Conditional Relatedness, Recombination, and The Chromosome Numbers of Insects

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ABSTRACT. If two polymorphic loci are out of phase equilibrium, a homozygote at one of these loci is more highly related to its kin, at the other locus, than is an equivalent heterozygote. As a result, selection can favor (1) phenotypic responses to relative heterozygosity, and (2) increased recombination between the loci inducing these responses. Selection is expected to have these consequences only to the extent that kin strongly affect each other's fitnesses. The chromosome numbers of social insects appear to be higher, on average, than those of allied solitary species, which is consistent with this model on the assumption that chromosome numbers are selected in part for their effects on recombination.

INTRODUCTION

Coefficients of relatedness are always conditional on something. In the usual formulations they are conditional on structural relatedness, that is, on the pedigree connections between the individuals whose relatedness is to be evaluated. Full siblings are related by one half, aunt and nephew by one fourth, first cousins by one eighth, and so on, in randomly mating diploid populations of infinite size. At first it seems strange to speak of structural relatedness as something that *conditions* genetic relatedness, since we usually treat it as that which *defines* genetic relatedness. Structural relatedness is certainly one of the most important determinants of genetic relatedness, but it is not the only one. For example, phenotypic similarity is treated as the conditioner of genetic relatedness in so-called "spotter gene" or "green beard" models. Genetic relatedness at a given locus may be conditional on both structural relatedness and gene frequency if the fitnesses of the related individuals are determined according to one of a number of nonadditive schemes (Seger, 1981, and references therein). Genetic relatedness may also be conditional on heterozygosity, in species with certain kinds of population structures (Seger, 1976). Here I show that where relatedness *is* conditional on heterozygosity, relatively high rates of recombination may be favored by natural selection. This idea survives a comparative test in which chromosome number is used as a proxy for the average rate of recombination.

Ernest E. Williams became my thesis adviser some time after I first saw the outlines of this argument, but some time before I first attempted to publish it. This is nearly the twentieth version of a paper that has had a long and erratic development. Ernest encouraged me to stay with it on many occasions when I found myself stalled and confused. Several important changes resulted directly from his criticisms. From time to time he asked me whether I thought the model might help to explain lizard microchromosomes. I always said that I supposed it might, but that I was still unable to see how. In fact, I found the question

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extremely aggravating. Micros obviously posed a beautiful problem, closely related to all my other interests, and yet I had no idea what to do with them. Couldn't he see that? Why then did he persist in bringing them up? I now suspect that Ernest sets out quite deliberately to tie knots like this in the minds of all of his students. Those who manage to get rid of the things, by solving them, will advance science. The rest will suffer from recurring and unusually vivid flashes of ignorance, no matter what progress they may make on other problems. In either case, Ernest succeeds as a teacher. We may not have been exploited as students, but if this suspicion is correct, most of us were thoroughly outwitted.

RELATEDNESS CONDITIONAL ON HETEROZYGOSITY

Consider two loci each with two alleles (A_1 and A_0 at A, B_1 and B_0 at B). Let p_1 be the frequency of A_1 at A, and let p_2 be the frequency of B_1 at B. Let x_1, x_2, x_3, and x_4 be the frequencies of the gametes A_1B_1, A_1B_0, A_0B_1, and A_0B_0, respectively. Then we can express the gametic frequencies as

$$\begin{aligned} x_1 &= p_1p_2+D, \\ x_2 &= p_1(1-p_2)-D, \\ x_3 &= (1-p_1)p_2-D, \\ x_4 &= (1-p_1)(1-p_2)+D, \end{aligned}$$

by letting

$$D = x_1x_4-x_2x_3.$$

It follows from the definition of conditional probability that

$$P(A_i=A_1|B_j=B_1) = p_1+D/p_2 \qquad [1]$$

and

$$P(A_i=A_1|B_j=B_0) = p_1-D/(1-p_2). \qquad [2]$$

Thus the presence or absence of allele B_1 affects the probability that A_1 occurs on the same gamete if D is anything other than zero.

One of the classic results of population genetics theory states that D decays to zero from any initial value, in a closed randomly mating population, if all four alleles are neutral and there is any recombination between the A and B loci (Robbins, 1981; Malécot, 1948). D is traditionally referred to as the "coefficient of linkage disequilibrium," because the rate of decay depends on the tightness with which the loci are linked. But D can take nonzero values for unlinked loci, even under random mating, and so it is now increasingly referred to as the "coefficient of gametic phase disequilibrium."

Kimura (1956) and many others have studied the ways in which epistatic fitness interactions can generate nonzero values of D, at gene frequency equilibrium, in an infinite randomly mating population. Hill and Robertson (1968) were the first to show that phase disequilibrium is also generated at gene frequency equilibrium in the absence of epistasis, even between neutral loci, if the population is finite in size. The process is difficult to model, but easy to grasp intuitively. Gametes are sampled out of the parental generation, and therefore some are inevitably chosen in proportions that do not perfectly reflect the frequencies of their constituent alleles. On average there will be either an excess of "coupling" gametes (D positive) or an excess of "repulsion" gametes (D negative). These random perturbations of the gametic frequencies tend to cancel, giving an expectation of D equal to zero. But since D is very seldom *exactly* zero, the expectation of D^2 is greater than zero.

The expected magnitude of D depends strongly on the gene frequencies p_1 and p_2. It can be shown that

$$D(p_1,p_2) = [\sigma_d^2 p_1p_2(1-p_1)(1-p_2]^{1/2} \qquad [3a]$$

gives a reasonable approximation to the expected absolute value of D if selection is weak (Seger, 1980). In equation [3a]

$$\sigma_d^2 = 1/[3+4N(c+k)-2/(2.5+N(c+2k))], \quad [3b]$$

where N is the effective population size, k is the sum of the four forward and backward mutation rates, and c is the recombination fraction (Ohta and Kimura, 1969). Since k is usually much smaller than c, mutation rates have little effect on σ_d^2 (and thus on $D(p_1,p_2)$), except for very tightly linked genes. Selection also has little effect unless it is very strong (Ohta and Kimura, 1969). Linkage increases the expected magnitude of D, but even for unlinked genes D takes values approximately 0.02, 0.10, and 0.20 times its possible maxima (given p_1 and p_2) in populations of sizes N=1,000, N=50, and N=10, respectively. Such population sizes appear to be typical (Jain, 1976; Lande, 1979; Levin and Kerster, 1969; Loukas *et al.*, 1979; Schaal, 1980; Schaal and Levin, 1978; Wright, 1978). Thus, although the *sign* of D is unpredictable unless there are epistatic fitness interactions between the A and B loci, the expected *magnitude* of D may be substantial, even when A and B are unlinked.

Given the conditional probabilities [1] and [2], it is easy to find the correlation between an individual's A-locus alleles, conditional on its B-locus genotype. Assign the arbitrary genic values 1 and 0 to alleles A_1 and A_0. The joint genic distribution of the gametes that united to form the individual can be written

$$\begin{matrix} a_{11} & a_{10} & M_1 \\ a_{01} & a_{00} & M_0, \end{matrix} \quad [4]$$

where a_{11} is the probability that the individual received A_1 from both parents, $a_{10} = a_{01}$ is the probability that it received A_1 from one parent and A_0 from the other, and a_{00} is the probability that it received A_0 from both. Letting X denote the genic value of the individual's maternal gamete, and Y that of the paternal, it follows immediately that

$$E(XY) = a_{11}$$

and

$$E(X) = E(X^2) = E(Y) = E(Y^2) = a_{11}+a_{10} = M_1.$$

Suppose the individual is known to be a heterozygote at the B locus. The probability of this event is $k=2p_2(1-p_2)$. The probability that the individual is both B_1B_0 and A_1A_1 is $2x_1x_2$. Thus the conditional probability that it is A_1A_1, given that it is B_1B_0, can be written as

$$\begin{aligned} E(XY) = a_{11} &= 2x_1x_2/k \\ &= 2(p_1p_2+D)[p_1(1-p_1)-D]/k \\ &= p_1^2-2D[D+p_1(2p_2-1)]/k. \end{aligned}$$

By similar reasoning

$$\begin{aligned} E(X) = M_1 &= (x_1+x_2)/2k \\ &= p_1-D(2p_2-1)/k. \end{aligned}$$

Thus

$$Cov(X,Y) = -[D^2/k][2+(2p_2-1)^2/k], \quad [5]$$

and

$$Var(X) = p_1(1-p_1)+[D(2p_1-1)/k][2p_1-1-D(2p_2-1)/k]. \quad [6]$$

Given any intermediate values of p_1 and p_2, Cov(X,Y) is negative or zero. Thus since we expect a nonzero value of D in a finite population, we expect a negative correlation between the A-locus gametes of a B-locus heterozygote. The correlation between an individual's gametes is the same as the coancestry of its parents, so the parents of a B-locus heterozygote have a negative coancestry and are negatively related at the A locus.

Where mating is at random, there can be no overall correlation between parents at the A locus or at any other. This

clearly implies that the parents of B-locus homozygotes are positively related at the A locus, by an amount sufficient to cancel the negative relatedness between parents of B-locus heterozygotes. By means of an argument exactly like that leading to equations [5] and [6] it can be shown that

$$\mathrm{Cov}(X,Y) = [D^2/k][2-(2p_2-1)^2/k] \qquad [7]$$

and

$$\mathrm{Var}(X) = p_1(1-p_1)+[D(2p_2-1)/k][1-2p_1-D(2p_2-1)/k], \qquad [8]$$

where $k=1-2p_2(1-p_2)$ is the overall probability of being a B-locus homozygote. In this case Cov(X,Y) is always zero or positive. Thus the parents of a B-locus homozygote are positively related at the A locus, in a finite population.

This implies, but does not prove, that a B_1B_0 heterozygote will be related to its full sibling by less than one half, across the A locus, while a B_1B_1 or B_0B_0 homozygote will be related to its sibling by more than one half. The explicit demonstration is tedious but straightforward (Seger, 1980). The conclusion can be summarized by saying that A-locus relatedness is *conditional* on B-locus genotype, for full siblings in a finite population. Figure 1 shows these conditional coefficients of relatedness as a function of population size, for the case in which $p_2=0.5$ and the A and B loci are unlinked. Linkage increases the expected magnitude of D, but decreases the extent to which the conditional coefficients diverge from one half. This is illustrated in Figure 2, for populations of effective size 10, 50, and 250.

Suppose the phenotype affected by A-locus genotype has fitness effects on both ego and its sibling. Then ordinary kin selection will favor A-locus alleles that maximize ego's inclusive fitness, given the siblings' nominal relatedness of one half, and given any well defined constraint on their possible joint fitnesses.

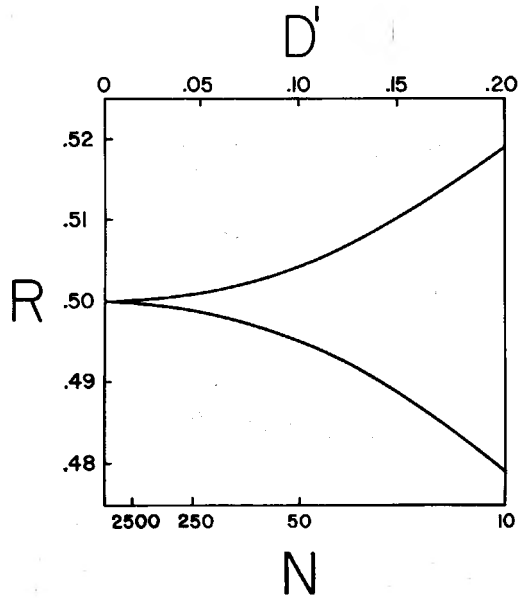

Figure 1. Conditional relatedness as a function of population size.

The upper curve shows R^+, a B-locus homozygote's A-locus relatedness to its sibling, as a function of N, the effective population size. The lower curve shows R^-, the corresponding relatedness of a B-locus heterozygote. The upper horizontal axis shows values of D', the standardized coefficient of gametic phase disequilibrium, corresponding to the values of N on the lower axis. Loci A and B are unlinked, and $p_2 = 0.5$.

But we know that ego's A-locus relatedness to its sibling is actually *conditional* on ego's B-locus zygosity. Thus it seems to follow that an A-locus allele that made its phenotypic effect appropriately conditional on ego's B-locus genotype could displace all alleles with unconditional phenotypic effects. This conjecture can be shown to hold under plausible assumptions regarding the constraints on joint fitness (Seger, 1980), and can be summarized by saying that an unconditional A-locus phenotype is *not* in general an ESS against a phenotype that is conditioned on the zygotic states of other polymorphic loci (Seger, 1976).

From the point of view of the A locus, the zygotic state of B is a signal that conveys information about the A-locus re-

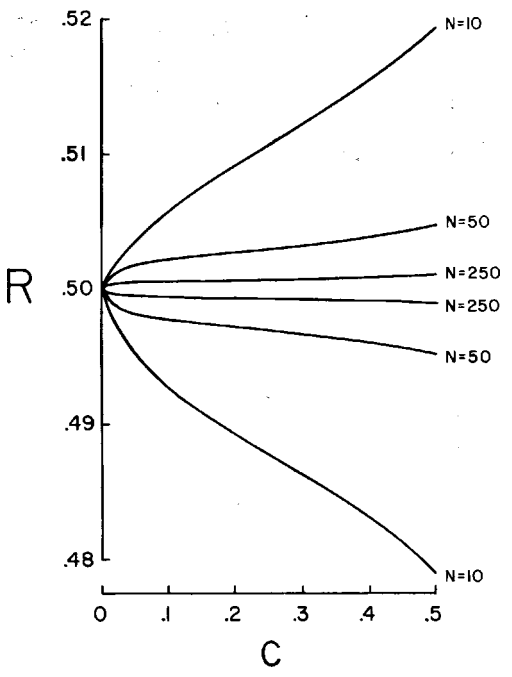

Figure 2. Conditional relatedness as a function of linkage between A and B.

Increasing the linkage between loci A and B increases the expected magnitude of D', but decreases the difference between R^+ and R^-. Here $p_2 = 0.5$, and c varies between 0 (no recombination) and 0.5 (free recombination). Three different population sizes are shown. Upper curves are R^+, lower curves are R^-, as explained in the legend to Figure 1.

latedness of ego and its sibling; genotypes B_1B_1 and B_0B_0 indicate an inflated A-locus relatedness, and B_1B_0 indicates a deflated relatedness, relative to the unconditional relatedness of one half. Thus we can refer to ego's membership in distinct *relatedness sets* which are defined by its B-locus genotype. In the example considered so far, in which there is only one signalling locus, there are only two conditional relatedness sets. But in principle there could be a large array of signalling loci and several distinct relatedness sets corresponding to different configurations of homozygosity and heterozygosity across the array of signalling loci.

Figure 2 shows that linkage between A and B reduces the distinctness of A-locus relatedness sets defined on B. Thus a gene that increased the rate of recombination between A and B would increase the distinctness of relatedness sets, and would thereby promote the spread of conditionally responding alleles at the A locus. But would selection also favor the gene for increased recombination?

SELECTION FOR INCREASED RECOMBINATION BETWEEN SIGNALLING LOCI

Consider a locus C, unlinked to either A or B, with alleles C_1 and C_0. C_1 promotes recombination between A and B, while C_0 suppresses it. Formally, B is a signalling locus from the point of view of C, just as it is from the point of view of A, despite the fact that C is unable to bring about any phenotypic response to the zygotic state of B. If A were unlinked to B, as C is, it and C would belong to the same relatedness sets defined on B and would therefore have the same inclusive fitness interests. This implies that C_1, which reduces linkage, might indeed be favored over C_0. Consider a slightly different model in which there are many polymorphic B loci, all linked to each other but unlinked to A and C. In the limit of complete linkage to each other they constitute a supergene with many pseudoalleles of different degrees of complementarity. It is clear that the average homozygosity over this set of B loci carries less information about the relatedness of an individual's parents when the loci are tightly linked than it does when they recombine freely, which implies that a gene promoting recombination between the B loci could be favored.

Like the fitnesses of sex ratio phenotypes, the fitnesses of recombination phenotypes do not manifest themselves until one or more generations following

their expression. Thus there seems to be no simple way to apply genotypic covariance methods (Price, 1970; Seger, 1980, 1981) to the evolution of recombination modifiers. But positive selection for increased recombination can be demonstrated by simulation of a particular model with many signalling loci. One such simulation is described below.

The model species is a diploid hermaphrodite in which self-fertilization alternates with random mating. The population is infinitely large, but the effective population size is finite, owing to the high frequency of selfing. Offspring are produced in pairs. Each pair is given one unit of resource (x), and an individual's fitness (W) depends on the amount of resource it consumes, according to the function

$$W(x) = 1.0 - e^{-x}.$$

Given this fitness function and total shareable resource, the fitness set for sibling pairs is slightly convex (Fig. 3). One sibling, called "left," divides the unit of resource between itself and the other sibling, called "right."

Locus A controls the left sibling's division of the unit of resource, and locus C controls the rate of recombination at B, a large array of signalling loci to which neither A nor C is linked. In the simulation, A and C are treated explicitly, but B is treated in the aggregate. Typical loci in B are imagined to be highly polymorphic, such that outbred individuals are always more heterozygous than inbred individuals, if recombination between B loci is free. C_1 causes free recombination between all B loci, and C_0 suppresses recombination between them. In some runs C_1 was dominant to C_0, and in others it was recessive.

The behavior of this genetic system can be described as follows. All of the selfed progeny of recombining parents are moderately homozygous at B, having received the same allele in egg and sperm at approximately half of their loci.

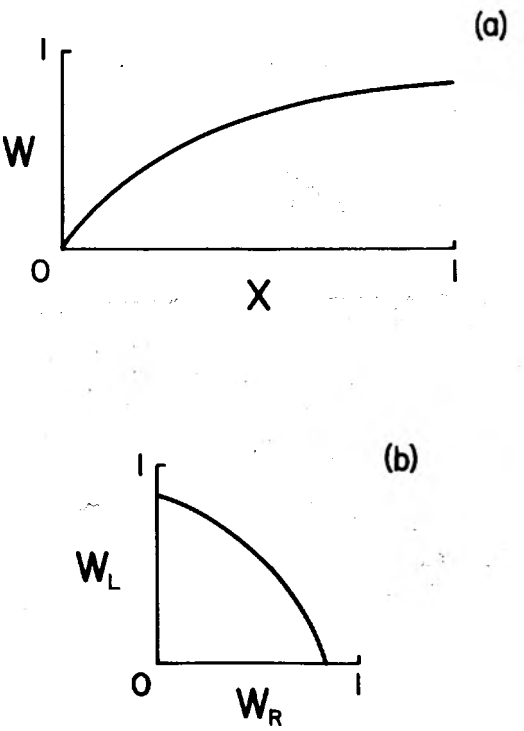

Figure 3. Fitness set for siblings.
The fitness of each sibling is determined by its consumption of resource (x), according to the function $W=1.0-e^{-x}$. This function is shown in (a). Given that two siblings share a total of one unit of resource, the set of their possible joint fitnesses is as shown in (b). W_L is the fitness of the "left" sibling, and W_R is the fitness of the "right" sibling, as explained in the text.

Half of the selfed progeny of nonrecombining parents are entirely homozygous, having received an intact copy of the same entire strand in egg and sperm, but half of them are only slightly homozygous, having received the parent's maternal strand in one gamete, and its paternal strand in the other. A_1 causes the left sibling to respond to its zygosity at B and is codominant with A_0, which when homozygous causes a fixed division of the resource. Left siblings of genotypes A_0A_0 keep 0.8 units of resource and give the remaining 0.2 units to their right siblings. This is the ESS nonresponding phenotype, which was found by playing

A_0 variants against each other in the absence of A_1. Left siblings of genotypes A_1A_0 and A_1A_1 keep relatively more for themselves when heterozygous at B (0.85 and 0.9 units, respectively), but keep less (0.75 and 0.7) when moderately or entirely homozygous at B. This level of response is derived from a simple inclusive fitness model, based on the formal relatedness of selfed siblings.

The simulation evolves as expected, when allowed to run freely for many generations. A_1 spreads from low frequencies, even in populations fixed for C_0, the gene that blocks recombination at B. This happens because the zygosity of B contains some information about the siblings' relatedness even when there is no recombination. Consider an outbred carrier of A_1, the responding gene. Such an individual is heterozygous at B, and therefore gives relatively little of the resource to its right sibling. This is appropriate, owing to the relatively low relatedness of outbred siblings. Now consider the inbred (selfed) carriers of A_1. Half of them are heterozygous at B, because there is no recombination, and mistakenly act as if they were outbred, but half of them are homozygous and appropriately give a relatively large share of the resource to their right siblings. Thus carriers of A_1 respond in a way that is appropriate to their actual relatedness three quarters of the time, and this is sufficient to give them a higher average inclusive fitness than that realized by the nonresponding A_0A_0 homozygotes, in this particular model.

When C_1 and A_1 are introduced together they both spread, and their progress appears to be mutually reinforcing. They go to fixation whether C_1 is recessive or dominant and whether the C and A loci are linked or unlinked (Fig. 4). This happens because the inbred progeny of recombining parents usually carry C_1, and those who also carry A_1 never make the inappropriate response made by half of the nonrecombinant inbred responders. C_1 increases the information about relatedness that is conveyed by the zygosity of the signalling B loci, and thereby improves the accuracy of the response to zygosity. This benefits both C_1 and A_1 because the siblings are equally related across all loci.

This model differs in several respects from previous models for the evolution of recombination. It does not require high fecundity, environmental variation, or epistatic fitness interactions. It does not require that the recombination modifier be linked to the sites it affects, or even that there be any average fitness differences between alleles at the affected loci. But this model does require that the affected loci (the B array in the realization discussed here) exhibit the form of overdominance that is brought about by the spread of genes that promote responses to the zygosity of the affected loci.

SOCIALITY, RECOMBINATION, AND CHROMOSOME NUMBERS

Selection for phenotypic responses to zygosity should be much stronger in some species than in others. Selection should be weak where relatives seldom compete or where there is never any significant inbreeding or gametic phase disequilibrium. Both of these conditions are probably found in many species with very large effective population sizes, for example, those with planktonic larvae. Selection should be strong where relatives often compete and where conditional relatedness sets are well differentiated. These conditions are probably found in a variety of species with small effective population sizes, for example, at all stages of the life cycle in many social species, during the larval stage in species with gregarious development, and during the embryonic stage in many self-compatible hermaphrodites. The fact that predominantly self-pollinating angiosperms tend to have higher chiasma frequencies than do their predominantly cross-pollinating relatives (Lewis and

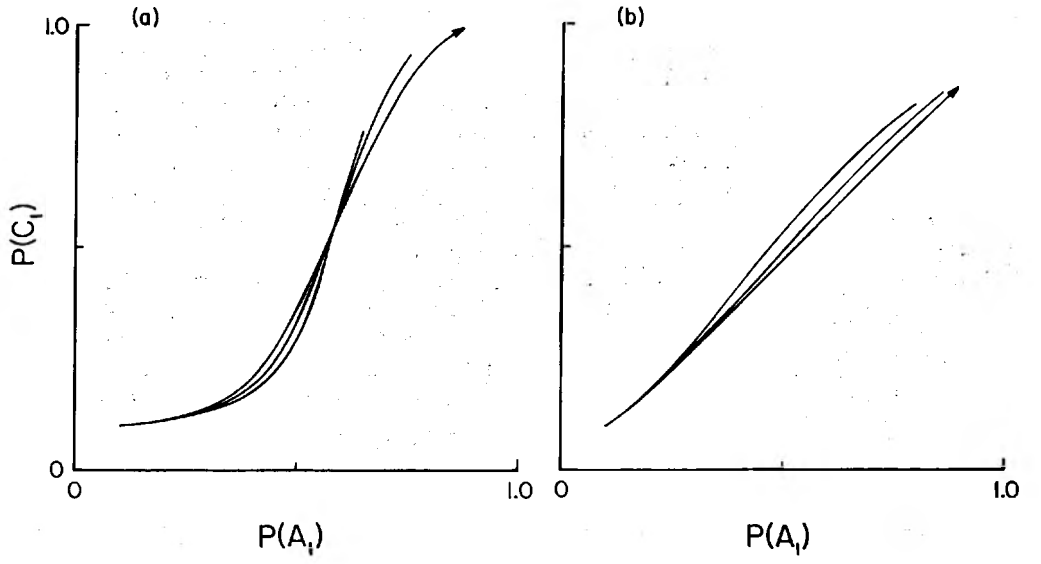

Figure 4. Gene frequency trajectories at loci controlling recombination and sibling behavior in a deterministic model.

A_1 promotes responses to zygosity, and C_1 promotes recombination. Figure 4.1a: C_1 is recessive to C_0. Trajectories terminate at generation 1350. Recombination fractions between A and C are zero (complete linkage), 0.005, and 0.5 (free recombination) in the longest, intermediate, and shortest trajectories, respectively. Figure 4.1b: C_1 is dominant to C_0. Trajectories terminate at generation 800. Recombination fractions are as in Figure 4.1a.

Both A_1 and C_1 spread more rapidly when linked to each other than when not linked, and both spread more rapidly when C_1 is dominant than when it is recessive. Rates of gene frequency change along the trajectories suggest that the marginal fitnesses of A_1 and C_1 change in complex ways during the course of selection. Apparent selective advantages for A_1 range between 0.003 and 0.012. Those for C_1 range between 0.003 and 0.025. C_1 benefits more from the advance of A_1 than A_1 does from the advance of C_1, which is expected because recombination affects only the responding phenotypes (those carrying A_1).

John, 1963; Murray, 1976; Ved Brat, 1965; Zarchi *et al.*, 1972) is consistent with this expectation, but it is also consistent with other models for the evolution of recombination under selfing (Charlesworth *et al.*, 1977; Maynard Smith, 1978).

In almost all social insect species there are aspects of larval development and of adult behavior that appear to reflect the distribution of coefficients of relatedness within colonies (Hamilton, 1972; Trivers and Hare, 1976). In some species there is also intense competition between colonies (Wilson, 1971; Michener, 1974). Many species of social insects appear to have populations small enough to generate significant gametic phase disequilibrium even under local random mating (Talbot, 1965; Wilson, 1958, 1963, 1971; Michener, 1974; Haskins and Wheldon, 1965; Haskins, 1970; Taylor, 1978). If so, Isoptera (termites) and social Hymenoptera (ants, bees, and wasps) should have rates of recombination higher on average than those of their closest solitary relatives.

Unfortunately, there are no comparative data on the recombination of markers, or even chiasma frequencies, for social insects and their relatives. But chromosome number is a major determinant of the average linkage within a genome, and chromosome numbers are known from many species of insects. Loci at the opposite ends of a long chromo-

some may recombine almost freely, if several crossovers usually occur between them. Nonetheless, they remain somewhat linked. A fission occurring between them reduces their linkage all the way to zero. More importantly, other pairs of loci at map positions between them but on opposite sides of the point of fission, which were formerly significantly linked, also become unlinked. Thus given an approximately constant number of crossovers per genome, the average linkage between loci is a strongly decreasing function of the chromosome number. This implies that chromosome number can be used as a proxy for the rate of recombination in comparative studies, as long as large numbers of species are compared.

As expected, the average chromosome numbers of eusocial taxa are consistently higher than those of the most closely related nonsocial taxa for which chromosome data exist (Sherman, 1979). Formicidae, Apidae, and Vespidae represent independent derivations of eusociality within the aculeate Hymenoptera (Wilson, 1971). Although only a few solitary aculeates have been karyotyped, their chromosome numbers are similar to those of the entirely solitary Symphyta and Parasitica (Table 1). This suggests that high numbers have evolved independently in the social families. The Isoptera are entirely eusocial, so no close comparisons are possible in their case. But their average chromosome number exceeds those of the other orthopteroid orders Blattodea, Orthoptera, Mantodea, Dermaptera, and Embioptera (Table 1).

It should be emphasized that the postulated selection in favor of increased recombination has nothing to do with sociality as such. Sociality is merely being used as an indicator of situations in which interactions are expected to be strongly conditioned on relatedness. In such situations natural selection should favor phenotypic responses to individual zygosity, if at the same time the population structure is such as to create significant gametic phase disequilibrium. Selection in favor of increased recombination is thus an expected secondary consequence of the establishment of systems of responding loci. It is therefore expected in some solitary species, for example, ones in which extended juvenile dependency leads to parent-offspring conflict over the allocation of resources (Trivers, 1974), and it is not expected in some social species, for example, those with very large effective population sizes. The range of haploid numbers in ants (3–46) exceeds that in all other Hymenoptera. If the argument made here is correct, some of this variation should be associated with differences of population structure. Unusually high chromosome numbers occur in *Rhytidoponera*, *Myrmecia*, and *Nothomyrmecia*. Aptery, brachyptery, and patchy distributions are common in these genera (Haskins and Wheldon, 1965; Haskins, 1970; Taylor, 1978), suggesting that many of their species typically experience small effective population sizes.

The average chromosome numbers of six species of blattid roaches believed to exhibit larval aggregation are 60% higher than the chromosome numbers of ten species believed not to exhibit larval aggregation (Sherman, 1979).

It is difficult to assign levels of statistical significance to the observed patterns of chromosome number variation, because it cannot be assumed that the chromosome numbers of related species are statistically independent. Thus it is not legitimate to perform an analysis of variance on the chromosome numbers of species, as was done by Sherman (1979). The analysis given here treats genus averages as primary observations. This improves matters somewhat, but since genera tend to be correlated within families, as do species within genera, genera cannot properly be treated as independent points in a comparison between families. Thus the significance levels given in the legend to Table 1 should be taken only as indicators of the relative strengths

TABLE 1. AVERAGE HAPLOID CHROMOSOME NUMBERS OF SOCIAL INSECTS AND THEIR ALLIES.*

	N	$\bar{n}$	s.d.
Orthopteroids			
DERMAPTERA	20	13.38	5.92
EMBIOPTERA	4	10.83	0.41
ORTHOPTERA	33	10.84	2.72
PHASMATODEA	40	20.14	4.45
MANTODEA	69	13.20	2.44
BLATTODEA	64	19.05	6.62
Blattidae	7	18.03	5.90
ISOPTERA*	27	20.71	1.95
Hymenoptera			
SYMPHYTA	27	9.41	3.96
APOCRITA			
PARASITICA	19	8.51	2.38
ACULEATA			
Vespoidea			
Formicidae*	69	15.76	6.82
Eumenidae	2	7.63	0.53
Vespidae*	2	14.30	2.40
Sphecoidea			
Halictidae	4	11.50	5.26
Anthophoridae	3	13.67	4.04
Megachilidae	1	16.00	—
Apidae*	18	16.23	2.86

*Sources of data are listed in Literature Cited B.

Social taxa are indicated in the table by an asterisk. Most of the analysis was conducted using genus averages rather than species counts, to increase the independence and representativeness of sample points, and to increase the normality of the resulting distributions. Thus N is the number of genera in a sample, and $\bar{n}$ is the mean of the genus mean haploid chromosome numbers. The average for Orthoptera, however, represents modal numbers for 28 families and subfamiles of Caelifera (based on hundreds of studied species [White, 1973]), and average numbers for five families of Ensifera (based on about 70 genera [Makino, 1951]). Data for Isoptera include nine unpublished species counts in five genera of Kalotermitidae (P. Luykx, personal communication). One-tailed U-tests were applied to comparisons of interest (t-tests with d.f. adjusted for unequal variances were also applied and gave similar results).

Isoptera exceed the blattid roaches, to whom they are believed to be most closely related (McKittrick, 1965), but the difference is not significant. The comparison with Blattodea as a whole is significant ($p<0.005$), as are those with Orthoptera, Mantodea, Dermaptera ($p<0.0005$), and Embioptera ($p<0.001$). The comparison with Phasmatodea is almost significant ($p<0.1$).

Formicidae (ants) and Apidae (eusocial bees) each exceed both Parasitica (chalcids, ichneumons, gall wasps) and Symphyta (sawflies, horntails) ($p<0.0005$). The small sample of halictid, anthophorid, and megachilid bees consists of five species with haploid numbers of 16, and three species with haploid numbers of 6, 8, and 9. The species with low numbers are communal to quasisocial (Michener, 1974), and three of the species with high numbers are solitary, contrary to expectation. Owing to the complexity of social evolution in the bees, a much larger and more representative sample of chromosome numbers is required to determine whether in fact they exhibit a pattern different from the one seen elsewhere in the Hymenoptera.

The most appropriate comparison is that between the Vespidae (eusocial paper wasps) and the closely related Eumenidae (solitary potter wasps). Their average numbers diverge in the expected direction, but too few genera have been studied to allow a statistical test of the generic averages. If all the published species counts (7 and 5, respectively) are used, the difference is significant ($U=5$, $p=0.024$, $t=2.8$, 8 adjusted d.f., $p<0.025$). Formicidae exceed Eumenidae ($p<0.005$) but not Vespidae, using species counts for the wasps.

of different associations, not as statistical tests.

A conservative test of the overall pattern can be carried out by comparing the signs of the differences between groups at the same taxonomic levels. There are at least six relevant comparisons. Apidae exceed all other bees. Vespidae exceed Eumenidae. Aculeata as a whole, who are mainly social in this sample, exceed Parasitica. Isoptera exceed Blattodea. *Rhytidoponera*, *Myrmecia*, and *Nothomyrmecia* exceed other ants. Gregarious blattids exceed nongregarious blattids. The comparison between Formicidae and Eumenidae could be added to this list, as could separate comparisons between Apidae and the other families of bees, but these will be omitted in the interests of conservatism. The probability that six independent comparisons all fall in the same unspecified direction is 0.03125, and the probability that they all fall in the same specified direction is 0.015625. Thus it appears that an association does exist between high chromosome number and circumstances favorable to the evolution of responses to zygosity.

DO BEES HAVE GREEN SETAE?

Sherman (1979) was the first to note the association between eusociality and high chromosome numbers. His explanation for the association, which differs from the one given here, can be summarized as follows. Parent and offspring are always related by exactly one half, because the parent transmits to the offspring exactly one member of each of its pairs of homologous chromosomes. Full siblings also expect to share half of their chromosomes identical by descent through the parents, but owing to the uncertainties of meiosis this expectation has a finite variance. Some siblings have more than half of their DNA in common, identical by descent, and others have less than half. Thus some siblings are related by more than one half, and others by less. If an individual could discriminate between the two kinds of siblings, favoring those to which it was related by more than one half, it could increase its inclusive fitness. In a social insect colony such discrimination is likely to work against the reproductive interests of the queen and her mate. The scope for such discrimination is a function of the variance of actual relatedness between siblings, and the variance of relatedness is an inverse function of the number of chromosome pairs. It is in the queen's interest to reduce the variance of relatedness as far as possible, so as to create a situation in which workers are selected to minister to their reproductive siblings "on the basis of their need rather than kinship" (Sherman, 1979). Increasing the chromosome number is probably one of the simplest and most effective ways to reduce the variance of relatedness. Thus chromosome numbers are expected to increase in eusocial lineages and in others where siblings interact intensely.

This argument turns on two critical assumptions. First, the variance of the proportion of DNA held in common identical by descent corresponds to a variance of relatedness on which kin selection could act. Second, although an increased variance of relatedness would be in the offspring's interest, a reduced variance will evolve because that is in the queen's interest. This second assumption is problematic, but it will not be considered here in any detail. The first assumption is almost certainly incorrect, at least as stated by Sherman (Dawkins, 1979). The idea seems to arise naturally from the identity-by-descent view of relatedness, which emphasizes amounts rather than covariances. The problem with it is that identity at one locus does not predict identity at another. As Dawkins puts it, "If I want to guess whether your hand of cards contains the ace of spades I would be quite wrong to say: I already know you have the 2, 3, 5, 6, 7, 9, 10, Jack and King of spades; therefore you have a strong hand in spades; therefore you probably have the ace!" A rational gene that controlled discrimination would gain no information regarding its presence in a sibling of its bearer by knowing that the sibling, like the gene's own bearer, had a green beard. If the discrimination gene were tightly linked to the green beard gene the argument might hold, except that selection at all unlinked loci would oppose the favorable discrimination. Few theoretical arguments are affected by the fiction of the single omnipotent gene, but this appears to be one that is. Any act as complex as a comparison of particular details of one's own and one's sibling's phenotypes must depend directly on scores or hundreds of genes, and indirectly on thousands. It is asking a lot to expect them all to be firmly linked to the same phenotypic marker and to be protected from interference on the part of genes elsewhere in the genome.

It was pointed out above that correlations of allelic state arise through drift in finite populations, and that these correlations can drive the evolution of phenotypic responses to zygosity. These same correlations can be shown to underlie a consistent version of Sherman's argument for the evolution of discrimination

based on phenotypic similarity. But as will be seen, this version of the argument may have implications somewhat different from those suggested by Sherman.

Consider a genetic system consisting of two unlinked loci, each with two alleles. Locus B determines a visible phenotypic attribute and has two distinct, fully penetrant, codominant alleles, B_1 and B_0. Locus A determines a response to the B-locus similarity of its bearer and another individual taken at random from the local population. With an adjustment for structural relatedness the argument can easily be adapted to the case of siblings, but the case of unrelated individuals is both simpler and more general than that of structurally related individuals. Locus A can be taken to represent every locus in the genome that is unlinked to B. We seek the A-locus relatedness of two individuals, conditioned on their B-locus similarity.

Let p_1 be the frequency of A_1, and let p_2 be the frequency of B_1. Then from equations [1] and [2] we have that

$$q_1 \equiv P(A_i=A_1|B_j=B_1) = p_1+D/p_2$$

and

$$q_2 \equiv P(A_i=A_1|B_j=B_0) = p_1-D/(1-p_2),$$

where D is the coefficient of gametic phase disequilibrium. Define as "alike" those pairs of individuals in which both are B_1B_1 or both are B_0B_0. Assuming Hardy-Weinberg proportions at both loci, and assuming that individuals meet at random, the probability that two individuals are both B_1B_1 is clearly

$$f_1 = p_2^4,$$

while the probability that both are B_0B_0 is

$$f_0 = (1-p_2)^4.$$

Consider the A-locus genotypes of the B_1B_1 pair. The probability of finding A_1 on either chromosome at the A locus in either individual is just q_1. Thus the probability that both individuals are A_1A_1 is q_1^4. Similarly, if both individuals are B_0B_0 the probability that they are also both A_1A_1 is q_0^4. Thus if two individuals are known to be "alike" at B, the probability that they are both A_1A_1 is

$$a_{22} = (f_1q_1^4+f_0q_0^4)/k,$$

where $k=f_1+f_0$. The probability a_{22} is the first element of the joint genotypic distribution

$$\begin{array}{cccc} a_{22} & a_{21} & a_{20} & M_2 \\ a_{12} & a_{11} & a_{10} & M_1 \\ a_{02} & a_{01} & a_{00} & M_0. \end{array} \qquad [9]$$

Proceeding in this manner it is easy to show that the other elements of the joint genotypic distribution can be represented as

$$\begin{aligned} a_{21} &= a_{12} = 2(f_1c_1q_1^2+f_0c_0q_0^2)/k, \\ a_{11} &= 4(f_1c_1^2+f_0c_0^2)/k, \\ M_2 &= (f_1q_1^2+f_0q_0^2)/k, \\ M_1 &= 2(f_1c_1+f_0c_0)/k, \end{aligned}$$

where $c_1=q_1(1-q_1)$ and $c_0=q_0(1-q_0)$. M_0 and the remaining a_{ij} are easily found by subtraction. Substitution of the key elements of [9] into

$$R = [a_{22}+a_{21}+\tfrac{1}{4}a_{11}-(M_1+\tfrac{1}{2}M_2)^2]/[M_1M_0+\tfrac{1}{4}M_2(1-M_2)]$$

(Seger, 1980) gives R^+, the A-locus relatedness of two randomly chosen individuals who are "alike" at B. If both loci are polymorphic and if D is nonzero then R^+ is positive (Table 2).

Define as "unlike" those pairs of individuals in which one is B_1B_1 and the other is B_0B_0. Define as "somewhat" those pairs in which at least one individual is the heterozygote B_1B_0. Then proceeding as above it is easy to find R^-, the relatedness of "unlike" pairs, and R^s, the relatedness of "somewhat" pairs. These are both negative.

Like the conditional coefficients dis-

TABLE 2. CONDITIONAL RELATEDNESS IN A SPOTTER GENE MODEL.*

D′=.05		p_2		
		.1	.3	.5
p_1	.1	.00001 −.00271 −.01430	.00076 −.00038 −.00596	.00499 0 −.00499
	.3	.00001 −.00269 −.01390	.00075 −.00038 −.00594	.00499 0 −.00499
	.5	.00001 −.00269 −.01385	.00075 −.00038 −.00594	.00499 0 −.00499

D′=.1		p_2		
		.1	.3	.5
p_1	.1	.00003 −.01115 −.06279	.00309 −.00154 −.02398	.01980 0 −.01980
	.3	.00003 −.01089 −.05575	.00302 −.00153 −.02361	.01980 0 −.01980
	.5	.00003 −.01086 −05501	.00301 −.00153 −.02357	.01980 0 −.01980

*The upper number in each set of three is the A-locus relatedness of two randomly chosen individuals that are "alike" at the B locus. The lower number is the relatedness of "unlike" pairs, and the middle number is the relatedness of "somewhat" pairs. Conditional relatedness is shown for three different frequencies of A_1 and B_1, at two different values of D′, the standardized coefficient of gametic phase disequilibrium. Each distribution is symmetrical about both p_1=0.5 and p_2=0.5.

cussed in the introduction, R^+ and R^- diverge as D increases (Table 2). But R^+ and R^- also diverge as the A and B loci become more tightly linked, unlike the conditional coefficients discussed in the introduction, which converge on the unconditional coefficient as linkage tightens. This implies that conflicts might arise between A-loci closely linked to B, and A-loci unlinked to B, because the former will tend to be farther out of phase quilibrium with B than will the latter. In view of this it is hard to see whether a high rate of recombination looks more like a way to frustrate discrimination than it does like a way to promote it. By reducing the expected gametic phase disequilibrium between B and a typical A locus, recombination reduces the difference between R^+ and R^-, thereby reducing the potential advantage of discrimination. But recombination also increases the fraction of the genome that is effectively unlinked to any given B locus. By increasing the similarity of the different values of R^+ evaluated at different loci, recombination might well increase the ease with which mechanisms inducing discrimination could evolve.

Phenotypic discrimination mechanisms of the form envisioned by Sherman and others require that an individual be able to assess its *own* similarity to another individual. Recent experimental work on discrimination by means of individual odor differences in *Lasioglossum* demonstrates that these bees compare strangers to the aggregate of their nestmates, and that they apparently do not perceive their own odors (Greenberg, 1980; C. D. Michener, personal communication).

CONCLUSIONS

It is possible that high chromosome numbers are caused by some feature of sociality that has nothing to do with kinship. For example, the genomes of social insects might be larger than those of solitary insects, and there might be an optimum distribution of chromosome sizes, at least within particular lineages. If this were true, chromosome number and genome size would correlate positively, but several lines of evidence suggest that they do not. Chromosome numbers and absolute DNA values for 45 species of insects representing six orders do not correlate either within orders or between them (Fig. 5). Imai, Crozier, and Taylor

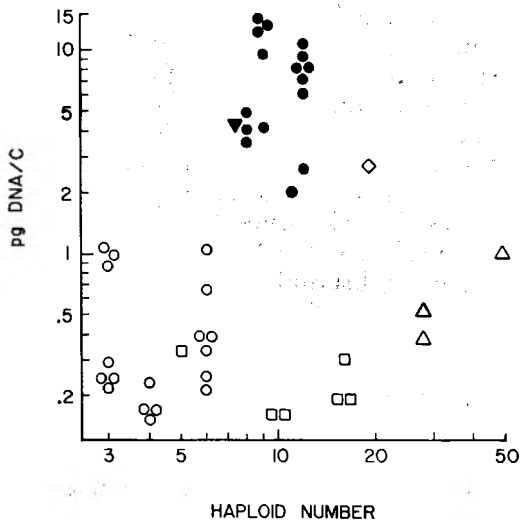

Figure 5. Relationship between genome size and chromosome number in six orders of insects.

Genome size is expressed as picograms of DNA per haploid genome. The orders are Orthoptera (●), Hemiptera (▼), Coleoptera (◇), Lepidoptera (△), Diptera (○), and Hymenoptera (□). Spearman rank correlations are nonsignificant within Orthoptera (0.097), Diptera (0.006), and Hymenoptera (zero). The entire sample is significantly correlated ($r_s=0.34$, $p<0.05$) because it is dominated by Orthoptera and Diptera, whose ranges do not overlap on either variable. Using mean values for each of the six orders, the overall correlation is negative and nonsignificant ($r_s=-0.086$).

There is a large average difference in genome size between the hemimetabolous and holometabolous insects in this sample (represented by filled and by open symbols, respectively). The only social species represented (*Apis mellifera* and *A. cerana*, n=16, 0.19 pg) have genomes that appear to be small even for Hymenoptera. Other Hymenoptera are *Megachile rotundata* (n=16, 0.3 pg), *Habrobracon juglandis* and *H. serinopae* (n=10, 0.16 pg), and *Mormoniella vitripennis* (n=5, 0.33 pg).

Sources of data are listed in Literature Cited B.

(1977) optically measured chromosome lengths in 16 species of myrmeciine and ponerine ants with haploid numbers of four to 42. They found that average chromosome length is inversely proportional to chromosome number, and concluded that the diversity of chromosome number in these subfamilies does not reflect any large or systematic differences of genome size. Data for angiosperms (Bennett and Smith, 1976) and for vertebrates (White, 1973) also fail to suggest an inherent relationship between genome size and chromosome number.

The first argument outlined here implies that selection will favor relatively high overall rates of recombination in species where it also favors phenotypic responses to heterozygosity. Such species are characterized by certain behavioral, ecological, and demographic parameters. To the extent that sociality indicates the required parameters, and to the extent that chromosome number indicates the rate of recombination, social insects are expected to have elevated chromosome numbers, as they do on average. The second argument, that of Sherman (1979), also leads to the expectation that social insects will have elevated rates of recombination. The two arguments invoke nearly identical sets of causal parameters and thus make similar comparative predictions. There seems to be no way to distinguish between them on the basis of available evidence.

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NOTE ADDED IN PROOF

Tooby (J. theor. Biol., **97**: 557–576 [1982]) has recently argued that eusocial

insects are expected to have high rates of recombination (and thus high chromosome numbers) because they live at high population densities and should therefore be subject to particularly intense selection for resistance to rapidly evolving pathogens.

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(FIGURE 5 AND TABLE 1)

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