Sex causes altruism. Altruism causes sex. Maybe.

Joel R. Peck

Centre for the Study of Evolution, School of Life Sciences, The University of Sussex, Brighton BN1 9QG, UK
(*j.r.peck@sussex.ac.uk*)

This study presents a mathematical model in which the fitness of an individual depends on the individual's genotype (individual effects) and on the genotypes of other members of the individual's local group (group effects). The findings suggest that, if phenotypes are a result of complex interactions between genes at different loci, then fitness-enhancing group effects may become common in sexual populations. The spread of fitness-enhancing group effects is facilitated when environmental conditions sometimes deteriorate temporarily. This is so even if the genotypes with the highest group effects also tend to have relatively low individual effects. In this sense, the process described here can lead to the evolution of altruism. By contrast, when populations are asexual it appears that group effects are much less important in determining the outcome of evolution. Thus, in nature, asexual populations may tend to be characterized by more antagonistic interactions than those that typically prevail when reproduction is sexual. This might help to explain why asexual lineages are prone to rapid extinction.

Keywords: evolution of sex; epistasis; altruism; population genetics; group selection

1. INTRODUCTION

The survival and reproductive success of living organisms typically depends, in part, on the characteristics of their neighbours (Wright 1969; Dugatkin 1997). Ever since Darwin, evolutionary biologists have been interested in the evolutionary dynamics of neighbour-affecting phenotypes (Darwin 1859; Wright 1969; Dugatkin 1997). Of particular interest has been the evolution of 'altruistic' phenotypes, which decrease the relative fitness of an individual, but which also enhance the fitness of the individual's neighbours.

One mechanism that can lead to the evolution of altruism is group selection. Essentially, group selection is just natural selection acting on differences in the genetic composition of groups. Natural selection on genetic composition can be effective only if there is sufficient genetic variation among the objects of selection (Darwin 1859; Fisher 1930). This is true for selection among groups (group selection), just as it is for selection among individuals. Traditionally, group-selection models have depended on genetic drift to generate the required between-group variation. However, genetic drift is most effective when groups are quite small, with some theoretical studies suggesting that less than 20 individuals per group are required for the evolution and maintenance of altruistic phenotypes that involve a substantial cost (Levin & Kilmer 1974; Wade 1978). Another possible mechanism for maintaining between-group variation is assortative migration, where altruistic migrants tend to migrate to altruistic groups, and non-altruists tend to migrate to non-altruistic groups (Wilson & Dugatkin 1997). Very small groups and assortative migration are both realistic possibilities, but neither is likely to be ubiquitous in nature.

This study considers the possibility that epistatic gene interactions can provide the necessary genetic variance for the evolution of altruism. Epistasis occurs when the effect on fitness of alleles at one locus depends on an individual's genotype at other loci. Epistasis appears to be very

common (Whitlock *et al.* 1995; Coyne *et al.* 1997; Elena & Lenski 1997; Zapata *et al.* 2002), and it is implicated in maintaining variation among subspecies, and among closely related 'sister' species that are capable of hybridization (Barton 1993; Coyne *et al.* 1997). As we shall see, with epistasis effective group selection is possible, even when group sizes are substantial and when individuals migrate at random.

The processes that operate in the model to be presented here are very similar (or identical) to those described by Sewall Wright in his discussions of the 'third phase' of his 'shifting-balance process' (Wright 1931, 1932, 1977, 1982), whereby superior genotypes spread through a metapopulation (i.e. through a collection of sites that are connected by migration). Wright pointed out that the shifting-balance process should favour genotypes that enhance the fitness of other individuals (Wright 1969; Barton & Hewitt 1989; Goodnight & Wade 2000) (i.e. that have relatively high group effects). This possibility has apparently not been considered in previous mathematical studies of the shifting balance, which have focused on the evolution of individual effects (Lande 1985: Barton 1992; Gavrilets 1996; Coyne *et al.* 1997). The results presented here provide support for Wright's idea about the evolution of group effects, and they show that Wright's idea can work even when the genotypes with the highest group effects have relatively low individual effects.

Wright's shifting-balance process has been the object of much negative criticism (Barton 1992; Gavrilets 1996; Coyne *et al.* 1997, 2000). The critics have claimed that the third phase of the process is particularly troublesome (Barton 1992; Gavrilets 1996; Coyne *et al.* 1997, 2000). They say that the third phase is unlikely to be effective when individuals rarely migrate. Instead, the metapopulation will simply preserve multiple genotypes, and none of them will tend to spread at the expense of others (Barton & Hewitt 1989; Gavrilets 1996; Coyne *et al.* 1997). Furthermore, according to the critics, even if one type does begin to spread, this spreading is likely to be

Proc. R. Soc. Lond. B (2004) **271**, 993–1000
DOI 10.1098/rspb.2003.2650
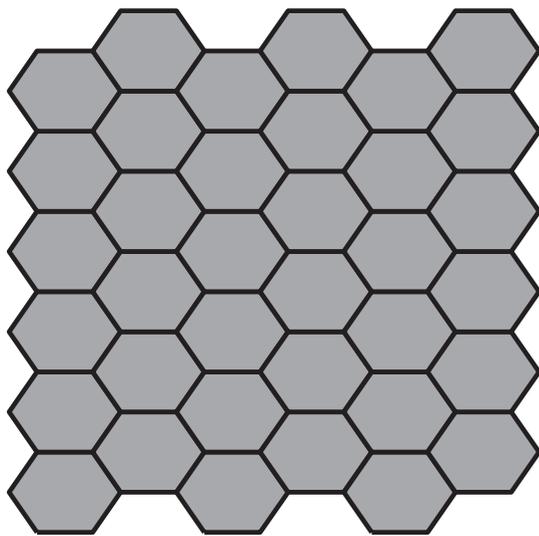
993

© 2004 The Royal Society

Figure 1. The configuration of the metapopulation. There are 36 habitable sites. Each site has six neighbours, except for those on the edge of the metapopulation.

caused by factors unconnected to superior fitness (Barton 1992; Gavrilets 1996; Coyne *et al.* 1997). However, in a recent theoretical investigation, Peck and Welch have shown that the central criticisms of the third phase of Wright's shifting-balance process lose their force if occasional widespread environmental deterioration causes changes in species range (Peck & Welch 2004). It appears that environmental changes of this sort can make phase three very effective. The present study will follow Peck and Welch's lead, and allow for widespread environmental changes. This seems reasonable, as changes in species range are very common (Parmesan & Yohe 2003).

This study will also focus on the effect of the mode of reproduction. The mechanism for maintaining the between-group genetic variation mentioned above depends on outcrossing. In an asexual (or highly inbred) organism, this mechanism does not apply. Thus, we might expect group selection to be much less effective in asexual (or inbred) organisms than in outcrossed sexual organisms. As we shall see, this expectation is fulfilled. Thus, there is good reason to expect that outcrossed sexual organisms will tend to evolve mutually beneficial phenotypes to a greater extent than will asexuals or inbreeders.

2. THE MODEL

The population is assumed to live in a metapopulation that consists of a 6×6 array of habitable sites, as illustrated in figure 1. Each site occupies a hexagonal area that is adjacent to the areas occupied by six other sites, unless it is on the edge of the metapopulation (as shown in figure 1). All the sites are equal in area. The sites are numbered (in arbitrary order) as 1, 2, … 36.

In their study of Wright's shifting balance, Peck and Welch used a diploid model with a simple fitness scheme (Peck & Welch 2004). The present study will focus on complex genetic interactions and it will be necessary to keep track of all possible genotypes and their associated fitness values. Therefore, to prevent the number of genotypes from becoming computationally unmanageable, let us assume that the population is haploid, with N loci

under selection, and with two possible alleles per locus. There are, therefore, 2^N possible genotypes.

Let us number the possible genotypes as 1, 2, …, 2^N, and the value of the individual and group effects associated with genotype i are denoted as X_i and Y_i, respectively. Both the X_i and the Y_i values lie on the interval between 0 and 1. The probability that an individual survives to reproductive age is equal to the X_i value for the individual's genotype multiplied by the mean of the Y_i values associated with the genotypes of all individuals on the site. Thus, the most viable individuals that can exist will have genotypes that confer a relatively high X_i value, and they will live on sites where the mean value of Y_i is relatively high.

Let us assume that, before the production of offspring, adults consume resources for a certain fixed period of time. At the beginning of this fixed period the quantity of resources is the same on all sites, and this quantity is denoted as R_0. Let $R_{t,i}$ represent the amount of resources on site i at time t after resource consumption has started. Let us assume that, at time t, all adults on site i are consuming resources at a rate $\alpha R_{t,i}$, where $\alpha > 0$. This means that the rate of resource consumption goes down as the resource becomes more scarce. This implies that, if we scale time so that resource consumption goes on for one unit of time per generation, then each adult on site i will consume a total amount of resources ($\bar{R}_i$) given by

$$\bar{R}_i = \frac{R_0[1 - \exp(-\alpha D_i)]}{D_i},$$

where D_i represents the number of adults on site i. Say that resource quantities are scaled so that the expected number of offspring for an asexual adult is equal to the amount of resources that she consumes. The actual number of offspring that a particular asexual adult produces is a random variable that follows a Poisson distribution.

Asexual offspring are genetically identical to their parents, except for new mutations. The probability that a particular locus will undergo a mutation is given by μ. Mutations transform the allele at a locus into the alternative allele.

When reproduction is sexual every adult is capable of producing offspring by 'female effort', which involves providing the resources required for development of the offspring (just as in asexual reproduction). For sexual adults, the number of offspring produced by female effort is regulated in exactly the same way as the number of offspring for an asexual population. Sexual adults can also produce offspring by male effort (they are hermaphrodites). This involves providing about half of the genetic material for offspring, but virtually no resources. When a sexual adult living on a particular site produces an offspring by female effort, the adult that contributes to the offspring by male effort is picked at random from among all the adults on that site. For each locus within the offspring's genome, the allele is picked from the same locus within one or the other parental genomes (each with a 50% probability). Thus, there is free recombination between loci. Mutations occur in the same way as under asexual reproduction.

After the offspring are born all members of the parental generation die. Next, viability selection occurs, so that some of the offspring die, depending on their genotypes,

and on the genotypes of others on their sites (as described above).

The final event during each generation is migration. Each individual migrates with probability m. A migrant moves across one of the six boundaries of its site (the particular boundary is picked at random). If no site is on the other side of the boundary (because the boundary is on the edge of the metapopulation) then the migrant returns to its original site.

Each computer-simulation trial was run for 10 000 generations. Preliminary studies of the model showed that the long-term outcome of evolution is often strongly influenced by the occurrence of episodes during which the availability of resources (R_0) is reduced substantially. This is so even if these episodes are rare and relatively brief. Episodes of this sort are common in nature (Diamond 1984; Lande 1985; Purvis *et al.* 2000). With this in mind, the value of R_0 was initially set to $R_0 = 1000$ on all sites for the first 4000 generations of each simulation trial. For the parameters studied, this generally led to population sizes in excess of 750 offspring per site during the first 4000 generations (for more details, see the notes associated with table 1).

Starting with generation 4001 the amount of resources available on each site (R_0) was decreased by 1.0 per generation. This continued until the mean number of offspring per site fell below 100. Because the initial value of R_0 was 1000, the required reduction in population size always occurred before generation 5000 (i.e. before R_0 reached zero). Once the required mean population size was achieved, the size of the metapopulation was maintained at a relatively low value by increasing the value of R_0 by 1.0 when the mean number of offspring per site was less than 100, and decreasing R_0 by 1.0 otherwise. Finally, after generation 5000, the value of R_0 was increased by 1.0 each generation until, sometime before generation 6001, it reached the initial value of $R_0 = 1000$, where it remained until the end of the trial (i.e. until generation 10 000). Thus, a period of relatively low-resource availability occurs in the middle of each simulation trial. This period will be called the *low-resource-availability generations*.

Each simulation trial was initialized by placing 1000 adults on each site. Each allele in each of these adults was independently set to one or the other of the two alternative alleles, each with a 50% probability.

3. RESULTS

To study the behaviour of the model several sets of simulation trials were run under a variety of conditions. Each of these sets consisted of 100 trials. To describe the outcome of evolution, let us define $\overline{X}$ and $\overline{Y}$ as the mean of X_i and Y_i in the metapopulation during some particular generation of a given simulation trial (when measured among newborn offspring). Let us also define $\overline{V}$ as the mean probability of surviving to reproductive age during the same generation. Finally, let us define $\overline{\overline{X}}$, $\overline{\overline{Y}}$ and $\overline{\overline{V}}$, respectively, as the mean values of $\overline{X}$, $\overline{Y}$ and $\overline{V}$, when averaged across all 100 trials in a set of simulation trials. As each trial carries on for 10 000 generations there are 10 000 values of $\overline{X}$, $\overline{Y}$ and $\overline{V}$ associated with each set of simulation trials.

Table 1. Numerical data from the computer simulations.

(The largest standard errors were associated with the $\overline{\overline{Y}}$ data and the $\overline{\overline{V}}$ data in STS.3S. The maximum of these standard errors was equal to 0.0196 (for $\overline{\overline{V}}$ at generation 4000 in STS.3S). For all the other data points in the table, the maximum standard error was equal to 0.009 29 (this was associated with $\overline{\overline{Y}}$ at generation 4000 in STS.1S). The mean number of juveniles per site was calculated for each trial, and then averaged across the 100 trials in each simulation trial set. In STS.2A the results of this calculation for generations 1, 4000, 5000 and 10 000 were 1000, 971, 99 and 970, respectively (rounding to the nearest integer). In STS.2S the analogous values were 1000, 957, 100 and 983. For STS.3A the values were 1001, 787, 103 and 786. For STS.3S the values were 1001, 905, 100 and 975.)

	generation 1			generation 4000			generation 5000			generation 10 000		
	$\overline{\overline{X}}$	$\overline{\overline{Y}}$	$\overline{\overline{V}}$	$\overline{\overline{X}}$	$\overline{\overline{Y}}$	$\overline{\overline{V}}$	$\overline{\overline{X}}$	$\overline{\overline{Y}}$	$\overline{\overline{V}}$	$\overline{\overline{X}}$	$\overline{\overline{Y}}$	$\overline{\overline{V}}$
STS.1A (additive genetic interactions, asexual)	0.498	0.488	0.243	0.664	0.493	0.327	0.663	0.504	0.330	0.664	0.493	0.327
STS.1S (additive genetic interactions, sexual)	0.505	0.497	0.251	0.669	0.495	0.332	0.668	0.504	0.337	0.669	0.495	0.332
STS.2A (complex genetic interactions, asexual)	0.500	0.500	0.250	0.786	0.504	0.396	0.786	0.505	0.397	0.786	0.505	0.397
STS.2S (complex genetic interactions, sexual)	0.500	0.500	0.250	0.698	0.505	0.352	0.725	0.608	0.440	0.727	0.596	0.433
STS.3A (high X_i associated with low Y_i values, asexual)	0.500	0.990	0.495	0.784	0.252	0.198	0.781	0.270	0.209	0.784	0.252	0.198
STS.3S (high X_i associated with low Y_i values, sexual)	0.500	0.990	0.495	0.699	0.577	0.395	0.667	0.998	0.660	0.675	0.904	0.607

(a) *Additive genetic interactions*

The first set of computer-simulation trials will be referred to as STS.1A (for simulation trial set number 1, asexual). STS.1A was used to consider the outcome of evolution in an asexual population when loci combine together in an additive fashion to produce the X_i and Y_i values. This procedure corresponds to the $K = 0$ case in Kauffman's *NK*-fitness-landscape model (except that the *NK* model would ordinarily involve X_i values only, and not the group effects represented by the Y_i values (Kauffman 1993; Altenberg 1997)). The $K = 0$ procedure consists of selecting one random number for each allele that can occur at each locus (a total of $2N$ numbers). These random numbers are called the *allelic effects*, and they are chosen from a uniform distribution on the interval (0, 1). Then, to get the value of X_i for genotype i, one simply takes the mean of the N allelic effects associated with that genotype. An identical (but independent) procedure was used to obtain the Y_i values. For STS.1A, and for the other simulations reported below, the parameters of the model were set as follows: $N = 10$, $m = 0.01$, $\alpha = 0.01$ and $\mu = 0.0001$. (Note that preliminary studies suggest that the qualitative nature of the results is relatively insensitive to moderate changes in these parameter values.)

The results from the 100 trials of STS.1A are shown in the first row of table 1 (the table also shows results from the other sets of simulation trials described below). In the first generation both $\overline{X}$ and $\overline{Y}$ were approximately equal to 1/2. This makes sense, as the expected value of X_i or Y_i for the initial randomly generated genotypes is equal to 1/2, and selection has not occurred in generation 1. By generation 4000 $\overline{X}$ was approximately equal to 2/3, which is the value expected under the assumptions of STS.1A if all population members have the genotype associated with the highest possible value of X_i (the *global-optimum genotype*). (The global-optimum genotype is associated with a different value of X_i in every trial, but over very many trials it will have a mean value of 2/3.) By contrast, at generation 4000, the group effects showed little sign of a response to selection, in that the value of $\overline{Y}$ was still close to its original value of (approximately) 1/2. The values of $\overline{X}$ and $\overline{Y}$ remained almost unchanged throughout the rest of the 10 000 generations, despite the occurrence, in the middle of each trial, of the low-resource-availability generations.

In the second set of simulation trials (STS.1S) the conditions were identical to those in STS.1A, except that reproduction was sexual, instead of asexual. The results were almost indistinguishable from those obtained in STS.1A. These results suggest that, regardless of the mode of reproduction, when phenotypes are determined by additive interactions group effects have little effect on the outcome of evolution. (Note that previous studies have shown that group effects can be influential when genetic interactions are additive, but this typically requires population sizes that are considerably smaller than those that occur during the low-resource-availability generations in the simulation studies presented here (Levin & Kilmer 1974; Wade 1978).)

(b) *Complex genetic interactions*

Although additive interactions may be important in nature (Lynch & Walsh 1998), it is clear that many loci interact in a complex way. That is to say, epistasis is common (Whitlock *et al.* 1995; Coyne *et al.* 1997; Elena & Lenski 1997; Zapata *et al.* 2002). Let us now consider the outcome of evolution when there are complex interactions among the 10 loci under study in the simulation trials. In STS.2A and STS.2S the same procedures were used as in STS.1A and STS.1S, respectively, except that a new set of allelic effects was chosen independently for each genotype. This corresponds to the $K = N - 1$ case in the *NK*-fitness-landscape model (Kauffman 1993). Under this scheme of fitness determination, the effect of changing one allele to another at one locus depends on the individual's genotype at all of the other $N - 1$ loci under selection. As a consequence, the 'fitness landscape' may be very complex. In particular there may be many genotypes for which any single change at any one of the 10 loci will cause a decline in the value of X_i. These genotypes are called *local-optimum genotypes* (Kauffman 1993). The probability that any given genotype is a local-optimum genotype is equal to $1/(N + 1)$. Thus, when $N = 10$, there are $2^N = 1024$ possible genotypes, and we can expect that *ca.* 93 of these are local-optimum genotypes (Kauffman 1993). One of these local-optimum genotypes is also the global-optimum genotype. With $N = 10$ the expected value of X_i for the global optimum is *ca.* 0.784.

In STS.2A reproduction was asexual, and the results are shown in figure 2a. By generation 4000 the value of $\overline{X}$ was equal to 0.786. This is close to 0.784, the value expected if, at generation 4000, all individuals have the global-optimum genotype. Indeed, averaging across the 100 trials in STS.2A, more than 99% of offspring had the global-optimum genotype at generation 4000. The value of $\overline{X}$ stayed virtually unchanged after generation 4000, despite the occurrence of the low-resource-availability generations. By contrast, the value of $\overline{Y}$ remained close to 1/2 throughout the 10 000 generations. These results are qualitatively similar to those in STS.1A and STS.1S, in that group effects seem to have little impact on the outcome of evolution.

Next, consider the results from STS.2S (see figure 2b). The conditions were identical to those in STS.2A, except that reproduction was sexual. The value of $\overline{X}$ was always smaller in STS.2S than under the asexual conditions of STS.2A (after the first few generations). Furthermore, when resource availability (R_0) began to decrease after generation 4000, the value of $\overline{X}$ increased. In STS.2S the mean group effect ($\overline{Y}$) was approximately equal to 1/2 until generation 4000, which is the same result found in STS.2A. However, in STS.2S, when population sizes began to fall after generation 4000 the value of $\overline{Y}$ increased sharply, and it only fell to a modest degree after resource availability increased again. The total rise in $\overline{Y}$ (comparing generations 1 and 10 000) was 19%. This compares with a 1% rise under the asexual conditions of STS.2A. These differences were reflected in the values of $\overline{V}$, which were higher in the sexual case (STS.2S) than in the asexual case (STS.2A) after the onset of the low-resource-availability generations.

(c) *Evolution when large individual effects are associated with small group effects*

In the simulations described so far, individual effects and group effects were assigned at random to genotypes.
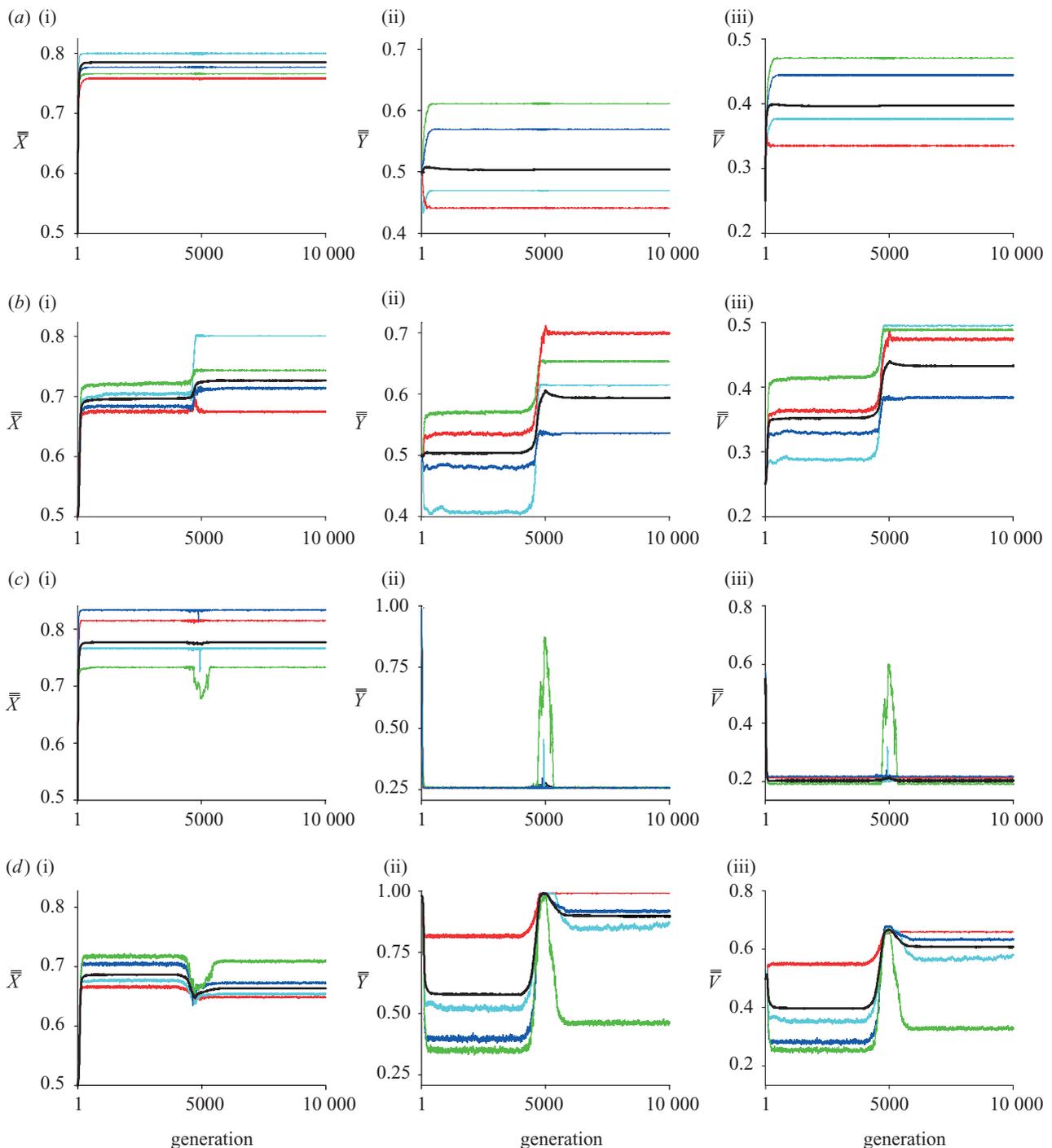
Figure 2. Results from the simulation studies. Each row of graphs gives the data from a different simulation trial set: (*a*) STS.2A, (*b*) STS.2S, (*c*) STS.3A, (*d*) STS.3S. The coloured lines represent the results from individual trials (to enhance visibility, only four of the 100 trials in a simulation trial set are shown). The black lines represent the mean outcome from the 100 trials that constitute a simulation trial set. (i) Individual effects (the coloured lines give the $\overline{X}$ values, whereas the black lines give the $\overline{\overline{X}}$ values). (ii) Group effects (the coloured lines give the $\overline{Y}$ values, whereas the black lines give the $\overline{\overline{Y}}$ values). (iii) The proportion of offspring that survived to reproductive age (the coloured lines give the $\overline{V}$ values, whereas the black lines give the $\overline{\overline{V}}$ values).

Thus, it is possible for a genotype to have a high individual effect and a high group effect. However, it seems likely that in many species those genotypes with the very highest individual effects will have relatively low group effects. This idea underlies much of the evolutionary theory of altruism (Wade 1978; Dugatkin & Reeve 1998), and there are some data to support it (Goodnight 1985; Griffing 1989; Goodnight & Wade 2000). These considerations

led to STS.3A and STS.3S. In these simulation trials sets, the same procedures were used as in STS.2A and STS.2S (respectively), except that, if the X_i value associated with any given genotype was less than 0.7, then the Y_i value was set to $Y_i = 1$. Otherwise, the Y_i value was set to $Y_i = 0.25$. (Note that, in STS.2A, STS.2S, STS.3A and STS.3S the global optimum for every trial always had an associated value of X_i in excess of 0.7. Furthermore, 0.7

is less than the value of $\overline{\overline{X}}$ at generation 10 000 in both STS.2A and STS.2S. Preliminary studies suggested that the difference in results between sexual and asexual populations was largest for threshold values in the region of 0.7. Thus, choice of this value helps to show the potential magnitude of the phenomena under study here.)

In STS.3A reproduction was asexual. After the first few hundred generations the value of $\overline{\overline{X}}$ was approximately equal to 0.784, the value expected if all individuals have the global-optimum genotype (see figure 2*c*). This is the same result found in STS.2A. The value of $\overline{\overline{Y}}$ was never elevated much above its minimum-possible value of 0.25 (except during the first few hundred generations). However, in a few trials the value of $\overline{Y}$ was elevated during the low-resource-availability generations. Data from two of these exceptional trials are shown in figure 2*c*.

In STS.3S reproduction was sexual, and this had a large effect on the results (see figure 2*d*). After the first few hundred generations, the value of $\overline{\overline{X}}$ was always substantially lower than under the asexual conditions of STS.3A. Conversely, under sexual reproduction (STS.3S), the value of $\overline{\overline{Y}}$ was much higher than under asexuality after the initial generations. Indeed, by generation 5000 the value of $\overline{Y}$ was in excess of 0.95 in 100% of the trials. After resource availability (R_0) began to increase again (i.e. after generation 5000) there was a modest decline in the value of $\overline{\overline{Y}}$. However, examination of the data on individual trials shows that, at generation 10 000, $\overline{Y}$ was still in excess of 0.95 in 67% of the trials. Thus, after the low-resource-availability generations, sexual reproduction allowed a nearly perfect long-term preservation of very high group effects in most trials. Furthermore, the value of $\overline{Y}$ was in excess of 0.5 (i.e. more than twice the minimum value for Y_i) in 95% of the trials at generation 10 000. Because sexuals were able to achieve and preserve high group effects, their viability at generation 10 000 was typically more than three times what would be achieved under the same conditions in an asexual population (see table 1).

4. DISCUSSION

The results show that, when genetic interactions are complex, a decrease in resource availability leads to higher group effects under sexuality than under asexuality, and these increased group effects are largely sustained even after resource availability increases again. This is so even when genotypes with the highest individual effects have low group effects.

The results suggest that sexual populations will often be characterized by much less antagonistic interactions than is the case for closely related asexual populations. Furthermore, when asexual lineages branch off from sexual lineages, the results suggest that the new asexual lineages will be prone to a decline in beneficial interactions among conspecifics, and to an increase in antagonistic interactions. It seems possible that changes of this sort could eventually cause a large decline in fitness within new asexual lineages, and this may help to account for the tendency of these lineages to die out relatively quickly (Maynard Smith 1978; Burt 2000).

What accounts for the results reported here? The answer has to do with the conditions under which a

low-frequency genotype can become common on a site when the migration rate is low. Under asexuality, such an increase tends to occur (absenting stochastic loss) when the low-frequency genotype has a higher fitness than the average fitness of individuals on the site in question. As all of the individuals on a site experience the same mean group effect, this sort of increase in frequency happens when the value of X_i for the low-frequency genotype is higher than the mean value of X_i on the site. As a consequence, genotypes with relatively high X_i values tend to spread through the metapopulation. Under sexuality the situation is very different. When a rare genotype is present on a site it is likely to broken apart through recombination with a common genotype in the following generations. This common genotype will typically be the result of a long period of natural selection, so that all of the alleles work reasonably well together, despite the complex genetic interactions that occur under the assumptions of STS.2A, STS.2S, STS.3A and STS.3S (i.e. common genotypes tend to be local-optimum genotypes). These same genetic interactions make it unlikely that a recombinant genotype will have a higher value of X_i than the common genotype, regardless of the values of X_i associated with its parents. Thus, a site on which most individuals have the same local-optimum genotype can be protected from invasions by other genotypes, regardless of the values of X_i associated with those genotypes, and regardless of whether the invaders arise by mutation, recombination or migration. Because locally rare alleles often remain rare, between-site diversity can be maintained in sexual populations. However, this mechanism depends on sexual reproduction and complex genetic interactions. Thus, it cannot operate in STS.1A, STS.1S, STS.2A or STS.3A.

Evidence in support of the foregoing interpretation of the results comes from calculating the mean value of Y_i on each site, and then calculating the range of values found (i.e. the difference between the largest and smallest mean values found among the 36 sites in the metapopulation). For example, at generation 4000, the mean value of this range (averaging across the 100 trials in a simulation trial set) was 0.015, 0.016, and 0.006 for STS.1A, STS.1S and STS.2A, respectively. By contrast, the mean value of this range for STS.2S was 0.252. Thus, complex genetic interactions, along with sexual reproduction, can allow for the preservation of much more between-site genetic variation than is possible with asexuality or with simple genetic interactions. Furthermore, at generation 4000, the mean value (averaging the 100 trials) of the highest mean value of Y_i on any of the 36 sites was 0.634 in STS.2S. This is higher than the value of $\overline{\overline{Y}}$ in STS.2S at generations 5000 or generation 10 000. Thus, we have evidence that much of the between-site variation required for effective group selection is generated before generation 4000, while population sizes are relatively large, and not during the low-resource-availability generations.

When the environment becomes worse some populations on some sites may become small, compared with their neighbours. This is particularly likely to happen on sites where the average group and/or individual effects are relatively low, and this allows migrants from neighbouring sites to take over (Peck & Welch 2004). If the population is sexual and the overwhelming majority of these migrants

are identical at the loci under selection, then they can mate in the new territory without a high chance of producing low-fitness recombinant offspring. Thus, although the spread of genotypes that confer a high mean fitness may be stymied when environmental conditions are good, they may spread readily through the metapopulation when environmental conditions deteriorate. This is why, in STS.2S, both the value of $\overline{X}$ and the value of $\overline{Y}$ increased during the low-resource-availability generations. Furthermore, because common local-optimum genotypes are hard to displace in sexual populations, there can be long-term persistence of the genotypes that become common during the low-resource-availability generations. Note that mechanisms similar to the one just described may operate in other evolutionary contexts (Boyd & Richerson 1990; Haig & Grafen 1991; Peck & Welch 2004).

It should be clear that processes similar to those discussed here may also act to enhance group effects in outcrossed populations (compared with inbred populations). In addition, the results suggest that relatively high rates of recombination should generally be associated with relatively high group effects.

In light of the results presented here, it seems possible that empirical investigations into the relationship between reproductive mode and the evolution of group effects would be fruitful. Indeed, there is some evidence to support this idea. In particular, among the insects, the species with the highest levels of apparent altruism also tend to have high numbers of chromosomes, which suggests a relatively large map length (Seger 1983). More compelling is a report from an investigation showing that, in honeybees (*Apis mellifera*), a recently discovered asexual strain is spreading in northern South Africa (Martin *et al.* 2002; this strain is asexual in the sense that it reproduces without mating). The asexual strain is a 'cheater' (i.e. it has relatively low group effects) in at least two ways. First, the asexuals seem not to do much foraging for food, relying instead on food collected by others. Second, the asexuals frequently lay eggs, an activity that is usually largely monopolized by the queen. The eggs laid by the asexual bees seem to be abnormal in some way, as they generally are not destroyed by workers, which is the usual fate of eggs laid by individuals other than the queen. From the results presented here, this sort of decline in group effects is exactly what would be expected during an increase in asexual reproduction within a population.

REFERENCES

Altenberg, L. 1997 NK Fitness landscapes. In *Handbook of evolutionary computation* (ed. T. Bäck, D. Fogel & Z. Michalewicz), pp. B2.7:5–B2.7:10. Oxford University Press.

Barton, N. H. 1992 On the spread of new gene combinations in the third phase of Wright's shifting-balance. *Evolution* **46**, 551–557.

Barton, N. H. 1993 Why species and subspecies? *Curr. Biol.* **3**, 797–799.

Barton, N. H. & Hewitt, G. M. 1989 Adaptation, speciation and hybrid zones. *Nature* **341**, 497–503.

Boyd, R. & Richerson, P. J. 1990 Group selection among alternative evolutionarily stable strategies. *J. Theor. Biol.* **145**, 331–342.

Burt, A. 2000 Perspective: sex, recombination, and the efficacy of selection—was Weismann right? *Evolution* **54**, 337–351.

Coyne, J. A., Barton, N. H. & Turelli, M. 1997 Perspective: a critique of Sewall Wright's shifting balance theory of evolution. *Evolution* **51**, 643–671.

Coyne, J. A., Barton, N. H. & Turelli, M. 2000 Is Wright's shifting balance process important in evolution? *Evolution* **54**, 306–317.

Darwin, C. 1859 *On the origin of species by means of natural selection, or the preservation of favoured races in the struggle for life*. London: John Murray.

Diamond, J. 1984 'Normal' extinctions of isolated populations. In *Extinctions* (ed. M. Nitecki), pp. 191–246. University of Chicago Press.

Dugatkin, L. A. 1997 *Cooperation among animals: a modern perspective*. Oxford University Press.

Dugatkin, L. A. & Reeve, H. K. (eds) 1998 *Game theory and animal behavior*. Oxford University Press.

Elena, S. F. & Lenski, R. E. 1997 Test of synergistic interactions among deleterious mutations in bacteria. *Nature* **390**, 395–398.

Fisher, R. A. 1930 *The genetical theory of natural selection*. Oxford: Clarendon.

Gavrilets, S. 1996 On phase three of the shifting-balance theory. *Evolution* **50**, 1034–1041.

Goodnight, C. J. 1985 The influence of environmental variation on group and individual selection in a cress. *Evolution* **39**, 545–558.

Goodnight, C. J. & Wade, M. J. 2000 The ongoing synthesis: a reply to Coyne, Barton, and Turelli. *Evolution* **54**, 317–324.

Griffing, B. 1989 Genetic analysis of plant mixtures. *Genetics* **122**, 943–956.

Haig, D. & Grafen, A. 1991 Genetic scrambling as a defense against meiotic drive. *J. Theor. Biol.* **153**, 531–558.

Kauffman, S. A. 1993 *The origins of order: self-organization and selection in evolution*. Oxford University Press.

Lande, R. 1985 The fixation of chromosomal rearrangements in a subdivided population with local extinction and colonization. *Heredity* **54**, 323–332.

Levin, B. R. & Kilmer, W. L. 1974 Interdemic selection and the evolution of altruism: a computer simulation study. *Evolution* **28**, 527–545.

Lynch, M. & Walsh, B. 1998 *Genetics and analysis of quantitative traits*. Sunderland, MA: Sinauer.

Martin, S. J., Beekman, M., Wossler, T. C. & Ratnieks, F. L. W. 2002 Parasitic Cape honeybee workers, *Apis mellifera capensis*, evade policing. *Nature* **415**, 163–165.

Maynard Smith, J. 1978 *The evolution of sex*. Cambridge University Press.

Parmesan, C. & Yohe, G. 2003 A globally coherent fingerprint of climate change impacts across natural systems. *Nature* **421**, 37–42.

Peck, J. R. & Welch, J. J. 2004 Adaptation and species range. *Evolution* **58**, 211–221.

Purvis, A., Jones, K. E. & Mace, G. M. 2000 Extinction. *Bioessays* **22**, 1123–1133.

Seger, J. 1983 Conditional relatedness, recombination, and the chromosome numbers of insects. In *Advances in herpetology and evolutionary biology* (ed. A. G. J. Rhodin & K. Miyata), pp. 596–612. Cambridge, MA: Harvard Museum of Comparative Zoology.

Wade, M. J. 1978 A critical review of the models of group selection. *Q. Rev. Biol.* **53**, 101–114.

Whitlock, M. C., Phillips, P. C., Moore, F. B. M. & Tonsor, S. J. 1995 Multiple fitness peaks and epistasis. *A. Rev. Ecol. Syst.* **26**, 601–629.

Wilson, D. S. & Dugatkin, L. A. 1997 Group selection and assortative interactions. *Am. Nat.* **149**, 336–351.

Wright, S. 1931 Evolution in Mendelian populations. *Genetics* **16**, 97–159.

Wright, S. 1932 The roles of mutation, inbreeding, cross-breeding and selection in evolution. *Proc. Sixth Int. Congr. Genet.* **1**, 356–366.

Wright, S. 1969 *Evolution and the genetics of populations*, vol. 2. University of Chicago Press.

Wright, S. 1977 *Evolution and the genetics of populations*, vol. 3. University of Chicago Press.

Wright, S. 1982 The shifting balance theory and macroevolution. *A. Rev. Genet.* **16**, 1–19.

Zapata, C., Nunez, C. & Velasco, T. 2002 Distribution of non-random associations between pairs of protein loci along the third chromosome of *Drosophila melanogaster*. *Genetics* **161**, 1539–1550.