

## A GENERALIZED MODEL OF PARASITOID, VENEREAL, AND VECTOR-BASED TRANSMISSION PROCESSES

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**Abstract.**—General models incorporating search behaviors are used to demonstrate the parallels between attack rates in host-parasitoid systems and transmission processes in sexually and vector-transmitted diseases. Density-dependent transmission, in which the probability of an individual's becoming infected is a function of the density of infectives,  $I$ , is the usual assumption in disease models. Frequency-dependent transmission, in which the probability of an individual's becoming infected is a function of the proportion of infectives,  $I/N$ , is often considered characteristic of venereal and vector-based systems. These two characterizations of the transmission process are shown to represent extremes of the Type II functional response curve. When there is vector-based transmission, and depending on the details of vector behavior, the probability of an uninfected host's becoming infected may range from being predominantly a function of  $I$  to being proportional to  $I/N^2$ . With a limited number of hosts visited per vector, transmission may decline with increasing overall density of the host population; this was observed in empirical data for a pollinator-transmitted disease. Unified, general models of the transmission process are essential for comparison of dynamic processes in different systems and for studies of the evolution of the transmission process itself.

Classic disease transmission models (Kermack and McKendrick 1927; Anderson and May 1979) have generally assumed that the probability of an uninfected individual's becoming infected is proportional to the density of infectives in the population. It has been pointed out that this assumption may not hold for vector-borne and venereal diseases (i.e., sexually transmitted diseases, or STDs), in which the transmission process is more likely to be dependent on the frequency of infectives in the population (May and Anderson 1979; Anderson 1981; Getz and Pickering 1983). A frequency-dependent transmission mode results in population dynamics that are quite different from those obtained with more classic density-dependent assumptions (Getz and Pickering 1983; Antonovics 1992, 1994; Thrall et al. 1993, 1995). Models based on either pure density-dependent or pure frequency-dependent transmission have the advantage of simplicity and are useful for comparison of the consequences of contrasting scenarios. However, they suffer from several difficulties. First, they are very phenomenological: the transmission coefficients are essentially "fudge factors," not being based on any explicit description of the biological process of transmission. This can lead to an intrinsic pathology in such models, whereby large values for the transmission

coefficients or disease incidence can result in more infectives being produced than there are uninfected individuals in the population (Thrall et al. 1995). Second, any real-world transmission process is likely to be somewhat intermediate between pure density dependence and pure frequency dependence. For example, while vectors may adjust their movement to compensate for changes in host density, such adjustment may not be perfect and will be subject to constraints implicit in any foraging behavior. Many diseases do not fall comfortably into one category or another. For example, pollinator-transmitted diseases (Alexander and Antonovics 1988) are a type of STD in which the transmission involves a vector moving from flower to flower; in cases in which pathogens time their release to coincide with lekking, or periods of sexual congregation (see, e.g., Tinsley 1989), the distinction between contact and noncontact transmission becomes blurred. Third, while the transmission parameter or coefficient is often denoted by  $\beta$ , the units of this coefficient are different in purely phenomenological models with frequency- and density-dependent transmission (Thrall et al. 1995).

Behavioral components of the transmission process have been extensively considered in host-parasitoid systems (Hassell 1978) in which models based on quite general, but realistic, aspects of parasitoid behavior have been developed. Behavioral aspects of transmission have also been an important component in models of venereal disease spread in humans (Hethcote and Van Ark 1987; Anderson et al. 1989), but these models have been designed in the context of specific human situations (e.g., a sexually active "pool") and may not be generalizable to a wider range of animal behaviors.

In the present study, we show how models incorporating pure density-dependent or pure frequency-dependent transmission parameters can be reconciled by simple consideration of processes implicit in the behavior of disease-carrying agents. We then extend this approach to examine the expectations with regard to vector-based disease transmission. Throughout, our goal is to develop heuristic, general formulations rather than to model any specific disease in great predictive detail.

#### THE TRANSMISSION PROCESS AND THE BEHAVIOR OF INFECTIVES

Infected individuals searching for mates, or vectors carrying disease, are likely to show complex behaviors. Generalized models for searching behaviors in relation to host densities have been developed in host-parasitoid and predator-prey systems (Hassell 1978). In this section we show how such models can be used to explicate the continuum between density-dependent and frequency-dependent transmission processes.

##### *Type I Functional Response and Density-Dependent Transmission*

We assume that there are  $I_t$  disease-carrying agents. These agents could be infected hosts, disease-carrying vectors, or parasitoids. In the case of microparasitic infections, hosts can be classified as susceptible and uninfected,  $S_t$ , or infected and infectious,  $I_t$  (where the total number of hosts [ $N_t$ ] is  $I_t + S_t$ ); we consider infectious individuals to be the disease-carrying agents. In the case of

parasitoids,  $S_t$  is assumed to represent the total number of hosts (all of which are assumed to be susceptible), and  $I_t$  is assumed to represent the total number of parasitoids, which are the disease-carrying agents. Numbers of parasitoids and their hosts are usually symbolized by  $P$  and  $N$ , respectively, rather than by  $I$  and  $S$ ; we use the latter because we wish to emphasize how models originally developed in a parasitoid context are generally applicable to disease systems.

We assume that each disease agent has time,  $T$ , available for search. If the number of encounters with hosts,  $N_e$ , rises linearly with host density, then the number of encounters with susceptible hosts per disease agent during any time interval is given by

$$\frac{N_e}{I_t} = aTS_t, \tag{1}$$

and the number of encounters per susceptible host is

$$\frac{N_e}{S_t} = aTI_t, \tag{2}$$

where  $a$  represents a constant proportional to the search rate of the infectious individual. If  $S_t$  and  $I_t$  are in units of numbers per unit area, then  $a$  represents the area searched by the infectious individual per unit time,  $T$ .

In disease models, particular attention has been given to the so-called force of infection, which in discrete-time models is the probability that a susceptible individual will become infected during a particular time period (Anderson and May 1991). If the per-encounter probability of disease transmission is  $\delta$  and the number of encounters per host is a stochastic variable following the Poisson distribution, then the probability that a susceptible individual becomes infected is

$$1 - \sum_{k=1}^{\infty} (1 - \delta)^k \frac{(N_e/S_t)^k}{k!} \exp\left(-\frac{N_e}{S_t}\right), \tag{3}$$

which equals

$$1 - \exp\left(-\delta \frac{N_e}{S_t}\right); \tag{4}$$

and when both  $\delta$  and  $N_e/N_t$  are small, this can be approximated as simply

$$\delta \frac{N_e}{S_t}. \tag{5}$$

It can be seen that the force of infection is equivalent to either equation (4) or (5), depending on the degree of approximation assumed regarding the magnitudes of  $\delta$  and  $N_e/S_t$ . In continuous-time models the force of infection is the rate at which susceptible hosts become infected, and this is equivalent to the form in equation (5). In all these equations, transmission is purely density-dependent, being a function of  $N_e/S_t$ , which from equation (2) is linearly proportional to  $I_t$ .

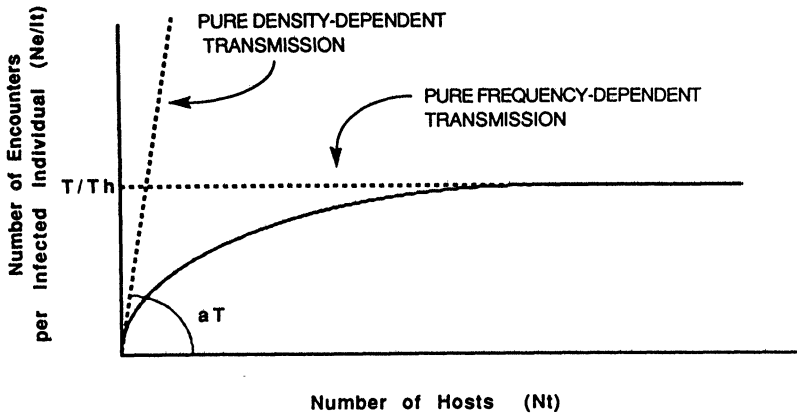


FIG. 1.—Graph of a typical Type II functional response (*solid curve*) showing the expected relationship between number of hosts encountered per disease-carrying agent (parasitoid, infected individual, or vector) and host density. Dotted lines show the expected relationships for extreme cases of pure density-dependent transmission (equivalent to a Type I functional response) and pure frequency-dependent transmission.  $T$ , Total time available for search;  $T_h$ , handling time of each disease agent-host encounter;  $a$ , constant representing the “area of discovery” or search rate. For further explanation, see eqq. (1), (2), (6), and (7).

The disease literature also usually invokes a transmission parameter (often denoted by  $\beta$ ) that, when multiplied by the density of infectives, gives the likelihood that a susceptible individual becomes infected (i.e., the force of infection). It follows that in equations (3)–(5),  $\beta$  is the force of infection divided by  $I_1$ , which in the continuous form gives

$$\beta = aT\delta. \quad (5a)$$

Equations (1)–(5) represent the so-called Type I response of Holling (1959), and equation (1) is shown in figure 1 as the dotted line labeled “pure density-dependent transmission.” There may be some confusion with the use of the term “density-dependent.” In the context of transmission, the term “density” refers to the density of infectives and not to the total population density or the density of susceptibles; in the context of host-population regulation, the term “density-dependent infection or parasitism” may also be used to imply that the probability of encounters with infected or parasitized individuals increases with increasing total density of hosts. It should be noted that when dealing with infected and susceptible hosts (as in a microparasitic disease), increasing the density of infectives while holding their frequency constant results in an increase in total population size. Therefore, under pure density-dependent transmission, the number of encounters per host will also increase with total population density; however, if only the number of susceptible hosts is increased, the number of encounters per susceptible host will be unchanged even though overall population density increases.

*Type II Functional Response and Frequency-Dependent Transmission*

The process of infection may involve some period of contact between the host and the infected individual, and/or the encounter may be followed by a period during which further encounters are avoided. These activities have come to be known (Holling 1959; Hassell 1978) as the "handling time,"  $T_h$ . This leads to a functional response of the form

$$\frac{N_e}{I_t} = \frac{aTS_t}{1 + aT_hN_t} \tag{6}$$

Note that here  $N_t = S_t + I_t$ , whereas in a parasitoid model it would be  $S_t$ , the total number of hosts (all of which are assumed to be susceptible). From equation (6) the number of encounters per susceptible host and infectious individuals becomes

$$\frac{N_e}{S_t} = \frac{aTI_t}{1 + aT_hN_t} \tag{6a}$$

Equation (6) is the so-called Type II response of Holling (1959) and is shown graphically as the solid curve in figure 1. The number of encounters per susceptible host rises with increasing host density to an asymptote,  $T/T_h$ . The rate of approach to this asymptote is determined by the magnitude of  $a$ . At the asymptote, for a large value for  $aN_t$ , the number of encounters per susceptible host is given approximately by

$$\frac{N_e}{S_t} = \frac{TI_t}{T_hN_t} \tag{7}$$

Therefore, the number of encounters per host by each infected individual becomes proportional to the frequency of infectives in the population. Pure frequency-dependent transmission, in which there are a fixed number of encounters by an infected host regardless of host density, is therefore represented by the horizontal line in figure 1. The number of encounters per susceptible host can be translated into the force of infection as in equations (3)–(5) above, depending on the desired level of approximation.

The general expectation that STDs should show frequency-dependent transmission follows from the fact that the numerical dynamics of these diseases are envisioned to encompass values of  $aT_h$  that place  $N$  in the asymptotic region of figure 1. This is because in many STDs, the handling time is likely to be large relative to the time interval. Handling time represents not just the period of copulation but might also include periods spent in intrasexual competition, pair formation, gestation, and parental care; and because transmission by definition is limited to the breeding season,  $T$  may also be small. Moreover, because of active and mutual searching, the parameter  $a$ , the area of discovery, may also be much larger in sexual encounters relative to situations in which encounters are incidental. A biological interpretation of a large value of  $a$  is that all hosts can be found regardless of density and that the number of encounters is determined only by the handling time.

## A MODEL OF VECTOR-BASED TRANSMISSION

We can partition the process of vector-based transmission into two phases, the first being the loading of the pathogen onto the vector and the second being the deposition of the pathogen and infection of the host. We assume that the loading of the pathogen onto the vector is a fairly rapid process that can be described in continuous time and in which equilibrium levels of the pathogen are achieved relatively rapidly. We then consider that infection of the host is a slower process that is best described in discrete time.

Throughout, we assume there is no vector-to-vector transmission and that the pathogen is passively transmitted, having no impact on the behavior or life history of the vector. We assume that the rate of supply of vectors is constant and independent of the number of hosts (i.e., their dynamics are effectively uncoupled during the time course of the transmission phase), that the vectors are unable to distinguish between susceptible and infected hosts, and that vectors either carry the pathogen or not (i.e., we ignore differences among vectors in the amount of the pathogen that they carry).

*Pathogen Loading onto the Vector*

We assume that vectors do not carry the pathogen when they first enter the population; for example, in a pollinator-transmitted disease, bees carrying spores may clear them in the hive prior to returning to a diseased population.

Let  $W$  be the number of vectors without the pathogen;  $Z$  is the number of vectors with the pathogen,  $V$  equals  $W + Z$  (i.e., the total number of vectors),  $I$  equals  $I_t$  (i.e., the number of infectious hosts), and  $N$  equals  $N_t$  (i.e., the total number of hosts). Then, the rate of change of  $W$  and  $Z$  over time is given by

$$\frac{dW}{dt} = \lambda - \frac{\delta_v aIW}{1 + aT_h N} - \mu W \quad (8)$$

and

$$\frac{dZ}{dt} = \frac{\delta_v aIW}{1 + aT_h N} - \mu Z, \quad (9)$$

where  $\lambda$  is the rate of supply of the vector (assumed to be a constant; units are numbers per time interval),  $\mu$  is the rate of departure of the vector from the population (units are numbers per time interval), and  $\delta_v$  is the per-encounter probability of pathogen loading onto the vector.

This model is identical to that used by Anderson (1981), except that it is more complex in assuming a Type II functional response on the part of the vectors visiting their hosts rather than a pure frequency-dependent loading rate (but it is simpler in assuming that the pathogen has no latent period in the vector). The ratio  $\delta_v/T_h$  determines the maximum rate at which the pathogens are picked up by the vector when all the hosts are infectious. The magnitudes of  $aT_h$  and  $a\delta_v$  determine the rate of approach to this asymptote; when  $1/aT_h = N$ , the rate of pathogen loading is half the maximum.

It can be readily shown from equations (8) and (9) that equilibrium values of vectors without spores and with spores are given by, respectively,

$$\hat{W} = \frac{\lambda}{\mu + \frac{\delta_v a I}{1 + a T_h N}} \tag{10}$$

and

$$\hat{Z} = \frac{1}{\mu} \frac{\delta_v a I}{1 + a T_h N} \hat{W}. \tag{11}$$

If the fraction of vectors with spores is small ( $\hat{Z} \ll \hat{W}$ ), then  $\hat{W} \approx \hat{V} = \lambda/\mu$ , and

$$\hat{Z} \approx \frac{1}{\mu} \frac{\delta_v a I}{1 + a T_h N} \hat{V}. \tag{12}$$

We assume below that these equilibria are established relatively rapidly within the time period over which disease transmission is possible.

*Pathogen Transfer from the Vector to the Host*

We now consider the probability that a susceptible host becomes infected by the pathogen. This will also be a function of the vector's Type II response and is proportional to the search efficiency of the vector, the rate at which the pathogens are deposited on the host, and the effectiveness of these pathogens in causing infections.

If the vector visits the host repeatedly, then in a fixed time interval (a year or a mating season) the probability that a susceptible host becomes infected is given by (from eqq. [4] and [6a])

$$1 - \exp\left(-\frac{\delta_h a T \hat{Z}}{1 + a T_h N}\right), \tag{13}$$

where  $\delta_h$  represents the per-encounter probability of pathogen unloading (i.e., the per-encounter infection) from the vector onto the host. Substituting  $\hat{Z}$  from equation (12) into equation (13), we get

$$1 - \exp\left(-\frac{a^2}{\mu} \delta_v \delta_h T \hat{V} \frac{I}{(1 + a T_h N)^2}\right). \tag{14}$$

Therefore, the force of infection is approximately proportional to  $I/N^2$  when  $a T_h$  is large and is approximately proportional to  $I$  when  $a T_h$  is small. Exact expressions can be obtained by substituting equations (10) and (11) into equation (13).

COMPLEXITIES IN HOST AND VECTOR BEHAVIOR

Complexities in the behavior of the vector and the host may change the simple scenario outlined above. Thus, vectors may experience a substantial latent period approaching their average survival time (Anderson 1981); or the number of en-

counters per vector may be very few, as in the case of mosquitoes that generally require one or a few blood meals. Hosts may, in turn, through their behavior severely limit the time during which they are exposed to vectors (e.g., by avoiding mosquito bites). Conversely, there can be pathogen-induced changes that increase the chance that the host becomes infected (Moore 1984).

In this section we explore the transmission consequences of a refractory period, a period immediately after a vector encounter during which the host does not attract or avoids the vector. The refractory period may result from behaviors to avoid the vector after one encounter. For example, in the *Silene-Ustilago* system, in which the disease is pollinator transmitted (Alexander 1987, 1990; Alexander and Antonovics 1988), female flowers will remain open and receptive to pollinators for up to 7 d if unvisited but will close within 24 h after pollination. In the model developed below, we ask how the transmission process might be affected, given a refractory period in which hosts are unavailable for search.

Let there be  $N$  (i.e.,  $I + S$ ) hosts and  $V$  vectors, searching over a time period. We assume that there is a refractory period,  $T_r$  (assumed to be a constant time interval), following a vector encounter, during which the host does not attract or avoids the vector. Correspondingly, the vector has a handling time during which it is not searching. Let there be  $N_e$  encounters between the host and the vector over the period  $T$ . The total susceptible host time is  $ST$ , of which  $N_e T_r$  is unavailable to the vector; correspondingly, the total vector time is  $VT$ , of which  $N_e T_h$  is unavailable for search. Then

$$\begin{aligned} N_e &= (a/T)(ST - N_e T_r)(VT - N_e T_h) \\ &= aVST \left(1 - \frac{N_e T_r}{ST}\right) \left(1 - \frac{N_e T_h}{VT}\right). \end{aligned} \quad (15)$$

Assuming that  $T_r$  and  $T_h$  are small relative to the time interval  $T$  (and/or that the number of encounters per host or per vector is small), we can obtain an approximate solution of equation (15) by assuming that the quadratic term is small. Then

$$\frac{N_e}{S} \approx aT \frac{V}{1 + aT_r V + aT_h N}. \quad (16)$$

Arguments similar to those in equations (8)–(12) give the equilibrium frequency of vectors carrying pathogens as

$$\hat{Z} \approx \frac{\delta_v}{\mu} \hat{V} \frac{aI}{1 + aT_r \hat{V} + aT_h N}. \quad (17)$$

If we assume that the probability of a susceptible host's receiving a visit is a stochastic variable that has a Poisson distribution (eq. [3]), the probability of a susceptible host's becoming infected by encountering a disease-carrying vector is a function of the per-encounter pathogen-unloading rate, the number of vector encounters per host, and the probability that the vector carries the pathogen:

$$1 - \exp\left(-\delta_h \frac{N_e \hat{Z}}{S \hat{V}}\right). \quad (18)$$

Substituting equations (16) and (17) into equation (18), we get

$$1 - \exp\left(-\delta_v \delta_h \frac{a^2 T}{\mu} \hat{V} \frac{I}{(1 + aT_r \hat{V} + aT_h N)^2}\right). \tag{19}$$

It can be seen that if the refractory period is short and the handling time is short, then the probability of becoming infected is a function of  $I$  and  $\hat{V}$ . If the refractory period is long but handling time is short, this probability is again a function of  $I$  but is independent of  $\hat{V}$  at high vector abundances. If the refractory period is relatively small or negligible, then equation (19) becomes

$$1 - \exp\left(-K \frac{I}{(1 + aT_h N)^2}\right), \tag{20}$$

where  $K = \hat{V} \delta_v \delta_h a^2 / \mu$ , and the probability of becoming infected is a function of  $I/N^2$ . If both the handling time and the refractory period are large, then the asymptotic relationships are more complex and depend on the relative magnitudes of  $aT_r \hat{V}$  and  $aT_h N$ .

DISCUSSION

The analyses presented above illustrate that formulations involving density-dependent disease transmission and frequency-dependent disease transmission are extremes on a continuum represented by a Type II functional response. However, in the case of vector transmission, details of host and vector responses may affect the form of the transmission. We only considered one complication, a refractory period in the host, but clearly other factors, such as a latent period in the vector, may affect the functional form of the transmission process. The above results are summarized in table 1, which shows the functional dependence of the force of infection for each transmission mode and the extremes of the Type II functional response.

Previously, the impact of behavior on disease transmission has been most extensively explored in host-parasitoid systems (Hassell 1978) in which the host releases parasitoids that themselves lay eggs on other hosts. Because the parasitoids actively transmit infectious stages from host to host, they resemble venereal disease systems in which infected hosts actively search for mates. There should therefore be a close parallel between models relating to venereal disease transmission and those pertaining to parasitoids. In both types of system, the force of infection is dependent on the searching efficiency and vagility of the disease-carrying agent. Thus, for the process to depend on relative abundances of host and parasitoid and not just on host density, parasitoids should be highly vagile and be able to detect hosts at considerable distances. Empirical studies, however, show that attack rates are highly dependent on host densities, and, therefore, the general assumption in host-parasitoid models is that host densities are effectively well below the numbers at which encounter rates asymptote (Hassell 1978). Moreover, in parasitoid transmission, parasitoid behavior is only a function of the number of extant susceptible individuals in the population; if it is assumed that

TABLE 1  
 FUNCTIONAL DEPENDENCE OF THE FORCE OF  
 INFECTION FOR PARASITIDS, SEXUALLY  
 TRANSMITTED DISEASES, AND VECTOR-MEDIATED  
 DISEASES

MODE OF TRANSMISSION	FUNCTIONAL RESPONSE	
	Large $aT_h$	Small $aT_h$
Parasitoid	$I/S$	$I$
Sexual	$I/N$	$I$
Vector borne:		
Small $aT_r$	$I/N^2$	$I$
Large $aT_r$	$f(I, N)$	$I$

NOTE.—The dependence is shown for two extreme types of the functional response curve of the transmitting agent. When  $aT_h$  has a large value, the number of encounters is limited by the handling time and asymptotes at high host density; when  $aT_h$  has a small value, the number of encounters is limited largely by the number of hosts;  $S$  indicates the number of susceptibles,  $I$  the number of infectives,  $N$  the total population size, and  $f(I, N)$  indicates no simple asymptotic form.

parasitoids do not avoid previously parasitized hosts, this is simply the number of hosts. In STDs the behavior of infected individuals also depends on the total number of hosts, but this now includes both infected and susceptible individuals.

A fundamental difference in the transmission process in STDs and host-parasitoid systems has been pointed out by Heesterbeek and Metz (1993). They argue correctly that the Holling-type argument cannot be strictly applied to STDs because sexual encounters (as opposed to, say, host-parasitoid encounters) occur not just between infected and susceptible individuals but also between individuals in the susceptible and in the infected categories. The handling time therefore influences the small percentage of the population involved in pair formation, while transmission only occurs among a subset of this percentage (susceptible-infected pairs). Using a mechanistic model based on rate of pair formation and disassociation, Heesterbeek and Metz derive an exact expression for the force of infection. A more heuristic derivation based on an approach involving handling time during pair formation gives an equivalent result, which they term "Holling Squared." However, their expression has asymptotic properties (for large or small values of  $T_h$  and  $N$ ) identical to the more naive Type II Holling response presented above and differs only in that the rate of approach to the asymptote is slower. Indeed, in STDs there are likely to be many additional complications not considered by either model. For example, males and females are likely to have different rates of partner exchange and different refractory periods, and there may be asymmetries in per-encounter transmission rates between males and females.

There are differences between parasitoid and STD systems in addition to those

directly affecting the transmission process, and these are likely to affect the overall dynamics. Thus, in parasitoid systems, the most general assumption is that the number of parasitoids (or infected individuals) present at any given time interval is only a function of the number of infected individuals in the previous season or time interval rather than, as in the case of vector-borne and venereal diseases, a function of the extant, current year's numbers of infected and susceptible individuals. This is because, in the case of parasitoids, parasitized hosts in the current year do not generally contribute to the current year's parasitoid load, nor do they normally overwinter and contribute to the transmission processes in the subsequent year.

In many models of human STDs (Anderson and May 1991), it is assumed that disease transmission depends on the number of new contacts per partner,  $c$ , independent of the host density. It can be seen from equation (6) that  $c$  could be considered equivalent to  $T/T_h$ , as  $a \rightarrow \infty$ . The force of infection is therefore a function of  $\delta$ ,  $c$ , and  $I_t/N_t$  (the functional form depending on the approximations represented in eqq. [4]–[5]), where  $\delta$  is the per-contact rate of transmission. It should be noted that usually in STD models, a contact is considered to be a sexual partner, in which case  $\delta$  is the per-partner rather than the per-copulation transmission probability. During sexual transmission, the force of infection will depend on the per-copulation transmission rate, on the number of copulations per partnership or mating, and on the number of new mates per unit time; the value for  $c$  should also be adjusted to take into account variance in the number of contacts resulting from individual heterogeneity in sexual behavior (Anderson and May 1991).

It is also possible that the transmission process in STDs is partly density-dependent because mating frequency may decline with decreasing population density. Indeed, if densities fall to such low values that contact rates decline, it is likely that there would be a concomitant fall in reproductive success. A number of sexually transmitted diseases may involve relatively passive transfer of infectious stages during sexual congregation (Tinsley 1989), or they may involve both contact and sexual transmission (e.g., many forms of brucellosis; Nielsen and Duncan 1990). The Type II functional response models clearly incorporate such "partial" density dependence in a general way. If we rewrite  $a = a'/(1 - a')$ , then  $a'$  will be zero for pure density dependence and unity for pure frequency dependence. This reparameterization is in and of itself rather trivial, but it helps shed light on the nature of the transmission parameter,  $\beta$ , when the force of infection is written as  $\beta I$  and  $\beta I/N$  in the density- and frequency-dependent cases, respectively. In such formulations,  $\beta$  is a composite value proportional to

$$\frac{\delta a T}{1 + a T_h N_t} \tag{21}$$

and does not directly represent the per-contact transmission probability. Therefore, when  $a' \rightarrow 0$ , the value of the density-dependent  $\beta$  is likely to be much smaller than the frequency-dependent  $\beta$  (when  $a' \rightarrow 1$ ) for any given per-contact transmission probability,  $\delta$ . This also leads to the  $\beta$ 's used in the two formulations having different units, as pointed out by Thrall et al. (1995). In the pure frequency-

dependent formulation, to calculate  $\beta$ , the per-contact transmission probability should be multiplied by  $aT/T_h$ , while in the density-dependent formulation, it should be multiplied by  $aT$ .

It has been remarked that "the measurement of the 'force of infection' is, in principle, easy. In contrast, direct measurement of 'beta' is essentially impossible for most infections" (Anderson and May 1991, p. 63). One reason for this may be that the quantity  $\beta$  is a highly composite measure that is itself a function of the density and frequency of infectives. Our analyses show that, given knowledge of contact number, the per-contact transmission rate, and the parameters that define the functional response of the infected individual, it should be relatively straightforward, in principle, to predict the force of infection, making recourse to the parameter  $\beta$  superfluous in both theoretical and empirical studies.

Parallels have also been drawn between a heterosexually transmitted disease and a two-species system with alternative hosts and vectors (Braun 1983). However, in classic STDs, the transmission process differs from that in vector-transmitted diseases because the vector itself is the host, and there is no pathogen-loading phase. Moreover, although the sex ratio may be affected by disease status (Zuk 1990), such differential susceptibility does not affect the primary sex ratio (Fisher 1930). Therefore, in an STD, the relative abundances of host and vector (i.e., the alternate sex) remain equal regardless of the dynamics.

The transmission process in vector-borne diseases is likely to depend in complex ways on both host and vector behavior. In the present study, we have encapsulated these complexities in a simple, but general, model. This model confirms that the assumption of pure frequency-dependent transmission in vector-borne diseases is unlikely to be correct (Getz and Pickering 1983). If the vector is not very vagile (i.e., small), then, as seems intuitively reasonable, disease transmission conforms to a density-dependent pattern: the denser the population, the easier it is for a nonvagile vector to move between hosts. If the vectors are large and vagile, they can compensate for increased host density by greater migration, and the risk of infection would be proportional to  $I/N^2$ . Assuming vector abundances are not dynamically coupled to host abundances, this would generate a functional response that initially increases with total population density and declines at higher densities. There is some empirical support for this scenario. It has been shown (Antonovics and Alexander 1992) that in experimental populations in which frequency and density of infectives were varied independently, spore deposition per flower decreased with increasing density. A nonlinear regression plot using the model in equation (20) provided a reasonable qualitative description of the data (fig. 2). Unfortunately, pollinator behavior was not studied, and per-encounter transmission rates are unknown; therefore, it is not possible to test estimated model parameters against independently obtained empirical estimates of those parameters.

There has been a multiplicity of formulations dealing with the disease transmission process. These have been based either on diverse assumptions and notation or on notation (as with the transmission parameter  $\beta$ ) that is identical even though it refers to subtly different processes. The present study has shown that vector-borne, sexual, and parasitoid transmission can be placed under a unifying concep-

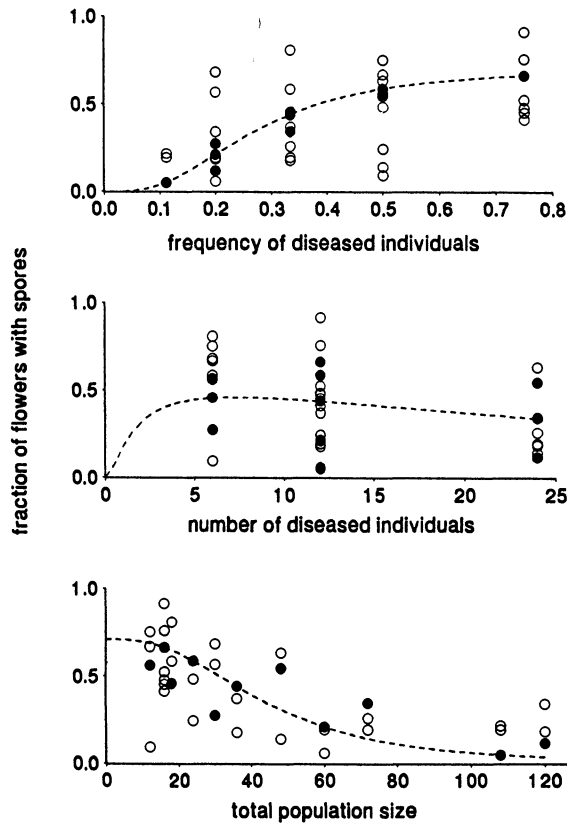


FIG. 2.—Graphs showing the relationships between spore deposition and frequency of diseased plants, density (number) of diseased plants, and total population size. Open circles indicate experimental data from an earlier study (Antonovics and Alexander 1992), solid circles indicate values predicted with the model of vector transmission with no refractory period (eq. [20]), and dotted lines indicate the predicted functional form of the relationships for specific numerical values. The values presented are the percentages of flowers with spores deposited on them (number of flowers sampled per plot = 5–22) in plots set out for 1 d with a given frequency and density of diseased plants. The date on which a plot was set up had a large effect on spore deposition, so the effect of date was removed by means of a weighted regression of the percentage of flowers with spores per plot (arcsine square-root transformed) against date; adjusted values were then back-transformed for presentation. The SAS (SAS Institute 1985) nonlinear regression procedure, NLIN (with the Gauss-Newton iterative method), was used to obtain parameter estimates for the model ( $K = 0.739$ ,  $aT_h = 0.045$ ; see eq. [20]) and to obtain predicted plot values. The number of predicted values is less than the number of observed values because the latter usually included two replicate plots for each frequency and density combination (see Antonovics and Alexander 1992 for details). The predicted functional relationships for frequency of diseased plants and total number of plants were calculated with the assumption of a constant density of diseased plants of 12; for numbers of diseased plants, the functional relationship was calculated with the assumption of a constant frequency of 0.33. These constant values represent intermediate values of the density and frequency treatments used in the experimental study.

tual umbrella in which their differences and similarities are seen as part of a continuum. Characterization of the transmission process in a conceptually unified manner is essential for comparative studies on how the transmission process affects population dynamics and for studies on the evolution of different transmission modes.

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