). This frequency-dependent process may be due exclusively to frequency-dependent effects—the most numerous prey provides a greater share of the diet of any predator—or by an active process of preferential prey selection which may be more suitable for the survival of a predator. The formalism introduced by the mixing probabilities describes both processes. Pimm (1982, 1988) observes rare as well as common species of prey in predators' diets, thus imposing a ranking in prey species selectivity. This ranking depends on each predator species but it is not transposable to communities. The modeling approach introduced here allows for the incorporation of these

effects. In the next sub-section we incorporate the mixing formalism in dynamic models of predator-prey interactions. These models form the basis of our approach to modeling food web dynamics.

4.b Predator Prey Interactions and Food Webs

The general model of predator (P)-prey (N) interaction is given by the system:

$$N'(t) = g[N(t)]N(t) - R(N,P); \quad P'(t) = P(t)G(N,P) - dP(t), \tag{19}$$

where $G(N,P)$ is the numerical response of the predator, $R(N,P)/P$ is the number of prey consumed relative to prey density per unit time or functional response of the predator, and the symbol '$\prime$' denotes derivative with respect to time. The term $g(N)$ models the *per capita* growth rate of a prey population when predators are absent and d is the density-independent mortality rate of the predator.

A generalized form of the predator-prey model (19) that allows for heterogeneity in prey and predator interactions is given by the following set of equations:

$$\begin{aligned} N_i' &= g\big(\{N_k\}_{k=1}^{l}\big)N_i - \sum_{k=1}^{n} p_{ki}c_k P_k, \\ P_k' &= e_k \sum_{j=1}^{m} r_j N_j q_{jk} - \delta_k P_k, \end{aligned} \tag{20}$$

where e_k represents the efficiency of the kth predators in transforming prey captured into predator biomass, and $i = 1\cdots, m$, $k = 1, \cdots, n$. In (20) the per-capita growth rate g may be a function of each of the basal prey types (thus assuming, in the absence of predation, interspecific competition between basal prey types). (p_{ki}, q_{jk}) is the matrix of mixing/contact probabilities whose eelements satisfy Definition 2.

Model (20) may also be used to describe the competitive interaction between species that share a spectrum of biotic resources distributed among themselves according to the mixing matrix (p_{ik}, q_{ki}). The first equation in (20) describes the ith prey population growing according to $g(\{N_k\}_{k=1}^{l})$ in the absence of predators. The term

$$\sum_{k=1}^{n} p_{ki}^{(0)} c_k P_k$$

represents the total consumption rate of N_j by all predators in the community. Analogously, the term

$$\sum_{j=1}^{m} r_j N_j q_{jk}^{(0)}$$

represents the total consumption rate of prey by predators of type i since these predators eat not only prey of type j but of all types. By virtue of the relations

$$c_k P_k p_{kj}^{(0)} = r_j N_j q_{jk}^{(0)},$$
$$\sum_{k=1}^{n} c_k P_k = \sum_{j=1}^{m} r_j N_j,$$

we have that the total rate of prey captured equals the total rate of prey consumed.

One of the simplest food web models considers only three trophic levels (basal, intermediate and top) each with m, n, and $\tilde{n}$ species, respectively. The dynamics are specified by the following food web transfer diagram:

$$N \to P \to T,$$

where $T = \{T_i\}_1^{\tilde{n}}$ denotes the *set* of top species, $P = \{P_k\}_1^n$ the *set* of intermediate species and $N = \{N_j\}_1^m$ the *set* of basal species. The model equations are

$$\begin{aligned} N_i' &= g(\{N_k\}_{k=1}^{l})N_i - \sum_{k=1}^{n} p_{ki}^{(0)} c_k P_k, \\ P_k' &= e_k^{(0)} \sum_{j=1}^{m} r_j N_j q_{jk}^{(0)} - \sum_{l=1}^{\tilde{n}} p_{lk}^{(1)} b_l T_l, \\ T_l' &= e_l^{(1)} \sum_{k=1}^{n} a_k P_k q_{kl}^{(1)} - \delta_l T_l, \end{aligned} \tag{21}$$

where $e_j^{(*)}$ are coefficients that measure the efficiency of conversion of captured prey into predator biomass, for the $(*)$ predator level. The term $g(\{N_k\}_{k=1}^{l})$ denotes the *per capita* growth rate of the kth basal species in the absence of predators. There are $(\tilde{n}+m)n$ links in this completely connected food web. The contact probabilities $(p_{ij}^{(1)}, q_{ji}^{(1)})$ (for encounters between T and P populations) and $(p_{ij}^{(0)}, q_{ji}^{(0)})$ (for encounters between P and N populations) satisfy the axioms in Definition 2 as well as the conditions

$$\begin{aligned} b_i T_i p_{ij}^{(1)} &= a_k P_j q_{ji}^{(1)}, \\ c_k P_k p_{kj}^{(0)} &= r_j N_j q_{jk}^{(0)}, \end{aligned} \tag{22a}$$

for $i = 1, \cdots, \tilde{n}$, $k = 1, \cdots, n$ and $j = 1, \cdots, m$. The rates a_k and b_k are defined analogously to r_j and c_k (see Definition 2 and the discusion that follows it on p. 8).

This model implicitly assumes that all species in level T are linked to all species in level P, and that all species in level P are linked to all basal species in N.

The following example assumes that, in the absence of predation basal species compete according to a Lotka-Volterra model (*e.g.*, Brauer, 1976) and that the mixing probabilities are separable. Thus,

$$\begin{aligned} N_i' &= s_i N_i\Big(1 - \frac{N_i}{K_i} - \frac{1}{K_i}\sum_{j=1}^{m}\alpha_{ji}N_j\Big) - \hat{c}N_i\frac{P_\bullet}{N_\bullet}, \\ P_k' &= q_k^{(0)}\hat{r}N_\bullet - \hat{b}P_k\frac{T_\bullet}{P_\bullet}, \\ T_j' &= q_j^{(1)}\hat{a}P_\bullet - \delta_j T_j, \end{aligned} \tag{22b}$$

where s_k represent the intrinsic growth rate of the kth basal species, α_{ji} represent the competition coefficients of the community matrix, and the subscript $\bullet$ represents the sum over all types in each of the trophic levels, *i.e.*, the total population of the corresponding trophic level. Also, in this case, the explicit forms of $q_k^{(0)}$ and $q_j^{(1)}$ are given by:

$$q_k^{(0)} = \frac{\tau_k^{(0)}P_k}{[1 + b(\sum \sigma_{kj}^{(0)}N_j)]\hat{r}}, \quad q_j^{(1)} = \frac{\tau_j^{(1)}T_j}{[1 + b(\sum \sigma_{ji}^{(1)}P_i)]\hat{a}},$$

where the superindex in the parameters relate to the position of the trophic level. The assumption of separable contact probabilities forces the contact rates in (22a) to be *independent of the type although not necessarily constant* within each trophic level, thus we write this as $c_i = \hat{c}$, $a_k = \hat{a}$, $\hat{b} = b_l$ $r_j = \hat{r}$ for all i, k, l and j respectively. However, separable contact probabilities allow dependence on total population density in the contact rates. Condition (22a) must be also satisfied for all time.

The mixing probabilities in (22) are assumed to have the simplest possible form, *i.e.*, they are assumed to be Ross solutions (separable solutions) describing proportionate mixing of captures. Thus, we have $p_{ij}^{(*)} = \bar{p}_j^{(*)}$ and $q_{ji}^{(*)} = \bar{q}_i^{(*)}$, where the superscript * denotes the trophic level as described above.

In model (22) we consider the case where the capture of prey by corresponding predators is given by Ross solutions (15) in both the top and the intermediate levels of the food chain. The capture of prey is essentially a random process where predators do not show preference for prey of any type. Furthermore, we have that the total population in each trophic level follows the dynamics specified by the system

$$\begin{aligned} N_\bullet' &= \sum_{i}^{m} g_i N_i - \hat{c}P_\bullet, \\ P_\bullet' &= \hat{r}N_\bullet - \hat{b}T_\bullet, \\ T_\bullet' &= \hat{a}P_\bullet - \sum_{k}^{\bar{n}}\delta_k T_k. \end{aligned}$$

This system is a representation of the dynamic behavior of the trophic species N, P, and T or, in other words, it represents a model for the dynamics of the trophic web as such. Only in very special cases, can the RHS of these equations be written in terms of N, P, and T, and hence be solved independently of the species that constitute each trophic level. Statistical models of food webs deal at this level of organization, therefore incorporating a particular aggregation structure.

If we assume that $g_k = s$ a constant for all basal prey types (*i.e.*, for all species belonging to the N level), and furthermore, if we choose $\hat{c} = cN_\bullet$, $\hat{r} = rP_\bullet$, $\hat{b} = bP_\bullet$, and $\hat{a} = aT_\bullet$ with a, b, c and r constants, we get

$$\begin{aligned} N'_\bullet &= sN_\bullet - cN_\bullet P_\bullet, \\ P'_\bullet &= rP_\bullet N_\bullet - bP_\bullet T_\bullet, \\ T'_\bullet &= aT_\bullet P_\bullet - \delta T_\bullet, \end{aligned}$$

which is a Lotka-Volterra predator-prey system.

Another important model that can be derived from our mixing framework under the assumption of separable mixing probabilities is the published by Hastings and Powell (1991). Here we need to assume the growth rate of each type to be a function of the total population, that is $g_k = s(1 - N_\bullet/K)$ where K stands for the carrying capacity of $N_\bullet$. Further we assume

$$\hat{c} = \frac{cN_\bullet}{D + N_\bullet}, \quad \hat{r} = \frac{rP_\bullet}{D + N_\bullet},$$

$$\hat{a} = \frac{aP_\bullet}{E + P_\bullet}, \quad \hat{b} = \frac{bT_\bullet}{E + P_\bullet},$$

where D and E are the prey population levels where the predation rate per unit prey is half its maximum value (Hastings and Powell, 1991). Substituting these definitions into (21), summing over i, k and l and using separable mixing probabilities as we did when the Lotka-Volterra predator prey system was derived we obtain

$$\begin{aligned} N'_\bullet &= s(1 - N_\bullet/K)N_\bullet - \frac{cN_\bullet}{D + N_\bullet}P_\bullet, \\ P'_\bullet &= \frac{rP_\bullet}{D + N_\bullet}N_\bullet - \frac{aP_\bullet}{E + P_\bullet}T_\bullet - \mu P_\bullet, \\ T'_\bullet &= \frac{bT_\bullet}{E + P_\bullet}P_\bullet - \delta T_\bullet, \end{aligned}$$

where μ is the mortality rate of the intermediate predator population. This food chain is known to present chaotic dynamics.

4.c Relation to Other Models

From model (22) we can obtain some interesting results that relate to ratio-dependent models (*e.g.*, Matson and Berryman, 1992). We do not want to expand in this aspect

whose discussion in the context of our approach will be published elsewhere. Here we only outline some preliminary ideas. Setting the RHS of (22b) to zero we find the equilibrium relations (* denotes values at equilibrium):

$$\begin{aligned}
\frac{P_\bullet^*}{N_\bullet^*} &= \frac{g_i^*}{\hat{c}^*}, \\
\frac{T_\bullet^*}{P_\bullet^*} &= \frac{q_k^{*(0)} \hat{r}^* N_\bullet^*}{\hat{b}^* P_k^*}, \\
\frac{T_j^*}{P_\bullet^*} &= \frac{q_j^{*(1)} \hat{a}^*}{\delta_j}.
\end{aligned} \tag{23}$$

The first equation predicts, at equilibrium, a proportional change in the ratio $P_\bullet^*/N_\bullet^*$ whenever a change in the growth rate of any basal type takes place. In the last level of the trophic chain, again at equilibrium, our model predicts a change in the density of the jth top predator species proportional to the total population size of its prey population, which is equivalent to

$$\frac{T_\bullet^*}{P_\bullet^*} = \hat{a}^* \sum_{j=1}^{n} \frac{q_j^{*(1)}}{\delta_j}, \tag{24}$$

by summing over all j in the last equation in (23). Thus, the ratio of predators to prey is simply proportional to the average probability of capture of the whole top predator population. These conclusions hold for specially aggregated *chains* of arbitrary length. Thus, this very simple model constructed under the assumption of separable mixing (contact) probabilities may serve as a framework for the theory of ratio-dependent predator-prey interactions. This possibility and the empirical verification of the predictions will be explored elsewhere.

4.d Remarks

Models of food webs can be divided into two categories: classical, or static, and dynamic. Dynamic models include those of Hastings and Powell (1991) and Tilman (1991), and they are the main topic of this section. Classical models include those that attempt to describe, from a statistical point of view, characteristic patterns common to sets of food webs. The cascade model of Cohen *et al.* (1990) provides a successful example of a classical or static model. It describes the plausible behavior of the population densities of trophically related species.

The concept of trophic species on which 'static' food web models are based is not a natural biological class but rather a theoretical classification. It is an equivalence class made of those organisms, regardless of the species to which they belong, that share the same predators and the same prey. Thus, when one tries to define a trophic species from data, sources of error, associated with the identification of common predators and common prey, arise.

The applicability of the concept of trophic species to the field of applied ecology depends heavily on the ability of experimentalists to eliminate errors in the classification process. This problem is not unique to the study of food web dynamics; it is quite common in the biological and social sciences. Once the researcher establishes what is a weak (negligible) interaction and what is a strong one, the trophic level of each organism is determined. However, as the study of Polis (1991) shows, it is difficult to decide what is a weak and what is a strong interaction in a trophic web.

Moreover, the existence of omnivory makes it difficult to justify the classification of organisms into trophic species. This is particularly important if one is interested in the population dynamics of the web. Omnivory implies a very diverse diet with some items being more frequent than others (a matter of taste, opportunity, or chance), and if a large share of a predator's diet is made up of rare organisms, their neglect on the basis of weak or strong interactions may be misleading. Consequently, the trophic structure dynamics of the food web cannot be defined exclusively in terms of strong interactions, but rather through a 'distribution' of interactions that reflects the composition of an organism's diet. In a recent study Paine (1992) concludes that in an species-rich herbivore guild there are mainly weakly negative or positive interactions with only a few strong negative ones.

A trophic web may be better described from a dynamical point of view if the description centers on what resources are used and the way these resources are used, that is, if the description is centered on guilds of species. A trophic web is a model of the interrelations between species that share common resources which have been shaped by natural selection through various mechanisms such as diffuse co-evolution (Maddox and Root, 1990). In fact, the mechanism of switching or apostatic selection has been recognized as one of the factors that may promote diversity in prey populations (Greenwood, 1984; Levin and Segel, 1982).

For the simple three-level food web discussed here, we have shown that the conservation principle of the mixing probabilities (in Definition 2) allows us to structure predator-prey interaction in terms of two main factors: the probability that a predator encounters its prey, and the conditional probability of capturing and eating the prey once it has been found. Moreover, the use of the mixing framework described in this work constitutes an approach to the modeling of switching predator behavior, of frequency-dependent mechanisms in prey selection, and interaction strength in food webs.

The models of food web dynamics based on our framework can be used, for example, to study the time evolution of any given initial food web configuration. It is possible to assign different 'strengths' to the interactions in the web through the initial contact rates satisfying conservation relations as the one shown in (22a), and then follow through time the fate of the individual species.

5. MODELS FOR VECTOR TRANSMITTED DISEASES

As Ross (1911) had noted, there is a close relationship between the mathematical formalism used to study sexually-transmitted diseases and the mathematical formalism used to study

vector-transmitted diseases. In this section, we outline the use of our framework in the context of vector-transmitted diseases.

5.a Host-Vector Mixing Frameworks

Mathematical models have played a very important role in the history and development of vector-parasite epidemiology. Sir Ronald Ross in 1911 developed a theory for parasite transmission mechanism while engaged in the study of malaria. He developed a simple mathematical model that provided him with the concept of transmission threshold. Later on, Macdonald (1957), based on the work of Ross, developed a new model from which he extracted the concept of vectorial capacity. The involvement of mathematical models in epidemiological theory has persisted along the years. In particular, we mention the mathematical model developed for the evaluation of control measures for malaria in the Garki Project (Molineaux and Gramiccia, 1980). This model was developed using the extensive research on the transmission of malaria in Northern Nigeria. It is based on the theory and results developed by Ross and Macdonald. Despite the construction of new models (*e.g.*, Aron and May, 1982; Rogers, 1988; Pacala and Dobson, 1990; Dietz, 1988b), all estimates and assumed mechanisms for parasite transmission from vector to host still rely on the assumptions implicit in the definition of vectorial capacity as presented by Macdonald (1957). The goal is to estimate it in order to understand and evaluate the strength and effectiveness of vectors for the transmission of parasites.

Transmission in vector-transmitted diseases depends on the life history of the vector species. malaria and dengue, for example, are transmitted by mosquitoes of several species including *Aedes spp.* for dengue and *Anopheles spp.* for malaria. Chagas' disease is transmitted by triatominae bugs of various genuses, *e.g.*, *Triatoma spp.*, *Rhodnius spp.*, and *Pastrongylus spp.* In malaria and dengue, transmisson of the parasite to human hosts involves only adult individuals since the larval stages are aquatic and have a completely different ecological niche. In Chagas' disease, however, the vector is a triatomine bug with 5 nymphal stages preceding reproductive maturity (Velasco-Hernández, 1991, 1993). All of them are hematophagous and all of them may be involved in the transmission process (Zeledon and Rabinovich, 1981). Thus when speaking about general models for vector-parasite-host interactions we must consider the age and stage structure of the vector population.

5.b Vectorial Capacity

The factors affecting transmission by the appropriate vector stages according to Molineaux (1988) are: (1) density of the vectors in relation to human hosts; (2) the effectivennes of the vector in acquiring and maturing the infection after feeding on an infective subject; (3) the frequency with which the vector takes a blood meal and the fraction of these blood meals taken on human hosts; (4) the duration of the parasite incubation period in the vector; (5) the longevity of the vector. Most of the above factors are included in the formula for the vectorial capacity defined as the capacity of the vector population to transmit the disease in terms of the potential number of secondary inoculations originating per unit time from

an infective person (Molineaux, 1988; Molineaux *et al.*, 1988). A formula for the vectorial capacity is derived form the concept of basic reproductive rate (not a rate) proposed by Macdonald (1957):

$$C = \frac{ma^2p^n}{-\ln p}, \tag{25}$$

where C denotes the vectorial capacity, m the number of vectors per human host, a the number of blood meals taken on a human host per vector per day (biting rate), p the proportion of vectors surviving per day, and n the length in days of the parasite incubation period in the vectors. Formula (25) is species specific since, for example, n varies from species to species.

If we denote by R_0 the basic reproductive number or ratio, then we have that

$$R_0 = \frac{C}{r},$$

where r^{-1} denotes the expected duration of infectivity (Molineaux, 1988). If we define μ as the death rate then $p = e^{-\mu}$ and

$$C = \frac{ma^2e^{-\mu n}}{\mu}, \tag{26}$$

then $R_0 = C/\mu r$, the form used by Aron and May (1982) in their models of Malaria transmission.

The assumptions on which (25) is based are, according to Molineaux (1988): (a) the vector is fully effective in acquiring and maturing the infection. This amounts to ignoring variability in susceptibility by the vector species; (b) vectors die at constant rate, independent of age, and senescence is ignored; (c) longevity is unaffected by the infection; (d) the probability of feeding on hosts is unaffected by the number of previous meals or by differences in host type, and (e) parasite presence does not affect preference by vectors (Dye, 1990).

Some of the important factors neglected in Formula (25) were described by Ribeiro *et al.* (1985) in their study on the blood finding strategy of *Aedes aegipty* and its interaction with the parasite *Plasmodium gallinaceum*. The probing behaviour by *Aedes aegipty* is complex and involves periodically repeated probing while searching for a blood meal (Ribeiro *et al.*, 1985): new attempts to feed depend on the success of the previous search. During each search the probability of feeding success may be interpreted as a function of the blood vessels on the skin. There is some evidence that the probability of desisting from feeding increases linearly with time while decreasing as a function of the previous number of attempts. Ribeiro *et al.* (1985) conclude that the dependence of the probability of feeding success on the density of blood vessels implies a preference for infected hosts since parasites induce an increase in the availability of blood vessels (see also Molyneaux and Jeffreries, 1986).

Feeding and probing behaviour of hematophagous arthropods may change during feeding depending on the infected status of the host, and this fact, according to Dye (1990), makes direct estimation of the vectorial capacity impossible. He argues that it is better to estimate the relative vectorial capacity before and after a control measure is applied.

In Chagas' disease, the feeding and developmental cycle can be broken into clear stages (see the study on the population of *Rhodnius prolixus*, Friend *et al.*, 1965). *Trypanosoma cruzi* is transmitted to susceptible hosts by contamination rather than by injection during biting. Triatomines urinate and defecate during or after a blood meal (Zeledon and Rabinovich, 1981), and this excretory behavior impacts the transmission dynamics of the parasite. Infective forms of *T. cruzi* go in the feces and are able to penetrate wounds or soft tissue—around the eyes and the mouth—infecting the host. The probability of infection increases with the duration of a blood meal (Zeledon and Rabinovich, 1981). Hess and Hayes (1970) have explored the potential of domestic animals to attract zoophilic species of mosquito (*Culex tarsalis* and other species of the same genus). It has been established that preferences do exist among vector populations in the selection of hosts; however, host preference in the field depends not only on the vector preferences but on the density and relative abundance of host types.

These results indicate that a careful modeling of the process of acquisition of blood meals by vectors is necessary in order to obtain a better estimate of the transmission probability. Searching and handling times must be explicitly considered as well as the functional form of the dependence of the transmission probability on these parameters. We cannot proceed to use formula (25) when the vector does not transmit the disease by biting, as in the case of Chagas disease, when there is more than one stage involved in the transmission process (Schofield, 1982; Zeledon and Rabinovich, 1981), or when handling, search times, host-preference, and frequency and density dependent effects are important.

5.c Contact Structure

The need for further theoretical work is therefore quite evident. We reinterpret the contact structure for frequency-dependent predation, completed in Section 4, in the context of host-vector interactions. We hope that our approach will provide a useful framework on which the questions raised may be systematically addressed.

Assume that vectors as well as hosts are subdivided into groups according to some variable of interest (geographical location, susceptibility to infection, species, etc.), and denote by C_{ij} the average number of contacts per day that vectors of group i have with hosts in group j. Consequently (for $i = 1, \cdots, M$ and $j = 1, \cdots, N$) we must have

$$V_i(t)\, C_{ij}(t) = H_j(t)\, C_{ji}(t)\,, \tag{27}$$

where $V_i(t)$ denotes the number of vectors of type i at time t and $H_j(t)$ denotes the number of hosts of type j at time t.

Let τ_i be the fraction of the total time available to an average vector of type i spent foraging, and let σ_{ij} denote average fraction of time spent by a vector of species i handling and probing hosts in group j, $i = 1, \cdots, n$, $j = 1, \cdots, m$. Then, as before,

$$\tilde{\sigma}_i = \sum_{k=1}^{N} \sigma_{ik} C_{ik} \tag{28}$$

denotes the mean *per capita* handling time of vectors of group i, and the proportion of time available for searching of vectors of type i to make contacts, that is, to find suitable hosts, is $\tau_i - \tilde{\sigma}_i$. Therefore, the average number of contacts that a typical vector of type i makes with hosts of type j per unit time is

$$C_{ij} = (\tau_i - \tilde{\sigma}_i)\, \beta_{ij} H_j, \tag{29}$$

where β_{ij} denotes the rate of successful contacts, *i.e.*, the actual biting rate of vectors of type i on hosts of type j. In many situations it is reasonable to assume that β_{ij} is a function of the total number of vectors of any type feeding at time t on hosts of type j. If Π_j denotes the average number of vectors of any type feeding on a host in group j, and if we assume that vectors are less efficient at biting very popular hosts (that is, hosts with many vectors feeding on them), then as a first approximation we have that

$$\beta_{ij} = \frac{r_{ij}}{\Pi_j}, \tag{30}$$

where the matrix (r_{ij}) can be interpreted as the matrix of maximum biting rates at low vector densities. We impose the condition $r_{ij} = r_{ji}$ for all $1 \le i \le M$, $1 \le j \le N$, and set $r_{ji} = 0$ otherwise. Substituting (29-30) into (28) one obtains as before

$$\tilde{\sigma}_i = (\tau_i - \tilde{\sigma}_i) \sum_{k=1}^{N} \sigma_{ik} r_{ik} m_k,$$

where $m_k = \frac{H_k}{\Pi_k}$ is the ratio of hosts of kth type to vectors of any type feeding on a host of type k. If p_{ij} denotes the proportion of effective contacts between vectors of type i and hosts of type j, then one easily sees (as in Section 2) that

$$p_{ij} = \frac{r_{ij} m_j}{\sum_{j=1}^{N} r_{ij} m_j}. \tag{31}$$

Similarly, if q_{ji} denotes the proportion of effective contacts between hosts of type j with vectors of type i then

$$q_{ji} = \frac{\tau_i r_{ij} V_i}{1 + \sum_{k=1}^{N} \sigma_{ik} r_{ik} m_k} \Big/ \sum_{i=1}^{M} \frac{\tau_i r_{ij} V_i}{1 + \sum_{k=1}^{N} \sigma_{ik} r_{ik} m_k}. \tag{32}$$

Therefore the matrix (p_{ij}, q_{ji}) is a mixing contact matrix; that is, it satisfies properties (di)-(div). Solutions (31-32) are not Ross-solutions, however, they become Ross solutions if we require that $r_{ij} = r$ for all indices (see Section 4, Equations 15a and 15b).

Remark. From equations (31) and (32) we see that the mixing of susceptible hosts depends on the ratio m_i—measuring how the host population is allocated to each vector type—while the mixing of susceptible vectors depends on the foraging time invested in 'capturing' a host. In this sense, solutions (31) and (32), allow for the possibility of modeling asymetric forms of transmission as those discussed in relation to Chagas' disease (see Velasco-Hernández and Castillo-Chavez, 1993).

In a classical contact epidemic model, which is quite appropriate for host-vector interactions, the incidence rate for infective hosts, or the number of newly infected hosts per unit time, has the general form

$$S_{h_j} \sum_{i=1}^{M} p_{ij} g_i \frac{I_{v_i}}{S_{v_i} + I_{v_i}}, \tag{33}$$

where g_i is a parameter that measures the infectivity of vectors of type i, S_{vi} and I_{vi} are the susceptible and infective subpopulations respectively of vectors of type i, and S_{hi} is the susceptible host population of type j. Similarly, the incidence rate of infected vectors has the general form

$$S_{v_i} \sum_{i=1}^{M} q_{ji} f_j \frac{I_{h_j}}{S_{h_j} + I_{h_j}}, \tag{34}$$

where f_j is a parameter that measures the infectivity of hosts of type j, and S_{hj} and I_{hi} are the susceptible and infective host populations respectively. The full equations are then readily written. Some preliminary work using these equations has begun to be carried out by Velasco-Hernández and Castillo-Chavez (1993).

6. THE DETERMINISTIC-STOCHASTIC CONNECTION

Classical deterministic models for the sexual spread of STD's such as gonorrhea among heterosexual populations can be found in Hethcote and Yorke (1984) while classical and pair formation models under a unified mixing framework for the spread of STD's can be found in Blythe *et al.* (1991), and references therein. A stochastic version of one of the deterministic models found in Blythe *et al.* (1991) is provided below. This formulation uses the modeling approach common to interacting particle systems (for details see Luo and Castillo-Chavez, 1991, 1992). Hence, it has great generality and flexibility.

6.a General Notation

Let $X = \{0, 1, \cdots, L\} \times \{0, 1\} \times \{0, 1, \cdots, N\} \times \{0, 1\} - \{0\} \times \{0, 1\} \times \{0\} \times \{0, 1\}$ and consider a stochastic process $\xi_t : X \to \{0, 1, 2, \cdots\}$, $t \geq 0$. For $x = (i, \mu; j, \nu) \in X$, our interpretation of this process is as follows:

1) The labels μ and ν represent the epidemiological status of the individuals. Specifically, 0≡susceptible and 1≡infected. The labels i and j represent groups of males and females.

2) For $i > 0$ and $j > 0$, $\xi_t(i, \mu; j, \nu)$ gives the number of pairs where the male is of the ith type and has epidemiological status μ and the female is of the jth type and has status ν at time t.

3) Singles are labeled by triplets. However, to keep the domain fixed we use four coordinates and set either i or j equal to zero. Specifically, if $i > 0$ and $j = 0$, then $\xi_t(i, \mu; 0) \equiv \xi_t(i, \mu; 0, 0) \equiv \xi_t(i, \mu; 0, 1)$ denotes the number of single males with status μ in the ith subpopulation at time t. Similarly, if $i = 0$ and $j > 0$, then $\xi_t(0; j, \nu) \equiv \xi_t(0, 0; j, \nu) \equiv \xi_t(0, 1; j, \nu)$ denotes the number of single females with status ν in the jth subpopulation at time t.

Let $S = \{0, 1, 2, \cdots\}^x$ and let $c : S \times S \to (0, \infty)$ be a real-valued function—the flip rate—to be specified later. We view $\{\xi_t : t \geq 0\}$ as an S-valued Markov process with flip rate $c(\cdot, \cdot)$, *i.e.*, if $\xi_t = \xi$ for some $t \geq 0$ then $c(\xi, \eta)$ denotes the instantaneous rate at which ξ_t may change to the state η. The generator of this process is

$$\Omega f(\xi) = \sum_{\eta} c(\xi, \eta)(f(\eta) - f(\xi)), \tag{35}$$

where f is a continuous function on S. Thus,

$$\frac{d}{dt} E f(\xi_t) = E \sum_{\eta} c(\xi, \eta)(f(\eta) - f(\xi_t)).$$

We assume the existence of an underlying mixing/pair formation matrix $(p_{ij}(\xi), q_{ij}(\xi))$ of the type described in Section 2. To specify the flip rates we use the following notation. For $\xi \in S$, $A \subset X$, $B \subset X$ and $A \cap B = \emptyset$, we define $\xi_B^A \in S$ as

$$\xi_B^A(x) = \begin{cases} \xi(x) + 1 & if\ x \in A \\ \xi(x) - 1 & if\ x \in B \\ \xi(x) & otherwise \end{cases}.$$

If we change the notation slightly and now use the letters m and f to denote the parameters associated with uninfected males and females and M and F to denote those associated with infected males and females then one defines the flip rate $c(\cdot, \cdot)$ as follows (here γ, δ, and σ are constant parameters):

a) Pair formation. For $i > 0$, $j > 0$,

$$c\left(\xi, \xi^{(i,\mu;j,\nu)}_{(i,\mu;0),(0;j,\nu)}\right) = b_j^f(\xi)\,\xi(0; j, \nu)\,p_{ji}^f(\xi)\,\frac{\xi(i, \mu; 0)}{\xi(i, \mu; 0) + \xi(i, 1 - \mu; 0)}.$$

b) Pair-dissolution (σ denotes the pair-dissolution rate). For $i > 0$, $j > 0$,

$$c\left(\xi, \xi^{(i,\mu;0),(0;j,\nu)}_{(i,\mu;j,\nu)}\right) = \sigma_{\nu\mu}\xi\left(i,\mu;j,\nu\right).$$

c) Transmission (δ denotes the transmission coefficient—transmission may occur only while paired). For $i > 0$, $j > 0$,

$$c\left(\xi, \xi^{(i,1;j,1)}_{(i,0;j,1)}\right) = \delta_F\xi\left(i,0;j,1\right), \quad c\left(\xi, \xi^{(i,1;j,1)}_{(i,1;j,0)}\right) = \delta_M\xi\left(i,1;j,0\right).$$

d) Recovery (γ denotes the recovery rate). For $i > 0$, $j > 0$,

$$c\left(\xi, \xi^{(i,0;j,0)}_{(i,0;j,1)}\right) = \gamma_F\xi\left(i,0;j,1\right), \quad c\left(\xi, \xi^{(i,0;j,0)}_{(i,1;j,0)}\right) = \gamma_M\xi\left(i,1;j,0\right),$$
$$c\left(\xi, \xi^{(i,1;j,0)}_{(i,1;j,1)}\right) = \delta_F\xi\left(i,1;j,1\right), \quad c\left(\xi, \xi^{(i,0;j,1)}_{(i,1;j,1)}\right) = \gamma_M\xi\left(i,1;j,1\right),$$

while for single infected individuals we have

$$c\left(\xi, \xi^{(i,0;0)}_{(i,1;0)}\right) = \gamma_M\xi\left(i,1;0\right), \quad c\left(\xi, \xi^{(0;j,0)}_{(0;j,1)}\right) = \gamma_F\xi\left(0;j,1\right);$$

furthermore, for $i > 0$, $j > 0$ we have the combined recovery rate

$$c\left(\xi, \xi^{(i,0;j,0)}_{(i,1;j,1)}\right) = \gamma_{FM}\xi\left(i,1;j,1\right).$$

e) Removal (μ denotes the removal rate from sexual activity). For $i > 0$, $j > 0$, μ, ν,

$$c\left(\xi, \xi^{(i,\mu;0)}_{(i,\mu;j,\nu)}\right) = \mu_f\xi\left(i,\mu;j,\nu\right), \quad c\left(\xi, \xi^{(0;j,\nu)}_{(i,\mu;j,\nu)}\right) = \mu_m\xi\left(i,\mu;j,\nu\right),$$

while for the removal rate of single individuals we have that

$$c\left(\xi, \xi_{(i,\mu;0)}\right) = \mu_m\xi\left(i,\mu;0\right), \quad c\left(\xi, \xi_{(0;j,\nu)}\right) = \mu_f\xi\left(0;j,\nu\right).$$

f) Recruitment (all recruited individuals are susceptible). For $i > 0$, $j > 0$,

$$c\left(\xi, \xi^{(i,0;0)}\right) = \Lambda^m_i, \quad c\left(\xi, \xi^{(0;j,0)}\right) = \Lambda^f_j.$$

g) Other. For other $\eta \neq \xi$, we assume

$$c(\xi,\eta) = 0 \text{ and } c(\xi,\xi) = -\sum_{\xi\neq\eta} c(\xi,\eta).$$

(More details are found in Luo and Castillo-Chavez, 1991, 1992).

As the time t changes, singles may form pairs, pairs may dissolve, and the disease may be transmitted (usually only within clearly specified pairings). The system $\{\xi_t\}$ consists of a series of changing elements in the set X, the set of all functions on S. The dynamics of the system is described by the rates at which the system changes. These rates are given as a set of nonnegative numbers $\{c(\xi,\eta) : \xi \neq \eta, \xi, \nu \in X\}$. Specifically, each $c(\xi,\eta)$ is the rate at which the system changes from ξ to η, that is,

$$P(\xi_{t+h} = \eta \mid \xi_t = \xi) = c(\xi,\eta)\,h + o(h), \quad \forall t \geq 0.$$

The deterministic model that corresponds to the above stochastic model in the context of a STD such as gonorrhea (susceptible→infected→susceptible) but that incorporates couples (transmission can only occur in a couple where one of the partners is infected) is described below (for more details see Blythe *et al.*, 1991).

Consider a population of sexually active heterosexual individuals divided into subpopulations by such factors as sex, race, socio-economic background, and average degree of sexual activity. There are N^f female and N^m male subpopulations, each divided into two epidemiological classes for single individuals: $f_j(t)$ and $m_i(t)$ (single susceptible females and males, at time t), and $F_j(t)$ and $M_i(t)$ (single infected females and males), all for $j = 1, \cdots, N^f$ and $i = 1, \cdots, m$. Hence the sexually-active single individuals of each sex and each subpopulation are given by $T_j^f = f_j + F_j$ and $T_i^m = m_i + M_i$. The epidemiological classes for pairs are given by π_{ji}^{fm}, π_{ji}^{Fm}, π_{ji}^{fM}, π_{ji}^{FM}, which are respectively the *numbers of pairs* of f-with-m, F-with-m, f-with-M, and F-with-M individuals. Transmission can only occur among those individuals in pair types π_{ji}^{Fm} or π_{ji}^{fM}. Since $\pi_{ji}^{fM} = \pi_{ij}^{Mf}$, we need only consider four types of pairs. We assumed that the transmission probability per unit time is constant within each pair containing one infected individual. We let δ_M and δ_F be the rates for male-to-female and female-to-male transmission, respectively. The *per capita* recovery rates are γ_M and γ_F for infected males and infected females, respectively, when their partner is uninfected. When both partners are infected (F-with-M pairs), simultaneous treatment of both is the norm for gonorrhea, so we incorporate 'combined' recovery rate γ_{FM}, with both parties moving directly to the f-with-m (no infection) pair type. The *per capita* dissolution rates are σ_{fm}, σ_{fM}, σ_{Fm}, and σ_{FM} for the different types of pairs, and the *per capita* removal rates from sexual activity due to death or other causes are μ_f and μ_m for all females and all males respectively. Let Λ_j^f and Λ_i^m denote the 'recruitment' rates (assumed constant) of single (assumed uninfected) individuals in the female and male populations respectively. We use the notation

$$p_{ji}^{xm} \equiv \frac{m_i}{M_i + m_i} p_{ji}^f, \quad p_{ji}^{xM} \equiv \frac{M_i}{M_i + m_i} p_{ji}^f, \quad p_{ij}^{yf} \equiv \frac{f_i}{F_i + f_i} p_{ij}^m, \quad p_{ij}^{yF} \equiv \frac{F_i}{F_i + f_i} p_{ij}^m,$$

($x = f$ or F and $y = m$ or M, for $i = 1, \cdots, N^m$ and $j = 1, \cdots, M^f$) for the fraction of pair formations between the specified sub-groups (i and j) which are of given infection status; for example, p_{ji}^{fm} and p_{ji}^{fM} give the fractions involving uninfected (m_i) and infected (M_i)

males respectively. Then the gonorrhea pair formation/dissolution model[2] is

$$\frac{df_j}{dt} = \Lambda_j^f + \gamma_F F_j + [\mu_M + \sigma_{fM}] \sum_{i=1}^{N^m} \pi_{ji}^{fM} + [\mu_M + \sigma_{fm}] \sum_{i=1}^{N^m} \pi_{ji}^{fm} - \left[C_j^f + \mu_f\right] f_j,$$

$$\frac{dF_j}{dt} = [\mu_m + \sigma_{Fm}] \sum_{i=1}^{N^m} \pi_{ji}^{Fm} + [\mu_m + \sigma_{FM}] \sum_{i=1}^{N^m} \pi_{ji}^{FM} - \left[C_j^f + \gamma_F + \mu_f\right] F_j,$$

$$\frac{dm_i}{dt} = \Lambda_i^m + \gamma_M M_i + [\mu_f + \sigma_{Fm}] \sum_{j=1}^{N^f} \pi_{ji}^{Fm} + [\mu_f + \sigma_{fm}] \sum_{j=1}^{N^f} \pi_{ji}^{fm} - [C_i^m + \mu_m] m_i,$$

$$\frac{dM_i}{dt} = [\mu_f + \sigma_{fM}] \sum_{j=1}^{N^f} \pi_{ji}^{fM} + [\mu_f + \sigma_{FM}] \sum_{j=1}^{N^f} \pi_{ji}^{FM} - [C_i^m + \gamma_M + \mu_M] M_i,$$

$$\frac{d\pi_{ji}^{fm}}{dt} = C_j^f p_{ji}^{fm} f_j + \gamma_M \pi_{ji}^{fM} + \gamma_F \pi_{ji}^{Fm} + \gamma_{FM} \pi_{ji}^{FM} - [\mu_f + \mu_m + \sigma_{fm}] \pi_{ji}^{fm},$$

$$\frac{d\pi_{ji}^{Fm}}{dt} = C_j^f p_{ji}^{Fm} F_j + \gamma_M \pi_{ji}^{FM} - [\mu_f + \mu_m + \sigma_{Fm} + \delta_F + \gamma_F] \pi_{ji}^{Fm},$$

$$\frac{d\pi_{ji}^{fM}}{dt} = C_j^f p_{ji}^{fM} f_j + \gamma_F \pi_{ji}^{FM} - [\mu_f + \mu_m + \sigma_{fM} + \delta_M + \gamma_M] \pi_{ji}^{fM},$$

$$\frac{d\pi_{ji}^{FM}}{dt} = C_j^f p_{ji}^{FM} F_j + \delta_F \pi_{ji}^{Fm} + \delta_M \pi_{ji}^{fM} - [\mu_f + \mu_m + \sigma_{FM} + \gamma_M + \gamma_F + \gamma_{FM}] \pi_{ji}^{FM},$$

with initial conditions $f_j(0) > 0$, $m_i(0) > 0$, $\pi_{ji}^{fm}(0) = 0$, $\pi_{ji}^{fM}(0) = 0$, $\pi_{ji}^{Fm}(0) = 0$, $\pi_{ji}^{FM}(0) = 0$, and at least one of the $F_j(0)$ and $M_i(0)$ greater than zero (for $i = 1, \cdots, N^m$ and $j = 1, \cdots, N^f$). We invoke the results of Kurtz (1970, 1971) and conclude that for large populations the deterministic and stochastic models will have the same asymptotic behavior.

6.b Simulations of the Process $\{\xi_t : t \geq 0\}$

The general approach for simulating jump Markov processes is as follows. From the construction of the flip rates we know that

$$c(\xi) = \sum_{\eta \in S} c(\xi, \eta) < \infty.$$

Hence, if we let the sequence $0 = \rho_0 < \rho_1 < \rho_2 < \cdots$ denote the jump times of the process. Then $\tau_n = \rho_n - \rho_{n-1}$ has an exponential distribution with rate $c(\xi_{\rho_{n-1}})$. We can simulate the process using the following procedure:

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[2] In general, this model should be modified using *Remark 3* and *Remark 5*. However, this modification implies that one gender makes the decisions (but see Castillo-Chavez *et al.*, 1993c, and Hsu-Schmitz, 1994).

i) Set initial state ξ_0 and assume a sequence of n jump times $0 = \rho_0 < \rho_1 < \cdots < \rho_n$ and their corresponding states ξ_{ρ_i}, $1 \leq i \leq n$ have been determined.

ii) Get τ_{n+1} from $\exp\{c(\xi_{\rho_n})\}$ and let $\rho_{n+1} = \rho_n + \tau_n$.

iii) Set $\xi_{\rho_{n+1}} = \eta$ with probability $\dfrac{c(\xi_{\rho_n}, \eta)}{c(\xi_{\rho_n})}$.

iv) Define $\xi_t = \xi_{\rho_n}$ for $\rho_n \leq t < \rho_{n+1}$.

We proceed to simulate the above stochastic model in a very special situation. We assume that the infection rates δ_M and δ_F are equal to zero or, equivalently, that there are no infected individuals in the population. Hence, we are simulating a purely demographic model. Individuals form and dissolve pairs. There is constant recruitment and we have individuals of several (economic, social, etc.) types. The simulation (described below) will have as its average dynamics the corresponding deterministic dynamics. However, the stochastic version allows for the study of changes in the variance as a function of time.

A 10,000-realization run was allowed to simulate the process up to time $t = 2.0$, and a 1,000-realization run was extended to $t = 32.0$. Simulations were carried out using four groups of single males and four groups of single females, resulting in 16 possible pair-types. The initial numbers used for single males, by group, were: $m_1 = 1000$, $m_2 = 900$, $m_3 = 800$, and $m_4 = 2700$. For females, the corresponding numbers were: $f_1 = 2000$, $f_2 = 1000$, $f_3 = 500$, and $f_4 = 3500$. The initial number of pairs was constrained to zero for all possible pair-types. Removal rates for individuals—whether single or in pairs—were held constant at 0.1. For this exercise, pair dissolution rates were also assumed to be invariant, but with a 5.0 value. Recruitment rates used for (single) males were the following: $m_1 = 100$, $m_2 = 90$, $m_3 = 80$, and $m_4 = 270$; the recruitment rates imputed for (single) females were: $f_1 = 200$, $f_2 = 100$, $f_3 = 50$, and $f_4 = 350$. Pair formation rates for males were set at: $m_1 = 3.5$, $m_2 = 3.0$, $m_3 = 2.5$, and $m_4 = 3.5$; and pair formation rates for females were set at: $f_1 = 2.5$, $f_2 = 2.45$, $f_3 = 2.3$, and $f_4 = 2.586$.

As shown in Figure 1, stability in absolute sizes is reached early—at $t < 0.5$ (relative distributions, not shown, expose the same trait). This pattern of very rapid stabilization is maintained when averages are computed from simulations based on as few as 30-to-100 realizations. Variance, expressed as standard deviations, for all possible pair-types also stabilizes at $t < 0.5$ (see Figure 2). However, variation for each of the groups of single individuals, male or female, continues to increase until $10 \leq t \leq 12$ (Figures 2 and 3). Further simulations will allow us to ascertain whether the pattern observed beyond $t = 12$ indeed reflects stability—or only smaller increments in variation. A simulation with 10,000 or more realizations and a time horizon of $50 \leq t \leq 100$ may be necessary to accomplish this. Further numerical studies of this framework have been carried out by Castillo-Chavez *et al.* (1993a,b).

7. CONCLUSIONS

In this article we have introduced a flexible framework for the modeling of contact structures in biology, which was built on our earlier work (see Blythe and Castillo-Chavez, 1989;

Castillo-Chavez and Blythe, 1989; Busenberg and Castillo-Chavez, 1989, 1991). It can be implemented in stochastic and/or deterministic frameworks. We have provided modeling applications in demography, including demographic models for pairs, epidemiology, inheritance of cultural traits, and food web dynamics. Recently, we have also estimated the contact structure, as modeled by our mixing matrices, of a student population as a function of sexual or dating activity (see Castillo-Chavez *et al.*, 1992; Hsu Schmitz and Castillo-Chavez, 1992). In addition, we have worked on methods for estimating the parameters associated with arbitrary mixing structures (see Rubin *et al.*, 1991; Blythe *et al.*, 1992). Furthermore, the representation theory of mixing matrices as a function of the preference structure of a population, as first developed by Busenberg and Castillo-Chavez (1989, 1991), has allowed us to begin studying the role of preference in two-sex mixing populations (see Hsu and Castillo-Chavez; Hsu *et al.*, 1993). In fact, we designed and conducted a behavioral survey of a college population in order to estimate the preference and mixing structure of two-sex mixing populations.

This research represents our initial efforts in understanding the role of 'social' structures in disease dynamics. Most models in the past assumed a fixed social/behavioral structure. The serious study of the transmission dynamics of HIV pointed out the serious limitations of this approach. We have observed a large number of theoretical advances over the last few years (see Castillo-Chavez, 1989; Jewell *et al.*, 1992; Anderson and May, 1991; and Hethcote and Van Ark, 1992). However, we have just begun to understand the effects of changing contact structures in population dynamics. This is what happens when biology meets mathematics!

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Figure 1. Population by Sex, Class, and Group. Simulation with $\sigma = 5.0$, $t <= 2.0$, and 10,000 Realizations.

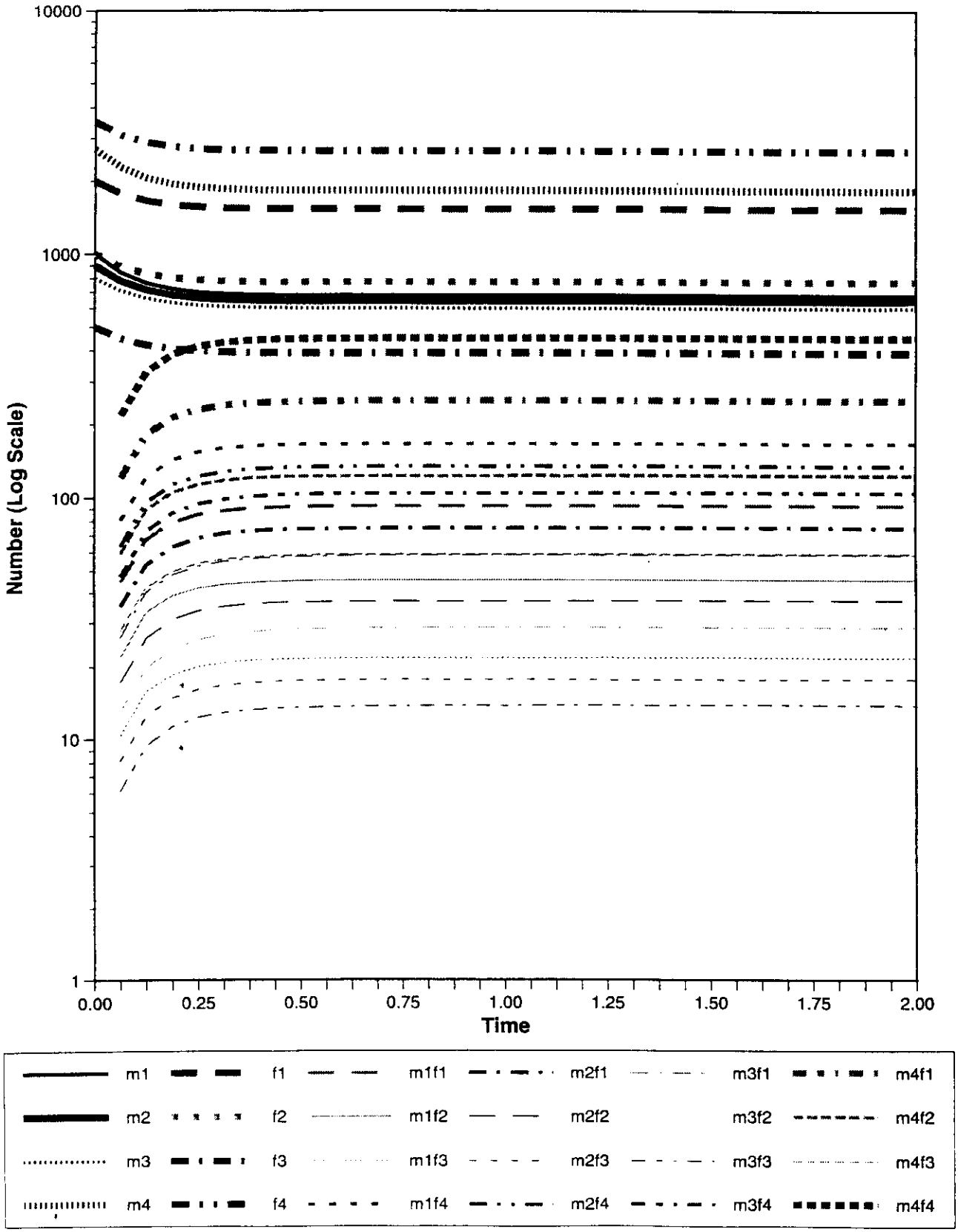

Figure 2. Standard Deviation, Total Population by Sex, Class, and Group. Simulation with σ = 5.0, t <= 2.0, and 10,000 Realizations.

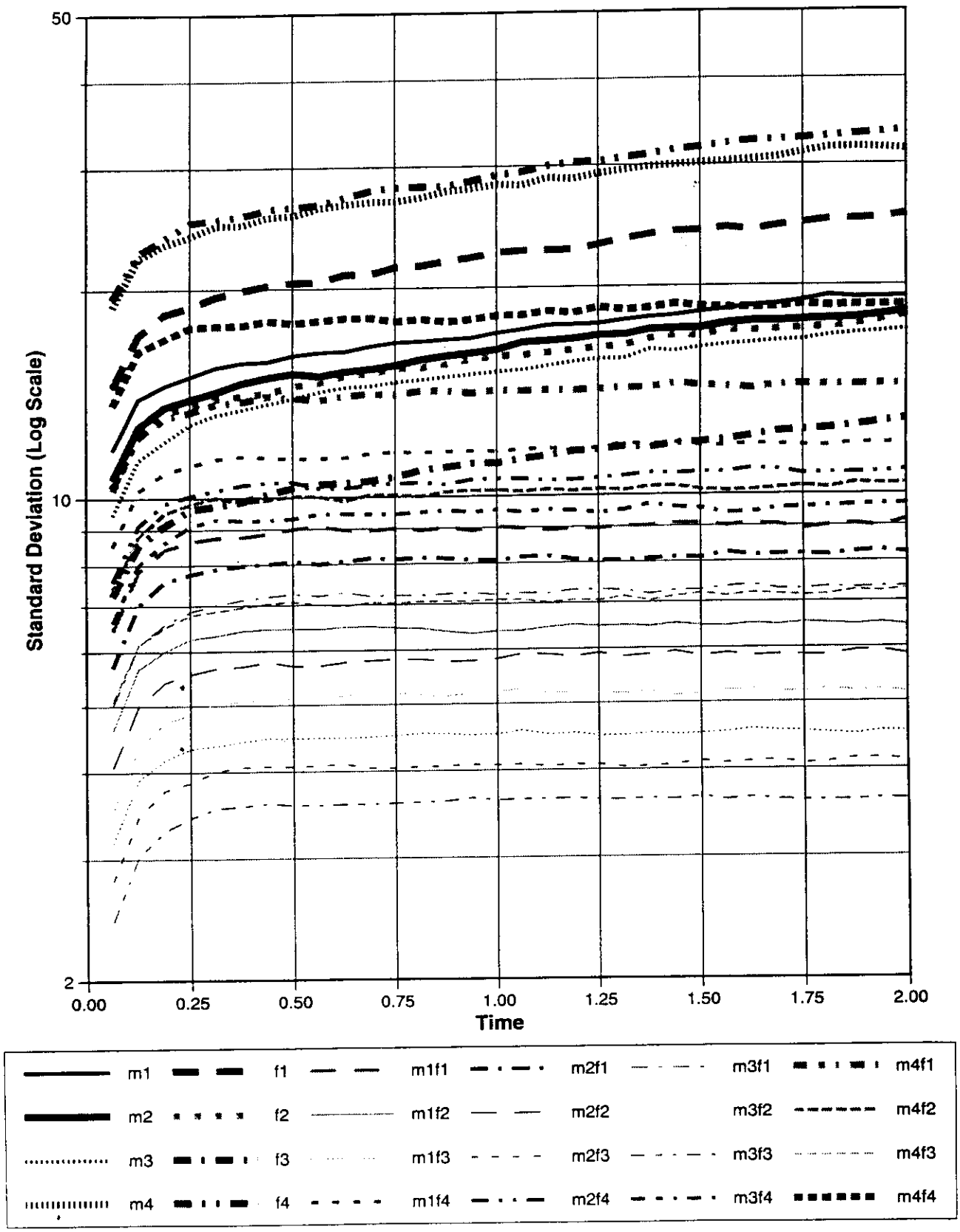

Figure 3. Standard Deviation, Total Population by Sex, Class, and Group. Simulation with σ = 5.0, t <= 32.0, and 1,000 Realizations.