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On the evolutionary ecology of marking pheromones

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Summary

Many parasitic insects mark hosts with a pheromone after oviposition. The evolutionary ecology of such marking pheromones was studied to determine (i) under what ecological and behavioral conditions such pheromones could evolve and (ii) why so many of these marking pheromones are water-soluble and thus short-lived. We used a number of different techniques. First, the fitness values of individual normal (non-marking) and mutant (marking) insects foraging for hosts were computed using dynamic state-variable models. Second, population level models were used to study when a population of non-marking individuals can be invaded by marking individuals. Third, behavior-rich simulations (developed originally for apple maggot, *Rhagoletis pomonella*) were used to test 'experimentally' some of the hypotheses generated using the individual and population-level models. Finally, we developed a model for the 'benefit' over time to an individual by marking. This model shows that when benefit is measured in terms of larval survival, nearly all of the benefit to a mother is obtained from short-lived marks. Genetical theories of pheromone evolution and the connection between our results and existing theories of altruistic behavior are discussed.

Keywords: Pheromones; altruistic behavior; parasitic insects; *Rhagoletis pomonella*.

Introduction

Parasitic insects must make several decisions while searching for and exploiting hosts. These decisions include: (1) how long to continue searching within a host-containing patch (Morrison and Lewis, 1981); (2) directionality of search (Strand and Vinson, 1982); (3) how many offspring to commit to a host once one is discovered (Charnov and Skinner, 1985); and (4) what sex those offspring might be (for those parasites that can control offspring gender) (Waage and Ng, 1984). All four decisions contribute directly to parasite reproductive success; thus we expect that such decisions are strongly shaped by natural selection and that parasite decisions are optimal relative to the parasite's current information state. When such states vary, however, so might the decisions parasites make (Mangel and Roitberg, 1989).

Decisions 3 and 4 above are often highly dependent upon a parasite's ability to recognize the presence of conspecifics. Because hosts represent a limited discrete resource the fitness function for eggs/host decelerates and in many cases the optimal response to encountering an already parasitized host is rejection of that host (Iwasa *et al.*, 1984; Parker and Courtney, 1984; Mangel, 1987a,b). Such responses require that the parasite somehow assesses the previous presence of conspecifics, a phenomenon often referred to as host discrimination (van Lenteren, 1981). Host discrimination is, in fact, commonly observed in many parasitic insects, both the more traditional

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parasitic wasps and the taxonomically different but functionally similar plant parasites (*sensu* Price, 1977; van Lenteren, 1981; Prokopy, 1981).

Host discrimination is frequently mediated though employment of marking pheromones, chemical substances deposited by egg-laying females, that appear to function as display or epideictic (*sensu* Corbet, 1971) messages (Roitberg and Prokopy, 1987). And while substantial progress has recently been reported in both the determination of the chemical nature (e.g. Hurter *et al.*, 1987) and the response of individuals to these compounds (e.g. Hubbard *et al.*, 1987; Mangel and Roitberg, 1989) almost no one has considered how such information systems originated. We believe, however, that such a consideration is crucial to understanding how marking systems operate in contemporary time. In this paper, we attempt to explain how and why employment of marking pheromones by parasitic insects is likely to have evolved, apparently independently across several orders of insects (Prokopy, 1981).

Marking pheromones present an intriguing problem for evolutionary theory because they appear to function simultaneously at several levels. For example, while we have argued that females deposit marking pheromones to signal to themselves where their own offspring reside (Roitberg and Prokopy, 1987), others have suggested that these marks may function to prevent conspecifics from superparasitizing such occupied hosts: thus, the term oviposition-deterring pheromone is commonly employed (Prokopy, 1972). Some researchers have also suggested that these compounds allow parasite populations to utilize more efficiently host populations by mediating a more even spread of parasite offspring across those hosts (Bauer, 1986). Marking pheromones have also been termed kairomones (Nordlund, 1981) because on some occasions second-order parasites or hyperparasites employ these compounds to locate and parasitize parasite offspring. These examples illustrate the confusion surrounding the functional aspects of marking pheromones. Furthermore, some of the viewpoints are group selectionist. Clearly, an evolutionary assessment of how such communication systems arise and persist in nature is necessary to alleviate the confusion.

Our approach to understanding marking pheromone evolution is to elucidate those ecological and behavioral parameters necessary for successful invasion and maintenance of individuals that mark within insect populations. This approach is excellently described by Charnov and Stephens (1988):

> Our model does not deal with the genetics of evolution directly; like other life-history theorists, we suppose that natural selection has molded the decision process to maximize an appropriate measure of Darwinian fitness . . . we focus on the fitness measure, and on constraints or tradeoffs among its component parts. It is our opinion that such a focus will lead to an understanding more fundamental than, say, the insights gained by the currently popular 'polygenic' approach.

In this paper, we consider two scenarios that are likely to describe the majority of cases where marking pheromone systems have evolved. In the first scenario, we consider a population composed of 'normal' individuals, i.e. individuals that neither mark hosts nor recognize marked hosts. We then allow a 'double mutant' to enter the population. This mutant has the ability to both mark and recognize marks.

In the second scenario, we again consider a 'normal' population whose individuals do not overtly mark hosts. They do, however, sometimes leave behind information on their presence and activities (e.g. oviposition wounds that exude host fluids, wing scales, etc.). Since such 'weak' marks are sometimes produced, we assume that 'normal' individuals have evolved the ability to recognize those marks when present. Thus, 'normal' individuals employ an imperfect system (i.e. parasites do not always detect the presence of conspecific eggs) where mistakes are sometimes made. We then introduce a 'strong' marking mutant (e.g. one that spreads oviposition wound exudate over the surface of the host (e.g. *Dacus oleae*, Cirio, 1971)) into the population of

'normal' recognizers. In contrast to the first scenario, all individuals (i.e. both normals and mutants) will recognize the marks left by the mutants, but only the mutants produce 'strong' marks. Similarly, all individuals only sometimes recognize previous oviposition activities of normals (i.e. weak markers). This second scenario more likely describes the evolutionary process for many insects since: (i) it requires successive rather than simultaneous development of recognition and marking systems; and (ii) recognition machinery can be maintained in the absence of overt markers since there are still payoffs, albeit infrequent, for searching for and responding to weak marks.

The two scenarios provide feasible starting points for the evolution of marking behavior. We now ask what conditions are necessary for marking behavior to spread through the population. We employ three techniques to accomplish our aim:

1. **Individual fitness computations**. Here we calculate the relative fitness of mutant and normal individuals and thus the likelihood that mutant behavior will continue to spread through the population when present at different frequencies. That is, we determine ecological conditions under which the mutant individuals have higher fitness than the normal individuals.
2. **Population-level models.**. Here we consider changes in mutant frequencies across generations by employing classical foraging models that evaluate reproductive success of normals and mutants as a function of their oviposition rate and offspring survival.
3. **Behavior-rich simulations**. Here we employ detailed computer simulations of the foraging fruit-parasitic flies *Rhagoletis pomonella* to determine reproductive success of normals and mutants when present at different frequencies.

By identifying components critical for the successful invasion and then maintenance of host marking behavior we hope to provide a better understanding of the employment of those compounds by parasitic insects in contemporary time. Finally, we ask 'given that a population of individuals employs marking pheromones, how long should a mark on an individual host persist?' By answering this question we provide further support for our functional interpretation of benefits of host marking.

Individual fitness models: wasting eggs and wasting time

In this section, we show how the fitness of an individual parasite can be computed, using dynamic, state-variable models (McNamara and Houston, 1986; Mangel, 1987a,b; Houston *et al.*, 1988). Our approach is to compute the fitness of normal (weakly marking – scenario 2) and mutant (strongly marking) parasites as a function of various environmental parameters.

The parasitic fruit fly *Rhagoletis pomonella* may lay up to 10 or 11 eggs in a day and has about six hours for foraging for oviposition sites. Since each oviposition event takes only a few minutes, we begin by considering the case in which time is not a major constraint on the oviposition behavior. On the other hand, for a parasite that lays only 10 or 11 eggs, unintentional superparasitism may have a cost, depending upon the relative survivorship of successive eggs. We begin with the simple assumption that only one egg will survive in a host that has been superparasitized (Averill and Prokopy, 1987). This means, for example, that if two eggs are laid in near-simultaneity, then the expected fitness from either is about half the fitness expected if they were alone. If a female parasitizes a host that was previously attacked by a conspecific, then its egg is less likely to provide the same increment in fitness as if the host were clean (i.e. unparasitized). Even worse, if the female parasitizes a host that she previously parasitized, one of the two eggs is certainly wasted.

We model this situation in the following way. In a patch of S hosts, there are Π normal parasites

present and $\mathfrak{M}$ mutant parasites present. We assume that normal and mutant individuals detect fruit with equal capabilities. The normal individuals weakly mark hosts after oviposition, so that there is a probability p_d that a previously parasitized host is detected.

The mutant individuals strongly mark hosts after oviposition, so that a host parasitized by a mutant individual is detected with probability = 1. We assume that oviposition in a clean host provides an increment in lifetime fitness of f and that oviposition in a marked host provides an increment in lifetime fitness of $f'<f$. Finally we assume that if a parasite oviposits in a host that she previously parasitized, the increment in fitness from the second egg is zero. (This corresponds to 'wasting' an egg.)

Our objective is to compute the expected fitness accumulated during the course of foraging for hosts. To do this, we divided the parasite's lifetime into T 'foraging periods' and introduce state variables that characterize the parasite and the environment. Let $X(t)$ denote the egg complement of an individual at the start of period t. Let $N(t) = [N_0(t),N_1(t),N_2(t)]$ denote the state of the environment: $N_0(t)$ is the number of clean hosts at the start of period t, $N_1(t)$ is the number of hosts marked by normal individuals, and $N_2(t)$ is the number of hosts marked by mutant individuals.

We assume that the probability of encountering a host of type i during period t is given by:

$$\lambda_i = (1-e^{-\epsilon S})N_i/\Sigma_j N_j$$

where ϵ is a parameter characterizing the search effectiveness of the parasite. (See Roitberg (1985) for a discussion of search effectiveness of *R. pomonella*. Mangel (1985) provides a discussion of how ϵ might be estimated; basically random search for hosts is assumed.)

Let $F_n(x,n,t,T)$ denote the maximum expected fitness of a normal individual at the start of period T, based on oviposition decisions between t and T and given that $X(t) = x$, $N(t) = n$. No fitness after T (end of adult life) implies:

$$F_n(x,n,T,T) = 0$$

We let $\rho(t)$ denote the probability that a parasite survives to period $t+1$, given that she is alive in period t. To derive an equation for the fitness function, consider the following mutually exclusive events during period t when $X(t+1) = x$ and $N(t+1) = n = [n_0,n_1,n_2]$:

1. The parasite may not encounter a host of any type during period t. In this case $X(t) = x$ and $N(t) = n$.
2. The parasite may encounter a clean host. We assume that she always oviposits in a clean host, so that if one is encountered $X(t+1) = x-1$ and $N(t+1) = n_c = [n_0-1,n_1+1,n_2]$
3. The parasite may encounter a host that was parasitized by a mutant individual. In this case, the decision on whether or not to oviposit is made according to the behavior that maximizes her expected overall fitness. If she does accept the host, then $X(t+1) = x-1$, but the vector $N(t)$ does not change, since the mutant individuals mark more strongly than the normal individuals and the current host was first parasitized by a mutant individual.
4. The parasite may encounter a host that was parasitized by a normal individual and detect the previous parasitization. In this case, there is a probability $1/\mathfrak{N}$ that she parasitized the host previously and a probability $(\mathfrak{N}-1)/\mathfrak{N}$ that one of the other normal individuals parasitized the host.
5. The parasite may encounter a host that was parasitized by a normal individual, but not detect the previous parasitization. In this case, she treats the host as if it were clean and thus oviposits, but obtains fitness as in case 4.

These five cases are summarized in the following equation:

$$
\begin{aligned}
F_n(x,n,t,T) &= (1-\Sigma_i\lambda_i)\rho(t)F_n(x,n,t+1,T)\\
&\quad+\lambda_o\{f+\rho(t)F_n(x-1,n_c,t+1,T)\}\\
&\quad+\lambda_2\max\{\rho(t)F_n(x,n,t+1,T);f'+\rho(t)F_n(x-1,n,t+1,T)\}\\
&\quad+\lambda_1[p_d\max\{\rho(t)F_n(x,n,t+1,T);(1/\mathfrak{N})\rho(t)F_n(x-1,n,t+1,T)\\
&\qquad+((\mathfrak{N}-1)/\mathfrak{N})(f'+\rho(t)F_n(x-1,n,t+1,T))\}\\
&\qquad+(1-p_d)\{(1/\mathfrak{N})\rho(t)F_n(x-1,n,t+1,T)\\
&\qquad+((\mathfrak{N}-1)/\mathfrak{N})(f'+\rho(t)F_n(x-1,n,t+1,T))\}] \qquad (1)
\end{aligned}
$$

The five terms on the right-hand side of Eqn (1) correspond to the five mutually exclusive events listed above.

Let $F_m(x,n,t,T)$ denote the maximum expected fitness of a mutant individual, when $X(t) = x$ and $N(t) = n$. Once again, there are five mutually exclusive events. If $N(t) = n = (n_0,n_1,n_2)$ and a clean host is encountered, then $N(t+1) = n_c = (n_0-1, n_1, n_2+1)$; if a host marked by a normal individual is encountered and accepted, then $N(t+1) = n_m = (n_0,n_1-1,n_2+1)$, etc. Reasoning similar to the reasoning that leads to Eqn (1) leads to the following equation for $F_m(x,n,t,T)$:

$$
\begin{aligned}
F_m(x,n,t,T) &= (1-\Sigma_i\lambda_i)\ \rho(t)\ F_m(x,n,t+1,T)\\
&\quad+\lambda_o(f+\rho(t)F_m(x-1,n_c,\ t+1,\ T)\}\\
&\quad+\lambda_2\max[\rho(t)F_m(x,n,t+1,T);\ (1/\mathfrak{M})\ \rho(t)\ F_m(x-1,n,t+1,T)\\
&\qquad+((\mathfrak{M}-1)/\mathfrak{M})(f'+\rho(t)F_m(x-1,n,t+1,T))]\\
&\quad+\lambda_1[p_d\max\{\ \rho(t)F_m(x,n,t+1,T);\ (f'+\rho(t)F_m(x-1,n_m,t+1,T))\}\\
&\qquad+(1-p_d)\ (f'+\rho(t)F_m(x-1,n_m,t+1,T))\}] \qquad (2)
\end{aligned}
$$

These equations are solved 'backwards' in time, starting with $t = T-1$ and going until $t = 1$ (so-called 'backward induction').

Table 1 shows the results of computations using these equations. In this table, we consider the situation in which two of seven individuals in a patch of seven hosts are mutants and show the fitness of normal and mutant individuals for all possible starting combinations of clean and marked hosts. The most interesting case is the clean patch, corresponding to $n_1 = n_2 = 0$; this is the first line of the table. (If the mutant individuals do not have an adaptive advantage in a clean patch, then they are not likely to have an advantage in a partially parasitized patch.)

Other lines of the table show fitness values for different starting conditions in the patch. From a table such as this one, we can determine when the mutant individual has an adaptive advantage over the normal individual, in terms of expected fitness through oviposition in the patch. Similar results are obtained for other parameter values. For example, for parameters as in Table 1 except that probability of detection = $p_d = 0.5$ and $n^0 = (7,0,0)$ we find that $F_n(6,n^0,1,T) = 4.03$ and $F_m(6,n^0,1,T) = 4.17$. On the other hand, if the parameters are as in Table 1 except that the probability of surviving through period $t = \rho(t) = 0.4$, $f' = 0.1$, $p_d = 0.5$, then $F_n(3,n^0,1,T) = 1.47$ and $F_m(3,n^0,1,T) = 1.48$ when $n^0 = (7,0,0)$.

Table 2 shows how the fitnesses depend upon the number of normal and mutant individuals in the patch; holding the ratio constant at 10 normal to 1 mutant. We see that the relative fitness of the mutant individuals decreases as the density of parasites increases.

Figures 1–3 show how the fitnesses depend upon different environmental parameters, particularly the number of hosts in the patch, the probability of detection of a host marked by a normal individual, and the number of normal individuals. The conclusion that we draw from these figures is that the mutant may be at a competitive advantage over normal individuals when the probability of detection of oviposition by normal individuals is small, over a wide range of host and parasite densities. That is, as p_d increases, normal individuals become more 'mutant-like' in detection, but only mutants pay the cost. This robustness is exactly what is needed for the evolution of marking pheromone systems.

Table 1. Fitness of normal and mutant parasites. (Other parameters: $\mathfrak{N} = 5, \mathfrak{M} = 2, S = 7, T = 20, f_c = 1, f_m = 0.5, \rho(t) = 0.9, p_d = 0.1, \varepsilon = 100\,000.$)

n_1	n_2*	$F_n(6,n,1,T)$	$F_m(6,n,1T)$
0	0	3.96	4.17
0	1	3.73	3.75
0	2	3.52	3.33
0	3	3.29	2.91
0	4	3.06	2.48
0	5	2.82	2.05
0	6	2.59	1.61
0	7	2.34	1.17
1	0	3.66	3.87
1	1	3.44	3.45
1	2	3.21	3.03
1	3	2.98	2.60
1	4	2.74	2.17
1	5	2.51	1.74
1	6	2.28	1.30
2	0	3.36	3.57
2	1	3.14	3.15
2	2	2.91	2.73
2	3	2.67	2.30
2	4	2.44	1.87
2	5	2.21	1.44
3	0	3.07	3.27
3	1	2.84	2.85
3	2	2.60	2.43
3	3	2.37	2.01
3	4	2.14	1.57
4	0	2.77	2.98
4	1	2.54	2.56
4	2	2.31	2.13
4	3	2.08	1.71
5	0	2.47	2.68
5	1	2.24	2.26
5	2	2.01	1.84
6	0	2.17	2.39
6	1	1.94	1.97
7	0	1.8	2.10

*Once the values of n_1 and n_2 are given, n_0 is computed by $n_0 = 7 - n_1 - n_2$.

Table 2. Fitnesses of normal and mutant parasites in a 10:1 ratio (all parameters as in Table 1).

m	$F_n(6,n^0,1,T)$	$F_m(6,n^0,1,T)$	Ratio
2	4.118	4.2668	1.036
4	4.131	4.2669	1.033
6	4.135	4.2673	1.032
8	4.137	4.2697	1.032
10	4.139	4.2711	1.032

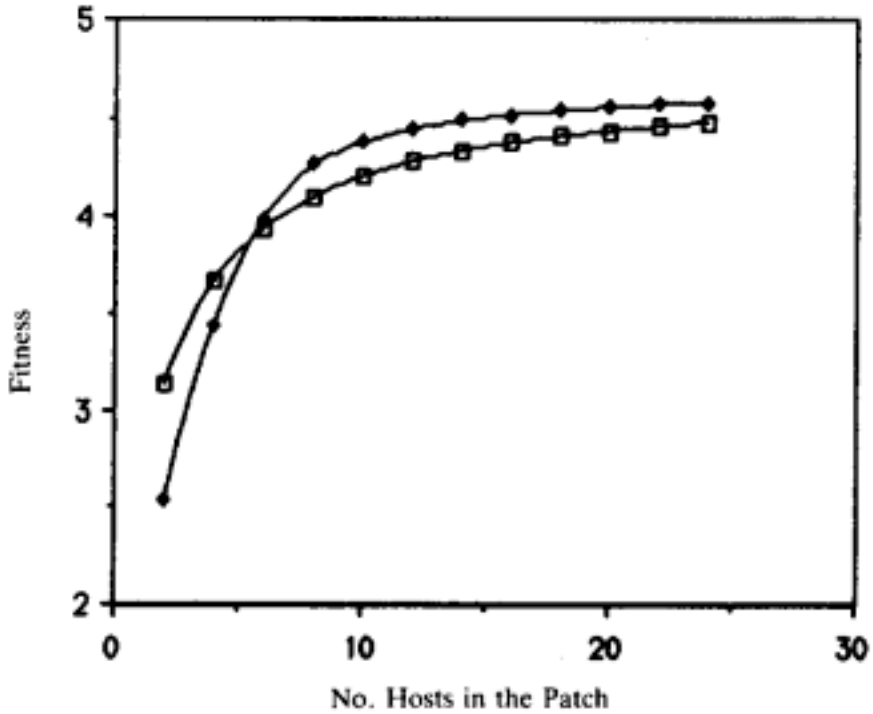


Figure 1. Fitness of normal and mutant parasites as a function of the number of hosts in the patch. Parameter values as in Table 1, except that $N = 14$, $M = 2$. The curve marked normal (⊡) gives $F_n(6,n^0,1,T)$ and the curve marked (◆) gives $F_m(6,n^0,1,T)$.

Let us now briefly consider the second situation, in which time may be more limiting than eggs. To do this, we consider three types of individual: non-marking parasites (normal) which simply oviposit and leave the host, weakly marking parasites which oviposit and leave a weak mark that is sometimes detected and strongly marking parasites which oviposit and leave a strong mark that is always detected. We assume that: (i) a parasite never re-encounters its own eggs; and (ii) marking uses up time that could be spent foraging. Because of the first assumption, a parasite never wastes an egg by ovipositing in a host that she already parasitized. Superparasitism can occur, however, with the concomitant decrease in fitness of the second egg. Because of the second assumption, the number of foraging periods in a day decreases with the strength of marking, since time is spent marking the fruit. Thus, if T_n, T_w and T_s respectively represent the time horizons for normal, weakly marking and strongly marking parasites, we have the relationship $T_n > T_w > T_s$. The equations for fitness through egg production can be derived in a manner similar to Eqns (1) and (2) and solved analogously. In this case, we also find that marking

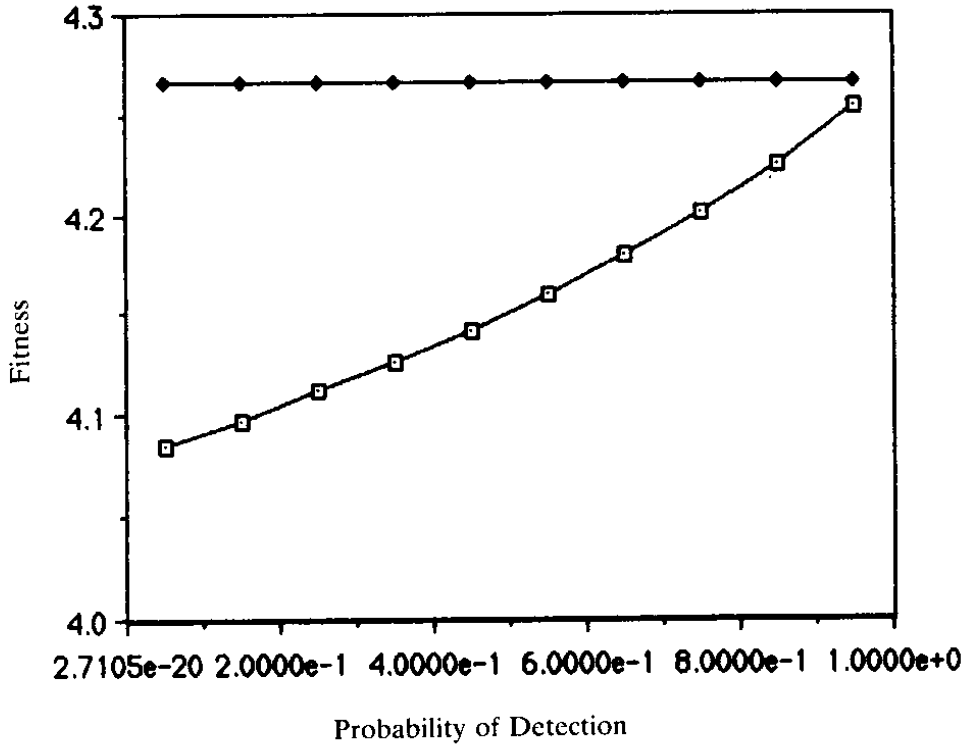


Figure 2. Fitnesses of normal and mutant parasites as a function of the probability of detection p_d of parasitization by a normal parasite. Parameter values as in Table 1, except that $N = 10$, $M = 2$, $S = 8$. The curve marked normal (⊡) gives $F_n(6,n^0,1,T)$ and the curve marked mutant (◆) gives $F_m(6,n^0,1,T)$

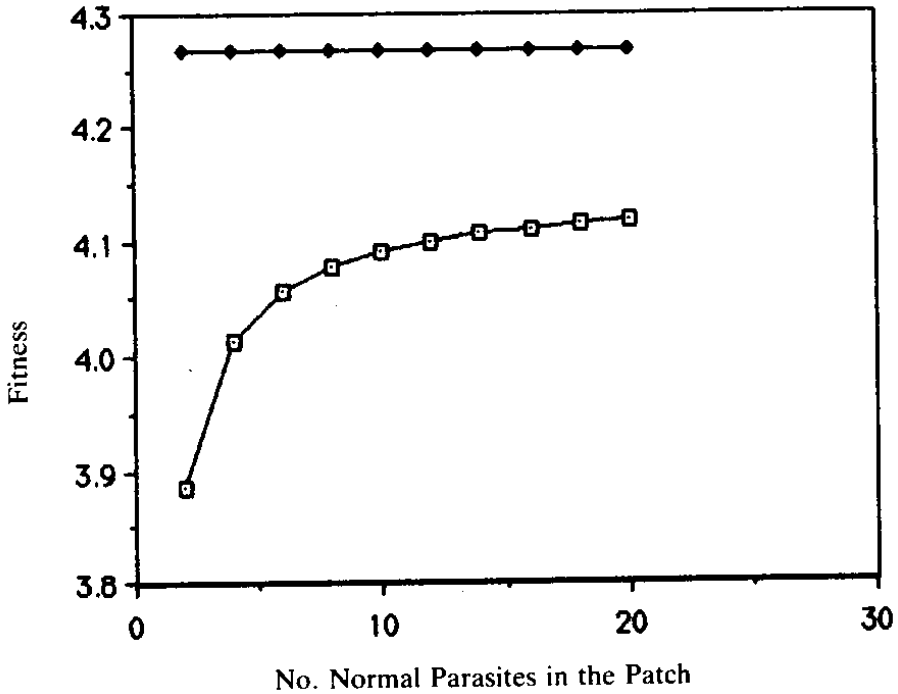


Figure 3. Fitnesses of normal and mutant parasites as a function of the number of normal parasites in the patch. Parameter values as in Table 1, except that $M = 2$, $S = 7$. The curve marked normal (⊡) gives $F_n(6,n^0,1,T)$ and the curve marked mutant (◆) gives $F_m(6,n^0,1,T)$.

can have an adaptive advantage. The markers typically have an adaptive advantage if the expected number of encounters with hosts exceeds the egg complement. In addition, computations show that whenever the stronger marker has an adaptive advantage relative to normal individuals, the weak marker also has an adaptive advantage. This last point has some bearing on our discussion of the lifetime of marking pheromones in the last section of the paper.

In summary, employment of state-variable models shows that the markers will out-perform non-markers when alternative hosts are available at sufficient densities.

Population-level models

In this section we employ models that differ from those discussed previously in that they are deterministic and focus more at the level of population or gene pool. Once again, however, we emphasize the role of individual behavior.

Scenario 1: Marker–recognition double mutants

Several hypotheses can be erected regarding both the origin and maintenance of host marking systems. The first considers the relative shortage of hosts available for exploitation. Under conditions where hosts provide limited food and/or shelter, per capita survivorship will be some decreasing function of larval crowding. Thus, individuals that spread their offspring evenly among hosts may be at an advantage in that, on average, their offspring are likely to face lower levels of competition. Such logic suggests that as resource availability declines, the advantage accrued to host-marking individuals and host recognizers should increase. To test this hypothesis we develop the following scenario. Consider a population of normal parasites that neither mark their hosts nor recognize the presence of conspecific (sibling or otherwise) eggs or larvae within hosts. Further, assume, as is common in many parasite systems, that, as a larva, the first egg deposited within a host kills any eggs subsequently deposited in that host (e.g. *Opius* sp. parasitoids – J. Nelson, pers. comm.; see Godfray (1987) for an excellent discussion of the evolution of larval fighting). Now assume a rare double mutant female (that both marks and recognizes the presence of marks) enters the population. The presence of such dual behavior means that marking individuals will avoid ovipositing in hosts that have been parasitized previously and marked by mutant individuals but will continue to oviposit in hosts containing normal eggs. By contrast, normal individuals will continue to oviposit indiscriminately in all hosts. Thus, while normal offspring compete with both mutant and normal larvae, mutants only compete with normals. To assess the likelihood that the host-marker mark-recognizer complex will invade a population of normals we track the reproductive success of mutant and normal females. If we assume that only one larva can survive within each host and that the older larva always kills the younger one, then reproductive success will be a function of the number of eggs laid in unparasitized hosts. In terms of offspring production and adult foraging success, four types of host must be considered:

N_0 = number of unparasitized (clean) hosts.

N_1 = number of normal hosts. The host contains at least one normal and no mutant eggs. Such hosts are susceptible to oviposition by both mutants and normals but will produce a single normal adult in the next generation.

N_2 = number of mutant hosts. The host contains at least one egg and the oldest one is a mutant. Such hosts are susceptible to oviposition by normals only and will produce a single mutant adult next generation.

N_3 = number of normal-first hosts. The host contains at least one of each of normal and mutant

eggs and the normal egg is the oldest. Such hosts are susceptible to oviposition by normals only but will produce a single normal offspring.

Finally, we assume that host resources become available over a very short period of time (e.g. fruit crop ripens simultaneously; larvae become susceptible to parasitism during a narrow window of vulnerability). Thus, the probability of invasion by the marker complex depends on the proportion of N_0 hosts that are converted to mutant hosts, relative to normals.

Figure 4 shows the various pathways for host dynamics. The rate of conversion from one host type to another can be evaluated by a model that employs the following parameters:

ε = search rate
H = habitat area
T_o = time to oviposit in a host
T_m = time to mark a host following oviposition
T_r = time to reject a marked host
$\mathfrak{N}$ = number of normal adults
$\mathfrak{M}$ = number of mutant adults (3)

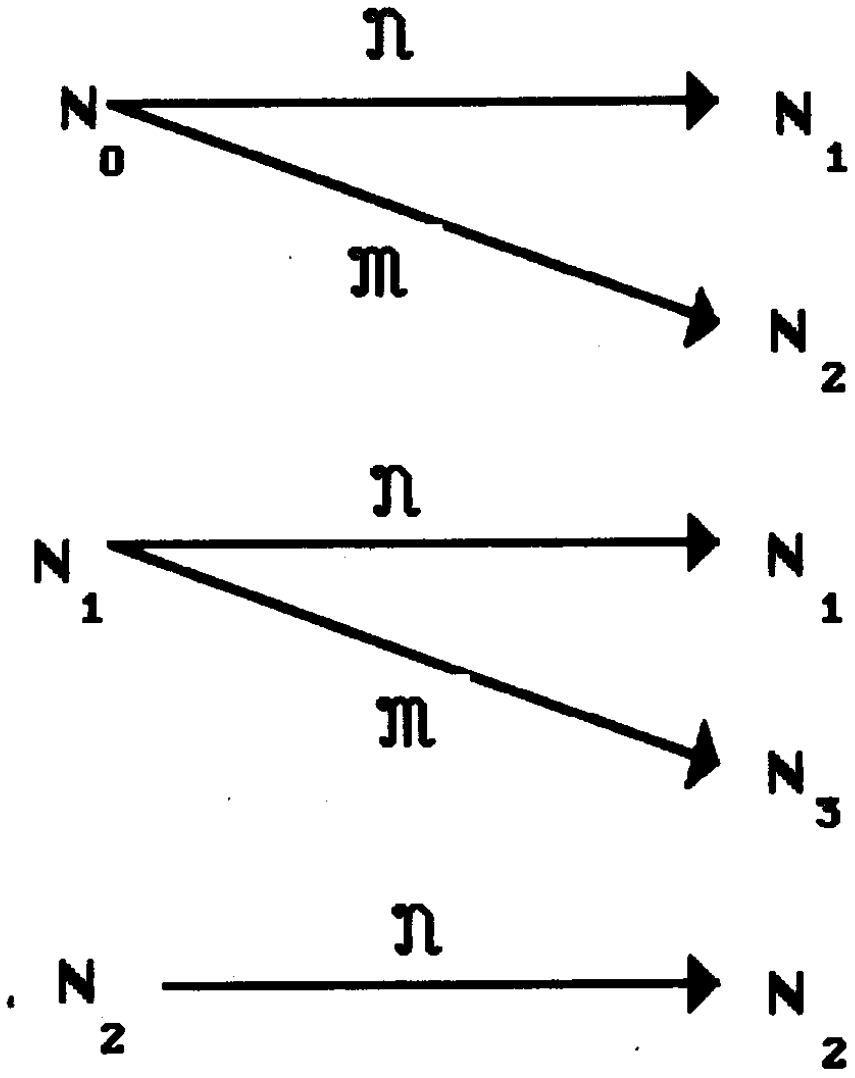


Figure 4. Transition pathways by which normal ($\mathfrak{N}$) and mutant ($\mathfrak{M}$) parasites turn empty hosts (N_0) into hosts that will successfully harbor normal (N_1 and N_3) mutant (N_2) offspring.

The model consists of four equations that utilize the three transitions illustrated in Fig. 4:

$N_1(t+\Delta t) = N_1(t)$ + [the number of empty hosts attacked by normals during Δt] − [the number of normal hosts attacked by mutants during Δt]

$$= N_1(t) + r_{N_0,N_1} - r_{N_1,N_3} \tag{4}$$

$N_2(t+\Delta t) = N_2(t)$ + [the number of empty hosts attacked by mutants during Δt]

$$= N_2(t) + r_{N_1,N_2} \tag{5}$$

$N_3(t+\Delta t) = N_3(t)$ + [the number of normal hosts attacked by mutants during Δt]

$$= N_3(t) + r_{N_1,N_3} \tag{6}$$

The transition rates $r_{i,j}$ are derived from classical foraging models such that a parasite's foraging time is divided into host search and host handling time (e.g. Hassell, 1979):

$$\begin{aligned} r_{N_0,N_1} &= \frac{\varepsilon(N_0(t)/H)\,\Delta t}{\{1+(\varepsilon T_o)\,(N_0(0)/H)\}}\,\mathrm{n} \\ &= \frac{(\varepsilon/H)\,N_0(t)\,\Delta t}{\{1+(\varepsilon/H)\,T_o\,N_0(0)\}}\,\mathrm{n} \\ &= \frac{N_0(t)}{\{(H/\varepsilon)+T_o\,N_0(0)\}}\,\mathrm{n}\,\Delta t \end{aligned} \tag{7}$$

$$\begin{aligned} r_{N_0,N_2} &= \frac{\varepsilon\,(N_0(t)/H)\,\Delta t}{\{1+(\varepsilon)[\{N_0(t)+N_1(t)\}/H]\,(T_o+T_m)+ (\varepsilon)[\{N_2(t)+N_3(t)\}/H](T_r)\}}\,\mathrm{m} \\ &= \frac{N_0(t)}{\{(H/\varepsilon)+[N_0(t)+N_1(t)](T_o+T_m)+ (N_2(t)+N_3(t))(T_r)\}}\,\mathrm{m}\,\Delta t \end{aligned} \tag{8}$$

and

$$r_{N_1,N_3} = \frac{N_1(t)}{\{(H/\varepsilon)+[N_0(t)+N_1(t)](T_o+T_m)+ (N_2(t)+N_3(t))(T_r)\}}\,\mathrm{m}\,\Delta t \tag{9}$$

The model shows that mutants face a tradeoff. They can avoid wasting time and eggs by not ovipositing in already infested, marked hosts but at a cost of time (and presumably some physiological cost of producing and depositing the mark) spent marking each host (compare the denominators for Eqns (7) and (8). Finally, note that host encounter rates are based solely on host density. This is a reasonable starting assumption since the hypothesis we are testing considers only host availability, not host distribution.

Mutant and normal reproductive success can be computed through computer solution of Eqns (4)–(6) by incorporating various values for host density, time to oviposit (T_0), time to mark (T_m) and time to reject (T_r). Results that employ both high (36 000) and low (1000) host densities and one mutant individual for every 1000 of normal individuals are shown in Fig. 5. The resulting curve is almost identical for both densities and is shown as a single line. The figure demonstrates that success of mutants relative to normals is inversely related to T_m. Further, however, only when T_m approaches zero do mutants fare as well as normals, but never better. In addition, although not shown in the figure, mutant fitness is almost totally insensitive to T_r. The explanation for this relative independence is that when mutants are rare and hosts are encountered at random, then encounters with, and the associated benefits from, marked hosts are also rare and thus of little impact. As noted earlier, similar results are obtained when hosts are either abundant or rare. Thus, even without a consideration of the genetic constraints that

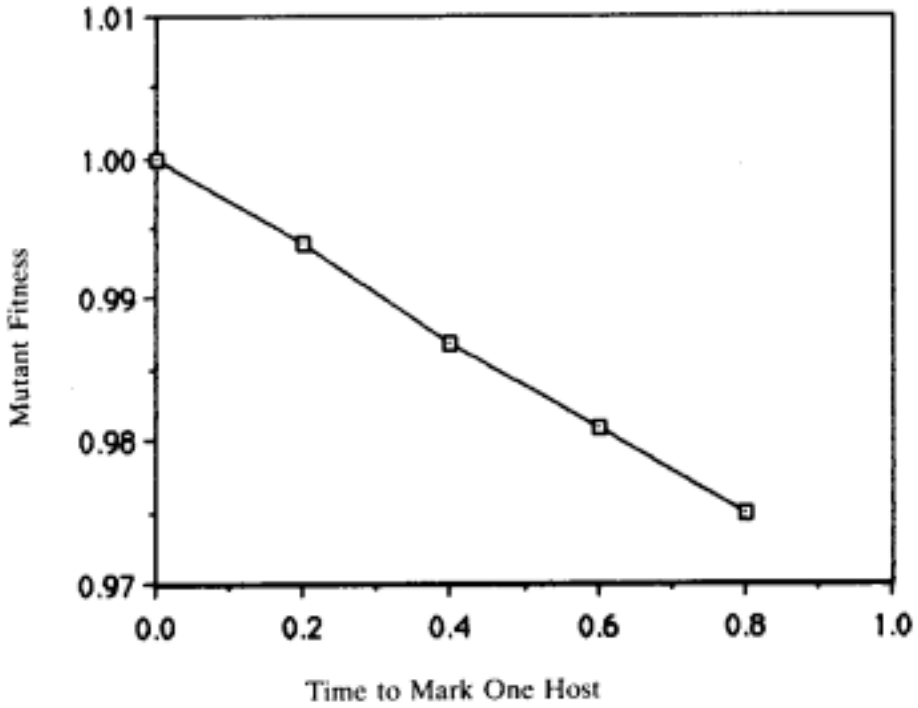


Figure 5. Relative reproductive success for double mutants that mark and recognize vs. normals which can do neither when host marking time varies. The ordinate shows the marking time, the abcissa shows relative numbers of markers and non-markers in generation $t+1$, given equal numbers in generation t.

might operate to oppose the invasion of marking behavior into the population, we conclude that resource shortage alone cannot explain the origin of host marking in parasite populations.

The model we have described does not consider a spatial structure. Hosts are considered to be randomly distributed and encountered; thus host encounter rates are wholly dependent upon global host density. In fact, hosts are often clumped in distribution and many parasites aggregate at patches of high density (Lessels, 1985). Under such conditions, local density of marked and unmarked hosts may have great impact on relative mutant performance, even when such mutants are globally rare. To test the hypothesis that marking is advantageous when hosts are clumped and parasites aggregate at high-density clumps, we employ a modified version of the previous model. The modifications include:

1. Dividing the host habitat into cells and assigning hosts to cells, as determined by a clumping factor.
2. Allowing parasites to spend different amounts of their foraging time in cells with different densities of hosts.

To achieve the latter, we use the following aggregation submodel (Hassell and May, 1973) and define:

β_i = the proportion of foraging time spent in cells i
α_i = the proportion of hosts found in cells i
u = the tendency for parasites to aggregate in high host density cells (10)

and set:

$\beta_i = c\alpha_i^u$: where c is a normalization constant so that $\Sigma\beta_i = 1$ (11)

Thus, parasites with $u = 0$ distribute their foraging time equally among cells whereas parasites with $u = 1$ will aggregate in cells in direct proportion to the host density within those cells. Eqns (4)–(6) can now be modified to include aggregation by multiplying π and μ by β_i for each cell:

$$\begin{aligned} N_1(t+\Delta t) &= N_1(t) + \Sigma\, \beta_i(r_{N_{0i},N_{1i}} - r_{N_{1i},N_{3i}}) \\ N_2(t+\Delta t) &= N_2(t) + \Sigma\, \beta_i\, (r_{N_{0i},N_{2i}}) \\ N_3(t+\Delta t) &= N_3(t) + \Sigma\, \beta_i\, (r_{N_{1i},N_{3i}}) \\ N_0(t+\Delta t) &= \Sigma[N_{0i}(0) - \{N_{1i}(t+\Delta t) + N_{2i}(t+\Delta t) + N_{3i}(t+\Delta t)\}] \end{aligned} \tag{12}$$

In these equations, the summations extend over all cells in the habitat. By varying the clumping factor, we establish host distributions that vary from random to clumped. Next we assume mutants to aggregate in cells with high proportions of hosts without marks (i.e. N_0 and N_1) while normals, because they cannot recognize marks, aggregate in cells of high host density (i.e. all classes of hosts treated equally). Mutant aggregation is computed by weighting the availability of unmarked hosts by the density of marked hosts. That is, we replace the number of unmarked hosts by $(N_{0i} + N_{1i})\, e^{-\omega(N_{2i}+N_{3i})}$ where ω is a weighting parameter. (See Waage (1979) and Roitberg and Prokopy (1984) for discussions on how and why such weighting systems might operate.) Finally, we vary u, the parasite tendency to aggregate.

Results that consider habitats containing 10 000 hosts distributed among 1000 cells are shown in Fig. 6. The hosts are distributed at three different clumping levels, as denoted by the parameter k from the negative binomial distribution. In the figure, we show 'equal performance isoclines' for mutants relative to normals. Thus, the area below each curve represents the

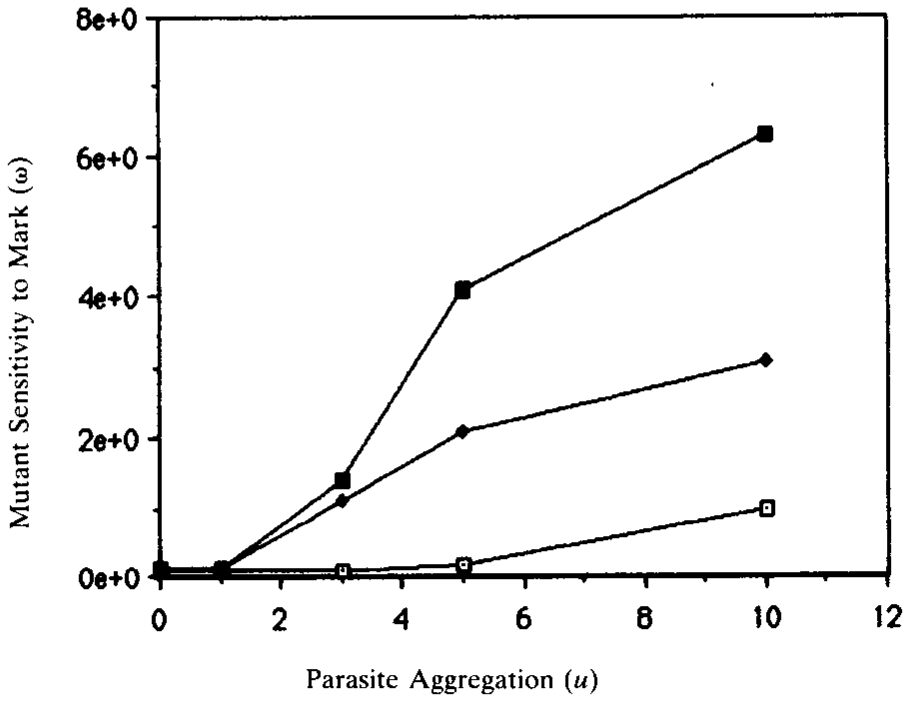


Figure 6. Equal performance isoclines for double mutants when all parasites aggregate (u) at high host density cells and only mutants alter aggregation according to presence of marked hosts. The area below each curve shows combinations of normal aggregation and mutant response (ω) to marks under which mutants reproduce at higher rates than normals. The three curves represent three host distributions as indexed by k of the negative binomial where $k = 150$ (■), $k = 10$ (◆) and $k = 0.8$ (⊡).

combination of u and ω values for which mutants out-perform normals at the three different host clumping levels. We interpret these results to mean that the more likely a parasite is to intensify search within highest density cells (high u) the more likely a mutant is to out-perform normals by spending less time (measured by ω) in those cells with marked hosts. When hosts are highly clumped (e.g. $k \rightarrow 0$), however, high sensitivity to marks will cause mutants to search in very low-density cells where their reduced search efficiency will never be compensated for by higher offspring survival, relative to normals. This results in a range of host clumping, parasite aggregation values that lead to mutant advantage (Fig. 6).

Mutants may be at a considerable advantage when hosts are moderately clumped and parasites intensify their search in the patches of highest density (high u). Under such conditions, mutants will tend to forage in high, though not the highest, density patches, and attain high rates of oviposition and low rates of egg wastage, while normals will suffer very high offspring death rates. Thus, we conclude that in habitats where hosts are clumped and marking allows mutants to avoid areas of high parasite oviposition activity, marking may spread through a population.

The models employed thus far are general and heuristic but also very complex. Further, we have implicitly assumed that aggregation leads to higher re-encounter rates between parasites and hosts than would occur were parasites to forage randomly. We now employ a more tractable model which retains most of the features of the previous model but explictly employs assignable re-encounter rates. As before, we track changes in hosts of type N_0, N_1, N_2 and N_3 by solving four simultaneous equations, but we introduce several new parameters:

γ = probability of re-encountering a host immediately following an oviposition in that host
λ_n = host encounter rate for normals
λ_m = host encounter rate for mutants
$\text{Success}_N(t)$ = number of normal ovipositions during period t
$= N_0(0)\,\lambda_n\,\Delta t$
(Note: normal success rates are constant for a given density of hosts)

Mutant success and reject values are computed by:

$$\begin{aligned}\text{Success}_M(t) &= \text{number of mutant ovipositions during period } t\\ &= \{\text{Success}_M(t-1)(1-\gamma)[(N_0(t)+N_1(t))/N_0(0)]\}\\ &+ \{\text{Reject}_M(t-1)[(N_0(t)+N_1(t))/N_0(0)]\}\end{aligned}$$

$$\begin{aligned}\text{Reject}_M(t) &= \text{number of mutant rejections during period } t\\ &= [\text{Success}_M(t-1)\gamma]+\{\text{Success}_M(t-1)(1-\gamma)[(N_2(t)+N_3(t))/N_0(0)]\}\\ &+ \{\text{Reject}_M(t-1)[(N_2(t)+N_3(t))/N_0(0)]\}\end{aligned} \tag{13}$$

where

$$\begin{aligned}\text{Success}_M(0) &= N_0(0)\lambda_N\Delta t\\ \text{Reject}_M(0) &= 0\end{aligned} \tag{14}$$

The assumptions underlying these equations are: (i) normals oviposit indiscriminately and (because total host density does not change) they deposit the same number of eggs each time period; (ii) mutants reject all marked hosts (N_2 and N_3) and the probability that they will encounter such hosts depends upon the probability of re-encounter following an oviposition in either N_0 or N_1 (i.e. γ) and the likelihood of encounter when randomly encountering hosts i.e. following a rejection. Thus we assume that, following encounters with marked hosts, mutants move from that local site and randomly encounter host types as a function of their density. We also assume that parasites that don't re-encounter hosts, encounter new hosts with probability equal to their relative density.

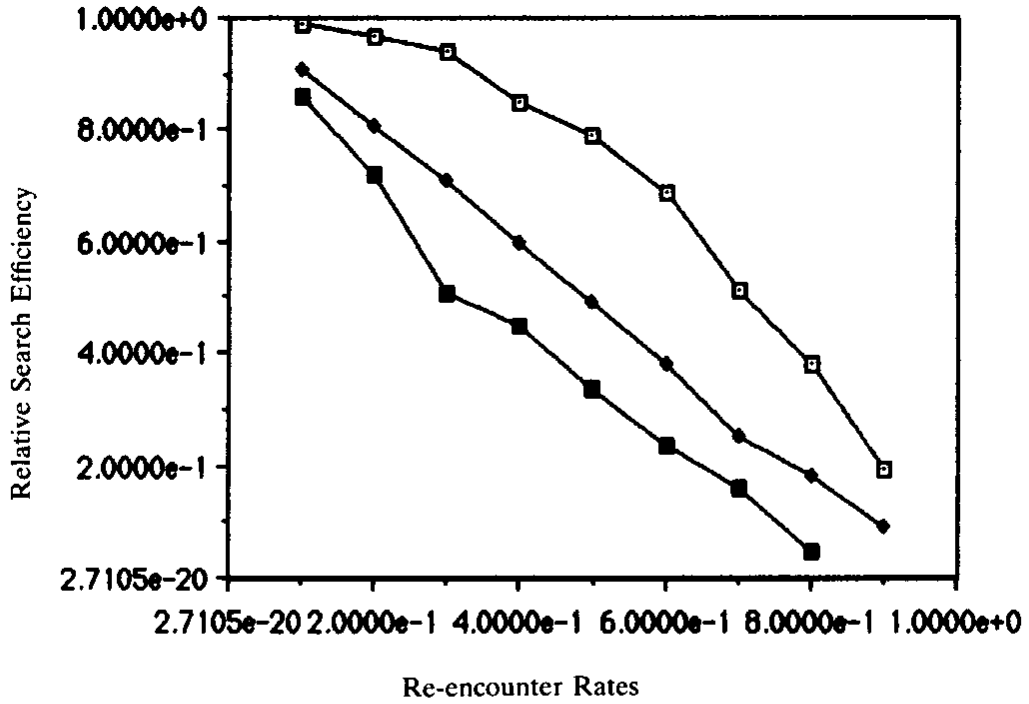


Figure 7. Equal reproductive performance isoclines for double mutants when re-encounter rates (α and γ) and mutant relative search efficiency (λ) vary. The area above each curve shows the combinations of α, γ and λ for which mutants out-perform normals at three frequencies of mutants, 0.001 (□), 0.5 (◆) and 0.9 (■) within the population.

We again track changes in host types over time as a function of transitions between host types:

$$N_1(t+\Delta t) = N_1(t)+[\text{Success}_N(t-1)(1-\gamma)(N_0(t)/N_0(0))] - [\text{Success}_M(t-1)(1-\gamma)(N_1(t)/N_0(0))] - [\text{Reject}_M(t-1)(N_1(t)/N_0(0))]$$

$$N_2(t+\Delta t) = N_2(t)+[\text{Success}_M(t-1)(1-\gamma)(N_0(t)/N_0(0))] + [\text{Reject}_M(t-1)(N_0(t)/N_0(0))]$$

$$N_3(t+\Delta t) = N_3(t)+[\text{Success}_M(t-1)(1-\gamma)(N_1(t)/N_0(0))] + [\text{Reject}_M(t-1)(N_1(t)/N_0(0))]$$

$$N_0(t+\Delta t) = N_0(0) - N_1(t+\Delta t)-N_2(t+\Delta t) - N_3(t+\Delta t) \quad (15)$$

In these equations, we assume that encounter rates for mutants (λ_m) are lower than for normals (λ_N) because some of the search time of mutants must be spent marking hosts and searching hosts for marks. It is now possible to ask how small the ratio λ_m/λ_n can be and yet mutants still invade the population of normals. Figure 7 shows equal performance isoclines for three different mutant frequencies for varying re-encounter probabilities (γ). The region above each isocline indicates combinations of γ and λ_m/λ_n under which marking will spread. When re-encounters are frequent, the reduced encounter rate for mutants is compensated for by their higher rates of egg-laying in unparasitized hosts. In addition, the figure shows that as mutants become more common the conditions under which marking may spread are less restricted. The interpretation of this result is that as mutants become more common (and because they respond to each other's

marks) they will tend to distribute their eggs throughout the habitat, thus increasing the chance that a normal parasite will unknowingly lay her eggs in an already occupied host.

Finally, when hosts are patchily distributed those behaviors that cause high re-encounter rates (e.g. area-restricted search, Vinson (1976)) may also lead to high encounter rates. Thus, marking individuals may actually enhance their host encounter rates without suffering concomitant high superoviposition rates.

Scenario 2: Weak and strong markers

We now consider the second scenario with weak and strong markers. We employ models of similar structure to those described earlier in this section, except that normals and mutants both have a probability of detecting = $p_d \leq 1.0$ and ovipositing in weakly marked hosts. Similarly, all individuals will detect (and reject) hosts ($p_d = 1.0$) parasitized and marked by mutants. The detailed foraging Equations (4)–(6) must now be modified to include the detection process. The modified transition rates model is given in Appendix 1.

The modified rates r_{N_m,N_1} and r_{N_m,N_2} are almost identical to one another because all individuals now recognize some weakly marked and all strongly marked hosts. The rates differ, however, in that only mutants spend some of their search time marking hosts. Thus, normals receive all of the payoffs from marking but none of the costs and, as expected, mutants never achieve equal or higher fitness than normals. In fact, this is true whether or not we break the habitat into cells, since normals would now also partition their time between different cells in exactly the same manner as would mutants. Thus, at the level considered by this model, when all individuals perceive marks, differential aggregation will not occur and overt marking will never spread through the population.

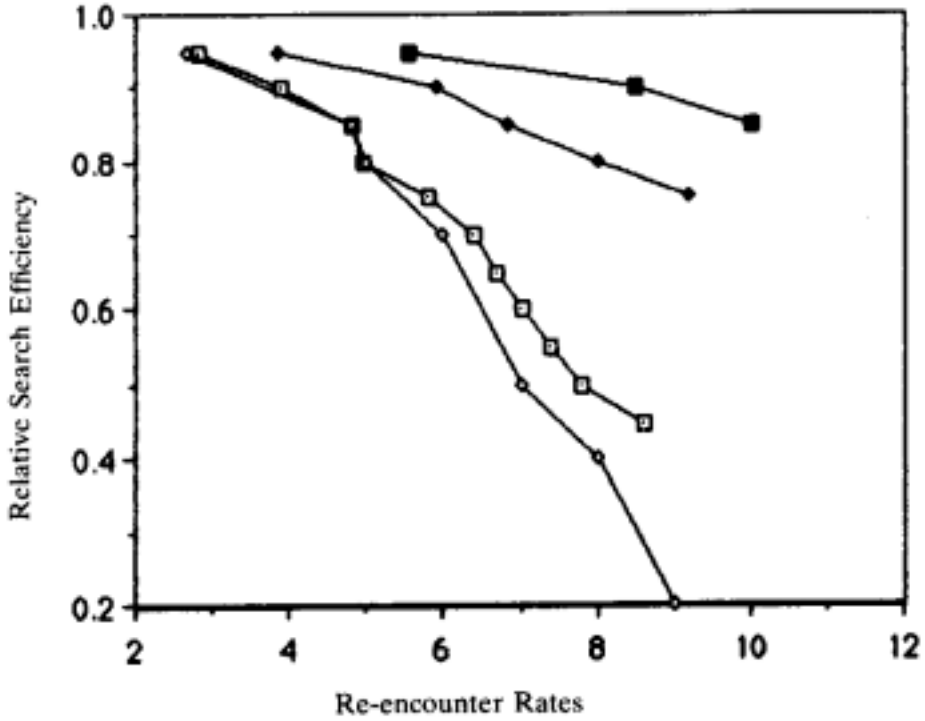


Figure 8. Equal reproductive performance isoclines for mutants when all parasites always recognize mutants' marks and sometimes (p_d) detect normals'. The curves are defined as in Fig. 7 for three probabilities of normal detection ($p_d = 0.1$ ⊡, 0.5 ◆, 0.7 ■) for a single frequency of mutants (0.001). A single isocline (◇) for a double mutant at the same frequency is shown for comparison.

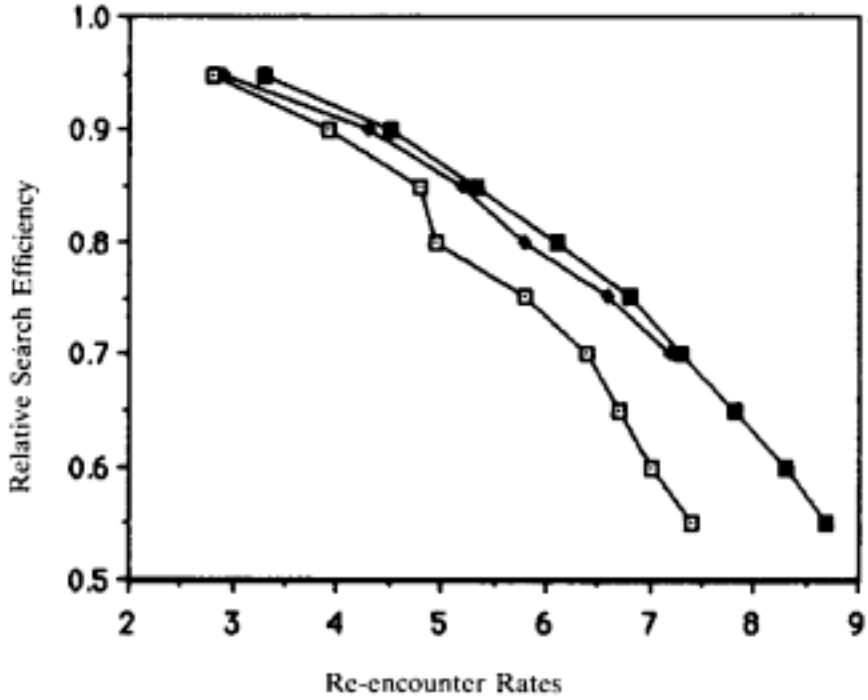


Figure 9. Equal reproductive performance isoclines for mutants when all parasites always recognize mutants' marks and sometimes (p_d) detect normals'. The curves are defined as in Fig. 7 for a single probability of normal detection ($p_d = 0.1$) for three frequencies of mutants 0.001 (⊡), 0.05 (◆) and 0.09 (■).

This model treats re-encounters at the level of cell or multihost patch. It is possible, however, to re-examine the system at the individual host/cell level by employing the re-encounter model described earlier. Thus, we consider those combined values of search rates, probability of re-encounter and probability of detection under which strong marking may spread through a population. The modified model is given in Appendix 2. It differs from the original in that rejection probabilities are incorporated for all parasitized hosts when encountered by both normals and mutants.

Note the similarity for success and reject equations for both mutants and normals (Appendix 2). The principal difference is that, while both types re-encounter the host they most recently oviposited in, normals reject such hosts with probability p_d and mutants always reject their previously parasitized hosts.

Results of the solution of the modified model are shown in Figs 8 and 9. Here, the marker performance isoclines are plotted for three different probability of detection(p_d) levels (Fig. 9) and for three different mutant frequencies (Fig. 8). Several points emerge. First, mutants outperform normals at low p_d and high re-encounter rates so long as search efficiency is not greatly decreased by the marking behavior. Second, in contrast to the previous re-encounter model results, the conditions for mutant spread become more restricted as marking becomes more common (compare Figs 7 and 8). Finally, although marking behavior can spread in a weak–strong marking system, the conditions are more restricted than when only mutants recognize marks. The last two points arise because, in the second scenario, all individuals potentially benefit from the marking behavior.

In summary, employment of the population-level models shows that marking can spread through the population so long as markers have a greater than average chance of encountering marked hosts, relative to non-markers, whether or not non-markers can recognize such marks.

Behavior-rich simulations

In this section, we model rates of successful ovipositions for 'double mutants' using a computer simulation that essentially mimics the habitat and behavior of *Rhagoletis pomonella* females. Our aim here is to show that when the details of a particular parasite's ecology are considered the same general conclusions regarding marking pheromones emerge.

The model we employ is a discrete-event stochastic computer simulation. A detailed description of a similar version of the model can be found in Tourigny (1985) and Mangel *et al.* (in prep.). The model accurately simulates the host foraging behavior of *Rhagoletis* sp. females under both natural and seminatural conditions. The model consists of five major components: (1) a spatial distribution of hosts (fruit) within discrete habitats (trees); (2) fly search; (3) fly oviposition; (4) physiological limits; and (5) larval competition.

Host distribution

Hosts are grouped into clusters consisting of between three and five individual host fruit. The number of hosts assigned to a given cluster is randomly chosen, as is the number of clusters on different branches within the tree. A clumping factor causes varying levels of cluster clumping within branches to occur.

Fly search process

Flies search randomly within branches and move to fruit clusters once such clusters fall within their reactive visual envelope (Roitberg, 1985). Once within a cluster, flies move randomly among individual hosts and the probability that a fly will continue searching within a cluster depends upon whether an egg was laid on the previous host visit (Roitberg and Prokopy, 1984). The probability that a fly will continue searching within a given branch increases with continued cluster-finding rates. Flies that emigrate from a branch are randomly re-assigned to another one. Each movement within a branch takes one unit of time and flight to another branch takes two units of time.

Fly oviposition

Upon locating hosts, flies lay eggs with some probability p_{ov}. For mutants, p_{ov} in host types N_2 and N_3 is always zero for the first eight days following mark deposition. A host encountered nine or more days after a previous oviposition is treated identically to hosts that are clean or previously parasitized by normal flies only. Normals treat all hosts identically and oviposit with the same non-zero probability as mutants. All flies expend a single unit of time ovipositing in hosts; mutants use a further two units of time assessing and marking hosts following oviposition.

Limits and parameter choices

Flies are limited to 75 moves per day. This value represents the product of observed movement rates (Roitberg *et al.*, 1982) and foraging bout length/day (Prokopy and Roitberg, 1984). At the start of each day, flies are randomly re-assigned to new positions within the tree. Adult flies may forage for hosts for an average of 12 days. At the end of each day, however, each fly is evaluated as alive or dead. The egg complement is 12 ± 3 mature eggs/day. Up to three eggs not utilized on a given day's foraging are recoverable during the next day's foraging.

Larval competition

The model conditions are set such that each host will generally yield a single offspring. Each offspring must survive for 14 days within a host to complete larval development. In the simulation, larvae within each host are examined for survival at the end of each day. Survival is

randomly determined by a survival probability p_s. Since larvae can be present at different densities and in different combinations of various aged individuals, we model survival as follows:

Conditions	Daily larval survival probability
Alone within a host	0.99
Larva present with equal age larvae	0.97 (no. competing offspring)
Larva present with older larvae	0.875 (no. competing offspring)
Larva present with younger larvae	0.985 (no. competing offspring)

In summary, the system we simulate is characterized by individuals that re-encounter hosts with high frequency because hosts are clumped and foragers concentrate their search in areas where hosts have been contacted. In addition, hosts rarely support more than a single offspring, thus, superparasitism can lead to wastage of time spent foraging and wastage of eggs.

Results of dynamic, stochastic simulations are shown in Figures 10, 11 and 12 for five cluster densities and three levels of cluster clumping. First note that when host densities decrease so do rates of oviposition for mutants (Fig. 10). Thus, the cost of host marking and recognizing appears in this system and is most evident at high frequencies of mutants. Normals, by contrast, do not suffer significant reduction in oviposition rate and, in fact, generally exhaust their egg supply on most days, at least at the host density levels we employed.

Second, the results are reversed when we consider the effects of host distributions on offspring

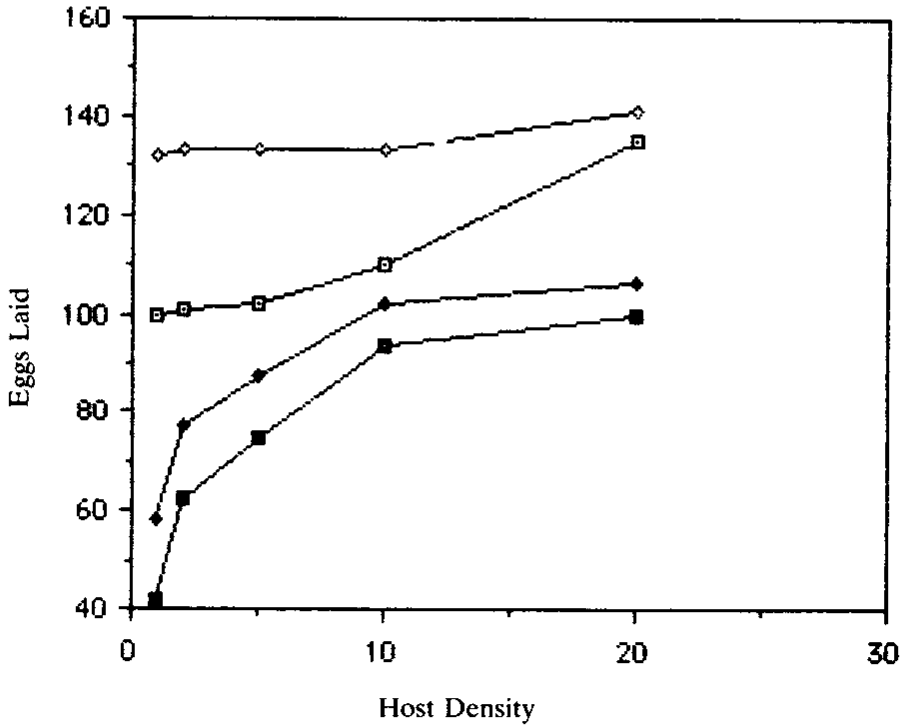


Figure 10. Behavior-rich simulation lifetime-egg-deposition values for individual mutants and normals at three normal to mutant ratios (9 n:1 m (⊡), 5 m:5 n (◆), 1 n:9 m(■)) at five host-cluster per branch densities. Note, a single line is shown for normals (◇) because egg deposition rates for normals are unaffected by mutant frequencies.

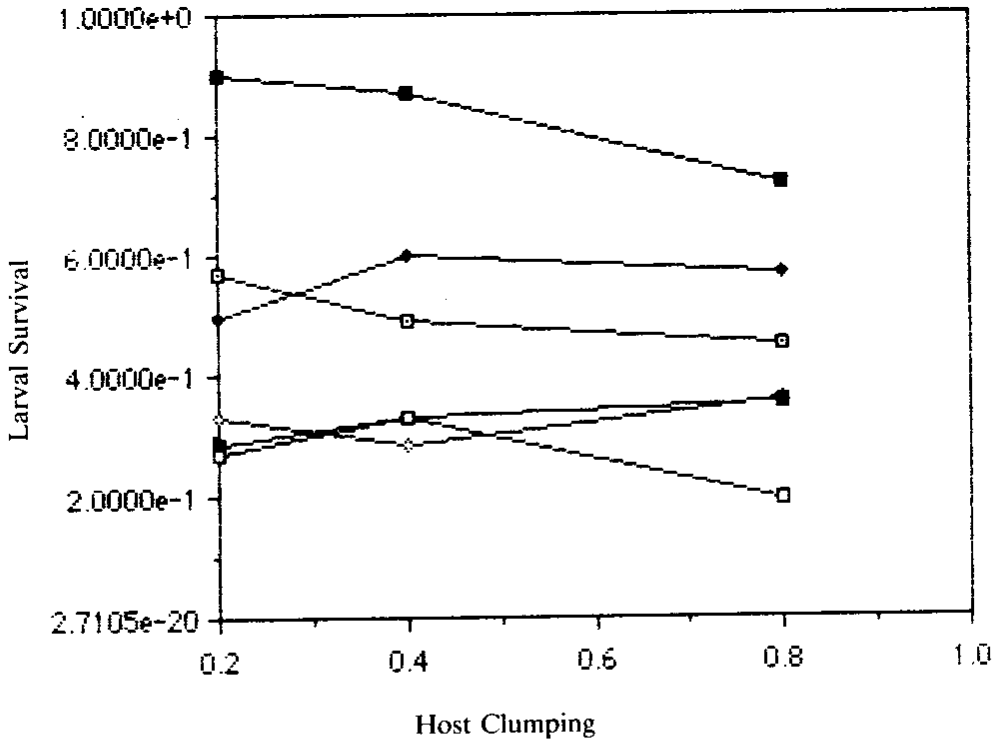


Figure 11. Behavior-rich simulation survival rates for mutant and normal larvae at three normal to mutant ratios, at three levels of host-cluster clumping. Mutant larval survival 9 n:1 m (⊡), 5 n:5 m (◆) and 1 n:9 m (■). Normal larval survival 9 n:1 m (◇), 5 n:5 m (■) and 1 n:9 m (□).

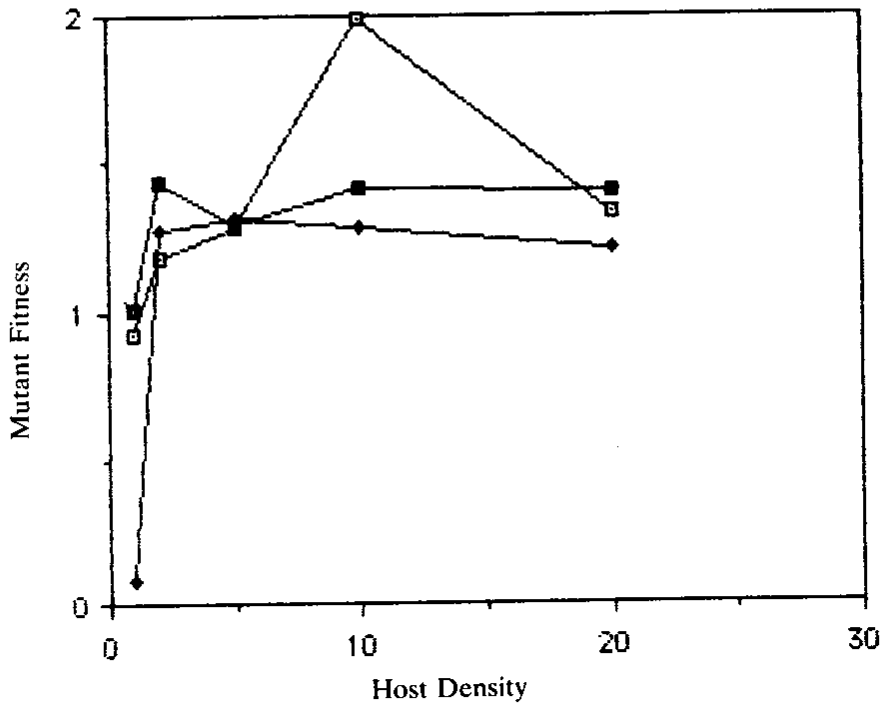


Figure 12. Behavior-rich simulation fitness values for mutants, relative to normals, at three normal to mutant ratios (9 n:1 m (■), 5 n:5 m (◆) and 1 n:9 m (⊡)) at five host-cluster per branch densities.

survival with mutants performing best at highest mutant frequencies (Fig. 11). Further, mutant offspring survival rates exceed those of normals at all three levels of host clumping at each of the three mutant frequencies. The within-behavior-type patterns are not as clear as in the previous category and can be attributed to the effects of mutants spreading their eggs among hosts more frequently at high mutant frequencies.

Finally, Fig. 12 shows mutant relative fitness at the five host densities. The point that emerges is that at nearly all host densities the cost of marking and recognizing marked hosts (as evidenced by lower oviposition rates) is almost always offset by increased survival rates for offspring. It is only when clusters are sparse, search time is limited and parasites readily emigrate from clusters harboring already-parasitized hosts that mutant behavior will not spread through populations. We have not yet conducted simulations with 'weak markers' but suggest that similar results would emerge, albeit with more restricted conditions under which mutants would out-perform normals. We suggest this because, in our simulations at least, individuals experienced high re-encounter rates with hosts (about 80% during the day the host was first encountered).

We believe that these simulations provide a kind of 'experimental' validation for some of the ideas developed in the previous sections. That is, even when rare, marking behavior may spread through a population, even when time- and energy-consuming, as long as there is a high probability that mutants obtain payoffs from such behavior.

The marking 'benefit curve'

In this section we show how a marking benefit curve can be modeled. Such a curve provides insight into why many marking pheromones are short-lived and water-soluble. We begin with the presumption that the benefit to an ovipositing female is measured as the probability that her egg hatches and, if there is more than one egg present in the host, that her larva wins the competition. Let $p_w(d)$ denote the probability that an egg laid on day 0 is victorious in a competition with a second egg laid in the host on day d. We assume that $p_w(d)$ takes the functional form:

$$p_w(d) = 1 - 0.5 \exp(-\gamma_w d) \tag{16}$$

where γ_w is a parameter. We assume that if two eggs are laid on the same day ($d = 0$), then there is an equal probability of victory ($p_w(0) = 0.5$); this appears to be a reasonable starting assumption.

We also assume that the first female marks the host after oviposition and that the amount of mark remaining τ time units later is given by:

$$M(\tau) = M_0 f^{\tau} \tag{17}$$

where M_0 is the amount of mark originally placed on the host and $f < 1$ is a decay rate. From f, we can compute the half-life of the mark; measured in days. Note that τ should be measured in units less than days (e.g. oviposition periods). In the computations that follow, we assume that each day is divided into 14 oviposition periods. The probability of detecting a host that is marked, when the level of marker is $M(\tau)$ is assumed to be given by:

$$p_d(M(\tau)) = 1 - \exp(-\gamma_m M(\tau)) \tag{18}$$

where γ_m is a parameter. For simplicity of calculation, we will assume that the mark decays overnight (e.g. is washed off with condensation), so that the level of mark is constant during each day.

We assume that the female marks mainly so that her own eggs are not wasted, i.e. so that she does not oviposit in the same host twice. To simplify the computations, we assume that the

female leaves the patch of hosts after a single day and does not return. Thus the benefit of marking involves two aspects: (i) the probability that a female detects a host in which she has oviposited; and (ii) the probability that her larva wins a competition if the host is superparasitized by a different female.

First consider the computation of the probability that a female detects her own mark, given that she has oviposited in a host at time 0. The probability that the female stays in the patch will depend upon the number of hosts in the patch, which we still denote by S. Rather than modeling the exit decision as a dynamic optimization problem, we simply assume that:

$$\text{Prob(exit the patch after } t \text{ time units}|S \text{ hosts in the patch)} = p_e(t) = 1 - \exp(-\gamma_e t/S) \tag{19}$$

where γ_e is a parameter.

Suppose that the female stays in the patch for t units of time after an oviposition. Assuming that she randomly visits hosts, the probability that she will visit the host in which she has oviposited is given by $p_v = 1/S$. The probability that she visits the host j times during $[0,t]$ and recognizes the mark each time she visits is then given by a binomial distribution (characterizing the j out of t visits to the fruit) times $p_d(M_0)^j$, which characterizes the probability of j detections. Averaging this quantity over the exit distribution gives the probability that the female recognizes her own mark on the day that she oviposits.

A similar approach can be used to compute the probability that her larva wins a competition if a superparasitism by a different female occurs. That is, consider other parasites encountering the

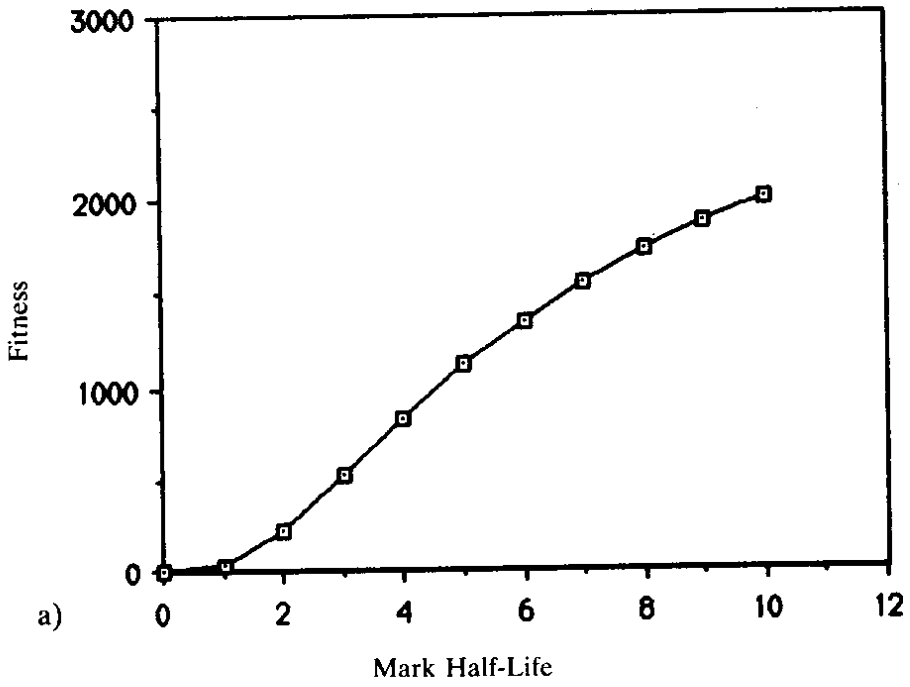
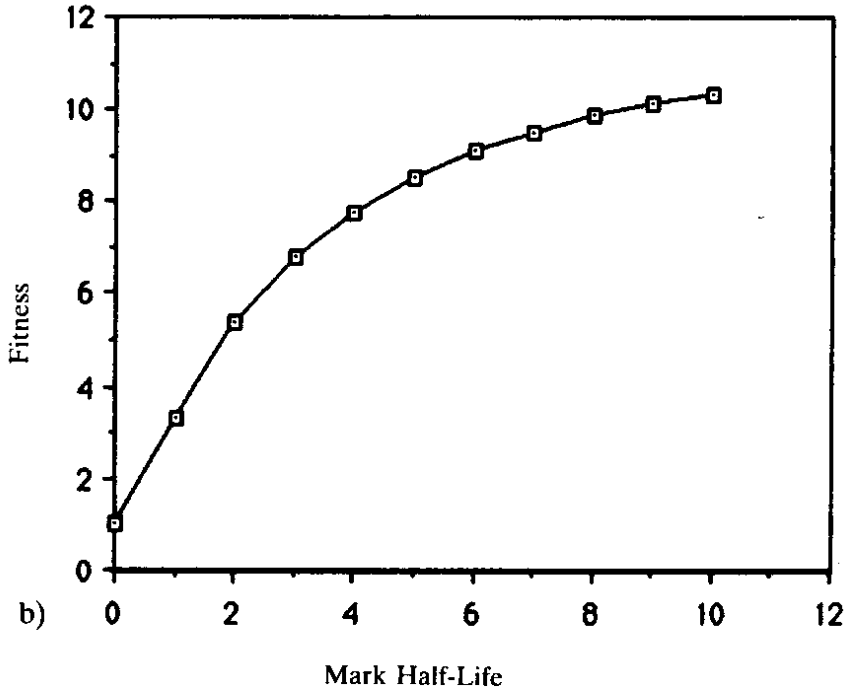


Figure 13. The marking benefit curves. The fitness accrued to a female from a single marking event, indexed by larval survival (R_w = Prob(her larva lives)/1 −Prob(her larva lives)) is shown as a function of the half-life of the mark. Parameters common to all three curves are: 14 oviposition periods per day, $S = 16$, $\gamma_w = 0.3$. Other parameters are: (a)$\gamma_m = 5$, $M_0 = 2$, $\gamma_e = 5$, $D = 20$; (b) γ M = 3, γ_e =3, $M_0 = 1$, $D = 4$; (c) $\gamma_m = 3$, $\gamma_e = 3$, $M_0 = 1$, $D = 20$. The probability that the female recognizes her own larva is 0.9999 for (a), 0.993 for (b) and 0.984 for (c).



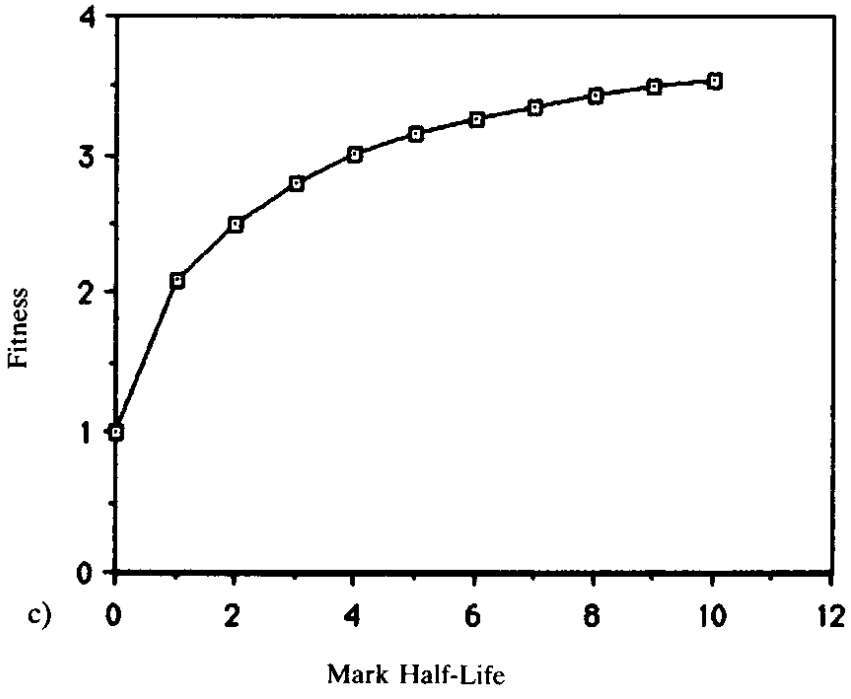


Figure 13 (*contd.*)

hosts between day 0 and some fixed time at which the larva is fully grown, denoted by D. After d days, there will be Td periods in which the host could have been visited. The probability of j visits is again given by a binomial distribution. The probability that her larva is alive on day D is then a sum of two terms. The first corresponds to a detection of the mark (and thus no superparasitism)

in each of the j visits (in which case her larva survives with certainty). The second corresponds to failure to detect her mark, a second parasitization, and her larva winning the competition.

A useful measure of the relative value of the mark is the ratio

$$R_w = \text{Prob(her larva lives)}/[1 - \text{Prob(her larva lives)}] \tag{20}$$

Figure 13 shows the results of such computations. Most of the advantage to the female occurs during the first two days after oviposition. This is due mainly to the probability of winning increasing with time. A mark with a relatively short half-life (e.g. a water-soluble mark lasting just two days) is nearly as effective as a much more costly (e.g. lipid-based) mark lasting a much longer time.

Discussion and conclusions

In this paper, we have developed a theory for the evolutionary ecology of marking pheromones by considering three different approaches to understanding the components of fitness associated with marking pheromones. We first used models based on individual, state-variable dynamics to compute the fitness of normal (non-marking) and mutant (marking) insects as a function of various environmental parameters and behavioral patterns. We then considered population-level models using what might be called 'classical host parasite' equations to determine when large populations of non-marking individuals could be invaded by marking individuals. Finally, we used behavior-rich simulations of the apple maggot fly to provide (as nearly as possible) 'experimental' confirmation of our hypotheses. We also addressed the question of the lifetime of marks and developed a simple model which suggests that water-soluble marks evolved because the main advantage of marking is that an insect can avoid wasting her eggs by ovipositing a second time in a host that she has already parasitized.

We have not cast our theory into an explicit genetic context. This could be done, for example, if one were willing to make additional (and at this time, *ad hoc*) assumptions about the genetics of marking systems. We believe, on the other hand, that the key to understanding the evolution of marking is in characterizing the nature of the fitness function. If F_n and F_m denote the fitness of normal and marking individuals in a certain environment and p_m denotes the fraction of marking individuals in the population, then under very general conditions the fraction of marking individuals in the next generation p_m' is:

$$p_m' \sim p_m(F_m/F_n) \tag{21}$$

and we believe that this tells as much of the story as a full-blown genetic model would.

We have also not used existing theories of altruistic behavior (e.g. Hamilton, 1964). It is clear that marking a host after oviposition can be viewed as altruistic behavior, since it provides information to everyone else in the population, at the expense of the individual who does the marking. For example, Charnov (1977) considered the spread of an 'altruistic act' in a population. Charnov's condition and its interpretation for our case is:

$$c/b < (m-p)/(1-p) \tag{22}$$

where c is the cost of marking, b is the benefit others receive from marking, m is the proportion of marking events benefiting marking individuals and p is the proportion of marking individuals in the populations. In our case, c is negative since the act of marking benefits the marker herself (i.e. she can avoid wasting eggs) and b is some positive value. Further, since everyone else in the population (mutant or not) has an equal chance of benefiting from the deposition of the mark, then $m > p$, because m includes the marker herself receiving benefit at some higher than average

rate. Note that $c/b < 0$ and $m - p > 0$, so that the condition in Eqn (22) always holds, suggesting that marking should spread through the population. That is, a behavior which benefits others will still spread through a population if it benefits the actor more than it benefits others.

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References

Averill, A. and Prokopy, R. J. (1987) Intraspecific competition in the tephritid fruit fly *Rhagoletis pomonella*. Ecology **68**, 878–86.

Bauer, G. (1986) Life history strategy of *Rhagoletis alternata* (Diptera: Trypetidae), a fruit fly operating a non-interactive system. *J. Anim. Ecol.* **55**, 785–94.

Charnov, E. L. (1977) An elementary treatment of the genetical theory of kin selection. *J. Theor. Biol.* **66**, 541–50.

Charnov, E. L. and Skinner, S. W. (1985) Complementary approaches to the understanding of parasitoid oviposition decisions. *Environ. Ent.* **14**, 383–91.

Charnov, E. L. and Stephens, D. W. (1988) On evolution of host selection in solitary parasitoids. *Amer. Natur.* (in press).

Cirio, U. (1971) Reperti sul meccanismo stimulorisposta nell' ovideposizione del *Dacus oleae* Gmelin. *Redia* **52**, 577–600.

Corbet, S. A. (1971) Mandibular gland secretions of larvae of the flour moth, *Anagasta kuehniella*, contains an epideictic pheromone and elicits oviposition movements in a hymenopteran parasite. *Nature* **232**, 481–4.

Godfray, H. C. (1987) The evolution of clutch size in parasitic wasps. *Amer. Natur.* **129**, 221–33.

Hamilton, W. D. (1964) The genetical evolution of social behaviour. Pts I & II. *J. Theor. Biol.* **7**, 1–52.

Hassell, M. P. (1979) *Dynamics of Arthropod Predator–Prey Systems*. Princeton University Press, Princeton, New Jersey, USA.

Hassell, M. P. and May, R. M. (1973) Stability in insect host–parasite models. *J. Anim. Ecol.* **42**, 693–726.

Houston, A., Clark, C. W., McNamara, J. and Mangel, M. (1988) Dynamic models in behavioural and evolutionary ecology. *Nature* **332**, 29–34.

Hubbard, S. F., Marris, G., Reynolds, A. and Rowe, G. W. (1987) Adaptive patterns in the avoidance of superparasitism by solitary parasitic wasps. *J. Anim. Ecol.* **56**, 387–402.

Hurter, J., Boller, E.F., Blattman, B., Buser, H.R., Bosshard, N.U., Damm, L., Kozlowski, M.W., Schoni, R., Raschadorf, F., Schlumpf, E., Ricter, W.J., and Schreiber, J. (1987) Oviposition-deterring pheromene in *Rhagoletis cerasi* L.: Purification and determination of chemical constitution. *Exper.* **43**, 155–64.

Iwasa, Y., Suzuki, Y. and Matsuda, H. (1984) Theory of oviposition strategy of parasitoids. I. Effect of mortality and limited egg number. *Theor. Pop. Biol.* **26**, 205–27.

Lenteren, J. C. van (1981) Host discrimination by parasitoids. In *Semiochemicals: Their Role in Pest Control* (eds D. A. Nordlund, R. L. Jones and W. J. Lewis), John Wiley & Sons, New York, USA, pp. 153–80.

Lessells, C.M. (1985) Parasitoid foraging: should parasitism be density dependent? *J. Anim. Ecol.* **54**, 27–41.

Mangel, M. (1985) Search models in fisheries and agriculture. *Lecture Notes Biomath.* **61**, 105–37.

Mangel, M. (1987a) Modelling behavioural decisions of insects. *Lecture Notes Biomath.* **73**, 1–18.

Mangel, M. (1987b) Oviposition site selection and clutch size in insects. *J. Math. Biol.* **25**, 1–22.

Mangel, M. and Clark, C. W. (1986) Towards a unified foraging theory. *Ecology* **67**, 1127–38.

Mangel, M. and Roitberg, B.D. (1989) Dynamic information and host acceptance by a tephritid fruit fly. *Ecol. Ent.* (In press).

McNamara, J. and Houston, A. (1986) The common currency for behavioural decisions. *Amer. Natur.* **127**, 358–78.

Morrison, G. and Lewis, W. J. (1981) Allocation of searching time by *Trichogramma pretiosum* in host-containing patches. *Entomologia Experimentalis et Applicatta* **30**, 31–9.

Nordlund, D. A. (1981) Semiochemicals: a review of the terminology. In *Semiochemicals: Their Role in Pest Control* (eds D. A. Nordlund, R. L. Jones and W. J. Lewis), John Wiley & Sons, New York, USA, pp. 13–28.

Parker, G.A. and Courtney, S. P. (1984) Models of clutch size in insect oviposition. *Theor. Pop. Biol.* **26**, 27–48.

Price, P. W. (1977) General concepts on the evolutionary biology of parasites. *Evolution* **31**, 405–20.

Prokopy, R. J. (1972) Evidence for a pheromone deterring repeated oviposition in apple maggot flies. *Environ. Ent.* **1**, 326–32.

Prokopy, R. J. (1981) Epideictic pheromones that influence spacing patterns of phytophagous insects. In *Semiochemicals: Their Role in Pest Control* (eds D. A. Nordlund, R. L. Jones and W. J. Lewis), John Wiley & Sons, New York, USA, pp. 181–213.

Prokopy, R.J. and Roitberg, B.D. (1984) Foraging behavior of true fruit flies. *Amer. Sci.* **72**, 41–50.

Roitberg, B. D. (1985) Search dynamics of fruit parasitic insects. *J. Insect Physiol.* **31**, 865–72.

Roitberg, B. D. and Prokopy, R. J. (1984) Host visitation sequence as a determinant of search persistence in fruit parasitic tephritid flies. *Oecologia* **62**, 7–12.

Roitberg, B. D. and Prokopy, R. J. (1987) Insects that mark host plants. *BioScience* **37**, 400–6.

Roitberg, B. D., Lenteren, J. C. van, Alphen, J. J. M. van, Galis, F. and Prokopy, R. J. (1982). Foraging behaviour of *Rhagoletis pomonella*, a parasite of hawthorn (*Crataegus viridis*), in nature. *J. Anim. Ecol.* **51**, 307–25.

Strand, M. and Vinson, S. B. (1982) Behavioural response of the parasitoid *Cardiochiles nigriceps* to a kairomone. *Entomologia Experimentalis et Applicata* **31**, 308–15.

Tourigny, G. (1985) A general dynamic and stochastic discrete-event simulation model of the foraging behaviour of true fruit flies (Tephritidae), with special reference to the apple maggot, *Rhagoletis pomonella* (Walsh). Masters Thesis, Simon Fraser University, 293pp.

Vinson, S. B. (1976) Host selection by insect parasitoids. *Ann. Rev. Ent.* **21**, 109–33.

Waage, J. K. (1979) Foraging for patchily distributed hosts by the parasitoid *Nemeritis canescens*. *J. Anim. Ecol.* **48**, 353–71.

Waage, J. K. and Ng, S. M. (1984) The reproductive strategy of a parasitic wasp. I. Optimal progeny and sex allocation in *Trichogramma evanescens*. *J. Anim. Ecol.* **53**, 401–16.

Appendix 1

Modified transition rate detailed foraging model that includes probabilities (p_d) that parasites will recognize weakly marked hosts.

$$r_{N_0,N_2} = \frac{\varepsilon\ (N_0(t)/H)\Delta t}{\{1+(\varepsilon)[\{((1-p_d)N_1(t))+N_0(t)\}/H](T_o+T_m) + (\varepsilon)[\{p_dN_1(t))+N_2(t)\ +\ N_3(t)\}/H](Tr)\}}\ \mathfrak{m}\ \Delta t$$

$$= \frac{N_0(t)}{\{(H/\varepsilon)\ +\ [((1-p_d)N_1(t))+N_0(t))](T_o+T_m) + [(p_dN_1(t))+N_2(t)\ +\ N_3(t)](T_r)\}}\ \mathfrak{m}\ \Delta t$$

$$r_{N_0,N_1} = \frac{N_0(t)}{\{(H/\varepsilon)\ +\ [((1-p_d)N_1(t))+N_0(t)](T_o)\ +\ [(p_dN_1(t))+N_2(t)+\ N_3(t)](T_r)\}}\ \mathfrak{n}\ \Delta t$$

$$r_{N_1,N_3} = \frac{N_1(t)}{\{(H/\varepsilon)\ +\ [((1-p_d)N_1(t))+N_0(t)](T_o+T_m) + [(p_dN_1(t)+N_2(t))\ +\ N_3(t)](T_r)\}}\ \mathfrak{m}\ \Delta t$$

Appendix 2

Modified transition rate re-encounter model that includes probabilities (p_d) that parasites will recognize weakly marked hosts.

$$N_1(t+\Delta t) = N_1(t) + [\text{Success}_N(t-1)(1-\gamma)(N_0(t)/N_0(0))] + [\text{Reject}_N(t-1)(N_0(t)/N_0(0))] - [\text{Success}_M(t-1)(1-\gamma)[(1-p_d)N_1(t)/N_0(0))] - [\text{Reject}_M(t-1)[(1-p_d)N_1(t)/N_0(0))]$$

$$N_2(t+\Delta t) = N_2(t) + [\text{Success}_M(t-1)(1-\gamma)(N_0(t)/N_0(0))] + [\text{Reject}_M(t-1)(N_0(t)/N_0(0))]$$

$$N_3(t+\Delta t) = N_3(t) + [\text{Success}_M(t-1)(1-\gamma)\{(1-p_d)N_1(t)/N_0(0)\}] + [\text{Reject}_M(t-1)[(1-p_d)N_1(t)/N_0(0))]$$

where:

$\text{Success}_N(t)$ = number of normal ovipositions during period t
$= \{\text{Success}_N(t-1)(1-\gamma)[((N_1(t)(1-p_d))+\ N_0(t))/\ N_0(0)]\}$
$+\ \{\text{Success}_N\ (t-1)\ \gamma\ (1-p_d)\}\ +$
$[\text{Reject}_N(t-1)[((N_1(t)(1-p_d))+N_0(t))/N_0(0)]\}$

$\text{Success}_M(t)$ = number of mutant ovipositions during period t
$= \{\text{Success}_M(t-1)(1-\gamma)[((N_1(t)(1-p_d))+\ N_0(t))/\ N_0(0)]\}$
$+\ \{\text{Reject}_M\ [((N_1(t)(1-p_d))+N_0(t))/N_0(0)]\}$

$\text{Reject}_N(t) = \{(\text{Success}_N(t-1)\ \gamma\ p_d\}+$
$\{\text{Reject}_N(t-1)[((N_1(t)p_d)+(N_2(t)+N_3(t)))/N_0(0)]\}$

$\text{Reject}_M(t) = \{(\text{Success}_M(t-1)\ \gamma\}+$
$\{\text{Reject}_M(t-1)[((N_1(t)p_d)+(N_2(t)+N_3(t)))/N_0(0)]\}$