THE MANIFOLD GAIN OF SEXUAL REPRODUCTION

Marion Cook
En Vignette, Echenevex, 01170 Gex, France

Received 8 September 1992

ABSTRACT. **A sexual population in which males contribute only genes to the next generation should experience a twofold reproductive cost when competing with an asexual, all-female, population and be rapidly driven to extinction[1,2]. Some short-term advantage must accrue to sexual reproduction[2]. One possibility is that the male could shelter a gene from selection when currently less fit in a polymorphism expressed only by the female. Indications are that under fluctuating selection gene sheltering by one sex can protect a polymorphism expressed by the opposite sex in both diploid and haploid populations. This refutes orthodox opinion that temporal variation in fitness has no tendency to maintain polymorphism in the haploid case[3]. When progeny numbers are directly involved such buffering by the male can give a sexual population a manifold advantage over an asexual population. This population when of low fecundity can be more stable than any competing asexual population. Low recombination rates can be advantageous if the fecundity locus is linked to the sex locus. Stablility can also be enhanced by male competition.**

* * *

Sexual reproduction and recombination still raise unsolved problems in evolutionary biology (for a review see Roughgarden[4]). Roughgarden[4] has shown an advantage for diploid sex under a fluctuating environment, but requiring a strong selection advantage when only one locus with two alleles is involved.

In this paper one-locus, two-alelle models for both haploid and diploid populations are presented that require only moderate selection advantages which can be lowered by recombination.

Large slow-breeding organisms, such as birds and mammals, mostly exhibit sexual reproduction, whereas small fast-breeding organisms tend to be asexual. Blueweiss et al[5] have shown intrinsic rate of increase between species to be strongly negatively correlated with body size. Within species there is probably a similar effect, and some heritability of progeny number, even assuming females can adjust numbers of young according to breeding conditions. Taking birds for example, in only one[6] out of five wild species studied has no significant heritability in clutch size been demonstrated, variation in clutch size in the other four species showing h^2 values ranging from 0.23 to 0.50 [7,8,9,10,11].

* * *

Evolutionary Theory 10: 129-143 (December, 1992).
The editors thank B. Charlesworth and M. Uyenoyama for help in evaluating this paper.

In a population the genotype giving rise to the progeny number with the greatest longterm geometric mean fitness (LGMF) would be expected to go to fixation. However, a range in progeny values is the norm. With birds there is usually an optimal clutch size each breeding season, and in bad seasons small clutches can be more successful than large ones[12].

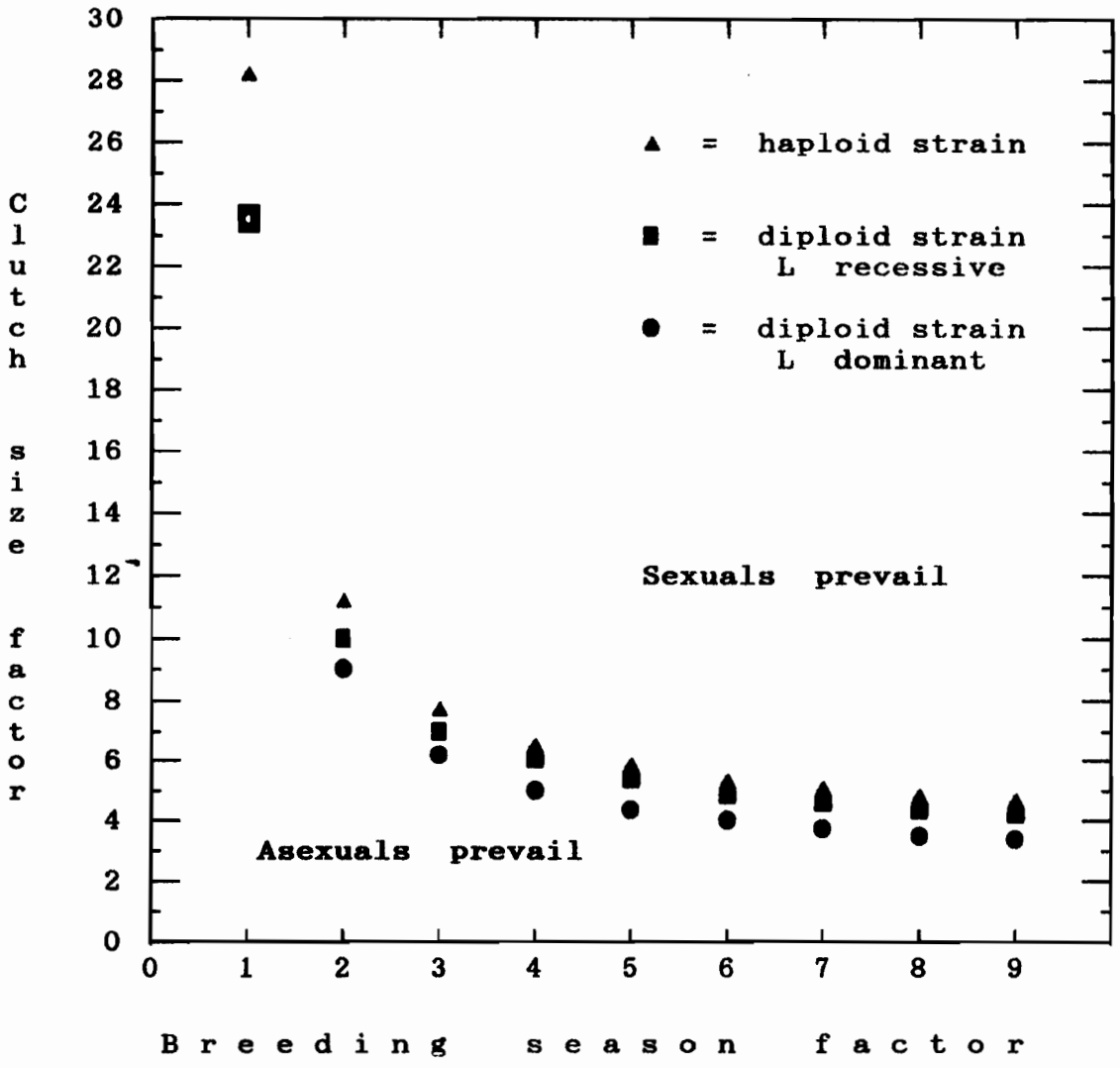

FIG. 1. In the region above each plot the sexual strain always prevails over the asexual strain when breeding conditions cycle regularly since the sexual LGMF > 2S, and prevails most of the time when conditions are stochastic.

Temporal fluctuation can create additive variance in fitness[13], and Hamilton[14], Hamilton et al[15] and Lloyd[16] have proposed that this may favour sexual reproduction in a single locus system. Computer simulations were written in Turbo Pascal to study further this effect on sexual reproduction. The models presented here are general and do not only apply to birds, the terms "clutch size" and "brood" being used for convenience.

The simplifying assumption was made[15] that each clutch size clone had the same LGMF as had the equivalent sexual clutch size genotype. This de-emphasised competition between clones, so allowing the effect of the LGMF of the sexual population as a whole to be assessed. Generations were considered non-overlapping and discrete.

The LGMFs for the asexual clutch size clones were computed for a complete cycle of environmental conditions consisting of one or more good breeding seasons to one bad breeding season, i.e. over n generations. Thus the environment cycles through n - 1 good seasons, in which S progeny are produced from each small clutch and L progeny from each large clutch (S < L), and one bad season, in which all S progeny are reared from small clutches but only B progeny from large clutches (S > B). Under clonal reproduction the value of B for which the genotypes specifying small and large clutches increase at equal rates is:

$$B = \frac{S^n}{L^{n-1}}$$

Males in the sexual strains are assumed to equal females in frequency and to contribute only their genes to the next generation, this implying an effective halving of natural rate of increase of sexuals relative to asexuals when other things are equal.

The main findings are summarised in Fig. 1. The clutch size factor, csf, determines the size of the largest clutch, L, in relation to the smallest, S, in a population, L = csf x S. The breeding season factor, bsf, determines the number of good seasons, gs, to bad seasons, bs, in a cycle of n generations, gs = bsf x bs. When S = 1 is assumed, the plot gives the minimum value of L able to maintain population size, since the LGMF = 2 for a sexual strain, whereas the LGMF = 1 for an asexual strain.

Haploid Populations

In the haploid strain the small clutch is determined by the gene A_1 and the large clutch by its allele A_2 (Table 1). The frequency of the small clutch size allele is expressed as a ratio, R, of the frequency of the large clutch size allele. It is to be noted that the fitness of a male in terms of F1 progeny depends on the genotype of his mate, and not on his own genotype. With random mating, and clutch size values that give each asexual clone a LGMF equal to the small clutch size, the sexual strain invades the asexual strain and drives it to extinction under cycling conditions, and prevails most of the time under stochastic conditions with the same mean proportion of good seasons to each bad season, when clutch sizes conform with the

clutch size factor in Fig. 1. Initial gene frequencies do not affect the outcome. Starting from gene frequencies of 10^{-4} the haploid strain usually drives the asexual strain extinct in less than 1000 generations.

This haploid polymorphism is strongly buffered since a clutch size allele escapes direct selection in the male as does a recessive allele in the heterozygote in a diploid polymorphism, but here half the complement of each allele avoids direct selection every generation no matter what its frequency (or the frequency of males if this differs from that of females).

TABLE 1. Haploid strain zygote frequencies from breeding in good seasons and bad seasons

Good season			Bad season		
♂ / ♀	$R\ A_1$	$1\ A_2$	♂ / ♀	$R\ A_1$	$1\ A_2$
$RS\ A_1$	$R^2S\ A_1A_1$	$RS\ A_1A_2$	$RS\ A_1$	$R^2S\ A_1A_1$	$RS\ A_1A_2$
$L\ A_2$	$RL\ A_1A_2$	$L\ A_2A_2$	$B\ A_2$	$RB\ A_1A_2$	$B\ A_2A_2$

The ratio after the first good season, R_1, of the frequencies of the small clutch size allele A_1 and the large clutch size allele A_2 for the haploid sexual strain is:

$$R_1 = R_0 \frac{R_0 2S + L + S}{R_0(L + S) + 2L}$$

in which R_0 is the ratio of the frequency of A_1 to the frequency of A_2 in the initial generation. This recursion is carried forward through the $n - 1$ good seasons and after the n^{th} season, in which bad conditions prevail, the ratio at the beginning of the next n-generation cycle is:

$$R_0' = R_n = R_{n-1} \frac{R_{n-1} 2S + B + S}{R_{n-1}(B + S) + 2B}$$

Since the functions on the right in these two equations are both monotone functions of the frequency ratio, the full recursion over n seasons is also monotone in the initial ratio R_0. This property of monotonicity ensures departure from unstable states and convergence to the nearest equilibrium state.

For sufficiently low initial frequencies of the small clutch size allele (low R_0) the frequency ratio after a full cycle is approximately:

$$R_0' = R_0 \left(\frac{L + S}{2L}\right)^{n-1} \frac{B + S}{2B}$$

This recursion indicates that the allele that determines small clutches increases when rare if:

$$\left(\frac{L+S}{2}\right)^{n-1}\frac{B+S}{2} > BL^{n-1}$$

Most of the progeny carrying the common A_2 allele are produced by $A_2 \times A_2$ matings, and most of the progeny carrying the rare A_1 allele are produced by $A_1 \times A_2$ or $A_2 \times A_1$ matings. The clutch size produced by reciprocal mixed matings depends on the genotype of the maternal parent. Since the two matings of this kind occur with equal frequency, the average brood size of mixed matings is the arithmetic mean of the brood sizes of the two genotypes. The quantity on the right of the equation represents the number of A_2 progeny generated over the n-generation cycle by an A_2 lineage, and the quantity on the left the number of progeny generated over the same period by an A_1 lineage. Inequality indicates that the rare small clutch genotype invades the population only if it produces more progeny during a full cycle than the common large clutch genotype.

Similarly, the allele that determines the large clutch increases when rare if:

$$\left(\frac{L+S}{2}\right)^{n-1}\frac{B+S}{2} > S^n$$

For B as given here, these last two equations correspond to:

$$\left(\frac{AM(S, L)}{GM(S, L)}\right)^{n-1}\left(\frac{AM(S^{n-1}, L^{n-1})}{GM(S^{n-1}, L^{n-1})}\right) > 1$$

in which AM(x, y) represents the arithmetic mean of x and y and GM(x, y) the geometric mean. Since this equation is always satisfied, AM > GM, the alleles that determine small and large clutch sizes both increase in frequency when rare. This finding, together with monotonicity of the recursion function over the full n-season cycle, implies convergence to a stable polymorphic equilibrium state.

The model does not have to be symmetrical. As long as the proportion of young surviving from a large clutch after a bad season is some value between 0 and 0.3S when n = 2, 0 and 0.1S when n = 10 for example, polymorphism is maintained. If $n < 7$ the equivalent of overdominance of the heterozygote is present.

With L sufficiently large, a LGMF > 2S is clearly possible:

$$\bar{W} = \sqrt[n]{\frac{(R_0S+L)(R_0+1)\ldots(R_{n-2}S+L)(R_{n-2}+1)\ (R_{n-1}S+B)(R_{n-1}+1)}{(R_0+1)(R_0+1)\ \ldots\ (R_{n-2}+1)(R_{n-2}+1)\ (R_{n-1}+1)(R_{n-1}+1)}}$$

$$= \sqrt[n]{\frac{(R_0S+L)\ldots.(R_{n-2}S+L)\ (R_{n-1}S+B)}{(R_0+1)\ \ldots.\ (R_{n-2}+1)\ (R_{n-1}+1)}}$$

The sexual and asexual populations increase in size in good seasons, then crash in bad seasons:

$$\textit{crash factor} = cf = \frac{\textit{population before the crash}}{\textit{population after the crash}}$$

This cost of evolution increases rapidly with n (Fig. 2):

$$cf = \frac{(R_0S + L) \ldots . (R_{n-2}S + L)(R_{n-1} + 1)}{(R_0S + L) \ldots . (R_{n-2}S + L)(R_{n-1}S + B)} = \frac{(R_{n-1} + 1)}{(R_{n-1}S + B)}$$

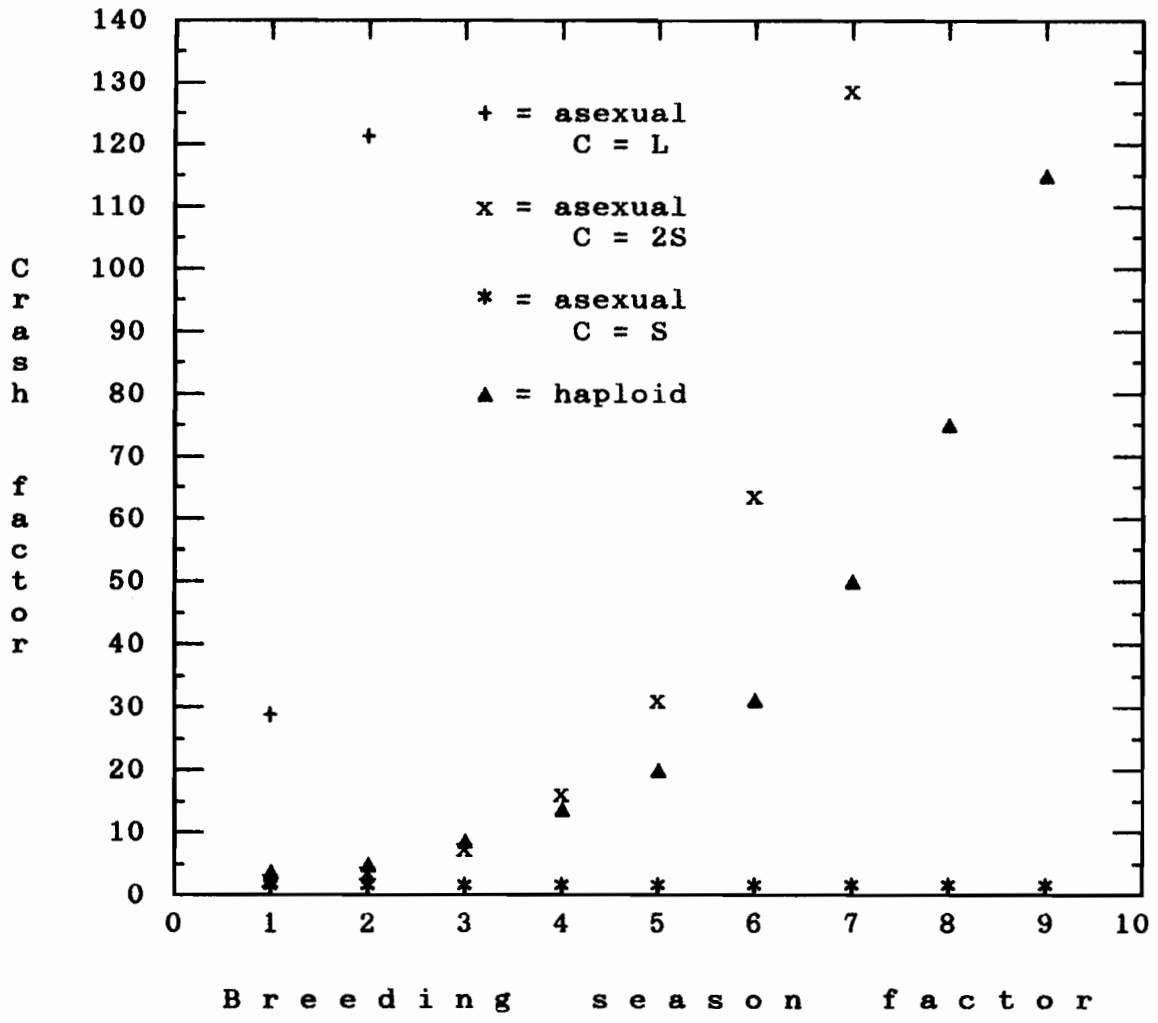

FIG. 2. Plots of the crash factors for the haploid sexual strain when the LGMF = 2S and for three asexual clones with different clutch sizes, C, and LGMF = 1S. The plot for the asexual clone with C = 2S is for a clone with csf = 2. The crash factor of the clone with C = S always equals one.

Although the crash factor for the haploid increases with n it is much lower than for any asexual clutch size clone with C > 1 and LGMF = 1, unless C = 2 and n ≤ 4, as is apparent since the $cf = C^{n-1}$ for an asexual clone. An asexual clone with C = 1

would not be viable in nature, once it fell in frequency it would have no ability to recover. If the asexual LGMF > 1 the sexual strain will not have such an advantage, the asexual clone with C = S probably being viable since S > 1.

The haploid strain can prevail over an asexual clone with a lower cf since it is more productive in a good season following a bad season due to the buffering effect of the male.

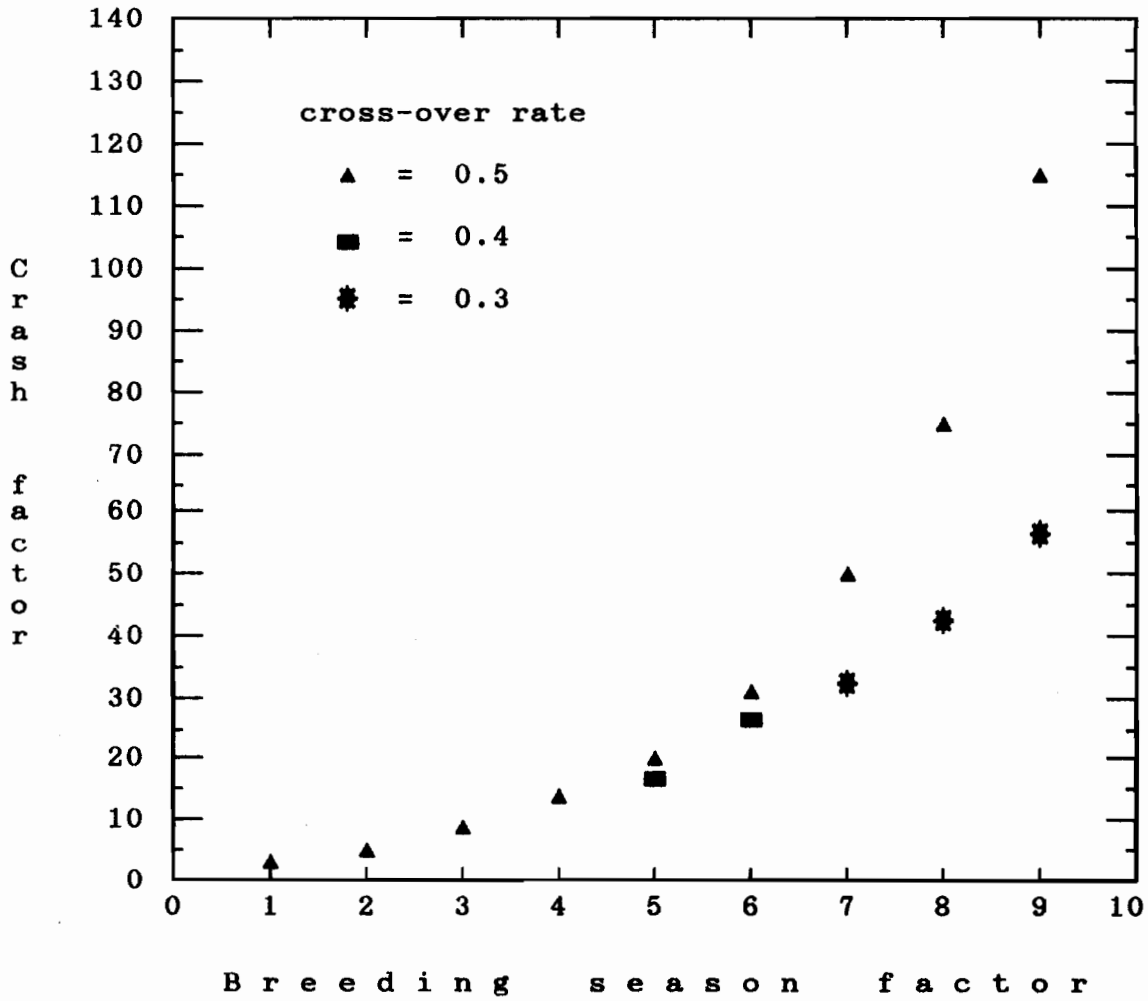

FIG. 3. Cross-over rates lower than 0.5 plotted are those requiring the lowest clutch size factor to give LGMF = 2S. Linkage of the clutch size and sex loci is disadvantageous where good breeding seasons are fewer than five in a cycle.

Recombination in a fluctuating environment has been studied by Charlesworth[17] and appears to be relevant here. If the clutch size genes are on the sex chromosomes there is linkage of the clutch size locus with the male and female "locus". Letting r represent the recombination fraction (0 indicating no crossing over to 0.5), and the subscripts f and m label allele frequencies for females and males, then after a good season:

$$R_{1f} = [R_{0f}(R_{0m}S + S - rS) + R_{0m}rL] \;/\; (R_{0m}L - R_{0m}rL + L + R_{0f}rS)$$

$$R_{1m} = [R_{0m}(R_{0f}S + L - rL) + R_{0f}rS] \;/\; (R_{0f}S - R_{0f}rS + L + R_{0m}rL)$$

For alelle frequencies after a bad season L is replaced by B, but the recombination fraction is kept the same.

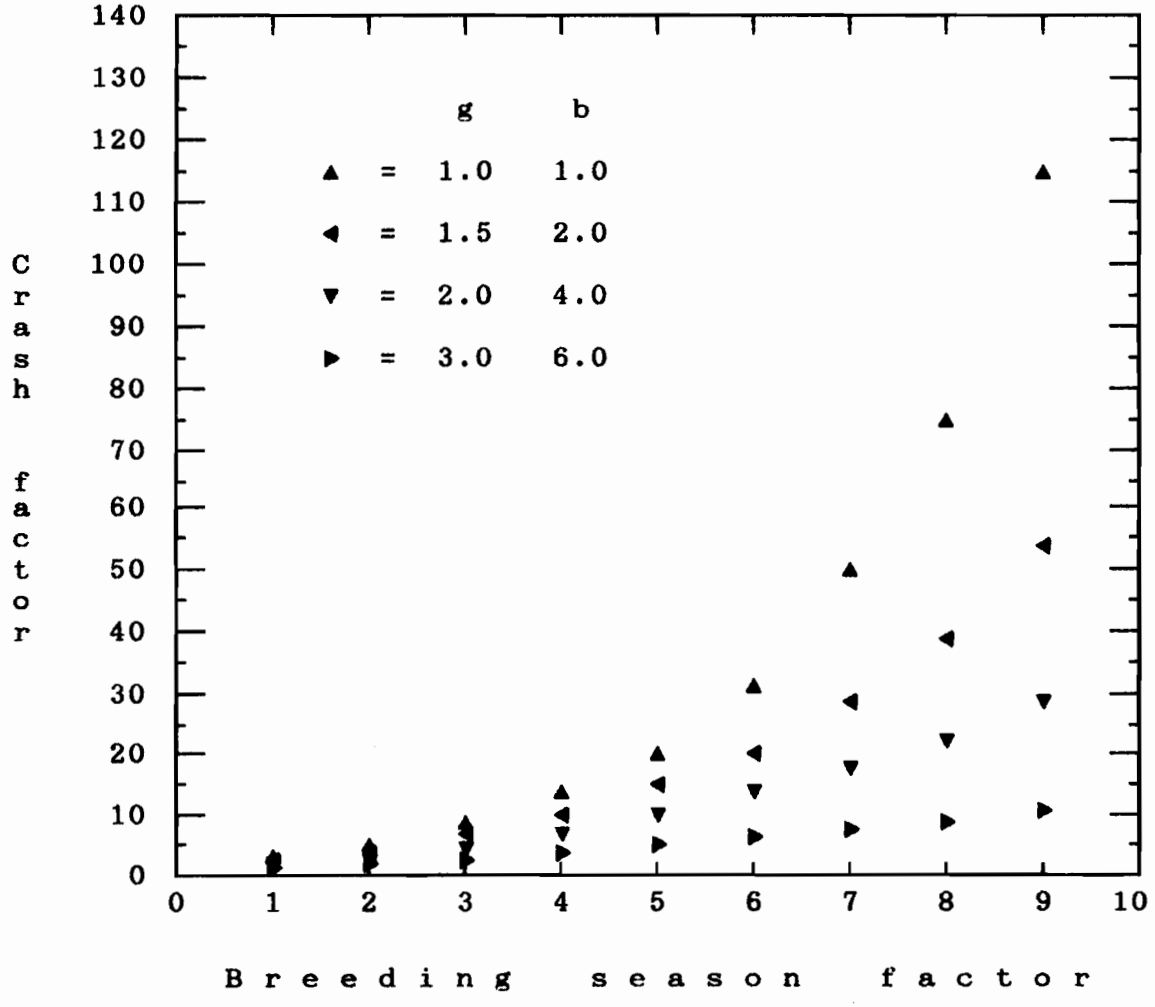

FIG. 4. Plots of the crash factors for the haploid strain when the LGMF = 2S with random mating and with three different probabilities of males from small clutches mating after a good season, g, and after a bad season, b. A male from a large clutch always has a mating probability = 1.0.

Linkage can be advantageous when there are more than four good seasons in a cycle. For instance, a cross over rate of 0.3 is optimal when there are nine good seasons to one bad, the LGMF being raised from 2.0 to 2.1 when S = 1 and the crash factor halved (Fig. 3). With stochastic conditions loose linkage is always favourable if good seasons exceed bad. Loose linkage with long cycles helps preserve the small clutch allele and further

favours the sexual strain in the presence of the asexual strain.

Male competition was introduced to see if the small clutch gene frequency could be further stabilised, males from small clutches tending to be larger in size[18] and thus more competitive. Presumably size differences between males would be more marked after bad seasons. A polygynous mating system was assumed. Various intensities of male competition were tried, small clutch males (α males) having a relative probability > 1.0 of mating, large clutch males (ß males) having a relative probability = 1.0 of mating (in the absence of competition the probability = 1.0 for both α and ß males).

Letting g denote the relative probability of α males mating following a good season, allele frequencies are:

$$R_{1f} = [R_{0f}(R_{0m}2S + S) + R_{0m}L] \;/\; (R_{0m}L + 2L + R_{0f}S)$$

$$R_{1m} = [R_{0m}(R_{0f}2gS + L) + R_{0f}gS] \;/\; (R_{0f}gS + 2L + R_{0m}L)$$

L is replaced by B in a bad season and g by b, the relative probability of α males mating following a bad season.

Male competition maintains more stable gene frequencies if the relative probability of α males mating is greater than ß males. When n = 2 and conditions cycle this is most marked if relative probabilities of α and ß males mating after good breeding seasons are both 1.0, the effect of competition after good seasons being to lower the LGMF as well as the crash factor, whereas competition after bad seasons raises the LGMF while lowering the crash factor. When conditions are stochastic, or n > 2, mating probability differences after good breeding seasons have the larger effect in raising the LGMF and lowering the crash factor. The more intense the competition after good and bad seasons, the lower the crash factor (Fig. 4), and the higher the LGMF, within the limits found if n = 2. These findings also hold in the presence of the asexual strain.

Female mate choice can allow similar results if choosy females prefer mating with larger males, the mate choice allele going from low values to fixation unless cost to choice is high. These mate choice results will be presented in a later paper.

Diploid Populations

In the diploid strains clutch sizes are determined by the dominant gene, A, and its recessive allele, a. Similar results to those for the haploid are obtained with random mating but clutch size factors are lower due to the additional buffering effect exerted by the heterozygous female (Fig. 1). From initial gene frequencies of 10^{-4} the diploid strain usually drives the asexual strain to extinction in less than 1000 generations.

With the same conditions and working as for the haploid, and subscripts d, h and r signifying whether an allele is from the homozygote dominant, heterozygote or homozygote recessive, the expected frequency ratio after a good season when the small clutch gene is dominant, $R = R_d + R_h$, are:

$$R_1 = R_0 \frac{R_0 2S + S + S_{h0} + L_{r0}}{R_0(S + S_{h0} + L_{r0}) + 2S_{h0} + 2L_{r0}}$$

$$1_{h1} = R_0(S + S_{h0} + L_{r0}) \;/\; [R_0(S + S_{h0} + L_{r0}) + 2S_{h0} + 2L_{r0}]$$

$$1_{r1} = (2S_{h0} + 2L_{r0}) \;/\; [R_0(S + S_{h0} + L_{r0}) + 2S_{h0} + 2L_{r0}]$$

$$R_0' = R_{n-1} \frac{R_{n-1} 2S + S + S_{hn-1} + B_{rn-1}}{R_{n-1}(S + S_{hn-1} + B_{rn-1}) + 2S_{hn-1} + 2B_{rn-1}}$$

Since most of the large clutch size alleles are in the homozygote recessive, L_r approximates to L, and for sufficiently low initial frequencies of the small clutch size allele (low R_0) the frequency ratio after a full cycle is approximately:

$$R_0' = R_0 (\frac{L + S}{2L})^{n-1} \frac{B + S}{2B}$$

This corresponds to the haploid case, except here most of the progeny carrying the common a allele are produced by aa x aa matings, and most of the progeny carrying the rare A allele are produced by Aa x aa or aa x Aa matings.

With similar working when the small clutch size is determined by the recessive gene, $R = R_h + R_r$, frequency ratios are:

$$R_1 = \frac{R_0(L + R_{h0} 2L + R_{r0} 2S) + R_{h0} L + R_{r0} S}{2L + R_0 L + R_{h0} L + R_{r0} S}$$

$$R_{h1} = (R_0 L + R_{h0} L + R_{r0} S) \;/\; (2L + R_0 L + R_{h0} L + R_{r0} S)$$

$$R_{r1} = R_0(R_{h0} 2L + R_{r0} 2S) \;/\; (2L + R_0 L + R_{h0} L + R_{r0} S)$$

$$R_0' = \frac{R_{n-1}(B + R_{hn-1} 2B + R_{rn-1} 2S) + R_{hn-1} B + R_{rn-1} S}{2B + R_{n-1} B + R_{hn-1} B + R_{rn-1} S}$$

For sufficiently low initial frequencies of the small clutch size allele (low R_0) the frequency ratio after a full cycle is approximately:

$$R_0' = R_0 (\frac{2L}{2L})^{n-1} \frac{2B}{2B}$$

Clearly the small clutch size allele when recessive and rare is unlikely to increase in a large population if cycles are short

unless there are local mating or group effects. If conditions are stochastic, or if the small clutch allele frequency is above 10^{-4} when conditions cycle, then the allele will invade. If good seasons are 2 to 31 in a cycle there is overdominance of the heterozygote and the population is very stable.

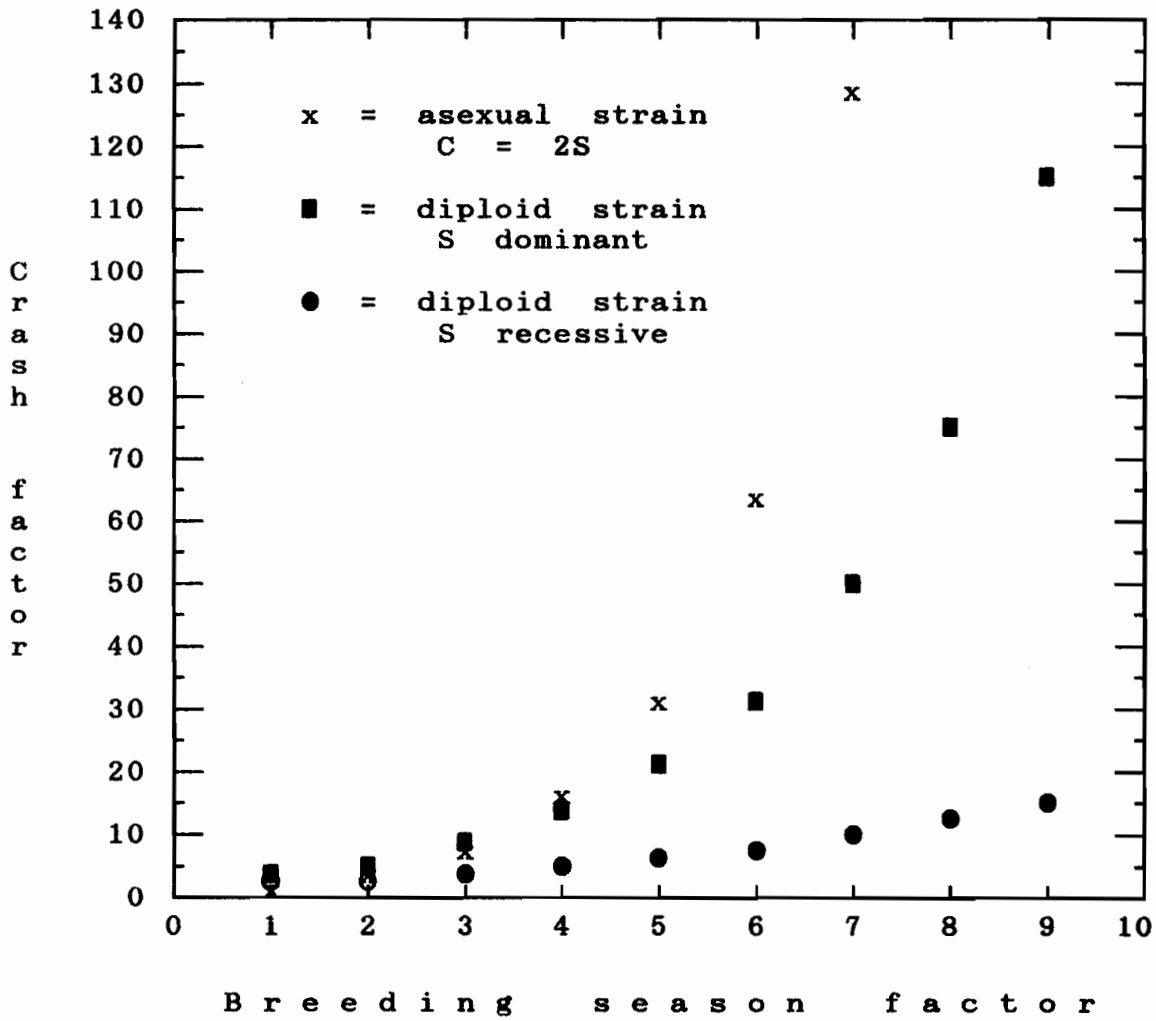

FIG. 5. Plots of the crash factors for the diploid strains resulting from random mating when the small clutch is determined by the recessive allele and when determined by the dominant allele, and for an asexual strain with C = 2S.

The LGMF and the crash factor when the small clutch size allele is dominant are:

$$W_d = \sqrt[n]{\frac{(R_0S + S_{h0} + L_{r0}) \ldots (R_{n-2}S + S_{hn-2} + L_{rn-2})(R_{n-1}S + S_{hn-1} + B_{rn-1})}{(R_0 + 1) \ldots (R_{n-2} + 1)(R_{n-1} + 1)}}$$

$$cf_d = \frac{R_{n-1} + 1}{R_{n-1}S + S_{hn-1} + B_{rn-1}}$$

and when the small clutch size allele is recessive they are:

$$W_r = \sqrt[n]{\frac{(L + R_{h0}L + R_{r0}S) \ldots . (L + R_{hn-2}L + R_{rn-2}S)(B + R_{hn-1}B + R_{rn-1}S)}{(1 + R_0) \ldots . (1 + R_{n-2})(1 + R_{n-1})}}$$

$$cf_r = \frac{1 + R_{n-1}}{B + R_{hn-1}B + R_{rn-1}S}$$

When the small clutch is dominant the crash factor increases as rapidly as in the haploid strain, but when it is recessive the crash factor increases slowly with n (Fig. 5), and is much lower if n > 2 than for any competitive asexual clone with LGMF = 1.

With the female as the heterogametic sex as in birds, and the small clutch dominant, linkage of the clutch size locus with the sex locus always lowers the crash factor and considerably raises the LGMF. The tighter the linkage the more advantageous, e.g. when n = 2, r = 0.01 and S = 1 the cf is lowered from 2.7 to 1.5 and the LGMF raised from 2.0 to 3.2, so allowing a reduction in large clutch size from 24 to 9 to give an LGMF > 2. If the small clutch is recessive linkage is only advantageous if strong and n < 6, e.g. when n = 2, r = 0.01 and S = 1 the cf is lowered from 2.2 to 1.5 and the LGMF raised from 2.0 to 3.2.

With the male as the heterogametic sex, and the small clutch dominant, linkage raises the LGMF and reduces the cf if n = 2, e.g. when r = 0.01 and S = 1 the cf is lowered from 2.7 to 1.9 and the LGMF raised from 2.0 to 2.5. When n = 3-5 linkage is unfavourable under cycling conditions. If n > 5 linkage is again favourable if weak, but the crash factor is reduced less than in the haploid case. If the small clutch is recessive linkage is only favourable when n = 2, e.g. with r = 0.01 and S = 1 the cf is lowered from 2.2 to 1.9 and the LGMF raised from 2.0 to 2.5.

Male competition lowers the crash factor and raises the LGMF when the small clutch is determined by the dominant allele. If n > 2 the crash factor decreases as competition intensifies to a similar extent as for the haploid, and the LGMF increases.

When the small clutch is determined by the recessive allele competition is no help if n ≤ 4. If n > 4, weak competition (probability of an a male mating < 3 after a good season and < 6 after a bad season when n = 5, < 4 and < 8 respectively when n > 5) lowers the cf and raises the LGMF.

Female mate choice in both diploid cases can allow similar results. These mate choice results will be presented later.

Completely stable polymorphisms with genotype frequencies of 0.25AA : 0.50Aa : 0.25aa, which occur if the homozygote dominant and the homozygote recessive determine the large clutch and the heterozygote the small clutch, or vice versa, give a cf ≈ 2 when n = 2 (L = 14 if S = 1). Such a polymorphism does not require the relative fitnesses of the homozygotes to be perfectly correlated as do those of other workers[15,19,20]. It is to be noted

that when n > 4 the diploid strain with the large clutch dominant can prevail over a stable polymorphism with the same clutch size values, even though it shows greater variance in mean fitness.

Conclusion

These models accord with the original findings of Haldane[21] and Haldane and Jayakar[13] that in a diploid population a stable polymorphism is possible in the absence of overdominance of the heterozygote if there is temporal variation in selective intensities, or if the same gene substitution affects the fitness of the two sexes in opposite directions. These two factors act together here to allow stable polymorphisms in both haploids and diploids, but with the difference that the sexes have the same mean fitness, the complement of any clutch size gene carried by males receiving each generation the mean fitness under current breeding conditions of clutch size genes expressed by females.

The findings are not only applicable to birds. They accord with the observation that asexual invertebrates and vertebrates, as well as plants, tend to be restricted to temperate regions which have low disease pressure and to regions with an unpredictable environment[22]. If there are many good breeding seasons the asexual strain tends to take over, whereas if good and bad seasons are equally frequent the largest clutch usually has to be unrealistically greater than the smallest, more than 20 times, for the sexual strain to prevail.

Indications are that two to six good seasons to one bad are most favourable for sexual reproduction. Epidemic disease cycles could also be a driving force here, not just climatic conditions. Epidemic years tend to occur when the population has built up to the point where there are many susceptible young. Young from large "clutches" may be more prone to infection if they are less well fed or if they have more contact with other young. Infectious diseases often show two to six year cycles, longer on remote islands. Interestingly, island birds tend to have smaller clutches than mainland birds of the same species[23,24].

The cost of evolution is low compared to other models[25], there being a 33% to 55% crash in population size after a bad season when good and bad seasons alternate. When good seasons increase in frequency lower clutch size factors are necessary, even though crash factors increase. Fitness and crash factor values are consistent with the findings of Grenfell et al[26] that up to 70% of Soay sheep on St. Kilda die from starvation in population crashes occuring at three to four year intervals.

The crash factor phenomenon need not entail explosive population growth. If non-breeding season conditions were stringent, then the greater the crowding of the population the greater would be the fall-off in numbers. All individuals would

suffer. Large individuals may compete more successfully for food, but they need more than small individuals to survive. The population could be comparable in size at the start of most breeding seasons after fall-off, and just show an occasional poor breeding result. With red deer density dependent constraints on juvenile mortality help prevent a significant overshoot of the population equilibrium[27,28].

Roughgarden[4] stated a sexual population has a higher LGMF than an asexual population in a fluctuating environment since the restoration of its genotype frequencies to the Hardy-Weinberg curve each generation lessens its variance through time in mean fitness. This does not address the case of a haploid population. In the present findings, although even stronger buffering of variance can result from the repartition of genes between males and females each generation, the greater rapidity of a sexual population to recover from changed conditions is more important. The sheltering by the male of the currently rarer gene from direct selection makes this rapid recovery possible.

Hamilton et al[15] found promising short-term advantage for sexual reproduction with low fitness differences, but requiring unrealistic overdominance in the fitness patterns when only one locus was involved. Another single locus model by Hamilton[14] did not have overdominance, instead conditions were such that asexuals on their own could not constitute a viable population. Weinshall[25] presented a multiple allele single locus model, but the environment had to have three or more states. Kirkpatrick and Jenkins[29] showed a benefit for sex in diploids relying on segregation and selective load. Roughgarden[4] demonstrated advantage for sex dependent on recurrent mutation and assortment under fluctuating conditions. These earlier models were either not applicable to haploids or required two or more loci. The present models apply to both diploid and haploid populations. They do not require more than one locus in haploids, unrealistic overdominance, or recurrent mutation, they only require two alleles, a two-state environment and low fitness differences.

ACKNOWLEDGEMENTS

I thank the University of Geneva, B. Levrat and J. Mouttet for computer programming tuition, the University of Vienna and the Zoology Department of the University of Oxford for library facilities, E.M. Backett, P.H. Harvey, J. Maynard Smith and B.G. Murray Jr for constructive criticism of groundwork and W.D. Hamilton for many helpful comments on earlier manuscripts. I especially thank M.K. Uyenoyama and B. Charlesworth, both of whom when reviewing the manuscript suggested improvements for the analysis and its interpretation .

LITERATURE CITED

1. Williams, G.C. 1975. Sex and Evolution (Princeton University Press).
2. Maynard Smith, J. 1978. The Evolution of Sex (Cambridge University Press).
3. Felsenstein, J. 1976. A. Rev. Genet. 10 : 253-280.
4. Roughgarden, J. 1991. Am. Nat. 138 : 934-953.
5. Blueweiss, L., Fox, H., Kudzma, V., Nakashima, D., Peters, R. & Sams, S. 1978. Oecologia 37 : 257-272.
6. Gibbs, H.L. 1988. Evolution 42 : 750-762.
7. Perrins, C.M. & Jones, P.J. 1974. Condor 76 : 225-229.
8. Flux, J.E.C. & Flux, M.M. 1981. Naturwissenschaften 69 : 96-97.
9. van Noordwijk, A.J., van Balen, A.J. & Scharloo, W. 1981. Neth. J. Zool. 31 : 342-372.
10. Gustafsson, L. 1986. Am. Nat. 128 : 761-764.
11. Findlay, C.S. & Cooke, F. 1987. Evolution 41 : 453.
12. Boyce, M.S. & Perrins, C.M. 1987. Ecology 68 : 142-153.
13. Haldane, J.B.S. & Jayakar, S.D. 1962. J. Genet. 58 : 237-242.
14. Hamilton, W.D. 1980. Oikos 35 : 282-290.
15. Hamilton, W.D., Henderson, P.A. & Moran, N.A. 1981. In Natural Selection and Social Behavior : recent research and theory (eds Alexander, R.D. & Tinkle, D.W.) 363-381 (Chiron Press, New York).
16. Lloyd, D.G. 1980. Ev. Biol. 13 : 69-111.
17. Charlesworth, B. 1976. Genetics 83 : 181-195.
18. Perrins, C.M. 1965. J. anim. Ecol. 34 : 601-647.
19. Hartl, D.L. & Cook, R.D. 1973. Theor. Popul. Biol. 4 : 163-172.
20. Karlin, S. & Liberman, U. 1974. Theor. Popul. Biol. 6 : 355-382.
21. Haldane, J.B.S. 1962. Nature 193 : 1108.
22. Levin, D.A. 1975. Am. Nat. 109 : 437-451.
23. Lack, D. 1968. Ecological adaptations for breeding in birds. (Methuen, London).
24. Cody, M.L. 1966. Evolution 20 : 174-184.
25. Weinshall, D. 1986. Am. Nat. 128 : 736-750.
26. Grenfell, B.T., Price, O.P., Albon, S.D. & Clutton-Brock, T.H. 1992. Nature 355 : 823-826.
27. Clutton-Brock, T.H., Major, M. & Guinness, F.E. 1985. J. Anim. Ecol. 54 : 831-846.
28. Clutton-Brock, T.H. & Albon, S.D. 1989. Red Deer in the Highlands (Blackwell Scientific, Oxford).
29. Kirkpatrick, M. & Jenkins, C.D. 1989. Nature 339 : 300-301.