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Infection and colony variability in social insects

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SUMMARY

The average relatedness among colony members in the social insects, such as bees, wasps and ants, is often low, contrary to the expectations of kin selection theory. Lower relatedness results from multiple mating by the queens (polyandry) or from the presence of more than one functional queen (polygyny). Among the proposed advantages for such mating systems, selection by parasites for within-colony genetic variability is discussed. Empirical studies of this problem are few, but several lines of evidence suggest a role for parasites, such that genetic diversity reduces the rate of within-group transmission. Theoretical considerations indicate that multiple mating is advantageous under conditions of low mating costs relative to parasite pressure and when intermediately sized colonies have a disproportionately large share of reproductive success in the population. In this view, mating strategies (as in polyandry) and strategies of female associations (as in polygyny) that lead to an increased genetic diversity among offspring are, at least in part, an instance of variance reduction in relation to parasitism.

1. PARASITES, SOCIAL LIFE AND MATING SYSTEMS

Parasites can be contracted and subsequently threaten many individuals in socially living species. Therefore, Alexander (1974) and Freeland (1976) suggested that seemingly unrelated phenomena in social animals, such as group aggression towards new members, the maintenance of territories, or troop size in primates, may all have evolved to avoid novel infections. For directly transmitted diseases, positive correlations between group size, number of parasite species per host and infection intensity have been demonstrated in several animal species (see, for example, Davies *et al.* 1991); negative correlations are found in vector-transmitted diseases (Poulin & Fitzgerald 1989). Once acquired, disease can be transmitted more easily within than between groups, as suggested for human smallpox in African households (Becker & Angulo 1981). In several cases, fitness effects of parasites for group-living animals have been demonstrated (see, for example, Brown & Brown 1986). This reasoning clearly demonstrates the role of parasites as selective agents in social species.

Several hypotheses have been formulated that connect parasitism with mating systems. For example, polyandry (females mating with several males) produces increased genetic diversity among offspring. Polygyny (males mating with several females) is also common and produces similar results. Particular benefits may accrue when mating is selective, reflecting the tendency of females to garner resistance alleles for offspring from high-quality males (Hamilton & Zuk 1982). Interestingly, the degree of polygyny and exposure to severe pathogens correlates positively across a number of human societies (Low 1988). Social life and mating systems are particularly intertwined in social insects: polyandry and 'polygyny' (here meaning the presence of more than one queen per colony) correlate with the evolution of sociality and the potential for cooperation or conflict. From the perspective of the parasite the primary importance of sociality is that it aggregates individuals both in space and by genotype, increasing the chance of transmission and successful infection and establishment in a new host.

2. PARASITES AND VARIABILITY IN SOCIAL INSECTS

(*a*) *Social organization*

Social insects – essentially the social Hymenoptera (ants, bees, wasps) and the termites – are a large and diverse group. In the social Hymenoptera, mother queen(s) and their daughters (the workers) live together in colonies and cooperatively raise young that become either new (sterile) workers or sexual individuals. The organization of work in social insect colonies, based on different degrees of division of labour and polymorphism, affects parasitism and is in turn affected by parasites in ways yet to be explored (Schmid-Hempel & Schmid-Hempel 1993). For example, acute bee paralysis virus in honey bees is transmitted by trophallaxis (when workers transfer liquid food to one another) (Bailey & Gibbs 1964). When the colony increases foraging activity, trophallaxis is reduced and the disease dies out (Bailey *et al.* 1983). On the other hand, infection by the microsporidian parasite *Nosema apis* causes workers of

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the honey bee to start foraging earlier in life and thus alters the entire work profile of the colony (Wang & Moeller 1970). In the fire ant *Solenopsis*, parasitic flies (Phoridae) attack the majors (the large worker caste) which are costly to produce (Feener 1987, 1988). Feener thus suggested that such parasitism may select against caste differentiation. Workers infected by a fungal pathogen in termites (Kramm *et al.* 1982) or by a virus in the honey bee (Waddington & Rothenbuhler 1976) are more intensively groomed by their nestmates, which are thus more likely to become infected. This is suggestive of parasites being able to manipulate secretions and/or the social behaviour of their host to their own advantage. Finally, infected workers could threaten their colony by transmitting a disease to their queen. This should be particularly relevant in species where the colony depends on a single queen, as in the leaf-cutting ants. Interestingly, in all species of social insect, it is the young workers that attend the queen while the older ones forage (Wilson 1971). As the probability of being infected by a disease must increase with age and novel diseases can be picked up by the foragers, young workers should be less likely to be an infection risk. Thus, parasitism provides additional hypotheses for the adaptive value of the standard age-dependent division of labour (Schmid-Hempel & Schmid-Hempel 1993).

Transmission to larvae is necessary to complete the life cycle for some parasites of social insects. The microsporidian *Burenella* in fire ants has to infect the fourth instar larvae (Jouvenaz *et al.* 1981), while the honey bee mite *Varroa* must develop on young (Kraus *et al.* 1986). Transmission to such specific classes of host individuals depends on colony organization: in particular, on age-dependent division of labour. For example, when infection of a worker occurs early in life and growth of the pathogen within the host is fast, the parasite is transmitted by young workers that spend most of their time attending the tasks within the nest, such as brood care. When infection is late or parasite development is slow, the infection will be spread by older workers that spend most of their time foraging outside the nest. This timing thus affects the rate of transmission to other colonies in the population (horizontal transmission). Such transmission could occur when foragers get lost or intrude into other colonies (as is the case in nectar-robbing bees), or when transmission is linked with resource use.

The latter has recently been demonstrated in the case of bumble bees and their trypanosome parasite *Crithidia bombi*: Durrer & Schmid-Hempel (1994) experimentally showed that foragers become infected when they visit a flower that has previously been visited by an infected worker. *C. bombi* is common in natural populations of its host *Bombus* spp. (Shykoff & Schmid-Hempel 1991*b*). It causes a retardation of ovarian development (Shykoff & Schmid-Hempel 1992) and a loss in the production of daughter queens for a given size of the colony (P. Schmid-Hempel & C. B. Müller, unpublished observations). Workers, males and queens become infected by ingestion of infective cells. These can readily be picked up inside the nest through contact with contaminated larvae and nest material. Wu (1994) further showed that colonies of the bumble bee *B. terrestris* are infected by several strains of *C. bombi* at the same time. In standard medium, and presumably also in hosts (Wu 1994), these strains differ in their growth rate. Fast strains should thus easily outcompete slow strains within a single host. Schmid-Hempel & Schmid-Hempel (1993) hypothesized that strain variability might nevertheless be maintained if: (i) fast strains were generally less infective to new hosts, i.e. had a disadvantage in between-host transmission; or (ii) if workers from different colonies vary in the time when they are most infectious to others. The experimental tests showed that expectation (i) was not met, because transmission shortly after infection (1–3 d) was always more successful than transmission at a later time (4–12 d after infection). However, expectation (ii) was supported, because the time period after infection during which transmission to new hosts was most effective varied significantly among colonies, with some colonies acting as 'filters' for short transmission intervals and others for longer ones (Schmid-Hempel & Schmid-Hempel 1993). Hence, strain variability could be maintained through colony-level variation in the most effective transmission interval after infection.

(*b*) *Mating systems*

The breeding system of social insects has received increased attention because it provides information basic to the understanding of social evolution and speciation (see, for example, Ward 1989) and the evolution of sex ratios in natural populations (Boomsma & Grafen 1991). The current hypothesis is that sociality has evolved because of the effects of kin selection (Hamilton 1964). It is therefore expected that individuals within groups of socially living animals, such as workers of social insect colonies, are typically more closely related to each other than the population average. In social Hymenoptera the haplodiploid reproductive system can indeed ensure high degrees of relatedness. However, genetic evidence shows that social insect colonies often do not consist of closely related individuals as a consequence of polyandry and polygyny. The latter includes primary polygyny, foundress associations, and secondary polygyny (when additional females are admitted to the colony).

Several hypotheses for the adaptive value of multiple mating (polyandry) have been formulated. Crozier & Page (1985) propose that polyandry may be beneficial if the resulting increase in genetic variation among workers in the colony allows the expression of a better colony phenotype. This is the case when, in a fluctuating environment, increased genotypic variation expands the range of tolerable conditions. The studies of Robinson & Page (1988) indicate that different worker patrilines within a colony differ in their response to task-specific stimuli. Polyandry thus provides the possibility to acquire the necessary alleles so that the many tasks are fulfilled

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that are essential for successful development and reproduction. Polyandry may minimize the genetic load associated with the production of less fertile diploid males (Page & Metcalf 1982). Furthermore, polyandry may evolve as a consequence of sperm limitation, i.e. when queens require sperm from more than one male to be capable of producing large colonies. In ants, polyandry is indeed positively correlated with colony size (Cole 1983). However, this hypothesis raises the intriguing question of why males are not capable of producing or of transferring more sperm in the first place, and why they sometimes, in polyandrous aggregates, transfer far more sperm than the queen can accommodate, as for example in honeybees (Koeniger 1991). Yet other hypotheses suggest that polyandry reduces the queen–worker conflict over sex allocation to the advantage of the queen (Queller 1993).

Polygyny has similarly been explained by the production of advantageous genotypic variation at the colony level (Crozier & Consul 1976). Other hypotheses concentrate on the evolution of polygyny as a result of high dispersal risks and/or a low probability of independent colony founding for the dispersing queens (Pamilo 1991). Polygyny may also evolve when queens are comparatively short-lived with respect to the colony such that other related queens can take over (Nonacs 1988).

Quite generally, parasites are suspected to play a role in the maintenance of genetic variability of their host populations, because the production of diverse offspring helps the host to escape the constantly coevolving parasites (Hamilton *et al.* 1990; Ladle 1992). Offspring variability takes on a special meaning when dispersal is limited and sons or daughters stay at home to form social groups. Examples include the social insects, but also many other socially living organisms, such as social mammals or colonial Bryozoa. Consequently, several authors have suggested that within-colony genotypic variability in social insects, and hence polyandry and polygyny, may be the result of selection by parasites and pathogens (Tooby 1982; Hamilton 1987; Sherman *et al.* 1988).

(*c*) *Genotypic variability*

Variation in host resistance and/or parasite virulence has a genotypic component in almost any instance so far analysed (see, for example, Wakelin & Blackwell 1988). Although less intensively investigated than other organisms, social insects are no different in this respect. In the honey bee, for example, selection for increased resistance is possible against the microsporidian *Nosema* (Rinderer *et al.* 1983), acarine disease, hairless-black syndrome, or American foulbrood (Tanada & Kaya 1993). In addition, honey bees show natural variation in resistance, for example against the mite *Acarapis woodi*, foulbrood and microsporidia (Bailey & Ball 1991). For the corresponding pathogens and parasites, on the other hand, little is known about genotypic variation in infectivity or virulence.

Genotypic variation for resistance–virulence traits and genotype–genotype interactions are necessary preconditions for the parasite hypothesis of polyandry or polygyny to hold true. To test whether multiple mating actually reduces the effects of parasitic infection is straightforward in principle. However, to date only Shykoff & Schmid-Hempel (1991*c*) have tested whether the genotypic mix of bumble bee worker groups affects the spread of the parasite *Crithidia bombi*. The results demonstrated that transmission among relatives was indeed more likely than among non-relatives (figure 1*a*), suggesting that heterogeneous groups are less at risk. In addition, workers from nests that were naturally infected were more likely to become infected than those coming from nests that were not naturally infected, suggesting natural variation in susceptibility (figure 1*b*).

Further experiments support the idea that increased transmission among relatives is due not to behavioural interactions or any other kin effect, but to the matching of parasite strains and host genotypes involved. Shykoff (1991) compared transmission of *C. bombi* to relatives and non-relatives of bumble bee workers from two different nests infected with the same parasite. There was no difference in transmission success to related compared with unrelated target workers when confronted with the same strain of parasite. However, transmission success varied with the nest of origin of the parasite strain, indicating differences in the type of infection among colonies. More recently, Wu (1994) in a preliminary study found a colony–strain interaction effect in the survival of infected workers. Hence, in bumble bees infected by *C. bombi*, it is possible that a variety of parasite strains trickle into each colony through the foraging activities of the workers, for example with nectar brought

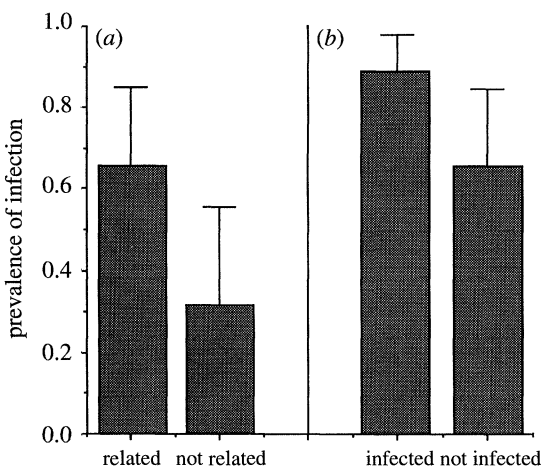

Figure 1. Success of transmission of *Crithidia bombi* in its host *Bombus terrestris* in relation to whether (*a*) the source of infection is a related or a non-related individual; (*b*) the exposed individual was from a colony that had or had not naturally acquired an infection. Bars denote average prevalence (95% c.i.) in worker groups exposed to the infection; small figures are numbers of groups tested. The differences are significant at $p < 0.05$ (from Shykoff & Schmid-Hempel 1991*c*).

back by the foragers (Durrer & Schmid-Hempel 1994). The particular genotypic mix of each colony will allow only certain strains to establish successfully; this would render polyandry potentially adaptive. In addition, the process must lead to an assortment of pathogen strains among colonies and creates the relatedness effect for the success of transmission (Shykoff & Schmid-Hempel 1991*a*).

3. THEORETICAL CONSIDERATIONS

(*a*) *Polyandry*

Consider a situation with ubiquitous horizontal transmission such that k different pathogen strains can infect colonies of hosts, and where the hosts have k different, corresponding resistance alleles. Colonies are supposed to contain a single queen mated randomly with a total of m males. Such a mating system will produce a mixture of homozygous and heterozygous workers in the colony. If the colony is infected by the corresponding pathogen strain (i.e. virulent with respect to the present host alleles), homozygotes are assumed to suffer and contribute less to the growth, survival and eventual reproduction of the colony with factor $w = 1 - s (w < 1)$, where s is the selection exerted by the pathogens against their individual hosts. Heterozygotes and those not infected by a virulent strain are assumed to contribute with factor 1. Imagine that these contributions affect the state, X, of the colony (e.g. its size) at time of reproduction. State X relates to fitness through a monotonically increasing 'fitness function', indicating the number of sexual individuals produced and their mating chances. As shown in the Appendix, with these assumptions, the expected state of a colony at time of reproduction is

$$\mathrm{E}(X) = 1 - \frac{sp}{2} \text{ and } \mathrm{Var}(X) = \frac{1}{4}\frac{s^2}{m}pq, \qquad (1)$$

where $p = 1 - q =$ the probability that a queen mates with a male having an allele identical to one of hers.

The fitness function is assumed to be the result of processes operating more or less independently of the parasites' effects. For example, large colonies may be disproportionately successful because they are well defended against predators, better able to survive drought periods, or ergonomically more efficient. For simple functions, fitness can be readily calculated. (i) Linear function: $W_{\mathrm{linear}} = \mathrm{E}(X) = 1 - sp/2$; fitness will be indifferent to mating number. (ii) Quadratic fitness function (see figure 2): $W_{\mathrm{quadratic}} = \mathrm{E}(X^2) = (s^2/4m)pq + (1 - sp/2)^2$. This will select against multiple mating with factor $1/m$. (iii) Truncation selection: colonies below a certain threshold size, $X < X_0$, are assumed to produce no reproductives, while those above all have fitness 1. This is the explicit case considered by Sherman *et al.* (1988). Fitness is proportional to the variance of the distribution as given by equation (1). Hence, multiple mating will be favoured if $X_0 < \mathrm{E}(X)$, and its benefits increase with $1/_m$, because this decreases variance and less is truncated. Conversely, multiple mating will be at a disadvantage if $X_0 > \mathrm{E}(X)$, because this reduces the tail of the distribution that is escaping truncation.

In the general case, fitness W scales with colony state X as $W = X^a$, where a is a shape parameter. Furthermore, the number of infections per colony (by the same or different strains) is now a random variable. Mating can be costly too: females in search of mates become exposed to predators and hazards of the weather, or simply have to expend a lot of time and energy. At least for the model situation, the costs of multiple mating need not only refer to actual costs incurred when mating. The potential for ergonomic inefficiency as a result of the resulting within-colony conflict, for example, would also qualify (Schmid-Hempel 1990). The appendix shows that for this general case, the expected fitness from mating strategy m is:

$$W_m = \mathrm{e}^{-c(m-1)}\left(\mathrm{e}^{-\mu f} + (1 - \mathrm{e}^{-\mu f}) \times \sum_{i=0}^{m}\left(1 - \frac{i}{2m}s\right)^a \binom{m}{i} p^i q^{m-i}\right) \qquad (2)$$

where $\mu =$ average number of infections per colony (a Poisson variable), $c =$ reduction of fitness for each additional mating, and $f =$ frequency of a particular, dangerous strain among the parasites. This equation has been evaluated numerically for a range of values of the parameters s, c, p, k, μ, and m. Figure 3 shows the characteristic result. Multiple mating is advantageous under conditions where costs of mating are low relative to parasite pressure (as indicated by a low ratio c/μ). More interestingly, multiple mating is favoured for values of a close to 0.5. The advantage of multiple mating is thus most evident when the shape of the fitness function implies diminishing returns from size, or in other words when colonies of intermediate size have a large share of the total reproductive

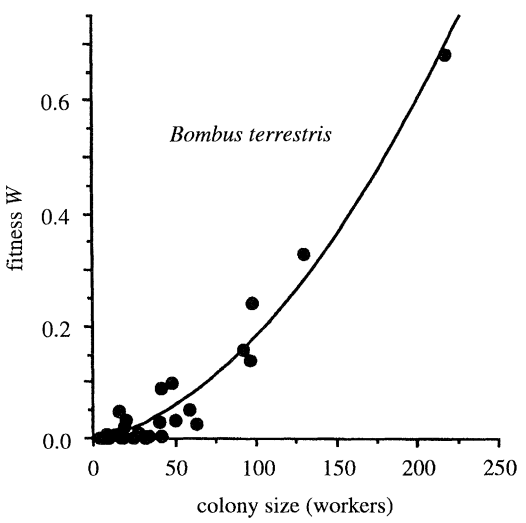

Figure 2. Estimated fitness function for the bumble bee *Bombus terrestris*, calculated according to the Shaw–Mohler equation from the number of sons and daughters produced in an experimental field population (from C. B. Müller & P. Schmid-Hempel, unpublished observations).

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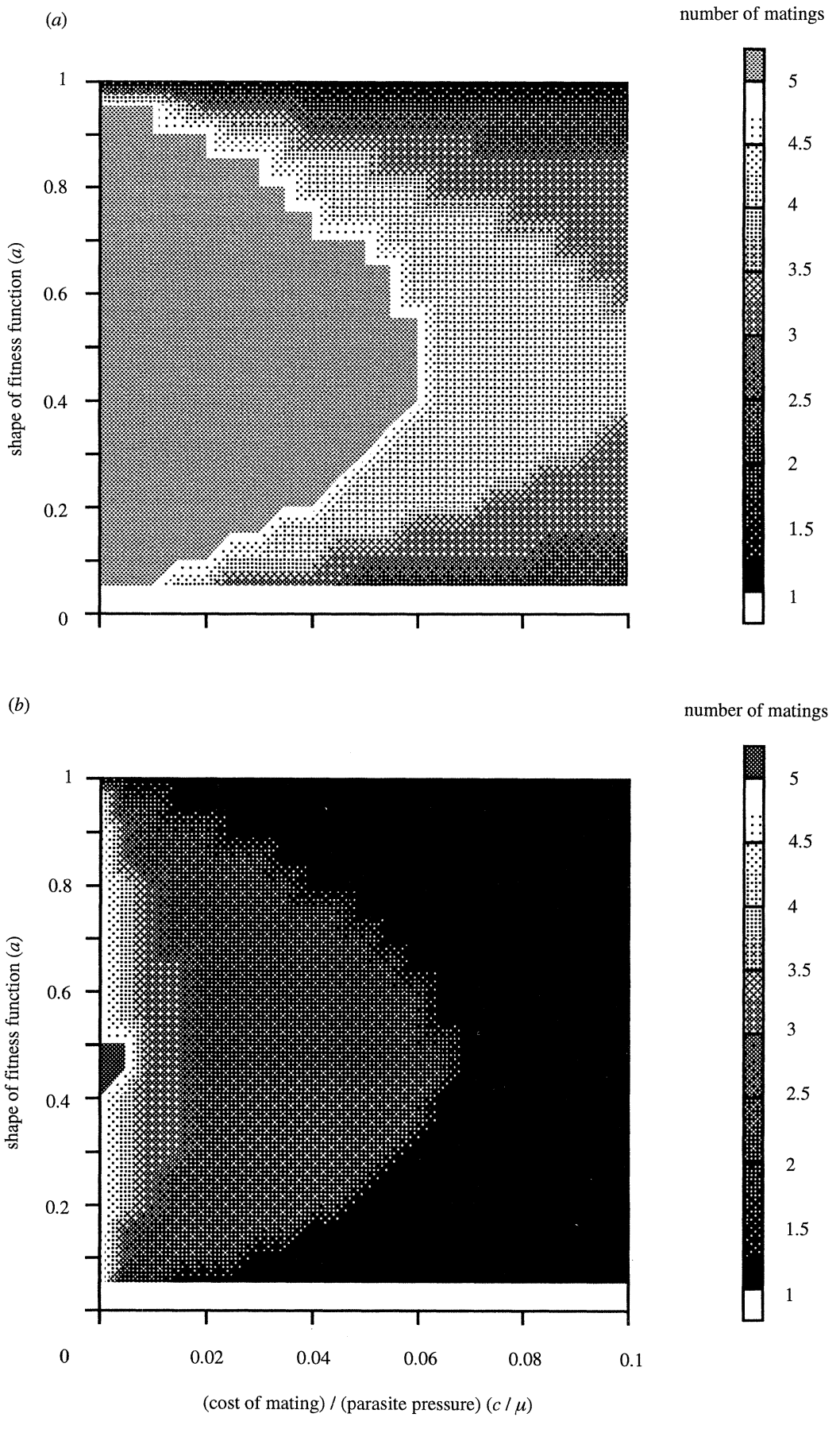

Figure 3. Number of matings (graded grey values) that yield maximum fitness as a function of the shape parameter of the fitness function (a), the costs per mating (c) and the average number of infections per colony (μ). Selection coefficient for both graphs, $s = 0.9$. (a) Small allelic diversity ($k = 4$); (b) large allelic diversity ($k = 16$).

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success in the population. This might occur, for example, because of limitation in the size of available nesting sites. Note also that if multiple mating is advantageous, the best number of matings is relatively low.

In the numerical analyses, further assumptions can be relaxed. For example, what happens if queens are allowed to mate with a random number of males, m, and colonies are subject to a Poisson-distributed number of infections, of which any number of strains, $0 \ldots n$, could be virulent (i.e. when a strain matches an allele among the different worker lines)? For these cases, numerical simulations based on populations of 500 colonies were run. The results suggested the same qualitative pattern.

(*b*) *Polygyny*

Polygyny is somewhat different from polyandry because associating with other females must increase rather than decrease the risk of contracting a disease for the colony. Hence, polygyny must be advantageous for other reasons. The requirement is that each queen within a multiple female association would on average enjoy a greater success than singly nesting queens. This could occur as a result of nest site limitation or predation pressure on incipient colonies. Given that polygyny evolves, parasites could counteract the kin-selected advantages of associating with related females for the same reasons as discussed before, i.e. increased transmission as a result of genetic homogeneity of the colony. Relatedness under polygyny does indeed vary considerably. In some epiponine wasps, for example, a high degree of relatedness is maintained by cyclical oligyny (Hughes *et al.* 1993), but in *Ropalidia* serial polygyny leads to relatively low degrees of relatedness (Gadagkar *et al.* 1993), whereas the multiple queens in the ant *Solenopsis invicta* seem not to be related at all (Ross 1993).

The simplest formal treatment of the polygynous case assumes that the expected fitness per queen is an increasing function, at least up to some point, of the number of females (F) present in the colony. If queens are not related to each other and are each mated to a single, unrelated male, the probability that a given queen has mated with a male that carries an allele matching one of the queen's is again $p = 2/k$. Among F queens in random association, the expected number of queens with matching males is $\mathrm{E}(F') = Fp$, with variance $\mathrm{Var}(F') = Fpq$, where $q = 1 - p$ as before. The expected frequency of homozygous workers in the colony is then $p/2$, that of heterozygotes $1 - (p/2)$. Similar to polyandry, equation (1) would hold if m were replaced by F, and expected fitness corrected by $1/F$. It follows that concave fitness functions would increase the benefits of polygyny whereas convex fitness functions (as in figure 2) would favour an intermediate degree of polygyny. The number of queens (F) that maximizes individual fitness can be calculated by combining equations (1) or (2) with assumptions about the increase in success with the size of the association.

4. REPERCUSSIONS

Hamilton & Zuk (1982) suggested that female mating preferences would ensure that offspring were endowed with good genes to resist parasites. The present analysis illustrates a further situation where the genetic composition of the group in terms of variability immediately affects the success of its members. The case of social insects can thus stand as a model system for highly structured populations consisting of family groups.

The formal analysis suggests that the benefit of multiple mating (and polygyny) depends on the shape of the 'fitness function' that relates colony state after parasite selection (e.g. size at reproduction) to reproductive success. Although it has been noted several times that colony size correlates positively with reproductive success (Schmid-Hempel *et al.* 1993), it is the actual shape of the distribution that is effective. In particular, polyandry is selected for when fitness is a concave (decelerating) function of colony size, as independently suggested by Sherman *et al.* (1988). The explicit case considered by Sherman *et al.* (1988), i.e. truncation selection, has also in a different context been identified as favouring genetic variability in relation to parasitism (see, for example, Hamilton *et al.* 1990).

This analysis has neglected a number of aspects of standard epidemiology, such as density-dependent transmission, immunity and heterogeneities (Anderson & May 1991), for reasons of practicability and simplicity, and concentrated instead on the problem of variance reduction (Gillespie 1977). In addition, immunity in the sense of providing long-lasting protection against repeated infections is virtually absent in insects (Gupta 1986). On the other hand, mating strategies involve not only the problem of how many males to mate with, but also the choice of mates. This can lead to a non-random assortment of genes in the population; the advantages of multiple mating should be enhanced by this. Polygyny similarly involves choice of associates. So far, this has been analysed only from the perspective of kin selection and reproductive conflict (see, for example, Keller 1993). It is clear that more detailed theoretical studies will be necessary to fully understand the long-term dynamics of the system.

Some of the predictions formulated with the present analysis are amenable to empirical verification. For example, the bumble bee *B. terrestris* depicted in figure 2 seems to be a singly mating species as expected (Röseler 1973; Estoup *et al.* 1994). Such figures for the production of males and daughter queens are rarely available for the social insects. Under certain circumstances, an argument could be made that cavity-nesters would be more likely to have a concave fitness function favouring multiple mating, whereas those species nesting in the open would attain convex curves (as in figure 2). In the genus *Apis*, *A. mellifera* and *A. cerana* are cavity nesters with a range of 10–12 matings. *A. florea*, with 3–4 mates, and *A. andreniformis*, with 7–8 matings, both nest on branches in the open (Koeniger 1991; Otis 1991). These differences are thus

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in line with the predictions. Note that a similar expectation would result if cavity nesting were associated with increased chances to contract durable spores of diseases, because cavities might be reused by colonies more often than the corresponding open locations on branches. As durability is also expected to lead to increased virulence of the pathogen (Ewald 1993), this too would make multiple mating more advantageous for cavity nesters. In addition, geographical variation in mating frequencies might occur in regions of the parameter space (figure 3) with steep clines such that small variation in parasite pressure or mating costs could tip the balance. Hence, the parasite hypothesis would also suggest that the degree of relatedness among members of a colony should vary locally in response to parasite pressure. It remains to be verified how important parasites are relative to the other hypotheses mentioned in the introduction.

Finally, parasites and genetic diversity within colonies are intriguing issues with respect to recognition and discrimination for or against certain classes of individuals. For example, workers infected by bee paralysis elicit aggressive responses in their nestmates (Drum & Rothenbuhler 1985). Presumably, this overrides kinship. Hence, kin can be a bane or boon with respect to disease, and discrimination for or against kin is a dilemma. Diseased relatives should be evicted from colonies, but at the same time the risk of contracting novel infections by admitting aliens should be minimized. If the main purpose of discrimination is to avoid new infections, we should expect that discrimination against workers from other colonies that represent untried and potentially dangerous associations would always be stronger than within-colony discrimination against members of different patri- (under polyandry) and matrilines (polygyny), regardless of the actual degrees of relatedness. This pattern seems to emerge from many independent studies (see, for example, Carlin *et al.* 1993). It has been pointed out that the genetic polymorphism underlying kin recognition seems to be inherently unstable and may only be maintained if linked, for example, with loci under selection by parasites (Crozier 1987).

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APPENDIX

The worker's contribution is to increase the size (or state) of the colony, X, each proportional to the effort she is able to give. However, for convenience the colony sizes reached if all workers are uninfected or infected are scaled to unity and to $w = 1 - s$ respectively, s being a measure of the average fractional weakening caused by the pathogens to a susceptible host. Each colony is exposed to a random sample of the k pathogen strains, but at most one of the infections is assumed to be dangerous for the homozygotes (this latter assumption can be relaxed in numerical simulations).

The weakening caused to individual workers is presumed to be additive in reducing the size of the colony. Each queen through her polyandrous mating can be considered to be sampling the genes of the population at random. Under the assumed additivity

the mean achievement of queens building their colonies, $E(X)$, will obviously be independent of the number of males (m) they sample and will depend on (i) the chance that a sampled male has an allele the queen carries (p) (the number of alleles, k, in the population being large, the frequency of homozygous queens are taken as negligible), and (ii) the independent chance that the queen's gamete contributed to the worker carries the same allele as the male (1/2). Mean colony size is therefore $E(X) = 1 - sp/2$.

The variance of achievements, $\mathrm{Var}(X)$, however, reduces with m. If colony sizes are large and queens mate with single males, the variance of colony size is that of a binary variate taking values 1 and $1 - s/2$ with frequencies $q = 1 - p$ and p; thus $\mathrm{Var}(X) = (s^2/4)pq$. For the case where the queen mates with m males, the variance of the mean of m such varying sets is therefore $\mathrm{Var}(X) = (s^2/4m)pq$. For the case of quadratic dependence of colony fitness on colony size we need: $E(X^2) = \sum_i X_i^2 P(X = X_i)$. This expression can be found by reversing the 'correction' that provides a variance from a simple sum of squares: $E(X^2) = \mathrm{Var}(X) + [E(X)]^2$. Hence, $E(X^2) = (s^2/4m)pq + (1 - sp/2)^2$.

In the general case, when colony fitness depends on colony size with a power function, exponent a, it is easier to consider the explicit formulation. The queen's achievement when she mates with m males of which i have an identical allele to one of hers is

$$X_i = \frac{1}{2}\left(\frac{i}{m}w\right) + \frac{1}{2}\left(\frac{i}{m}1\right) + \left(1 - \frac{i}{m}1\right) = 1 - \frac{i}{2m}s.$$

Under these conditions, the expected colony fitness, W_m, is

$$W_m = E(X) = \sum_{i=0}^{m} X_i P(X = X_i)$$

$$= \sum_{i=0}^{m}\left(1 - \frac{i}{2m}s\right)^a \binom{m}{i} p^i q^{m-i}. \qquad \text{(A1)}$$

Colonies are infected by an average of independently acquired, Poisson distributed parasite strains. The probability of the colony not being infected is therefore $P_0 = e^{-\mu}$. Suppose that among the strains the frequency of a particular one (or a set of several) dangerous for a colony is f. The number of infections by this strain (or set) will also be Poisson distributed with mean μf. Hence, the probability of a colony receiving no infections by this strain (or set) is $P_A = 1 - e^{-\mu f}$. Since infection is assumed to build up rapidly within a colony, all other cases than zero dangerous infections can be considered equivalent, i.e. the colony becomes totally infected. Moreover, P_1 = probability that the colony is infected but the corresponding strain to which workers are susceptible is absent, hence $P_1 = e^{-\mu f} - e^{-\mu}$. The expected fitness for the colony is therefore: $W = (P_0 \cdot 1) + (P_1 \cdot 1) + (P_A \cdot W_m) = (e^{-\mu f}) + (1 - e^{-\mu f})W_m$. The qualitative conclusions are the same if infections are, for example, negatively binomially distributed.

Suppose, in addition, that mating has an independent cost such that a single mating is scaled to unity and each additional mating reduces fitness by a constant amount c. In this case, the expected colony fitness for a queen accepting m matings is:

$$W = e^{-c(m-1)}\left[e^{-\mu f} + (1 - e^{-\mu f}) \times \sum_{i=0}^{m}\left(1 - \frac{i}{2m}s\right)^a \binom{m}{i} p^i q^{m-i}\right]. \qquad \text{(A2)}$$

Dominant-recessive genetic system

In seeking the mean colony size for the whole population, recall that, in whatever colony they are produced, all worker contributions in the population are additive. Thus since susceptible *aa* workers have frequency q^2, to obtain the mean, the standard colony fitness (1) is decreased proportional to this: $E(X) = 1 - sq^2$. In a linear fitness function, this is also the mean colony fitness. In the quadratic fitness function, we need first to find the variance; for this we need to know the frequencies of the different kinds of female broods that can emerge from matings with a single male. Given Hardy–Weinberg frequencies, *aa* broods have frequency q^3, broods with '*A*' phenotypes and *aa* in 1:1 ratios have frequency $2pq^2$, and all other broods are entirely of '*A*' phenotype, frequency $1 - 2pq^3 - q^3$. The sizes of those types of colony are $1 - s$, $1 - s/2$, and 1; on the basis of these, together with their frequencies, via some algebra, we find

$$E(X^2) = 1 + sq^2\left[s\left(\frac{1}{m}p + q\right) - 2\right].$$

Hence, similar conclusions would also follow for this genetic system.