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Haplodiploidy, Eusociality and Absence of Male Parental and Alloparental Care in Hymenoptera: A Unifying Genetic Hypothesis Distinct from Kin Selection Theory

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Haplodiploidy, eusociality and absence of male parental and alloparental care in Hymenoptera: a unifying genetic hypothesis distinct from kin selection theory

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SUMMARY

Beginning with Hamilton (*J. theor. Biol.* **7**, 1–52 (1964)), evolutionary biologists have attempted to explain the apparent predisposition for the haplodiploid Hymenoptera to evolve both eusociality and female workers. As an alternative to kin selective, pre-adaptational, or ecological explanations for this association, I propose a new genetic hypothesis, the *protected invasion hypothesis*: dominant alleles for maternal care in finite haplodiploid populations are more resistant to loss from genetic drift than are paternal-care alleles in haplodiploid populations or than are either maternal or paternal-care alleles in diploid populations. Similarly, dominant alleles for female alloparental care in finite haplodiploid populations are more resistant to loss from genetic drift than are male alloparental alleles in haplodiploid populations or than are (male or female) alloparental alleles in diploid populations. A Markov model of phenotypic evolution describing the step-wise progress of a population toward one of two adaptive peaks demonstrates that even small differences in fixation probabilities among these alleles can translate into large differences in the long-run probabilities of observing the corresponding parental or alloparental strategies. Thus the protected invasion hypothesis immediately explains all of the peculiar social features of the haplodiploid Hymenoptera, namely: (i) the overwhelmingly greater tendency for maternal care than paternal care in Hymenoptera; (ii) the greater propensity for eusociality (alloparental sibling care) in Hymenoptera than in diploid insects; and (iii) the greater likelihood for females than males to become alloparents (workers) in the Hymenoptera. The hypothesis also correctly predicts (iv) the apparently higher frequency of paternal care in diploid species than in haplodiploid species, and (v) the lack of a sex-bias among workers of eusocial diploid species.

The protected invasion hypothesis is distinct from relatedness-based explanations and provides a more comprehensive explanation for the repeated appearance of the distinctive social structures of the Hymenoptera than does the kin selection model. I show that the bias toward eusociality in Hymenoptera is produced by protected invasion effects even when there is no female-biased sex ratio and no asymmetry between a female's relatedness to its siblings and to its own offspring. In addition, protected invasion effects create a bias for female versus male workers within the Hymenoptera even when there is no asymmetry between a female's and male's relatedness to its siblings. Furthermore, protected invasion effects create a bias toward eusociality in haplodiploid versus diploid populations even when the queen mates an indefinite number of times and there is no difference between haplodiploid and diploid colonies in the relatednesses of workers to their tended brood. Finally, the protected invasion hypothesis explains a phenomenon that cannot be explained by kin selection theory: the surprising overwhelming preponderance of maternal over paternal care in the Hymenoptera (because male and female parents have the same mean relatedness to their offspring when the female mates singly). An important implication of the protected invasion hypothesis is that synergistic co-operation among siblings is more likely to evolve in haplodiploid than in diploid species.

1. INTRODUCTION

Much of the initial enthusiasm for Hamilton's (1964) inclusive fitness theory for the evolution of co-operation among relatives was fuelled by the elegant way in which the theory appeared to account for some of the distinctive social structures of the haplodiploid Hymenoptera. In particular, there was considerable intuitive appeal to the idea that, by causing full sisters (but not full brothers) to be more highly related to each other ($r = 3/4$) than to their own offspring ($r = 1/2$), haplodiploidy could account both for the apparent bias toward eusociality in the Hymenoptera (compared with diploid insects) and for the overwhelming

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tendency for eusocial hymenopteran workers to be female. Since its inception, this 'the three-quarters relatedness hypothesis' (West Eberhard 1975) for the link between eusociality and haplodiploidy has encountered two difficulties. First, the hypothesis requires that colonies exhibit female-biased sex ratios, so that workers can capitalize on their relatively high relatednesses to sisters. Trivers & Hare's (1976) theory of worker sex ratio control provided one such mechanism for achieving female-biased sex ratios, but it was pointed out that post-ovipositional sex-ratio control would most likely evolve after the evolution of eusociality (Crozier 1977, 1982). Even worse, early population-genetic models suggested that the female-biased sex ratios envisioned by Trivers & Hare in fact would not promote the evolution of eusociality (Craig 1979) because at the female-biased, population sex-ratio equilibrium, the value of a female's brothers (which includes the mating success of the latter) would still equal the value of her sisters, despite her lower relatedness to brothers. Additional population-genetic models of kin-selected altruism suggested that male workers should evolve as readily as female workers in haplodiploid species (Craig 1982), apparently further loosening the proposed coupling between haplodiploidy and the idiosyncratic social behaviours of the Hymenoptera. These results stimulated theorists to seek new mechanisms generating female-biased sex ratios that were truly capable of catalysing the evolution of eusociality and female workers. For example, Seger (1983) proposed that, in partially bivoltine species, the mating of first generation males with second generation females would increase the value of females relative to males in second-generation broods, leading to female-biased sex-ratios in second-generation broods. The locally female-biased sex ratios of second-generation broods could then give impetus to the evolution of eusociality. In a similar vein, Godfray & Grafen (1988) suggested the occasional occurrence of colonies with unmated, male-producing, queens would cause selection for female-biased broods in colonies with mated queens, thus facilitating eusociality. Although there is some empirical support for these recent attempts to revive the link between haplodiploidy and eusociality (e.g. Brockmann & Grafen 1992), questions remain about their general applicability, as specific life-histories or mating patterns are required for the explanations to work.

The second major problem facing the three-quarters relatedness hypothesis is that the relatedness asymmetry favouring altruistic sisters quickly breaks down when queens mate more than once (Alexander & Sherman 1977; Page 1986; Ross 1986) or multiple females reproduce within the colony (Hölldobler & Wilson 1990; Keller 1993). Indeed, a fairly large body of evidence now indicates that, because of the latter reasons, many if not most hymenopteran females tend brood that are no more (and often less) closely related to themselves than are their own offspring (Strassmann *et al.* 1989; Gadagkar 1990; Hölldobler & Wilson 1990; Ross & Carpenter 1991).

Within the past fifteen years the three-quarters relatedness hypothesis has generally fallen out of favour (although the general theory of kin selection certainly has not). The emphasis in explanations for the apparent bias toward eusociality in the Hymenoptera has shifted from their unusual genetic structure to unusual aspects of their ecology or to their distinctive biological attributes, such as their propensity for nest-building and maternal care (e.g. Alexander 1974; Lin & Michener 1972; West Eberhard 1975; Evans 1977; Crozier 1982; Andersson 1984; Alexander *et al.* 1991). Parallel approaches have been used to explain why male workers are so rare in the social Hymenoptera. For example, Starr (1985), following Alexander (1974), proposed that the stinger pre-adapted females for worker roles, while Kukuk *et al.* (1989) argued instead that ecological factors promoting multiple-mating strategies by males prevented the evolution of male workers.

The latter second-generation explanations for eusociality and female workers in Hymenoptera have some appeal, but have theoretical limitations of their own. For example, explaining the bias toward eusociality in Hymenoptera by the widespread, pre-adaptive, occurrence of maternal care, or explaining the predominance of female workers by their pre-adaptive possession of a stinger, ignores the possibility that selection for these attributes merely reflect selective factors that can in the extreme also favour eusociality. Maternal care, nest building and stinger possession are themselves phenotypes subject to evolution (see also Kukuk *et al.* 1989), and thus it may be that these attributes are better seen as *reflections*, rather than as *causes*, of the processes that also underlie the hymenopteran biases toward eusociality and female workers, respectively. Certainly, it seems that such attributes would be much more evolutionarily labile than would the genetic system (haplodiploidy), as evolutionary alterations of the latter would likely require passage through a deeper selective valley. More appealing are ecologically based explanations for the hymenopteran eusociality. However, purely ecological explanations for the peculiar social structures of the Hymenoptera generally have failed to show convincingly how the ecological pressures bearing on hymenopterans systematically differ from those bearing on diploid insects. The recent demonstration of eusociality and a possible tendency for female-biased worker behaviour in a different haplodiploid order, i.e. in Australian gall thrips (Crespi 1992), has placed a greater strain on purely ecological (and preadaptational) hypotheses for the haplodiploid bias toward eusociality and female workers.

I propose a new genetic hypothesis for the biases toward eusociality and female workers in the Hymenoptera. This hypothesis also can explain the relative lack of male parental care in Hymenoptera (in comparison with diploid species such as termites or dung beetles). My hypothesis refocuses our attention on the haplodiploid genetic system of the Hymenoptera, but in a way that is very different from a relatedness-based approach.

The key to my approach is a new way of addressing the general question: why are we more likely to see certain phenotypes rather than others in nature?

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Traditionally, this question has been answered theoretically by considering a set of conceivable alternatives to the observed phenotype and modelling the conditions under which the observed phenotype has a higher fitness than these assumed alternatives. In particular, we expect the observed phenotype to be observed with probability 1.0 if it exhibits the highest fitness. More formally, we might say that the probability of observing phenotype A over phenotype B is 1.0 if A's fitness exceeds B's fitness and is zero if the reverse is true.

Thus, eusociality has been claimed to be especially likely to arise in the Hymenoptera, because under certain conditions such as those mentioned above, the unusually high relatedness among females generated by the haplodiploid genetic system makes it more likely that an altruistic worker's inclusive fitness (or, in classical population genetic terms, its neighbour-modulated fitness (Maynard Smith 1982*a*)) will exceed that of a non-altruistic female. When the compared inclusive fitnesses are subtracted from each other, Hamilton's rule for the spread of worker altruism is obtained: i.e. $rb-c>0$, where r is the coefficient of relatedness of a female worker to the tended brood relative to its relatedness to its own offspring, b is the increase in number of brood produced due to the actions of the altruistic worker, and c is the cost to the worker in terms of the number of the worker's own offspring lost as a result of the altruistic acts (Grafen 1984). In this view, the magnitude of the difference $rb-c$ (or, in general, the magnitude of the fitness difference between any two strategies) is not deemed important for assessing the likelihood of observing one of the two strategies. Only the sign of the difference is considered relevant as the sign tells us which strategy spreads; the magnitude of the difference informs us only of the rate of spread (loosely, the strength of the selective force).

However, when populations are finite, the magnitude of the selective effect usually will influence the long-run probability of observing a phenotype. The simple reason for this is that a phenotype in a finite population may fail to spread to fixation either (i) because it is selectively disfavoured relative to the alternative(s) (i.e. the sign of the selective differential is negative), or (ii) because the phenotype is lost due to the chance effects of genetic drift (even if it is selectively favoured). The probability of losing a favoured phenotype through genetic drift will depend strongly on the magnitude of the selective difference between it and its alternative(s); the stronger are selective effects compared to genetic drift effects, the greater the likelihood that the phenotype will spread to fixation instead of being lost (Kimura 1962).

Focusing on the latter point, I propose that the haplodiploidy generates biases in favour of female sibling co-operation (i.e. eusociality) and against male parental and alloparental care because the haplodiploid genetic system ensures that dominant alleles for these traits are especially protected from random loss (female co-operation) or are especially vulnerable to random loss (male parental and alloparental care) when such alleles are rare. I refer to this general hypothesis as the protected invasion hypothesis.

The protected invasion hypothesis has two parts. First, I show that the probability of ultimate fixation of an advantageous, mutant dominant allele for female parental care in haplodiploid populations will be greater than that of mutant alleles for male parental care in haplodiploid species as well as that of alleles for either male or female parental care in diploid species. Second, I will show that the probability of fixation of a mutant allele for female sibling co-operation in haplodiploids will exceed the corresponding fixation probabilities for male or female sibling co-operation in diploids and male sibling co-operation in haplodiploids, markedly so when co-operation synergistically (nonlinearly) enhances colony output.

In a third part of this paper, I show how even small differences in fixation probabilities for two distinct strategies may result in large differences in the long-run probabilities of observing the two strategies. Thus, the low frequency of male parental and alloparental care and the relatively high frequency of female co-operation in haplodiploid species is explained ultimately by the way the haplodiploid genetic system alters the fixation probabilities of these social strategies. Furthermore, I will show that these effects do not depend on the asymmetries in genetic relatedness generated by haplodiploidy, i.e. that the invasion protection hypothesis stands as a genuine alternative to relatedness-based hypotheses for enhanced female–female sibling co-operation and reduced male–male sibling co-operation in haplodiploid versus diploid species. In addition, the invasion protection hypothesis, unlike a kin selection hypothesis, can account for the apparent rarity of male parental care in the Hymenoptera; thus, the former provides a more unified explanation for the repeated appearance of the peculiar social structures of the haplodiploid Hymenoptera.

2. THE PROTECTED INVASION MODEL

(*a*) *Fixation probabilities for advantageous alleles in finite populations*

Kimura (1962) and Crow & Kimura (1970) have shown that the probability of fixation of a new, advantageous mutant allele in a finite population of variance-effective size N_e can be derived from the Kolmogorov backward equation used in the physical theory of diffusion. In the application of diffusion theory to population genetics, a mutant allele is viewed as subject to two forces: a deterministic force arising from natural selection that tends to drive the allele to fixation, and a stochastic force arising from genetic drift that tends to change allele frequencies in random directions, sometimes leading to complete loss of the allele from the population. The probability u that a new mutant allele ultimately will spread to fixation in a population of effective size N_e under the joint influence of selective and drift forces, is approximately given by

$$u = \int_0^{\iota} G(x)\mathrm{d}x \Big/ \int_0^1 G(x)\mathrm{d}x, \qquad (1)$$

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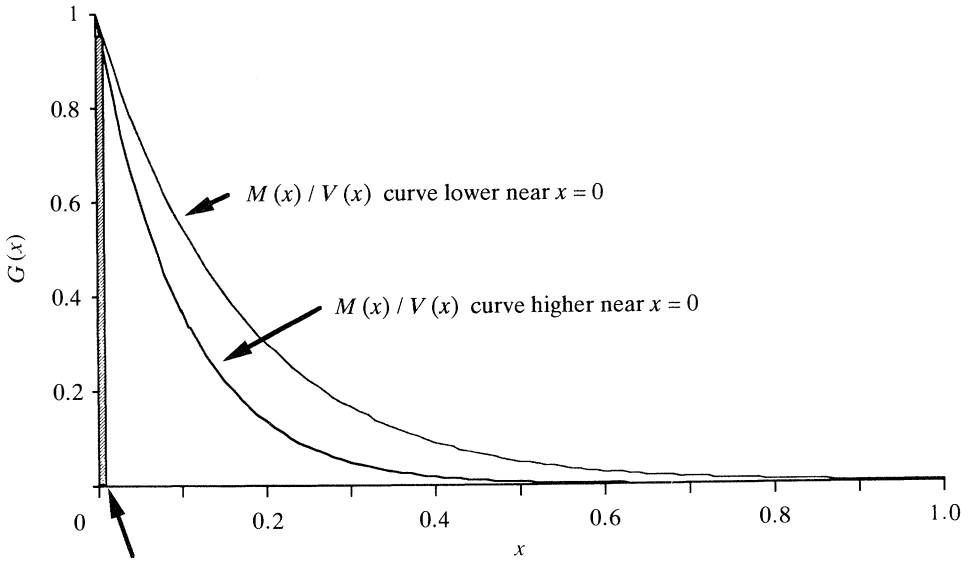

Figure 1. Graphical representation of equation (1) for the fixation probability of a new mutant allele.

Fixation probability $\approx t/\int_0^1 G(x)\mathrm{d}x$.

$M(x)$ is the deterministic change in gene frequency x due to natural selection, and $V(x)$ is the variance in gene frequency due to sampling error (genetic drift). $G(x)$ is $\exp(-2\int M(x)/V(x)\mathrm{d}x)$. The fixation probability is that fraction of the area under $G(x)$ from $x=0$ to $x=1$ that lies between $x=0$ and $x=t$, where $t=1/$(total number of alleles in the gene pool).

in which

$$G(x) = \exp\{-2\int(M(x)/V(x))\mathrm{d}x\}, \qquad (2)$$

and where $M(x)$ is the deterministic change in gene frequency due to selection at a gene frequency x, $V(x)$ is the variance in gene frequency due to genetic drift at a gene frequency x and t is the beginning frequency for a new mutant allele ($2/(3N_e)$ for haplodiploids and $1/(2N_e)$ for diploids).

Although equation (1) looks complicated, it has a simple graphical interpretation: the fixation probability is the area under the exponentially declining curve $G(x)$ between $x = 0$ and $x = t$, divided by the total area under the same curve between $x = 0$ and $x = 1$ (figure 1). The indefinite integral in the exponent of $G(x)$ measures the area under the curve $M(x)/V(x)$, and this area will be greater the higher on the y-axis is this curve. The greater the area under $M(x)/V(x)$ for a given interval, the larger will be the negative exponent in equation (2), and, consequently, the more steeply $G(x)$ will decline with increasing x (figure 1). The more steeply $G(x)$ declines, the larger will be the area of $G(x)$ that lies between 0 and t as a fraction of the area of $G(x)$ that lies between 0 and 1 (if N_e is not too small, the area between 0 and t will be nearly constant, approximately $G(0) \cdot t = t$), and therefore the larger will be the fixation probability (figure 1). Thus, the fixation probability (for a given t) increases with the height of the curve described by $M(x)/V(x)$, which is sensible because $M(x)/V(x)$ measures the strength of selection relative to the strength of genetic drift (at gene frequency x).

The first step in deriving ultimate fixation probabilities for the social strategies considered here is to find the function $M(x)/V(x)$ appropriate to each strategy and to the genetic system. $M(x)$ is simply the deterministic change in gene frequency as a function of the gene frequency x. $V(x)$ is simply the variance of a binomial proportion where the sample size is the size of the gene pool, i.e. $x(1-x)/(3/2)N_e$ for haplodiploid species and $x(1-x)/2N_e$ for diploid species. (Note that the effect of male haploidy in reducing overall effective population sizes of haplodiploid populations relative to diploid populations is accounted for by the coefficients of N_e, i.e. 3/2 (haplodiploid populations) and 2 (diploid populations). Thus, N_e in all formulae refers only to the number of breeding individuals, and thus comparisons of fixation probabilities in diploid versus haplodiploid populations with the same N_e assumes only that the two kinds of populations have a similar number of breeding individuals (as is reasonable if each species' ecological niche has a limited number of spaces for a limited number of individuals). Below, it will be further demonstrated that differences in fixation probabilities between haplodiploid and diploid populations are not sensitive to the differences in N_e between these populations if both N_es are sufficiently large.) I begin by examining fixation probabilities for male or female parental care strategies in haplodiploid and diploid species.

(*b*) *Male and female parental care in haplodiploids and diploids*

(i) *Haplodiploids*

I begin by imagining a dominant, advantageous, mutant allele A arises that causes haplodiploid, singly mated females to initiate (or exhibit enhanced) maternal care. A dominant (or at least a codominant) advantageous allele is assumed because such an allele

Table 1. *Mating frequencies and associated relative reproductive outputs for parental and alloparental strategies for males and females in haplodiploid species*

(Singly mated queens, no worker reproduction and equal sex investment ratios are assumed. The parental or alloparental allele A (allelic to non-parental or non-alloparental allele (a)) is assumed to be dominant and occurs with frequency $p = 1 - q$. Reproductive outputs (k) are relative to a reproductive output = 1.0 for non-parental or non-alloparental strategies. For sibling co-operation, k is a function $k(z)$ of the proportion z of (same-sexed) co-operators in the colony.)

mating type (female × male)	freq.	offspring genotypes		reproductive output			
		female	male	maternal care	paternal care	sibling care (females)	sibling care (males)
$Aa \times a$	$2pq^2$	1 *Aa*: 1 *aa*	1 *A*: 1 *a*	k	1.0	$k(0.5)$	$k(0.5)$
$Aa \times A$	$2p^2q$	1 *AA*: 1 *Aa*	1 *A*: 1 *a*	k	k	$k(1.0)$	$k(0.5)$
$aa \times a$	q^3	*aa*	*a*	1.0	1.0	1.0	1.0
$aa \times A$	pq^2	*Aa*	*a*	1.0	k	$k(1.0)$	1.0
$AA \times a$	p^2q	*Aa*	*A*	k	1.0	$k(1.0)$	$k(1.0)$
$AA \times A$	p^3	*AA*	*A*	k	k	$k(1.0)$	$k(1.0)$

is much more likely spread than a recessive advantageous allele (as we are interested in the long-run probability of observing maternal care, we must consider the events most likely to produce maternal care, because such events will dominate on evolutionary timescales.) For example, suppose that the multiple loci potentially promoting maternal care in a population lacking maternal care are divided into those which predispose this behaviour by means of recessive alleles and into those which do so by dominant alleles (letting n_r and n_d represent, respectively, the numbers of each kind of locus). Let u_r and u_d be the probabilities of appearance and fixation of (advantageous) mutant recessive and dominant maternal-care alleles, respectively, over a long period T. The overall probability that at least one maternal care allele will become fixed in a population by the end of the period T will be approximately equal to $1 - e^{-n_r u_r - n_d u_d}$. Now because $u_r \ll u_d$, this probability is roughly equal to $1 - e^{-n_d u_d}$ (if n_r is not much larger than n_d), meaning that the overall probability of maternal care depends primarily on probabilities of fixation of dominant alleles. Indeed, this is a possible explanation for why favoured alleles in natural populations (such as alleles underlying melanism in pepper moths) generally tend to be dominant (Hedrick & McDonald 1980; Hedrick 1983). (Analogously, assume that there are multiple switches that will turn on a light. A few switches turn on the light with a relatively high probability; the rest turn on the light with very low probability. The overall probability that the light will be turned on after random flippings of switches will depend primarily on the number of times that the high-probability switches are flipped.)

We thus seek the probability that the advantageous, dominant mutant parental-care allele A ultimately will be fixed in a finite population. (In all models below, mutations are assumed to occur in the germ line subsequent to soma formation and up to the first divisions of gametogenesis; mutations occurring too early in development would cause the behaviour to occur when there is no chance of the same gene occurring in the affected kin.) I will assume for simplicity that (for both this calculation and all those below) selection is weak enough that the mating frequencies by genotype are approximately those expected from random mating and Hardy-Weinberg genotype frequencies. The different mating types by genotype and their frequencies are given in table 1. When a female possesses an A allele, she cares for her offspring and increases her offspring output by a factor k compared to the output of non-caring females.

The change in the frequency of A after selection (Δp) is equal to [(2/3) (mean frequency of A alleles in females after selection) + (1/3)(mean frequency of A alleles in males after selection)]/(total frequency of alleles after selection) $- p$, where p is the frequency of A before selection. From table 1,

$$\Delta p = \frac{(2/3)\sum_i f_i m_{i,\mathrm{F}} w_i + (1/3)\sum_i f_i m_{i,\mathrm{M}} w_i}{\sum_i f_i w_i} - p, \qquad (3)$$

where f_i is the frequency of the ith mating type, $m_{i,\mathrm{F}}$ is the proportion of A alleles among the female offspring of the ith mating type, $m_{i,\mathrm{M}}$ is the proportion of A alleles among the male offspring of the ith mating type, and w_i is the reproductive output (1.0 or k) for the ith mating type.

When the appropriate values from table 1 are substituted into equation (3), we obtain, after simplification,

$$\Delta p = \frac{p[k(2p - p^2 + 2) + (1 - p)^2]}{3[kp(2 - p) + (1 - p)^2]} - p. \qquad (4)$$

Using the same procedure for the case of paternal care, we obtain for the paternal-care allele frequency change:

$$\Delta p = \frac{p[(1 + 2p) + 2(1 - p)]}{3(p(k - 1) + 1)} - p, \qquad (5)$$

where k is now the relative increase in offspring output due to paternal care.

Expressions (4) and (5) are equal to the $M(x)$ term of the diffusion formulae (equations 1 and 2); i.e. they measure the force of selection operating on maternal and paternal-care alleles, repectively. Before calculat-

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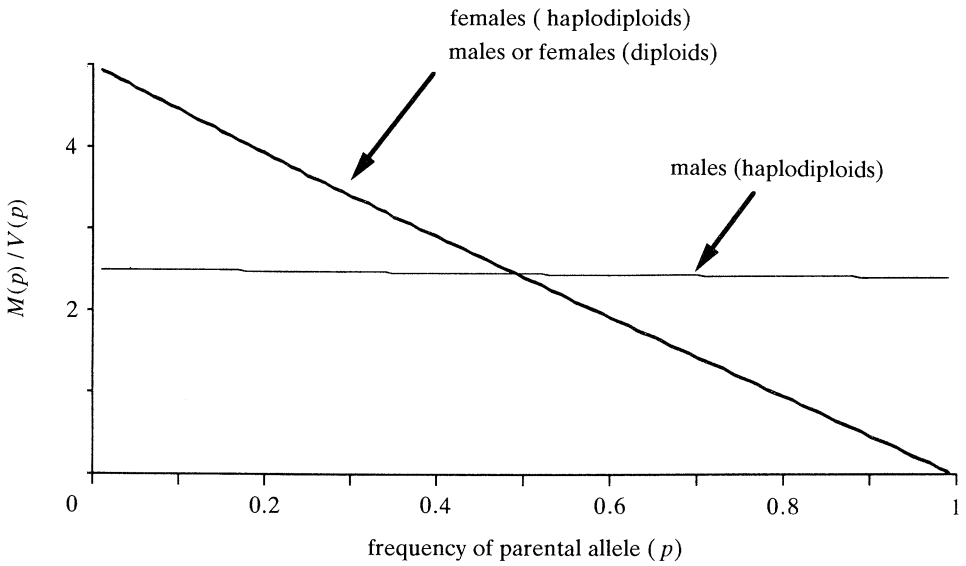

Figure 2. Relative forces of selection versus genetic drift (i.e. $M(p)/V(p)$) as a function of the gene frequency p for maternal and paternal alleles in haplodiploid and diploid populations. (k is assumed to equal 1.05; see text).

ing the probabilities of fixation of these alleles, it is intuitively useful to compare the selective forces favouring maternal and paternal-care alleles when these alleles are rare and thus most vulnerable to loss from random genetic drift. When the maternal allele is rare, expression (4) becomes, approximately, $2p(k-1)/3$, whereas when the paternal-care allele is rare, expression (5) is only $p(k-1)/3$. In other words, when the maternal-care allele is rare, the strength of selection favouring it is approximately twice the strength of selection favouring the paternal-care allele. This means that the maternal-care allele is less likely to be lost by random genetic drift than is the paternal-care allele in finite haplodiploid populations. This effect arises because the A allele for maternal care is more frequently exposed to positive selection than is the A allele for paternal care when each allele is rare. When either is rare, approximately 2/3 of the matings involving the A allele are $Aa \times a$, and only about 1/3 are $aa \times A$: the maternal-care allele is expressed (and enjoys a selective boost) in the former, more-frequent mating combination; the paternal-care allele is expressed only in the latter, less-frequent mating combination and consequently receives a more-diluted overall selective benefit. I will later argue that the resulting difference in probability of random loss can result in a much higher long-term probability of observing maternal care than paternal care in haplodiploid species.

Because we know both $M(x)$ and $V(x)$ for maternal-care and paternal-care alleles ($V(x) = V(p) = p(1-p)/(3/2)N_e$, as discussed above), we are now able to compare the relative force of selection and drift $M(p)/V(p)$ for maternal and paternal-care alleles over a wide range of gene frequencies p. In figure 2, we see that the $M(p)/V(p)$ curve for maternal-care alleles is above that for paternal-care alleles when the gene frequency is low, indicating greater protection for rare maternal-care alleles from loss through genetic drift.

The ultimate probabilities of fixation are easily calculated from equations (1) and (2) after some simple approximations to $M(x)/V(x)$ are derived for maternal and paternal-care alleles. In particular, the function $G(x)$ for maternal-care alleles is, under weak selection, closely approximated by

$$e^{-2N_e(k-1)(x-x^2/2)}, \tag{6}$$

and, similarly, the function $G(x)$ for paternal-care alleles is approximated by

$$e^{-N_e(k-1)[x-(k-1)x^2/2k]}, \tag{7}$$

(see Appendix 1). Expressions (6) and (7) can be substituted in equation (1) to obtain the probabilities of ultimate fixation for new mutant maternal and paternal-care alleles. These probabilities are shown in figure 3 for various values of k and an effective population size of 100. The fixation probability for maternal-care alleles always exceeds and can be nearly twice that for paternal-care alleles, as we might have guessed from the relative forces of selection on the two alleles when they are rare.

(ii) *Diploids*

Inspection of table 2 reveals that the reproductive outputs are symmetrical for paternal and maternal-care alleles; i.e. when a mating combination arises for which the reproductive output of one sex is k and the other sex is 1.0, there exists a mating combination of identical frequency for which the reproductive outputs are reversed. Thus, no sex bias in the fixation of parental alleles is expected for diploid species.

(iii) *Comparison of haplodiploids and diploids*

The deterministic change in gene frequency for (male or female) parental-care alleles in diploids is equal to

$$\Delta p = \left\{ \sum_i f_i m_i w_i / \sum_i f_i w_i \right\} - p, \tag{8}$$

where f_i is the frequency of the ith mating type, m_i is

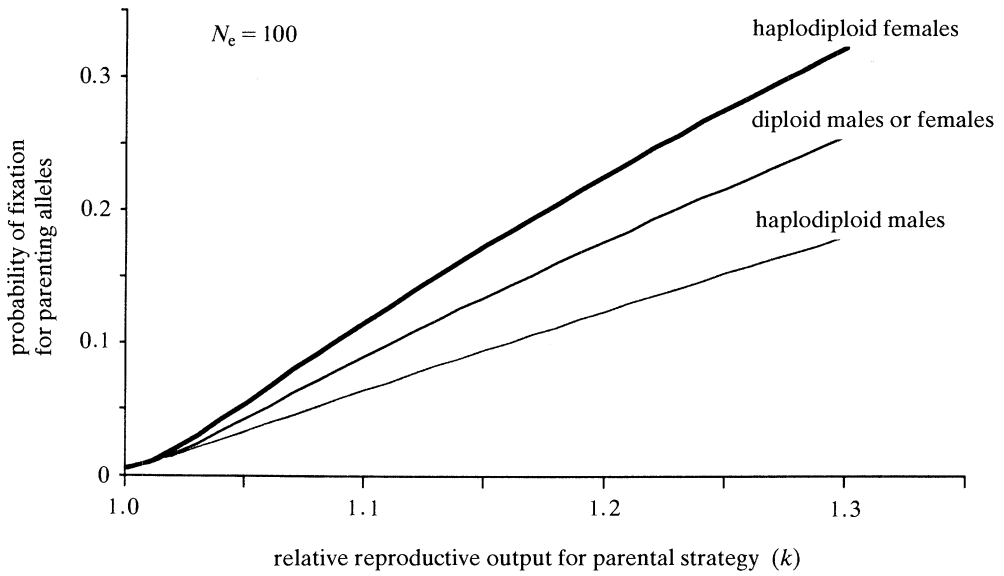

Figure 3. Fixation probabilities for new mutant parental-care alleles expressed in haplodiploid females, diploid males and females, and haplodiploid males, as a function of the relative reproductive output k for parental care.

the proportion of A alleles among the offspring of the ith mating type, and w_i is the relative reproductive output (1.0 or k) for the ith mating type. From equation (8) and table 2, the gene frequency change for (male or female) parental-care alleles in diploids is found to be

$$\Delta p = \frac{p[(1-p)^2 + k(1-(1-p)^2)]}{(1-p)^2 + kp(2-p)} - p. \qquad (9)$$

This is equivalent to $M(x)$ for diploid species (when x is substituted for p). When the parental-care allele is rare, the change in gene frequency due to selection is approximately $p(k-1)/2$, which lies exactly intermediate between the corresponding values for haplodiploid maternal-care alleles ($2p(k-1)/3$) and for haplodiploid paternal-care alleles ($p(k-1)/3$), which would lead us to expect that the fixation probability for a diploid maternal-care allele should also be intermediate between those for haplodiploid maternal and paternal-care alleles.

Interestingly, the $M(x)/V(x)$ function for parental-care in diploid species turns out to be identical to that for maternal care in haplodiploid species (see Appendix 2; figure 2). However, this does not mean that the corresponding fixation probabilities will be the same. In fact, the fixation probability for the maternal-care allele in haplodiploids will (for a given effective population size) be approximately 4/3 greater than the fixation probability for a parental-care allele in a diploid species. The reason for this is that the limit of integration t in the numerator of the right-hand expression in equation (1) is different for haplodiploid ($t = 2/3N_e$) and diploid species ($t = 1/2N_e$). As shown in figure 1, the fixation probability–when N_e is not too small and selection is not too strong–is approximately equal to t divided by the integral of $G(x)$ from 0 to 1. The latter integral is the same for haplodiploid

Table 2. *Mating frequencies and associated relative reproductive outputs for parental and alloparental strategies for males and females in diploid species*

(Parameters are as in table 1.)

mating type (female × male)	freq.	offspring genotypes: female or male	reproductive output: maternal care	paternal care (females)	sibling care (males or females)
$aa \times aa$	q^4	aa	1.0	1.0	1.0
$Aa \times aa$	$2pq^3$	1 Aa: 1 aa	k	1.0	$k(0.5)$
$aa \times Aa$	$2pq^3$	1 Aa: 1 aa	1.0	k	$k(0.5)$
$Aa \times Aa$	$4p^2q^2$	1 AA: 2Aa: 1 aa	k	k	$k(0.75)$
$AA \times aa$	p^2q^2	Aa	k	1.0	$k(1.0)$
$aa \times AA$	p^2q^2	Aa	1.0	k	$k(1.0)$
$Aa \times AA$	$2p^3q$	1 AA: 1 Aa	k	k	$k(1.0)$
$AA \times Aa$	$2p^3q$	1 AA: 1 Aa	k	k	$k(1.0)$
$AA \times AA$	p^4	AA	k	k	$k(1.0)$

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maternal-care alleles and diploid parental-care alleles (as $M(x)/V(x)$ is the same for both), so the ratio of their respective fixation probabilities is approximately $2/3N_e : 1/2N_e = 4/3$.

Intuitively, the latter result means that, for a given population size, a mutant maternal-care allele will be closer to fixation upon first appearance in haplodiploids than in diploids because there are only $(3/2)N_e$ slots to be filled in the former and $2N_e$ slots to be filled in the latter. (Of course, the probability that the appropriate mutant will appear in diploids is greater than the corresponding probability in haplodiploids, because there are fewer opportunities for mutation in the latter, but in a later section I will show that differences in mutation rates do not lead to differences in the long-run probabilities of observing the encoded strategies.) Thus, there will be a slightly greater tendency for maternal care to evolve upon first appearance in haplodiploid than in diploid species. Parental care alleles in diploid species will nevertheless tend to have higher fixation probabilities than paternal-care alleles in haplodiploid species (figure 3), as expected from the above examination of the forces of selection when parental-care alleles are rare (see also figure 2). Thus, the ordering of propensities toward parental care appears to be: haplodiploid maternal care > diploid maternal or paternal care > haplodiploid paternal care.

(*c*) *Male and female alloparental (worker) care in haplodiploids and diploids*

(i) *Haplodiploids*

Consider a dominant allele A that causes its bearers (potential workers = alloparents) to initiate or enhance care for its reproductive sisters and brothers in a matrifilial colony. The allele is assumed to be dominant for the same reasons as given in the section on the evolution of parental-care. The lone queen is assumed to be singly inseminated, and the colony sex investment ratio is unbiased. (Strictly speaking, for this model to represent the initiation, not just enhancement, of alloparental care, we must assume that queens produces eggs that hatch asynchronously, such that alloparents can help rear younger siblings from the same clutch before themselves dispersing into the mating pool. This assumption is made solely for computational convenience; other models for the initiation of alloparental care (eusociality) give qualitatively similar results; H. K. Reeve, unpublished results). Under these assumptions, a female worker is, on average, no more closely related to the siblings it cares for ($0.5 \times 3/4 + 0.5 \times 1/4 = 1/2$) than it is to its own offspring (also 1/2). In addition, a female worker is not more closely related to its siblings than is a male worker, so relatedness alone cannot explain why workers overwhelmingly tend to be female. Despite the absence of these relatedness asymmetries, the analysis below demonstrates that haplodiploidy makes co-operation among female siblings more likely to evolve than co-operation among male siblings by better protecting female-alloparental alleles from loss through random genetic drift. Further below, I will also show that female co-operation is more likely to evolve in haplodiploid species than in diploid species for a similar reason.

The relative reproductive output $k(z)$ of a colony with a proportion z of female co-operators among the female offspring is given for each parental mating combination in table 1. (For computational convenience, the relative reproductive output $k(z)$ is assigned to production of both males and females within a colony containing a fraction z female co-operators. This is a reasonable approximation even if alloparenting by females differentially reduces the total output of females (e.g. due to increased mortality or reduced fecundity), because $k(z)$ can be regarded as the mean of the different outputs of males and females from the colony when this difference is not too large. The latter interpretation of $k(z)$ holds exactly when the alloparental-care allele is rare, and as discussed below and in Appendix 2, the fixation probability is determined principally by the selective force on a rare allele.)

To find the deterministic change in the frequency of a female-alloparental allele undergoing positive selection, we again use equation (3) and the appropriate information in table 1. The gene frequency change is equal to

$$\Delta p = \frac{2k(0.5)p(1-p)^2 + k(1.0)p(1+4p-2p^2)}{3[2k(0.5)p(1-p)^2 + k(1.0)(1+p-p^2) + (1-p)^3]} - p. \quad (10)$$

We similarly find the gene frequency change for male-alloparental alleles:

$$\Delta p = \frac{2k(0.5)p(1-p^2) + k(1.0)p(2+p) + p(1-p)^2}{3[k(0.5)2p(1-p) + k(1.0)p^2 + (1-p)^2]} - p. \quad (11)$$

When female-alloparental alleles are rare, the gene frequency change (as a measure of the force of selection) is approximately $p(2k(0.5) + k(1.0) - 3)/3$, whereas that for male-alloparental alleles is only $p2(k(0.5) - 1)/3$. The relative magnitudes of these two quantities depends on the function $k(z)$ relating the relative reproductive output to the proportion of (same-sexed) co-operators z. If this function is linear, i.e. $k(0.5) = 1 + \phi$ and $k(1.0) = 1 + 2\phi$, then the selective force Δp will be twice as great for a female-alloparental allele as for a male-alloparental care allele when each is rare. If the function $k(z)$ is nonlinear, such that co-operation synergistically enhances colony output, the relative force of selection on rare female-alloparental alleles will be even stronger. In general, if $k(0.5) = 1 + \phi$ and $k(1.0) = 1 + s\phi$, where s measures the degree of synergy ($s \geqslant 2$), the selective force for rare female-alloparental alleles will be $(s+2)/2$ times that for rare male-alloparental alleles. For example, if $s = 8$, the selective force favouring rare female-alloparental alleles will be five times that for male-alloparental alleles. Another possible kind of nonlinearity would be a step-function

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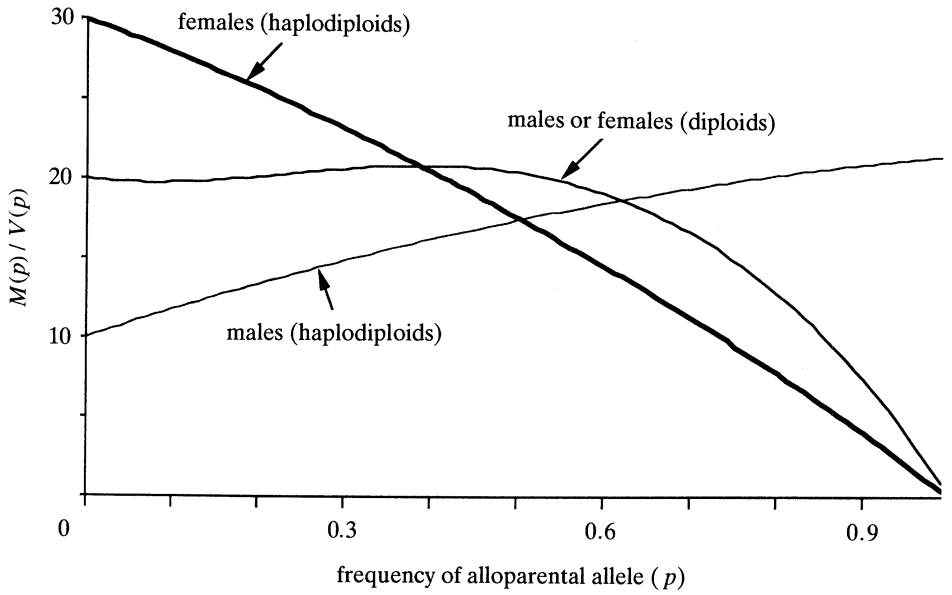

Figure. 4. Relative forces of selection and genetic drift ($M(p)/V(p)$) as a function of the gene frequency p for alloparental alleles expressed in haplodiploid females, diploid males or females, and haplodiploid males. ($k(0.5) = 1.10$; $k(0.75) = 1.10$; $k(1.0) = 1.30$; see text.)

in which there exists a threshold proportion of co-operators below which there is no increase in colony output. If this threshold is greater than 1/2, there would be no selective force favouring rare male-alloparental alleles (because $k(0.5) = 1$), rendering such alleles extremely vulnerable to random loss.

The intuitive meaning behind the above results is as follows: when an alloparenting allele A is rare, approximately 2/3 of the A-containing colonies will be produced by $Aa \times a$ matings and 1/3 will be produced by $aa \times A$ matings. The former matings result in colonies in which one-half of the sons exhibit genotype A and one-half of the females exhibit genotype Aa; both female and male alloparents would increase colony output by $k(0.5)$ in such colonies. However, colonies produced by $aa \times A$ matings would contain no male alloparents but would contain 100% female (Aa) alloparents; the female alloparents increase colony output by $k(1.0)$, hence the female-alloparental allele receives a big selective boost in such colonies and becomes more resistant to random loss (especially if co-operation produces synergistic benefits). Comparisons of the curves $M(p)/V(p)$ for female-alloparental haplodiploids and male-alloparental diploids show that the relative strength of selection versus drift indeed is greater for female-alloparental alleles at low allele frequencies (figure 4).

Thus, we should expect that the probability of ultimate fixation for female-alloparental alleles should be greater than that for male-alloparental alleles in finite haplodiploid populations, perhaps markedly so when co-operation synergistically increases colony output. To derive these fixation probabilities, I set the Δps in equations (10) and (11) equal to $M(x)$ (converting the ps to xs) and then divided by $V(x) = x(1 - x)/(3/2)N_e$ to obtain the functions $M(x)/V(x)$, which are then substituted into equations (2) and (1). Unfortunately, there is no robust linear approximation to the $M(x)/V(x)$ function for alloparental alleles, as was the case for parental alleles, so a two-tiered numerical method must be used to solve equation (1) for the former.

The fixation probabilities for male and female-alloparental alleles in a haplodiploid population of effective size = 100 are presented in table 3 for several colony output functions $k(z)$. As expected, fixation probabilities for female-alloparental alleles range from nearly two to over 15 times that for male-alloparental alleles, with the ratio increasing for increasingly nonlinear colony output functions. In sum, female alloparents (workers) are expected to evolve more frequently than male alloparents (workers) in haplodiploids, even when haplodiploidy does not create relatedness asymmetries favouring the evolution of altruistic female workers.

(ii) *Diploids*

In diploid species the frequency of the alloparental allele A in female offspring is the same as in male offspring for each colony type defined by the mating types (table 2). Thus, the fixation probabilities for male and female-alloparental care alleles must be equal and thus there should be an equal propensity for male and female workers to evolve.

(iii) *Comparison of haplodiploids and diploids*

To compare fixation probabilities for male and female alloparents in diploids and haplodiploids, we must first calculate the deterministic change in gene frequency for diploid (male or female) alloparents. Using equation (8) and the information in table 2, we obtain

$$\Delta p = \frac{k(0.5)pq^3 + k(0.75)2p^2q^2 + k(1.0)p^2(1 + pq)}{q^4 + k(0.5)4pq^3 + k(0.75)4p^2q^2 + k(1.0)p^2q^2} - p. \quad (12)$$

When the diploid alloparental allele is rare, Δp is

Table 3. *Approximate fixation probabilities for mutant alleles specifying sibling co-operation (alloparenting) for haplodiploid and diploid males and females as a function of the relationship k(z) between relative colony output and the proportion z of (same-sexed) co-operators in the colony*

(The $k(z)$ function is linear in the top row and becomes increasingly nonlinear (synergistic) in successively lower rows. $N_c = 100$.)

				fixation probability		
$k(0)$	$k(0.5)$	$k(0.75)$	$k(1.00)$	haplodiploid female alloparents	diploid alloparents	haplodiploid male alloparents
colony output						
1.00	1.10	1.15	1.20	0.22	0.17	0.12
1.00	1.00	1.05	1.10	0.07	0.03	0.03
1.00	1.10	1.10	1.30	0.28	0.17	0.13
1.00	1.00	1.10	1.40	0.24	0.06	0.05
1.00	0.90	1.00	1.40	0.15	< 0.01	< 0.01

approximately $p(k(0.5) - 1)$. This value is 1.5 times that for a male-alloparental care allele in haplodiploids, but it is only 3/4 that for a rare female-alloparental care allele in haplodiploids if the colony output function $k(z)$ is linear. More generally, the force of selection on a rare female-alloparental care allele in haplodiploid populations will be $((2 + s)/3)$ times that for a rare alloparental care allele in diploids, where s is the measure of co-operative synergy discussed in the section on haplodiploid alloparental care. For example, if $s = 8$, the selective force on rare female-alloparental alleles in haplodiploids will be 10/3 times that for rare alloparental alleles in diploids. The force of selection on female-alloparental alleles is greater in haplodiploids because, when the A allele is rare, 1/3 of A-containing colonies will consist of 100% co-operating female alloparents in haplodiploids, whereas virtually all A-containing colonies in diploids will have at most 50% co-operating female or male alloparents.

Of course, the greater the number of matings by the queen, the less likely it is that there will be positive selection for female workers (as in the case of kin selection). However, given that there is positive selection, it can be shown that the bias toward female workers in haplodiploids versus diploids remains regardless of the effective number of matings by a queen, and that this bias persists even in cases where there is no difference between haplodiploid or diploid colonies in the relatednesses of workers to their tended brood. If the queen mates randomly m times with different males and uses their sperm equally, and if colony sex ratio is equal, in both diploid and haplodiploid species females will be related to their siblings by an average value of $1/4 + 1/4m$. When an alloparental A allele is rare under these conditions, it is readily seen from equations (3) and (8) that the selective force on A (i.e. Δp) is approximately equal to $p[(2/3)k(0.5) + (1/3)k(1/m) - 1]$ for haplodiploids and to $p[(1/2)k(0.5) + (1/2)k(1/2m) - 1]$ for diploids. The latter quantity is less than the former quantity under the likely condition: $k(0.5), k(1/m) > k(1/2m)$. In the case where the benefit of co-operation is a linear function of the proportion of co-operators, the ratio of these selective forces (haplodiploid:diploid) is equal to 4/3, regardless of queen mating frequency. The ratio is even higher and is a decreasing function of m (approaching 4/3 for large m) when there are synergistic benefits from sibling co-operation. Thus, a prediction emerging from this analysis is that eusociality should tend to be more often compatible with multiple-mating by queens in haplodiploid species than in diploid species, especially if co-operative synergism at least sometimes occurs.

The intuitive reason behind the above results is that, when the queen mates m times, an approximate fraction $m/(2 + m)$ of A-containing haplodiploid colonies will contain a fraction $1/m$ co-operating female alloparents (the rest will contain 50% alloparents), whereas a larger fraction $m/(1 + m)$ of A-containing diploid colonies will contain only $1/(2m)$ co-operating alloparents (the rest will contain 50% alloparents). Thus, in haplodiploid (versus diploid) populations, a smaller fraction of families receive the rare alloparenting allele from one of the fathers, in which case the alloparental effort is largely wasted on a brood containing few copies of the alloparenting allele. As a result, there should be an overall stronger selection in haplodiploids on A when A is rare, whatever the mating frequency m. (Another analysis reveals that force of selection on rare, advantageous female-alloparental alleles will always exceed that on rare, advantageous male-alloparental alleles within haplodiploids, regardless of the number of matings by the queen.)

Based on the the analyses of selective forces on rare alloparental alleles, we might predict that fixation probabilities for (female or male) alloparental alleles in diploids should be less than that for female-alloparental alleles in haplodiploids but more than that for male-alloparental alleles in haplodiploids. The curves $M(p)/V(p)$ for these three cases would seem to bear out this prediction; the relative force of selection versus drift at low allele frequencies is highest for female-alloparental alleles in haplodiploid populations, the same or intermediate for alloparental alleles in diploid populations, and lowest for male-alloparental alleles in haplodiploid populations (figure 4). Fixation probabilities for alloparental alleles in diploid populations, calculated from equation (1), indeed

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are generally intermediate between those for female-alloparental alleles and male-parental alleles in haplodiploid populations (table 3). When the colony output function $k(z)$ is linear, it can be shown that (as in the parental case), the $M(x)/V(x)$ function is the same for female-alloparental alleles in haplodiploid populations and for (male or female) alloparental alleles in diploid populations. However, for the reasons given in the section on parental-care, the fixation probability will tend to be slightly higher for the former, by a factor of approximately 4/3. When the colony output function is nonlinear, reflecting synergistic benefits of co-operation, the fixation probability for female-alloparental alleles in haplodiploid species can be markedly greater than that for alloparents in diploid species. In sum, when the benefits of sibling co-operation are linear, the ordering of propensities toward alloparental care appears to be: haplodiploid females > diploid males or females > haplodiploid males. When the benefits of sibling co-operation are nonlinear and synergistic, the ordering of these propensities appears to be: haplodiploid females $\gg$ diploid males or females > haplodiploid males.

The above analyses assume that alloparental-care alleles have a sex-limited expression. It might be argued that an allele causing both males and females to become alloparental will always experience stronger selection than would sex-limited alleles for alloparental care. From this argument we would predict no sex bias in alloparental care in either diploid or haplodiploid species and reduced bias in haplodiploid versus diploid species. There are at least two arguments against this line of reasoning. First, for both haplodiploid and diploid species, it seems unlikely that a single gene would cause alloparental care in both sexes, as the two sexes have previously experienced long histories of selection for very different reproductive strategies; e.g. a mutant allele that predisposes maternal care in females may cause a very different (or no) effect in males, who have evolved a different neural machinery. Furthermore, the bias toward maternal care expected (see above) and observed in haplodiploids would seem to promote further sex-limitations on expression of alloparental-care alleles. Finally, even if an allele that caused alloparental care in both sexes spread to fixation in a haplodiploid species, subsequent modifiers that preferentially enhanced female-alloparental care would tend to accumulate faster (due to protected invasion effects) than would modifiers that enhanced male-alloparental care. If, as seems likely, the differential exaggeration and refinement of alloparental care in one sex reduces the benefits of alloparental care in the other sex (e.g. when returns for increased alloparental care begin to diminish), a point eventually may be reached at which males would do better to cease alloparental care altogether and instead devote all of their resources to mate acquisition. Hence, equal participation in alloparental care by the two sexes would be unstable in haplodiploid populations.

It is interesting to note that the relative probabilities of ultimate fixation for female versus male parental or alloparental alleles and the relative probabilities of fixation for parental and alloparental alleles in haplodiploid versus diploid populations appear to be approximately equal (when selection is weak) to the ratios of the corresponding selective forces when the alleles are rare, regardless of the effective population sizes. In other words, if $M(0)$ and $M(0)'$ are, respectively, the selective forces favouring a rare allele in two different contexts, it appears that the ratio of the ultimate fixation probability of a mutant allele in the first context to that of the same allele in the second (primed) context is roughly equal to $M(0)/M(0)'$. An informal proof of this approximation is provided in Appendix 2. Importantly, this approximation generalizes the numerical results obtained earlier, demonstrating that the biases toward parental and alloparental care observed so far do not depend critically upon the specific effective population sizes assumed in our numerical examples. Indeed, the biases are virtually independent of the effective population sizes if the latter are not too small (Appendix 2), indicating that the biases are robust against variation in N_e within haplodiploid populations or between haplodiploid and diploid populations.

3. MAPPING PROBABILITIES OF FIXATION ONTO LONG-TERM PROBABILITIES OF OBSERVING PHENOTYPES

So far I have discovered that the probabilities of ultimate fixation for new mutant (advantageous) parental and alloparental alleles are generally higher when these alleles are expressed in haplodiploid females than when they are expressed in diploid males or females or in haplodiploid males. However, I have not yet resolved the important question: will these different fixation probabilities translate into noticeably different, long-run probabilities of observing the different strategies encoded by these alleles? An affirmative answer to the latter question, is, after all, necessary for adequately explaining why eusociality is more common in haplodiploids than in diploids and why female parental or alloparental care is much more common than male parental or alloparental care within haplodiploids.

Before a model of the long-run probability of observing a parental or alloparental care strategy can be based on the fixation probabilities of the underlying alleles, the effects of mutation rates at the relevant loci must be considered. One apparent problem is that, by the assumptions, the mutation rates for alleles encoding female (parental or alloparental) strategies will tend to be of lesser magnitude than the mutation rates for alleles encoding male (parental or alloparental) strategies in haplodiploid populations. This occurs because, in the case of females, I have assumed that the relevant alleles are dominant or codominant (because these are most likely to spread); in haploid males, on the other hand, we must consider both dominant and recessive alleles, because either kind of allele will always be expressed. Because mutant recessive and dominant (advantageous) alleles are therefore equally likely to spread in the case of haploid males, the overall rate of mutations producing advan-

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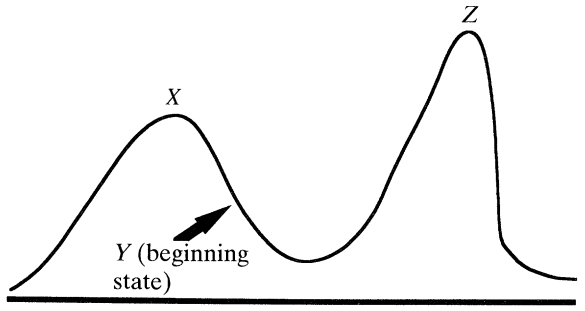

Figure 5. Adaptive landscape showing the beginning (non-equilibrium) state of a population (phenotype Y) located between two fitness peaks associated with the phenotypes X and Z.

tageous, male-strategy alleles should exceed that for advantageous, female-strategy alleles. This difference in overall mutation rates may seem to work against the expectation from fixation probabilities alone that male parental or alloparental care strategies are less likely to be observed in haplodiploid populations than are female parental or alloparental strategies. However, it must be remembered that male-strategy and female-strategy alleles are not in competition with each other at the same locus, but rather are in competition with alternative-strategy alleles at different loci. For example, a male-alloparental allele is best viewed as in competition with a male-non-alloparental allele at a locus distinct from that controlling the disposition to female alloparental care. As mutant male-non-alloparental alleles, like mutant male-alloparental alleles, are equally likely to spread whether dominant or recessive, the rates of mutation to the two kinds of alleles can be taken to be equivalent and thus these rates should not affect the long-run probability of 'seeing' male-alloparental care. (Similarly, differences in mutation rates between haplodiploid and diploid populations will not lead to differences in the long-run probabilities of observing a parental or alloparental strategy between the two kinds of populations: the long-run probabilities depend only on fixations of alternative alleles that compete within the same population and thus plausibly exhibit similar mutation rates.)

In the following model, I will show how even small differences in fixation probabilities of alleles can lead to marked differences in the long-run probabilities of sampling the phenotypes encoded by those alleles, regardless of assumed mutation rates. I begin by envisioning a population which exhibits some beginning (ancestral) strategy Y (e.g. males pursuing multiple-matings with females, at the expense of paternal care). Now suppose the environment changes, such that Y is suboptimal with respect to two strategies X and Z, i.e. Y lies between two adaptive peaks associated with X and Z (figure 5). A specific mutation at one locus of a type L_x takes the population in the direction of X and a specific mutation at another locus of type L_z takes the population in the direction of Z. If the X-enhancing allele at the L_x locus becomes fixed first, one of two events then occurs: either the fixation of an X-enhancing allele at a modifier locus of type L_x pushes the population one additional step closer to the X strategy, or the fixation of a Z-enhancing allele at a type L_z modifier locus might nullify the effect of the first X-enhancing allele, in effect taking the population back to the starting state. The latter possibility might be less likely if the nullifying allele moves the population back down into the valley. (Note: selection always promotes X or Y alleles; the 'valley' occurs not because of selection against these alleles, but because the fixation probability for one kind of allele can exceed that of the other over certain regions.) This stochastic process of successive or alternating fixation of X-enhancing and Z-enhancing alleles continues (with the two kinds alleles cancelling each others effects) until the population finally arrives at either the X or the Z peak. If the population moves to the peak associated with X, it will tend to stay there for long periods of time, hence the population is especially likely to exhibit strategy X in the long run. Thus, our critical question becomes: what is the probability that a population beginning at Y will end up at X (or Z)?

The above model for the long-run probability of observing a particular strategy can be made very simple if we make a discrete approximation to the adaptive landscape (figure 6) and assume that: (i) the numbers of L_z and L_x loci are large; (ii) the beginning Y strategy is H steps away from becoming full-blown strategy Z (i.e. the total number of fixations of Z-enhancing alleles at L_z loci minus the total number of fixations of X-enhancing alleles at L_x loci must be at least H for the population to move from strategy Y to strategy Z); and (iii) the Y strategy is N steps away from becoming full-blown strategy X (i.e. the total number of fixations of X-enhancing alleles at L_x loci minus the total number of fixations of Z-enhancing alleles at L_z loci must be at least N for the population to move from strategy Y to strategy X). Further suppose that, once the population hits either strategy X or strategy Z, the population irreversibly exhibits that strategy (i.e. the strategies X and Z are both evolutionarily stable (Maynard Smith 1982*b*) unless the environment again changes).

At any step (i.e. population state) i between the fitness peaks, let the fixation probability for a mutant X-enhancing allele at an L_x locus be a_i and the fixation probability for a mutant Z-enhancing allele at an L_z locus be b_i (figure 6). Thus, if there are approximately equal mutation rates for X and Z-enhancing alleles, the probability that the population heads for the X strategy at step i is $a_i/(a_i + b_i)$, and the probability that it heads for the strategy Z is $b_i/(a_i + b_i)$. (Remember that the assumption of equality of mutation rates is justified because: (i) male-strategy alleles are competing with other male-strategy alleles, diploid female-strategy alleles are competing with other diploid female-strategy alleles, etc., as discussed above; and (ii) mutation is plausibly assumed to be random with respect to fitness effects.) This Markov model for phenotypic evolution is diagrammed in figure 6, where Z is now explicitly recognized as the parental or alloparental-care strategy and X is seen as a non-parental or non-alloparental reproductive strategy (e.g. for males, seeking multiple mates; for females,

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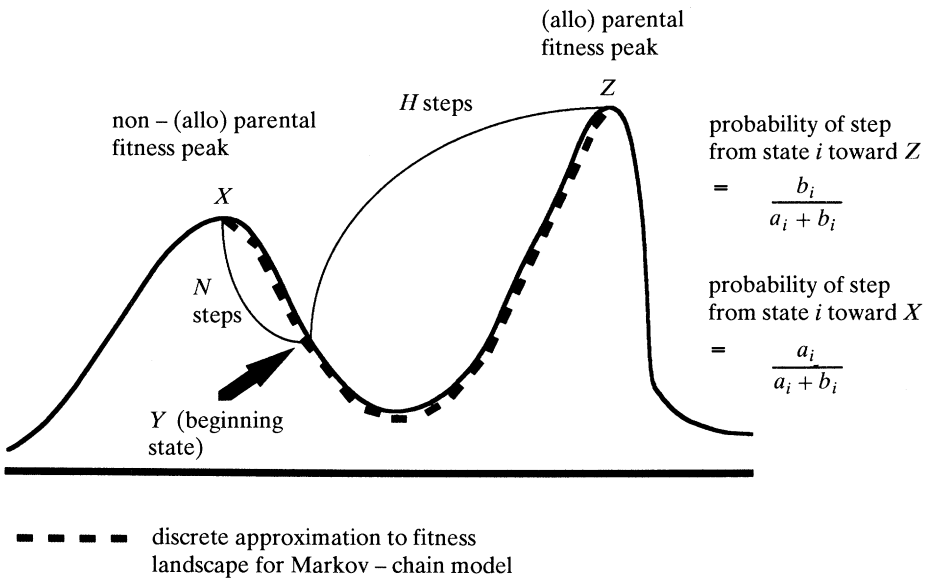

Figure 6. Discrete-step approximation to the fitness landscape in figure 5 used for construction of a Markov model of the transitions from Y to X or Z. Also shown are the one-step transition probabilities throughout the fitness landscape (see text); b_i is the fixation probability for a mutant parental or alloparental-care enhancing allele, and a_i is the fixation probability for a mutant non-parental or non-alloparental-care enhancing allele. The ancestral or beginning state of the population is N steps away from the non-(allo)parental fitness peak and H steps away from the (allo)parental fitness peak.

leaving an egg-filled nest to construct a new nest (as an alternative to maternal care), or nesting solitarily (as an alternative to alloparental care)).

The Markov model just outlined is formally equivalent to the gamblers ruin model in probability theory, in which it is supposed that, with each gamble, a perpetual gambler heads for one of two absorbing states, financial ruin or winning all of the opponent's money (Taylor & Karlin 1984). The probability of ending up in one of the absorbing states, or, in our context, the probability Q that our initial population ends up displaying the parental or alloparental strategy, is given by

$$Q = \left(1 + \sum_{i=1}^{N-1} \psi_i\right) \Big/ \left(1 + \sum_{i=1}^{N+H-1} \psi_i\right), \qquad (13)$$

where

$$\psi_i = (a_1 a_2 \ldots . a_i)/(b_1 b_2 \ldots . b_i),$$

(see Taylor & Karlin 1984, p. 106). (It should be noted that the above solution also satisfies a more general random walk model in which it is assumed that the one-step probability of transition is va_i toward X, vb_i toward Z and $1 - va_i - vb_i$ for remaining in intermediate state i, where v is the probability of mutation (summed over all potentially contributing loci) in a step. Thus, mutation rate does not affect Q even when the possibility of remaining in the initial state is included. Although the mutation rate does affect the mean time until an absorbing state is 'hit' in this case, it does not affect the probability that absorbing state Z is hit first. The probability of absorption in state Z seems most relevant for evolutionary prediction, as the time spent in transient states should be short compared with the time spent in an absorbing state.)

Equation (13) now provides us with a way to estimate the long-run probability of observing parental or alloparental care in male or female haplodiploid or diploid species given the fixation probabilities b_i for the alleles predisposing these strategies. As we would expect, Q increases as any b_i increases.

Of particular interest is the comparison of Qs for parental or alloparental care between the two different sexes, or for a given sex between the two genetic systems (haplodiploidy and diploidy). Thus I define Q^* as the ratio Q/Q', where Q is the probability of observing parental or alloparental care in one context and Q' is the same probability for another context.

The following is a simple, but revealing, case. Suppose that the beginning state of the population is on the right- or left-hand fitness slope in figure 6, that this slope is linear (i.e. the fixation probabilities are constant), and that the alternative fitness peak occurs at the bottom of this slope (e.g. there is no fitness basin and the left slope is vertical). It can be shown that Q^* is equal to

$$\left[\frac{1 - (a/b)^N}{1 - (a/b')^{N+H}}\right]\left[\frac{1 - (a/b')^{N+H}}{1 - (a/b)^{N+H}}\right], \qquad (14)$$

where b and b' are the fixation probabilities for parental or alloparental-care alleles in the two contexts. The important point is that a large value of Q^* can be produced by a small difference in the fixation probabilities b and b'. For example, suppose that the fixation probability for female-alloparental care in a haplodiploid population is 0.07 and that for a male-alloparental care allele is 0.03 (as in line 2 of table 3).

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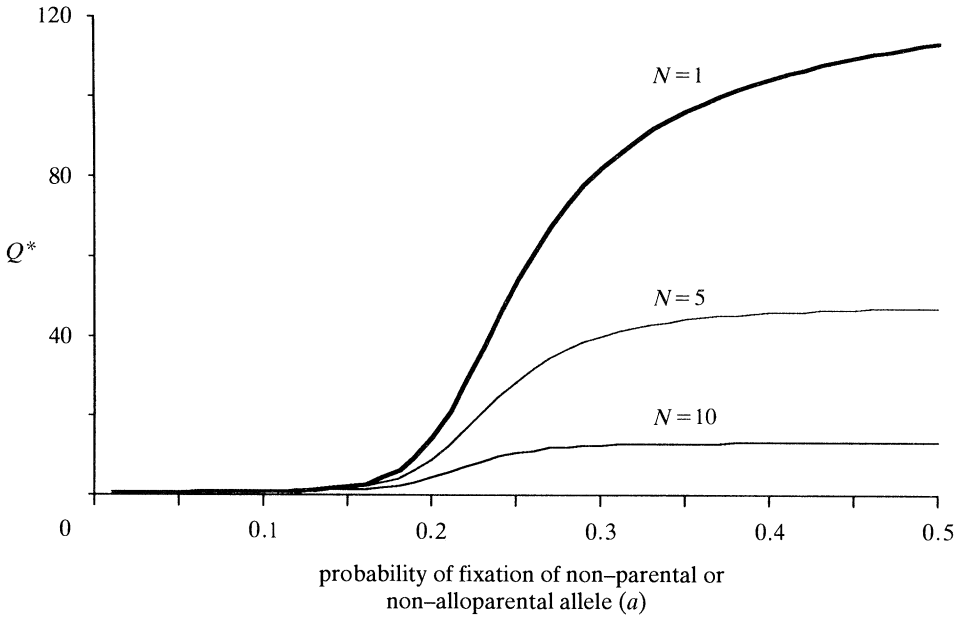

Figure 7. Relative probability Q^* of observing parental or alloparental strategy in the long run as function of the probability of fixation of a non-parental or non-alloparental allele (a) and the number of steps N from the ancestral strategy to the non-parental or non-alloparental strategy. (Total number of steps is fixed at 20.) Here, the probabilities of fixation of parental or alloparental alleles in the two compared contexts are 0.22 and 0.17, respectively (e.g. see line 1 of table 3).

If there are ten steps from the beginning strategy to the alloparental strategy and four steps to the non-alloparental strategy and if a (the probability of fixation for non-alloparental alleles) = 0.09, then Q^* equals approximately 3165! That is, the probability of observing female-alloparental care would 3165 times that for observing male-alloparental care within haplodiploid species!

Various values of Q^* are presented for comparisons of males or females in haplodiploid or diploid species in figure 7 and 8, based on the relative probabilities of fixation derived in the earlier sections of this paper. Several patterns emerge: first, the relative probability of observing the parental or alloparental strategy in one context versus a second context (Q^*) will increase as the number of steps N to the non-parental or alloparental strategy decreases and as the number of steps H to the parental or alloparental strategy increases. (These results are formally proved by noting that $dQ^*/dN < 0$ and $dQ^*/dH > 0$ when $N + H$ is held fixed.) For example, if the beginning strategy much closer to the non-alloparental strategy than to

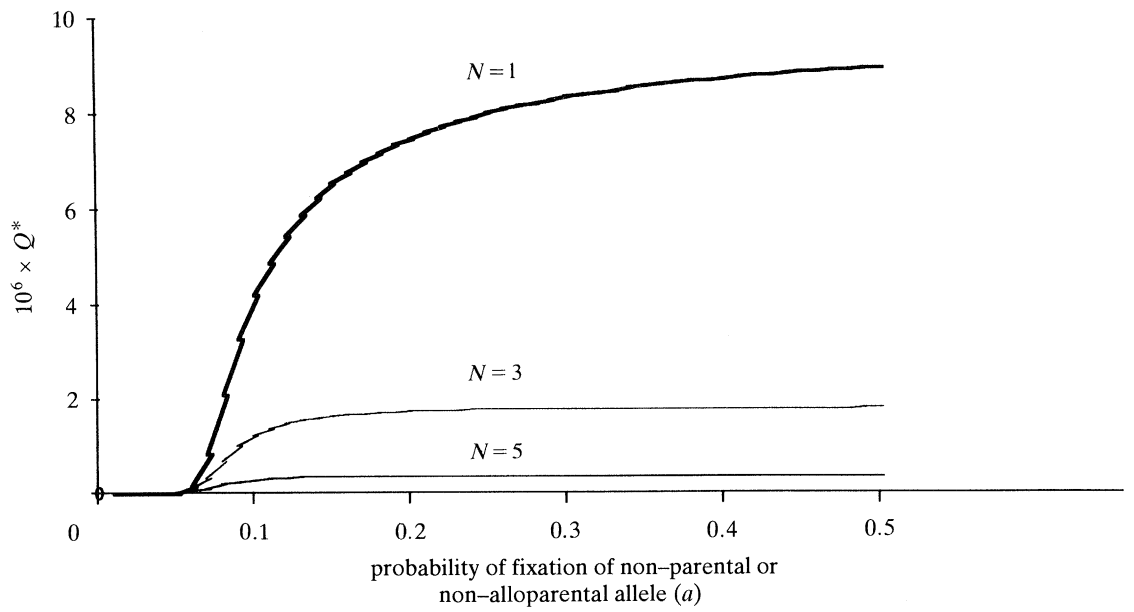

Figure 8. Relative probability Q^* of observing parental or alloparental strategy in the long run as function of the probability of fixation of a non-parental or non-alloparental allele (a) and the number of steps N from the ancestral strategy to the non-parental or non-alloparental strategy. (Total number of steps is fixed at 20.) Here, the probabilities of fixation of parental or alloparental alleles in the two compared contexts are 0.07 and 0.03, respectively (e.g. see line 2 of table 3).

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the alloparental strategy in the Markov model, there will usually be a much greater likelihood of observing alloparental care in the context with the higher fixation probability for alloparental-care alleles. This should often be the case, because only a few steps will be necessary to fine-tune the non-alloparental strategy when non-alloparenting is the beginning, or ancestral state, whereas potentially many steps may be necessary for the elaboration of alloparental care. In addition, Q^* increases as the probability of fixation for non-alloparental or non-parental alleles a increases (proved by noting that $dQ^*/da > 0$). In other words, the more likely are steps from the beginning strategy toward the non-parental or non-alloparental strategy, the greater the relative likelihood of observing parental or alloparental-care in the context associated with the higher fixation probabilities for parental or alloparental-care alleles. Again, this circumstance is precisely what we expect to occur when the ancestral strategy is a non-parental or alloparental one. In sum, in the long-run Markov model, small differences in fixation probabilities are amplified into marked (in some cases gigantic) differences in long-run probabilities of observing parental or allo-parental care, especially when the ancestral state is relatively close to the non-parental or non-alloparental fitness peak and step-wise transitions to the latter are more probable than step-wise transitions to the parental or alloparental fitness peak.

This Markovian model of phenotypic evolution is potentially of general importance in understanding the propensities of different kinds of populations to display different combinations of phenotypes. These propensities are controlled primarily by the fixation probabilities of alleles underlying the phenotypes (not by their mutation rates). It is important to note that, by this model, a relatively low fixation probability for an advantageous allele at some locus does not necessarily mean that the population will be relatively maladapted with respect to the phenotype affected by that locus; the population is simply less likely to make a transition to the more-distant adaptive peak and thus is more likely to settle at the adaptive peak closest to the ancestral condition. For example, consider advantageous alleles expressed only in haplodiploid males: although such alleles have smaller fixation probabilities than corresponding advantageous alleles expressed in diploid females, it does not follow that males will in general be less well adapted than females. Rather, phenotypes of haploid males will be less likely to move away from adaptive peaks closest to their ancestral states. Thus, in the context of parental and alloparental care, we are led to expect that females will be more likely to evolve parental or alloparental behaviour than would males in haplodiploid populations, given ancestral states of non-parental or non-alloparental behaviour for both sexes.

4. CONCLUSIONS

In finite haplodiploid populations, dominant alleles for female-parental (or alloparental) care are more resistant to loss by genetic drift than are male parental (or alloparental) alleles or than are male or female parental (or alloparental) alleles in diploid populations. The Markov model of phenotypic evolution demonstrates that the resulting differences in fixation probabilities among these alleles can translate into large differences in the long-run probabilities of observing the corresponding parental or alloparental strategies. Thus the protected invasion hypothesis immediately explains all of the peculiar social features of the haplodiploid Hymenoptera, namely: (i) the overwhelmingly greater tendency for maternal care than paternal care in Hymenoptera; (ii) the greater propensity for eusociality (alloparental sibling care) in Hymenoptera than in diploid insects; and (iii) the greater likelihood for females than males to become alloparents (workers) in the Hymenoptera. The hypothesis also correctly predicts (iv) a higher frequency of paternal care in diploid species than in haplodiploid species, and (v) the lack of a sex-bias among workers of eusocial diploid species (e.g. termites (Wilson 1971) and naked mole-rats (Jarvis 1981; Lacey & Sherman 1991)).

The protected invasion hypothesis is distinct from, and more generally applicable than, relatedness-based explanations for the distinctive social structures of the Hymenoptera. I showed that the bias toward eusociality in Hymenoptera is produced by protected invasion effects even when there is no asymmetry between a female's relatedness to its siblings and to its own offspring. In addition, protected invasion effects create a bias for female versus male workers within the Hymenoptera even when there is no asymmetry between a female's and male's relatedness to its siblings. Furthermore, protected invasion effects create a bias toward eusociality in haplodiploid versus diploid populations even when the queen mates an indefinite number of times and there is no difference between haplodiploid and diploid colonies in the relatednesses of workers to their tended brood. Finally, the protected invasion hypothesis accounts for a phenomenon that cannot be explained by relatedness-based explanations: the preponderance of maternal over paternal care in the Hymenoptera (because male and female parents have the same mean relatedness to their offspring when the female mates singly).

I must be clear here that I am not saying that kin selection is theoretically flawed or is unimportant. I simply propose that the protected invasion hypothesis provides a more robust and comprehensive explanation for the observed association between genetic system and kinds of social organization. The protected invasion hypothesis could, however, be viewed as a (not mutually exclusive) alternative to Hamiltons (1964) special version of kin selection theory known as the three-quarters relatedness hypothesis (see West Eberhard 1975).

Neither am I claiming that ecological factors are unimportant in the evolution of eusociality and parental strategies, as such factors must affect the strength of selection on, hence fixation probabilities of, parental and alloparental-care alleles. Thus, my thesis is not intended to tip the balance in favour of genetic or intrinsic factors (*sensu* Evans 1977) versus extrinsic or

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ecological factors as explanations of eusociality. The latter distinction is in some ways misleading, as it can be likened to arguing about whether the length or the width is more important in determining the area of a rectangle. The proper issue here is not whether the length or width is more important, but is (continuing the analogy) whether variation in length (i.e. variation in genetic factors) or variation in width (i.e. variation in ecological factors) is more important in explaining variation in the areas of different rectangles (i.e. the propensity for eusociality in different kinds of populations). I have merely attempted to argue that the genetic system can affect the propensity to evolve parental or alloparental care. Haplodiploidy is neither a necessary nor a sufficient cause for the evolution of eusociality (Alexander *et al.* 1991), but it is a predisposing factor.

Finally, it is desirable to extract some novel predictions (rather than retrodictions of previously known phenomena) from the protected invasion hypothesis. One prediction, admittedly difficult to test, is that alleles predisposing parental or alloparental care in diploids or in haplodiploid females should tend to be dominant over their alternative alleles (an assumption of the protected invasion hypothesis). Another prediction, already derived above, is that eusociality should tend to be more often compatible with multiple-mating by queens in haplodiploid species than in diploid species, especially if co-operative synergism is not uncommon. Superficially, this prediction is compatible with the observation that eusociality in diploid insects (i.e. termites) appears to be associated with queen monandry, whereas eusociality in the Hymenoptera is frequently associated with queen polyandry (Page 1986). A third prediction is that synergistic interactions should be more likely to occur and more elaborate in the haplodiploid social groups than in diploid social groups (e.g. see table 3). The sophisticated patterns of communication and co-operation in the advanced eusocial Hymenoptera (Wilson 1971; Oster & Wilson 1978; Deneubourg & Goss 1989; Wilson & Hölldobler 1988; Hölldobler & Wilson 1990), many of which have promoted superorganismic views of colony function (Oster & Wilson 1978; Seeley 1989; Ratnieks & Reeve 1992), are *prima facie* evidence of such elaborate synergisms, but rigorous tests will require examination of the colony-level fitness consequences of experimentally varying the proportion of co-operators engaged in a specific task. The invasion protection hypothesis predicts that haplodiploid social systems will be more frequently characterized than will diploid social systems by nonlinear, synergistic, relationships between colony fitness and proportion of co-operators.

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APPENDIX 1. APPROXIMATIONS OF $G(x)$ FOR MATERNAL-CARE AND PATERNAL-CARE ALLELES FOR HAPLODIPLOIDS AND DIPLOIDS

Haplodiploids: maternal-care alleles

$M(x)$ is given by expression (4) (x is substituted for p) and $V(x) = x(1-x)/(3/2)N_e$. $M(x)$ simplifies to

$$\frac{2x(k-1)(1-x)^2}{3[kx(2-x)+(1-x)^2]}, \tag{15}$$

so that $M(x)/V(x)$ becomes

$$\frac{N_e(k-1)(1-x)}{(kx(2-x)+(1-x)^2)}. \tag{16}$$

For weak selection (i.e. k close to 1.0), the denominator of (16) is approximately equal to $x(2-x) + (1-x)^2 = 2x - x^2 + 1 - 2x + x^2 = 1$, so (16) is closely approximated by the linear function $N_e(k-1)(1-x)$. Substitution of this expression into equation (2) yields equation (6).

Haplodiploids: paternal-care alleles

$M(x)$ is given by expression (5) (x is substituted for p) and $V(x) = x(1-x)/(3/2)N_e$. $M(x)$ simplifies to

$$\frac{x(k-1)(1-x)}{3(x(k-1)+1)}, \tag{17}$$

so that $M(x)/V(x)$ becomes

$$\frac{N_e(k-1)}{2(x(k-1)+1)}, \tag{18}$$

We can rearrange (18) as $N_e(1/2)(k-1)[1 - x(k-1)/(x(k-1)+1)]$. For weak selection (i.e. k close to 1.0), $1 + x(k-1) \approx k$, so that (18) is closely approximated by the linear function $(N_e/2)(k-1)[1 - x((k-1)/k)]$. Substitution of the latter expression in equation (2) yields equation (7).

Diploids: maternal or paternal-care alleles

$M(x)$ is given by expression (9) (x is substituted for p) and $V(x) = x(1-x)/(2N_e)$. $M(x)$ simplifies to

$$\frac{kx(1/2 - x + x^2/2) - x(1-x)^2/2}{[kx(2-x)+(1-x)^2]}, \tag{19}$$

so that $M(x)/V(x)$ becomes

$$\frac{N_e(k-1)(1-x)}{[kx(2-x)+(1-x)^2]}, \tag{20}$$

Note that this is identical to expression (16) for haplodiploid maternal care, so the linear approximation $N_e(k-1)(1-x)$ immediately applies and can be substituted into equation (2) to yield the appropriate formula for $G(x)$.

APPENDIX 2. RATIOS OF ULTIMATE FIXATION PROBABILITIES APPROXIMATED BY THE RATIO OF SELECTIVE FORCES ACTING ON RARE ALLELES

Let $M(0)$ and $M(0)'$ be, respectively, the selective forces for a rare (parental or alloparental) allele in

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two different contexts, and let t and t' be the corresponding limits of integration (see text and formula (1)). When selection is weak and the effective population size is not too small we have seen that the probability of ultimate fixation u in the first context is approximately

$$t/\int_0^1 G(x)\mathrm{d}x.$$

The expression for the second context is the same (except that the primed quantities are substituted), so that the ratio of fixation probabilities for the two contexts (u/u) is approximately

$$(t/t')\left\{\int_0^1 G(x)'\mathrm{d}x/\int_0^1 G(x)\mathrm{d}x\right\}. \quad (21)$$

Now, an examination of equations (1) and (2) reveals that the integral $\int G(x)\mathrm{d}x$ evaluated from 0 to 1 is approximately equal to

$$\Delta x\exp\{-2\Delta x[M(0)/V(0)]\} + \Delta x\exp\{-2\Delta x[M(0)/V(0) + M(0+\Delta x)/V(0+\Delta x)]\} + \ldots + \Delta x\exp\{-2\Delta x[M(0)/V(0) + \ldots + M(1-\Delta x)/V(1-\Delta x)]\} \quad (22)$$

where Δx is the length of one of n equal sub-intervals resulting from a partitioning of the interval [0,1]. As successive terms of this sum decay exponentially, only the first few terms in the sum will make significant contributions to the overall sum. Now $M(0)$ is always of the form x times c (see formulae for Δps in text), where c is always greater than one (because the rare allele always increases in frequency). Thus $M(0)/V(0)$ is equal to the limit of $xc/(tx(1-x))$ as the allele frequency x approaches 0, that is, to c/t. The latter expression typically will be quite large, because c is always greater than 1.0 and $1/t = 2$ or $1.5 \times N_e$ is usually much greater than 1.0 (usually by two or more orders of magnitude). Consequently, the exponential decay in successive terms will be rapid as the allele frequency moves away from zero (regardless of how the partitioning is chosen).

Now $M(0+\Delta x)/V(0+\Delta x)$ can be approximated from a first-order Taylor series expansion as $M(0)/V(0) + \Delta x \cdot \mathrm{d}(M(0)/V(0))/\mathrm{d}x$. As we assume weak selection, the latter derivative is close to zero, so we can treat $M(0+\Delta x)/V(0+\Delta x)$ as roughly equal to $M(0)/V(0)$. The latter approximation will be reasonable when x is close to zero. As x becomes much greater than zero, the approximation becomes poorer, but this will matter very little because the corresponding exponential terms in the sum (22) will be very small. Thus, a rough approximation to the sum in (22) is just

$$\Delta x\exp\{-2\Delta x[M(0)/V(0)]\} + \Delta x\exp\{-2\Delta x[2M(0)/V(0)]\} + \ldots + \Delta x\exp\{-2\Delta x[nM(0)/V(0)]\}, \quad (23)$$

which is in the limit equal to

$$\int_0^1 \exp\{-2x[M(0)/V(0)]\}\mathrm{d}x = -[V(0)/2M(0)]\exp\{-2[M(0)/V(0)]\} + [V(0)/2M(0)]. \quad (24)$$

Now $\mathrm{e}^{-2M(0)/V(0)} = \mathrm{e}^{-2c/t} \ll 1$, because $c > 1$ and $1/t \gg 1$ (see above). Thus, expression (24) is close to $V(0)/2M(0) = t/2c$. Substituting this value (and the corresponding primed values for the other context) into (21) yields $u/u' \approx (t/t')(2t'c/2tc') = c/c' = xc/xc' = M(0)/M(0)'$, as conjectured. Note that as the effective population size becomes large, the quantity $1/t$ becomes large, and thus the ratio of fixation probabilities becomes more accurately approximated by $M(0)/M(0)'$. This means that the ratio of fixation probabilities for two different populations is not sensitive to differences in the effective sizes of those populations if both populations are reasonably large.

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