Epidemiology and optimal foraging: modelling the ideal free distribution of insect vectors

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SUMMARY

Existing models of the basic case reproduction number (R_0) for vector-borne diseases assume (i) that the distribution of vectors over the susceptible host species is homogenous and (ii) that the biting preference for the susceptible host species rather than other potential hosts is a constant. Empirical evidence contradicts both assumptions, with important consequences for disease transmission. In this paper we develop an Ideal Free Distribution (IFD) model of host choice by blood-sucking insects, predicated on the argument that vectors must have evolved to choose the least defensive hosts in order to maximize their feeding success. From a re-analysis of existing data, we demonstrate that the interference constant, m, of the IFD can vary between host species. As a result, the predicted distribution of insects over hosts has 2 desirable and intuitively plausible behaviours: that it is heterogeneous both within and between host species; and that the intensity of heterogeneity varies with host and vector density. When the IFD model is incorporated into R_0, the relationship with the vector:host ratio becomes non-linear. If correct, the IFD could add considerable realism to models which seek to predict the effect of these ecological parameters on disease transmission as they vary naturally (e.g. through seasonality in vector density or host population movement) or as a consequence of artificial manipulation (e.g. zooprophylaxis, vector control). It raises the possibility of targeting transmission hot spots with greater accuracy and concomitant reduction in control effort. The robustness of the model to simplifying assumptions is discussed.

Key words: vector, behaviour, epidemiology, ideal free distribution, heterogeneity, basic case reproduction number.

INTRODUCTION

The pattern of contract between blood-sucking insects and their host animals is extremely heterogeneous: while most hosts are bitten relatively infrequently, a subset is generally heavily attacked. Quantifying the effect of patchiness using empirical data on the biting distribution of vectors of malaria and leishmaniasis, Woolhouse *et al*. (1997) estimated that on average 20% of any host population is contributing 80% of the net transmission potential. In other words, the heterogeneous distribution of vectors tends to create transmission 'hot' and 'cold' spots. Heterogeneous biting rates are therefore of considerable practical importance. If the pattern of biting is not predictable, and vector control is necessarily blanket, then the more heterogeneous the distribution the more critical it is that transmission hot spots are not overlooked, and the greater is the control effort required. Conversely, if the pattern of biting is predictable, then vector control can be targeted at the hot spots, and the more heterogeneous the distribution, the smaller the control effort required.

The classic measure of infectious disease transmission is the basic case reproduction number, R_0. For vector-borne diseases, R_0 models key features of vector and parasite ecology to predict the number of secondary cases arising over the infectious life-time of a single primary case in a population of susceptible hosts,

$$R_{0,x}=\frac{N(a\cdot h_x)^2 bcp^n}{H_x(-\ln p)r} \quad (1)$$

This equation, commonly referred to as the Ross–Macdonald model (Ross, 1911; Macdonald, 1957), was first derived for *Plasmodium* malaria transmitted between humans by *Anopheles* mosquitoes, and is still commonly used today. In this version, N denotes vector abundance, H_x denotes the abundance of the susceptible host population x, a is the daily biting rate of a vector, h_x is the proportion of bites by vectors on the susceptible host species, b is the per-bite probability that an uninfected vector acquires an infection from an infectious host, c is the per-bite probability that an uninfected host acquires an infection from an infective vector, p is the daily survival rate of the vector, n is the parasite extrinsic incubation period in days (i.e. the period from parasite ingestion to infectivity in the vector) and r is the daily recovery rate of infected hosts. When $R_0>1$, infection will persist once introduced, initially generating an increasing number of cases (the host population is initially fully susceptible);

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when $R_0 < 1$, the number of cases will fall to extinction. This last is the ultimate goal of vector control.

In the basic model of R_0, the preference of mosquitoes (or other disease vectors) for their hosts is dealt with in 2 stages. Firstly, the term h_x represents a fixed probability that an individual mosquito bites a human rather than any of the other available host species. The term is squared to account for the fact that at least 2 mosquito bites are required: one to acquire parasites, another to transmit them. This makes host choice between species particularly influential on R_0. Secondly, N/H_x describes a completely homogenous distribution of mosquitoes between all of the available human hosts (*not*, as is sometimes stated, a random distribution).

Treating preferences as fixed between species, and equal between individuals within species, ignores a large body of empirical evidence attesting to the contrary. Two examples illustrate the point. Firstly, in an elegant study in Tanzania, Knols *et al*. (1995) found that between 6 people conducting man-landing catches, mean individual catches showed consistent and significant differences in attractiveness to *An. funestus* and *An. squamosus*, and that the catches of these two species were inversely correlated, suggesting that they were responding to different cues. Secondly, in Argentina, Gurtler *et al*. (1997) found that the proportion of Chagas disease vectors, *Triatoma infestans*, feeding on humans as opposed to chickens or dogs decreased as the density of vectors on the host increased.

In response to these obvious discrepancies between model predictions and field data, in the late 1980s several workers developed more detailed mathematical descriptions of host choice that incorporate heterogeneous biting rates, and variable host preferences, both within and between host species (Dye & Hasibeder, 1986; Kingsolver, 1987; Hasibeder & Dye, 1988). However, these models concentrate on finding the best mathematical fit for an observed demographic pattern without considering the behavioural motivations that drive it. Without a theory to underpin them, extrapolation to make epidemiological predictions under novel ecological conditions is unreliable if not impossible. This is a critical short-coming in models which seek to inform thinking on vector control strategy, and one which is a conceptual block to further progress.

In the wider ecological literature of the 1980s and 1990s there has been a move to incorporate individual behaviour into models of animal population dynamics, with concomitant improvements in their predictive power (see, for example, Hassell & May, 1985; Lomnicki, 1988; Abrams, 1993; Sutherland, 1996). This approach looks beyond the proximate determinants of behaviour: ultimately, the behaviour of individual animals is motivated by the evolutionary pressure to maximize life-time fecundity. Until now these theoretical advances have not been applied to the population ecology of blood-sucking insects – that is the aim of this paper.

METHODS – MATHEMATICAL MODELS

The Ideal Free Distribution

Haematophagous insects (and other arthropods) depend on blood as a source of protein for egg production. Therefore fecundity is very closely linked to the amount of blood imbibed (e.g. Ready, 1979 for the sandfly *Lutzomyia longipalpis*, Takken, Klowden & Chambers, 1998 for the mosquito *Anopheles gambiae*). Volume for volume, the value of blood as a source of protein varies little between hosts (e.g. Ready, 1979; Briegel, 1985 for the hosts of the mosquito *Aedes aegypti*; Moloo *et al*. 1988 for hosts of the tsetse fly *Glossina morsitans morsitans*). In contrast, the absolute volume of blood that an individual insect obtains (what we subsequently call feeding success) can vary considerably from host to host. Webber & Edman (1972), for example, found that the proportion of *Culex nigripalpis* mosquitoes which obtained a bloodmeal varied from approximately 82 to 8%, depending on the species of ciconiiform bird on which they fed.

Feeding success depends principally on the defensive behaviour of the host animal: the more defensive the host, the more likely it is that a biting insect will be interrupted before it has fed to repletion. Studies with sandflies (Kelly, Mustafa & Dye, 1996), mosquitoes (e.g. Webber & Edman, 1972), horseflies (Waage & Davies, 1986), tsetse flies (e.g. Vale, 1977) and reduviid bugs (Schofield, 1982) all show that host defensiveness varies as a function of the intensity of biting: the greater the density of insects biting an individual host over a given time-period, the more defensive the host becomes. In the laboratory, density-dependent host defensiveness has been shown to drive both of the phenomena that we are interested in: heterogeneity in feeding rate between individual hosts (Anderson & Brust, 1997) and shifts in net host preference with changing biting density (Nelson *et al*. 1976).

Using Brazilian field census data from chicken sheds, Kelly *et al*. (1996) modelled density-dependent feeding success for the sandfly *L. longipalpis* on chickens as

$$G_i = \frac{Q_i}{N_i^{m_i}}. \quad (2)$$

This is directly analogous to Hassell & Varley's (1969) interference model for the searching efficiency of an insect parasitoid. The key features of this model are that the *per capita* blood gains, G_i, of a sandfly on a host, i, of a given intrinsic defensiveness, Q_i (defined as the feeding success on the host when $N_i = 1$), fall with increasing vector biting density, N_i.

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The rate of fall is a function of the interference constant, m_i. Thus were $1 > m_i > 0$, feeding success would depend on the density of biting vectors per host, and decay asymptotically to zero as vector density increases. Values greater than 1 imply an acceleration in the rate of increase in defensiveness with each bite, which is obviously unsustainable: at some point host defensiveness must reach a maximum. Biologically, it seems more likely that a host responds less to each new bite as the total number of bites sustained over a given time-period increases.

By regression analysis of a log-linear version of Equation (2), Kelly *et al*. (1996) estimated Q_i to be 0·966 (S.E. 0·083) (defined as the proportion of chicken-feeding sandflies which had taken full bloodmeals) and m_i as 0·69 (S.E. 0·053), with an explanatory power of 75%. At the highest natural sandfly densities, feeding success was reduced by up to 80%. In the only other study of this kind, feeding success of the mosquito *Culex tritaeniorhynchus* fell from over 90% to less than 10% as mosquito densities in cow sheds varied naturally from tens to thousands (Fujito *et al*. 1971).

These data argue for an intense evolutionary pressure for individual blood-sucking insects to maximize feeding success by optimizing their distribution over hosts. From Equation (2) it is clear that an individual blood-sucking insect can improve its feeding success by choosing a host with a higher intrinsic quality, a lower interference constant and a smaller number of blood-sucking competitors. Sutherland (1983) calculated the optimal distribution of a foraging animal under the pressure of conspecific interference. Interpreted with respect to blood-sucking insects, assuming m to be constant for all j hosts (an assumption which we challenge later), the Evolutionarily Stable Strategy for the proportion of an insect population, h_i, feeding on an individual host animal, i, is

$$h_i = \frac{Q_i^{1/m}}{\sum_j Q_j^{1/m}}. \tag{3}$$

This is known as the Ideal Free Distribution (IFD) (Fretwell & Lucas, 1970, reviewed and extended by Sutherland, 1996). The following models explore the epidemiological consequences of using IFD estimates of host choice.

The Ideal Free Distribution model of vector-borne disease epidemiology

The model makes the following assumption. (1) A vector population, N, feeds upon a host population, H, comprising 2 species, z: species H_x, which is susceptible to the parasite, and species H_y, which is not. (2) Within each host species, or defined subdivision thereof, the determinants of defensiveness – Q and m – are constant for all individuals. Therefore all members of the vector species have equal competitive abilities: all other things being equal, all individual vectors provoke an identical defensive response from the host. (3) All vectors in the population N can choose instantaneously between all hosts in the population H_z. There are therefore no travel costs associated with host-seeking by the vector. (4) For every individual host animal, i, all vectors have perfect knowledge of the innate defensiveness, Q_i, the interference constant, m_i, the density of vectors on the host, N_i, and therefore the actual defensiveness and value as a blood source of each host animal at any one time. They are therefore equipped with sufficient information to choose a host according to IFD (Equation 3). (5) No other species of biting insect is present. (6) The blood resource of all hosts is identical and inexhaustible. (7) The vector- and parasite-associated parameters a, b, c, p, n and r of the basic case reproduction number are constant.

Analysis of previous host choice models (e.g. Rogers, 1988; Van Buskirk & Ostfeld, 1995; Lord *et al*. 1996) with respect to the IFD model reveals two important assumptions. For example, Sota & Mogi (1989) (Equation 4) modelled biting preference for species x as

$$h_x = \frac{k_x H_x}{\sum_z k_z H_z}, \tag{4}$$

where the constant, k_x, is the innate host preference, defined as the instantaneous rate at which a single blood-sucking insect finds and then successfully bites an individual host of species x, and takes different values for each host species, z. The equivalent IFD model, from Equation (3) is

$$h_x = \frac{Q_x^{1/m} H_x}{\sum_z Q_x^{1/m} H_x} \tag{5}$$

Given the assumption of instantaneous choice between hosts we can set $k_x = Q_x$ and generally, $k_z = Q_z$. Thus Equation (4) is equivalent to Equation (5) only under the assumptions (i) that $m = 1$ and (ii) that m is constant for all host species.

In fact, there is no biological argument for setting m equal to 1. As already discussed, Kelly *et al*. (1996) directly measured m as 0·69 for *L. longipalpis*, and at the same time made a significantly different estimate of $m = 0{\cdot}11$ (S.E. 0·03) for a pooled estimate of mosquito species.

There is also no reason why m should be constant for all species. It is easy to imagine circumstances under which the defensive response of 1 host species (or indeed individuals within species) increases more rapidly than that of another. In a study unique in vector ecology, Fujito *et al*. (1971) measured the *per capita* feeding success of *C. tritaeniorhynchus*, a vector of Japanese Encephalitis virus (JE), biting at

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Table 1. Estimate of innate host defensiveness, Q, and the interference constant, m, for *Culex tritaeniorhynchus* feeding on cattle and pigs

(Data analysis in GliM as proportion blood−fed versus mosquito density in animal pens (pig or cow), using binomial errors and the logit link function. Data taken from Fujito *et al*. (1971).)

	Host species		
Parameter estimate	Pig	Cow	adj. $\chi^2(p)$
Q	0·46	0·58	0·53 ($>0·05$)
m	0·11	0·46	10·99 ($<0·001$)

different densities in pig and cattle sheds. A new analysis of these data yields significantly different estimates of m for each host species (Table 1), demonstrating empirically that m is not constant.

Allowing m to vary between host classes, the functional response for host class x becomes (cf. Eqn 2)

$$G_x = \frac{Q_x}{N_x^{m_x}}. \tag{6}$$

There is no analytical solution to this equation for the aggregative response if N on all host classes z is unknown (cf. Eqn 3). Rather, the IFD can be arrived at iteratively using an extension of the method described by Sutherland (1996, Chapter 13). Alternatively, it is possible to calculate the IFD number of insects on any and all host classes in terms of the number of insects on any one particular host class. If, for example, we know the number of malaria mosquitoes biting an individual human ($N_{x,i}$), then the number biting an individual cow ($N_{y,i}$) that keeps mosquito feeding success on both host species equal will be

$$N_{y,i} = \left(\frac{Q_y}{Q_x}\right)^{1/m_y} N_{x,i}^{m_x/m_y}. \tag{7}$$

In practical terms the need to know $N_{x,i}$ is not a serious drawback to the model: human and animal landing catches are commonly used to assay vector abundance, and such statistics could be substituted directly into Equation (7). Defining N, the total number of vectors in the 2-host species system, as

$$N = N_{x,i}H_x + \left[\left(\frac{Q_y}{Q_x}\right)^{1/m_y} N_{x,i}^{m_x/m_y} H_y\right] \tag{8}$$

we can write host preference as

$$h_x = \frac{N_{x,i}H_x}{N} = \frac{N_{x,i}H_x}{N_{x,i}H_x + \left[\left(\frac{Q_y}{Q_x}\right)^{1/m_y} N_{x,i}^{m_x/m_y} H_y\right]} \tag{9}$$

Equation (9) can be substituted into Equation (1) to estimate the impact of the full IFD model of species preferences with varying values of Q and m. The model could be extended to incorporate more host species, requiring only that Q and m be measured for each.

RESULTS

We test the effect of the 2 assumptions, that $m=1$ and m is constant for all hosts, on the relationship between R_0 and host and vector abundance.

When $m \neq 1$ *but constant for all host species*

Substituting Equation (5) into Equation (1) the value of $R_{0,x}$ for a range of values of m depends on the relative values of Q for the susceptible and non-susceptible host species. Standard formulations of R_0 can also produce a non-linear relationship with host abundance in an explicitly 2-host system ($m=1$, Fig. 1A, B), but this can be modified considerably as m departs from 1.

As m increases, the power $1/m$ tends to zero and the relationship between host abundance and R_0 becomes less non-linear. As m tends to infinity, the values of all $Q_z^{1/m}$ approach 1 and $Q_x^{1/m} = Q_y^{1/m}$ (solid line, Fig. 1A, B). However, under the biologically realistic assumption that $m<1$, the IFD model makes the departure from linearity more extreme as m decreases from 1 (Fig. 1A, B).

The result is either an underestimate of $R_{0,x}$ (Fig. 1A) or overestimate of $R_{0,x}$ (Fig. 1B), depending on the relative values of Q_x and Q_y. Perhaps more importantly for control, the shape of the relationship between $R_{0,x}$ and relative host abundance is more extreme the further m falls below 1. Thus the success of an intervention which seeks to manipulate the relative abundance of non-susceptible hosts (i.e. zooprophylaxis) would depend critically on the values of Q and m. When $Q_x > Q_y$ (Fig. 1A), the smaller the value of m the greater the relative abundance of non-susceptible hosts which must be achieved before there is a significant impact on transmission. Conversely, if $Q_x < Q_y$ (Fig. 1B), the smaller the value of m, the smaller the relative abundance of non-susceptible hosts which must be achieved.

When $m_1 \neq m_2 \neq m_i \neq 1$

It is evident from Equation (5) that when m is held constant for all host classes, host preference is independent of overall vector density, N. In contrast, when m varies between host species, host preference will be sensitive to the abundance of both hosts *and* vectors (Eqn 9). Changing vector:host ratios can result in an increase or decrease in biting preference for a particular host species, and may even drive a

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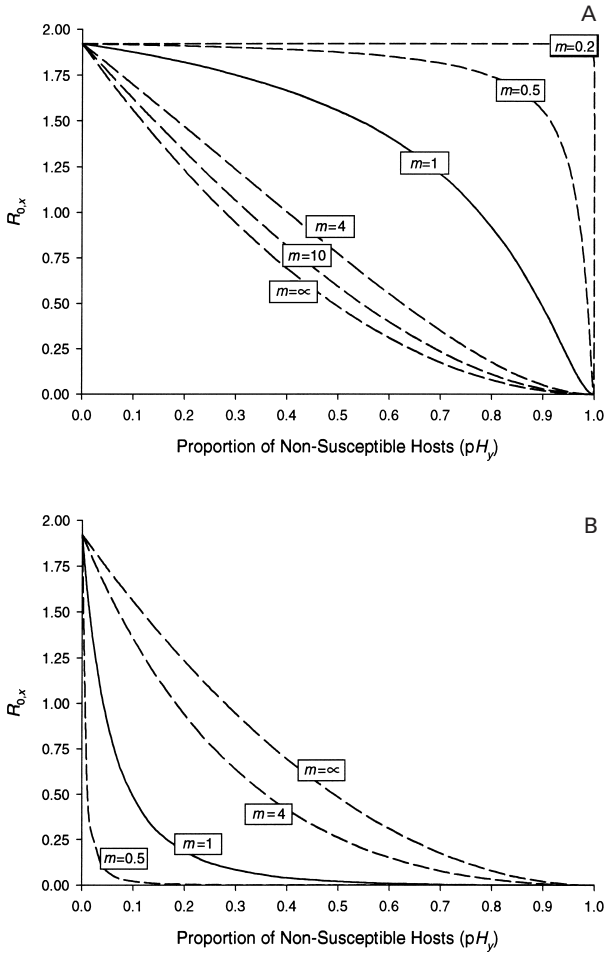

Fig. 1. The effect of the value of $m=1$ (——) and $m \neq 1$ (– – –) on the relationship between the basic case reproduction number of a vector-borne disease affecting species x ($R_{0,x}$) and the relative abundance of the non-susceptible host species y (H_y). (A) When $Q_x > Q_y$. (B) When $Q_x < Q_y$. Model parameters: (A) $Q_x=0{\cdot}9$; $Q_y=0{\cdot}1$. (B) $Q_x=0{\cdot}1$; $Q_y=0{\cdot}9$. Other model parameters: $N=10\,000$; $H_x=100$; $a=0{\cdot}333$/day; $b=0{\cdot}5$; $c=0{\cdot}5$; $n=3$ days; $p=0{\cdot}9$/day; $r=0{\cdot}1$/day.

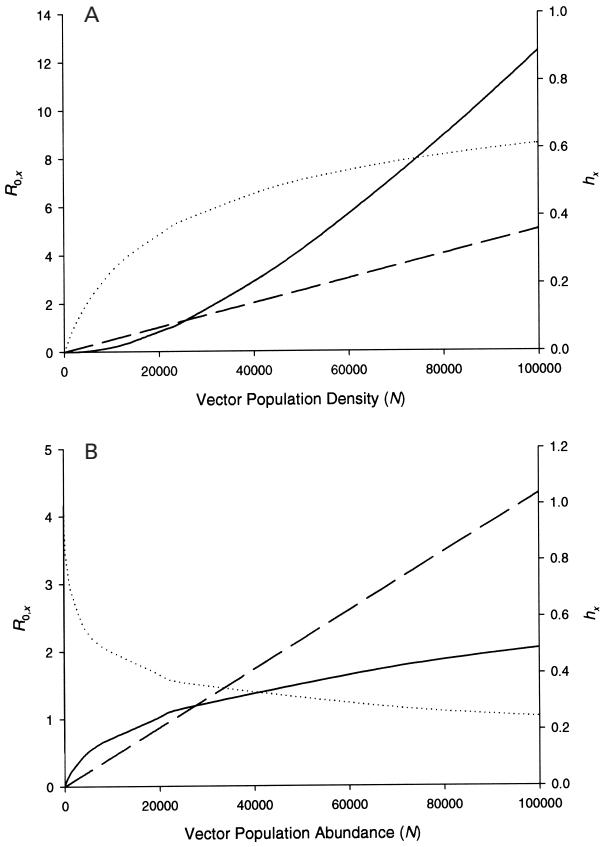

Fig. 2. The relationship between the basic case reproduction number (R_0, ——, – – –) and host preference (h_x, ……) and vector density (N). R_0 is calculated either using the IFD model of host choice (——) or with fixed host choice (– – –) taken to be the value of h_x calculated from the IFD model when $N_{x,i}=100$ (indicated by the crossover point of IFD and non-IFD lines). Model parameters: $a=0{\cdot}33$/day; $b=0{\cdot}5$; $c=0{\cdot}5$; $n=3$ days; $p=0{\cdot}9$/day; $r=0{\cdot}1$/day; $H_x=100$; $H_y=100$. (A) $Q_y=0{\cdot}9$; $Q_x=0.1$; $m_y=0.8$; $m_x=0{\cdot}4$. (B) $Q_y=0{\cdot}3$; $Q_x=0{\cdot}9$; $m_y=0{\cdot}5$; $m_x=0{\cdot}8$.

switch in net host preference from one species to another.

The change in host preference with vector density translates into a non-linear change in R_0. The presence and shape of the non-linearity depends upon the power term $N_{x,i}^{m_x/m_y}$. When the two values of m are the same, $m_x/m_y=1$, $N_{x,i}^{m_x/m_y}$ becomes $N_{x,i}$ and there is no non-linearity. When $m_x > m_y$, $m_x/m_y > 1$ and h_x increases with N, resulting in a faster than linear increase in R_0 (Fig. 2A). However, when $m_x < m_y$, $m_x/m_y < 1$ and h_x decreases with increasing N, causing R_0 to increase asymptotically (Fig. 2B). In these cases, where $m_x \neq m_y$, the rate of change in h_x with N – positive or negative – decays exponentially to zero as $N_{x,i}^{m_x/m_y}$ becomes infinitely large or vanishingly small. At extreme values of N, therefore, the change in h_x becomes simply the direct and linear effect of N on R_0 (Eqn. 1).

From a practical perspective, the IFD model of host choice predicts either greater or lower values for $R_{0,x}$ than the static model, depending on the relative magnitudes of m_x and m_y (Fig. 2A, B). It is important to note, again, that the non-linearity in the IFD model predicts that more (Fig. 2A, $m_x > m_y$) or less (Fig. 2B; $m_x < m_y$) vector control effort is required for the same reduction in R_0 than is the case under non-IFD assumptions.

The disparity between IFD and non-IFD models is also very sensitive to the prevailing vector density at the point when the sort of 'snapshot' value of h_x typically used in estimates of R_0 is measured (Fig. 3), such as from blood indices or host-choice experiments. Since h_x increases asymptotically, extrapolation of R_0 to novel conditions of host/vector density based on snapshot measurements of host preference are least inaccurate when taken under conditions of high vector density. Also of practical concern, the IFD model predicts that the man-landing rate, which is an empirical measure of the term Nah/H and used as a linear index of R_0, is in fact non-linear.

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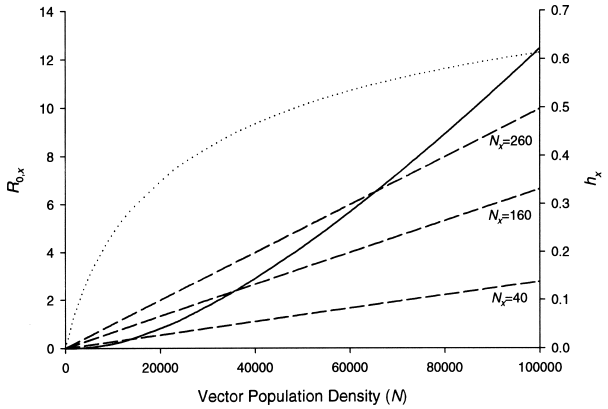

Fig. 3. The effect of estimating h_x at different vector densities on the non-IFD value of R_0 (– – –). The IFD relationship between the vector population density, N, and host preference, h_x, is calculated using Equation 11, for the parameter values: $Q_y = 0{\cdot}9$; $Q_x = 0{\cdot}1$; $m_y = 0{\cdot}8$; $m_x = 0{\cdot}4$. The IFD value of h_x is taken for $N_x = 40$, 160 and 260. These values of h_x are used to calculate 3 trajectories for R_0 versus N, assuming that h_x in each case is invariant (Eqn 1). The IFD relationship between R_0 and N (——) and the IFD value of h_x (····· line) are plotted for comparison. The point of intersection between the IFD and non-IFD values of R_0 represent the point when N corresponds to the value of N_x at which h_x was measured. Other model parameters: $a = 0{\cdot}333$/day; $b = 0{\cdot}5$; $c = 0{\cdot}5$; $n = 3$ days; $p = 0{\cdot}9$/day; $r = 0{\cdot}1$/day.

DISCUSSION

The IFD model of host choice captures, *a priori* from evolutionary theory, 2 desirable and intuitively plausible behaviours: that the distribution of vectors over hosts is heterogeneous, and that the intensity of heterogeneity can vary with host and vector density.

Since the level of interference depends in part on a power relationship with the density of other flies feeding on the individual host, the novel impact of IFD on parasite transmission is that R_0 can vary *non-linearly* with changing vector : host ratios, potentially driven by changes in host and/or vector density. Such a relationship has not been identified from field data, but this is hardly surprising given that the non-linearity is most pronounced at low vector density when field data have the greatest associated error.

The non-linearity in the model can result in substantially different predictions of the effect of changing vector and host abundance on the value of R_0 compared with the standard model. If correct, the model allows us to understand much better the effect of these ecological parameters on disease transmission as they vary naturally (e.g. through seasonality in vector density or host population movement) or as a consequence of artificial manipulation (e.g. zooprophylaxis or vector control). For example, a conservative interpretation of Fujito's data (given the non-significant difference in Q_{pig} and Q_{cow}) is that the preference for feeding on pigs should increase with the biting rate of *C. tritaeniorhynchus*. To the best of our knowledge, data in the form necessary to test such a prediction have never been collected, yet pigs act as reservoir hosts for JE, so any density-dependent, and therefore seasonal change in feeding on pigs might have important epidemiological consequences.

However, the principal appeal of modelling heterogeneities in host preference remains the possibility of predicting and targeting transmission hot spots with greater accuracy. Hosts could then be targeted by, say, focal insecticide spraying or vaccination programmes (either anti-disease or transmission blocking), with potential savings in time and money and greater assurance that persistent transmission hot-spots have not been left untreated.

Of critical importance for their practical value, these models contain only a small number of parameters, readily estimated from field data with considerable explanatory power ($r^2 = 75\,\%$ in the study by Kelly *et al.* 1996). The parameters are also potentially applicable across the range of host–vector systems, despite the idiosyncrasies of host recognition in each system.

IFD within host species

The between-species IFD model (Eqn 9) still contains the assumption that vectors are distributed evently between hosts of the same species. In reality, there is a considerable literature on the variation in both attractiveness and defensiveness of individuals of the same species (though nothing, yet, that links the two). Intriguingly, many consistent variations in attractiveness between individuals (most notably humans, but also other animals) have been found to correlate with epidemiologically significant demographic groups, such as age (e.g. Boreham, Chandler & Jolly, 1978), sex (Rahm, 1958), body size (e.g. Port, Boreham & Bryan, 1980) and disease status (e.g. Day & Edman, 1983). The effect of varying preference by demographic group can readily be incorporated into the IFD model of host choice to estimate R_0 for any demographic group within a species. The result is an elegant model of host choice as a continuum from species to individuals, rather than as 2 disjoint processes.

Criticisms of the model

The IFD model of host choice is, of necessity, a simplification of the real situation. It is intended to form a starting point for gathering field data that test model predictions and lead to appropriate modification. However, the power of the model, and therefore its usefulness, depends not on vector populations being truly IFD, but on the strength and consistency of the response of vectors towards IFD, which determines the explanatory power of the estimates of m and Q.

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When detecting or arriving at a host, the optimal decision to feed or move on depends on whether there are better hosts available and whether the time and effort involved in moving outweighs the benefits. The 2 fundamental assumptions of the ideal free distribution are therefore that vectors have perfect knowledge of the quality of all the available hosts (hence 'ideal') and that travel between them costs nothing ('free'). Most insect vectors range over a limited area. However, by their very nature, for most vector-borne disease systems of medical and veterinary importance a variety of alternative hosts are available within a reasonable distance, often aggregated in houses and animal pens. Here, travel costs will be negligible, and information on the relative quality of available hosts will be easier for the vector to gather.

The method by which individuals in a population of vectors acquire information on host quality will fundamentally influence their ability to approximate the IFD, and an evolutionary approach to host choice should provide us with new ways of thinking about the proximate determinants. We propose 3 main mechanisms by which vectors might gather information on travel costs and host quality and approximate the IFD. (1) *Evolved host preferences*. Of the major olfactory attractants identified, adaptive roles can be proposed for many. For example, ketones (e.g. Torr, Hall & Smith, 1995, with tsetse flies) could allow flies to select malnourished individuals which are likely to be more passive. Attraction to lactic acid (Geier, Sass & Boeckh, 1996) could serve as a marker for physical exhaustion. Heat and humidity, important short-range cues in some mosquitoes (Eiras & Jepson, 1994) increase with fever and therefore with lassitude. (2) *Learnt host preferences*. There has been considerable work on learning in insects, including long-term memory in hymenopterous parasitoids that have learned to respond to specific visual and olfactory host cues (Turlings *et al*. 1993). Despite this work, medical entomologists remain sceptical of the evidence that vectors memorize home ranges or learn host preferences, as reported for mosquitoes (e.g. Charlwood, Graves & Marshall, 1988) and sandflies (Kelly & Dye, 1997). (3) '*Naïve*' *mechanisms*. Perhaps the most immediately credible method of host 'choice' towards the IFD is by displacement from more to less defensive host animals, and has been shown to drive switches in host preference under controlled conditions (Nelson *et al*. 1976). This could combine with the more sophisticated mechanisms, evolved or learnt, to minimize the risks of choosing the wrong host. For example, the so-called 'invitation pheromones' apparently produced by actively blood-feeding insects may provide cues which guide other host-seeking insects to the most amenable hosts (Kelly, unpublished observations).

Modelling the proximate mechanisms by which foraging vectors approximate the IFD would clearly improve the predictive power of the present model. Several theoretical studies have included learning and travel costs in models of avian foraging dynamics. In this scenario, as travel costs increase, knowledge of patch quality becomes imperfect and the metabolic costs of moving are greater, such that foragers become more sedentary and will settle in patches of poor quality (e.g. Bernstein, Kacelnik & Krebs, 1991).

Finally, we assume that only one blood-sucking insect species is present. Differences between vector species in the magnitude of m can be dramatic, as was shown for *L. longipalpis* (0·69) and a pool of mosquito species (0·11) feeding on chickens (Kelly *et al*. 1996). This makes biological sense: mosquitoes have piercing mouthparts, while sandflies are pool feeders with considerably more painful rasping mouthparts. Models of the IFD with such unequal competitors predict that in the richer resource patches there should be more foragers, of greater competitive ability, and this pattern has been shown in many field studies of bird populations (see Sutherland (1996), Section 2.4).

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