THE MANIFOLD GAIN OF SEXUAL REPRODUCTION
II. Models not involving heritability of progeny number

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ABSTRACT. An advantage accruing to sexual reproduction not relying on differential genetic heritability of progeny number is modelled for species in which the product of fertilisation is dependent on one parent more than the other at some stage of development. In such species a genetic polymorphism affecting viability that continues to act after mating will result in different reproductive success for males and females of the same genotype, the fitness of the less involved parent in terms of progeny attaining independence largely relying on the viability of the more involved parent. This effect can maintain a protected polymorphism of two genes at a single locus under fluctuating selection in both haploid and diploid populations. Fitness differences necessary to give a short-term advantage to sex sufficient to overcome the two-fold reproductive cost experienced in competition with an asexual population are small. Additional loci allow even smaller fitness differences, and high levels of recombination can be advantageous. Fecundity can be low and there is no cost of evolution if environmental period is short.

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Part II of this paper develops further a hypothesis proposed in part I[1] to give an advantage to sex (for a recent review of hypotheses on the advantage of sex see Kondrashov[2]).

In part I genetic models were presented showing that under fluctuating selection gene sheltering by one sex could protect a polymorphism expressed by the opposite sex in both diploid and haploid populations. When a locus for fecundity was involved, buffering of gene frequencies by the male allowed a polymorphism for progeny number capable of giving a sexual population a manifold advantage over an asexual population.

Here in part II it is demonstrated that such a protected polymorphism need not depend on genetic heritability of progeny number. Genetic factors affecting viability would also tend to result in different reproductive success for males and females of the same genotype in species where progeny are dependent on one parent more than the other. The immediate fitness of a male and female of the same genotype could be identical, but their

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fitness in terms of progeny successfully raised would differ if the mate of the less involved sex were of a different genotype, the number of progeny attaining independence largely relying on the viability of the more involved parent.

In most species it is male reproductive success that is dependent on the fitness of the female. With mammals, for example, gestation and lactation make strong demands on the female and reproductive success is dependent on her viability. Even with insects extra demands beyond ova formation are made of the female who seeks out suitable sites for oviposition. There are exceptions where the male takes on more responsibility than the female in caring for the young, such as male South American poison frogs, but the same effect is to be expected, and the argument here will be confined to female responsibility.

Genetic models for haploid and diploid populations are presented to demonstrate this effect. Computer simulations were written in Turbo-Pascal to follow population trajectories. Generations were considered non-overlapping and discrete.

The locus for viability has two alleles. Both sexual and asexual populations consist of the same number of genotypes, the genotypes being genetically distinct clones in the asexual population. The assumption was made[3] that each asexual clone had the same fitness as had the equivalent female sexual genotype, and that asexual and sexual females were involved to an equal extent in caring for their young. This de-emphasised competition between clones so allowing the long term geometric mean fitness, *LGMF*, of the sexual population as a whole to be assessed.

To simplify the argument, and highlight the maternal effect, selection on all genotypes up to the time of mating was considered to be negligible, their fitness, F, being unity.

Males in sexual strains were 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 all else is equal. Mating was random, no male or sib competition, or kin selection, being involved.

The *LGMF*s for the asexual clones were computed for a complete cycle of environmental conditions consisting of n generations. The environment cycles through $0.5n$ generations in which a genotype sees a fitness of $F > 1$ if the environment is favourable to it, but a fitness lower than F if unfavourable, and $0.5n$ generations in which the genotype experiences the alternate environment. The reduced fitness value F^{-1} was used to aid comparison since the *LGMF* then equalled 1 for each clone.

The *LGMF* for the sexual strain was computed similarly. After mating, each sexual female genotype sees a fitness of F if the environment is favourable or F^{-1} if unfavourable, and the fitness of a male in terms of progeny depends on that of his mate.

Haploid Populations

In the haploid strain the viability gene A_1 confers a fitness of F in environment one and F^{-1} in environment two and its allele A_2 a fitness of F^{-1} in environment one and F in environment two. To allow the analysis, instead of Hardy-Weinberg frequencies, the frequencies of A_1, P, and A_2, Q, are expressed as ratios of the frequency of A_2, Q. This gives $A_1 = P / Q = R$ and $A_2 = Q / Q = 1$ for both males and females (Table 1).

TABLE 1. Haploid strain zygote values resulting from matings in different environments

Environment one			Environment two		
♀ \ ♂	R (A_1)	1 (A_2)	♀ \ ♂	R (A_1)	1 (A_2)
RF (A_1)	R^2F (A_1A_1)	RF (A_1A_2)	RF^{-1} (A_1)	R^2F^{-1} (A_1A_1)	RF^{-1} (A_1A_2)
F^{-1} (A_2)	RF^{-1} (A_1A_2)	F^{-1} (A_2A_2)	F (A_2)	RF (A_1A_2)	F (A_2A_2)

The ratio, R_1, of the frequencies of the allele A_1 and the allele A_2 after the first generation in environment one is:

$$R_1 = \frac{R_0^2F + \tfrac{1}{2}R_0F^{-1} + \tfrac{1}{2}R_0F}{\tfrac{1}{2}R_0F^{-1} + \tfrac{1}{2}R_0F + F^{-1}} = R_0\frac{R_02F + F^{-1} + F}{R_0(F^{-1} + F) + 2F^{-1}} \qquad (1)$$

in which R_0 is the ratio of the frequency of A_1 to A_2 in the initial generation. This recursion is carried forward through $0.5n$ generations in environment one. In environment two F is replaced by F^{-1} and F^{-1} by F in equation (1). The ratio at the start of the next n-generation cycle after the nth generation is:

$$R_0' = R_n = R_{n-1}\frac{R_{n-1}2F^{-1} + F + F^{-1}}{R_{n-1}(F + F^{-1}) + 2F} \qquad (2)$$

The functions on the right of equations (1) and (2) are both monotone functions of the frequency ratio, and the full recursion over n generations 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 gene A_1 (low R_0) the frequency ratio after a full cycle is approximately:

$$R_0' = R_0\left(\frac{F^{-1} + F}{2F^{-1}}\right)^{0.5n}\left(\frac{F + F^{-1}}{2F}\right)^{0.5n} \qquad (3)$$

This recursion indicates that gene A_1 increases when rare:

$$\frac{F^{-1} + F}{2} > 1 \tag{4}$$

The same argument and equation (4) apply to gene A_2 when rare. For F as given here, equations (3) and (4) correspond to:

$$\left(\frac{AM(F, F^{-1})}{GM(F, F^{-1})}\right)^n > 1 \tag{5}$$

in which $AM(x, y)$ represents the arithmetic mean of x and y and $GM(x, y)$ the geometric mean. Since equation (5) is always satisfied, $AM > GM$, except when $F = 1$, both genes increase in frequency when rare. This finding, together with monotonicity of the recursion function over the full n-environment cycle, implies convergence to a stable polymorphic equilibrium state.

Fitness values need not be symmetrical, nor the reduced fitness value the reciprocal of F, for the maintenance of polymorphism since the system is protected. Initial allele frequencies are immaterial, and polymorphism is maintained at the viability locus without recurrent mutation. The same results apply when the environmental period is irregular or stochastic.

The $LGMF$ in equation (6) is for the sexual population as a whole, $\overline{W}$. R_0 is the ratio of A_1 to A_2 in the mating formula for the initial generation, environment one, and R_{n-1} is the ratio of A_1 to A_2 in the mating formula for the final generation, environment two, in the complete n generation cycle. The new R value is obtained each generation by using the appropriate frequency ratio with either equation (1) or (2), depending on which environment is active. Where there are more than two generations, the intervening mating formulae with the relevant R and fitness values are inserted into (6). Male values can be cancelled out; they have been left for completeness.

$$\overline{W} = \sqrt[n]{\frac{(R_0F + F^{-1})(R_0 + 1)}{(R_0 + 1)(R_0 + 1)} \cdots \frac{(R_{n-1}F^{-1} + F)(R_{n-1} + 1)}{(R_{n-1} + 1)(R_{n-1} + 1)}} \tag{6}$$

The Hardy-Weinberg equation corresponding to (6) is:

$$\overline{W} = \sqrt[n]{(P_0F + Q_0F^{-1})(P_0 + Q_0) \cdots (P_{n-1}F^{-1} + Q_{n-1}F)(P_{n-1} + Q_{n-1})} \tag{7}$$

The equations corresponding to (1) and (2) are:

$$P_1 = P_0\frac{P_0 2F + Q_0F^{-1} + Q_0F}{2(P_0F + Q_0F^{-1})(P_0 + Q_0)} \qquad Q_1 = Q_0\frac{P_0F^{-1} + P_0F + Q_0 2F^{-1}}{2(P_0F + Q_0F^{-1})(P_0 + Q_0)} \tag{8}$$

$$P_0' = P_{n-1}\frac{P_{n-1}2F^{-1} + Q_{n-1}F + Q_{n-1}F^{-1}}{2(P_{n-1}F^{-1} + Q_{n-1}F)(P_{n-1} + Q_{n-1})} \qquad Q_0' = Q_{n-1}\frac{P_{n-1}F + P_{n-1}F^{-1} + Q_{n-1}2F}{2(P_{n-1}F^{-1} + Q_{n-1}F)(P_{n-1} + Q_{n-1})} \tag{9}$$

The new *P* and *Q* values are obtained each generation by using the allele frequencies from the previous generation with either equation (8) or (9), depending on which environment is active. Where there are more than two generations in a cycle, the intervening mating formulae with the relevant allele frequencies and fitness values are inserted into (7).

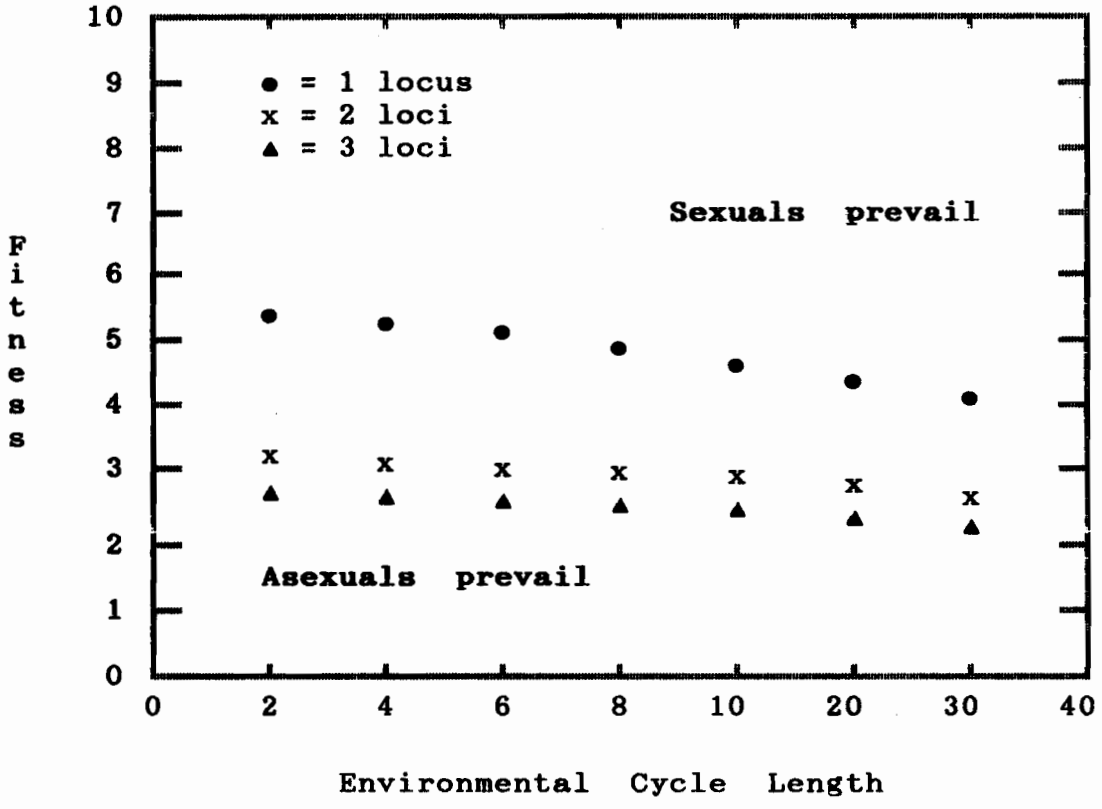

FIG. 1. Fitness values necessary for the haploid sexual strain to overcome the asexual strain. Environmental cycle length is in generations. The loci are not linked.

It is clear from (6) and (7) that a *LGMF* > 1 is possible for the sexual population as a whole (*LGMF* > 2 for sexual females, the fitness of a male in terms of progeny depending here on the viability of his mate). Starting from gene frequencies of 10^{-4} the sexual strain invades the asexual strain and drives it to extinction within 200 generations under cycling conditions, and prevails most of the time under stochastic conditions, if *F* is sufficiently large.

The value *F* = 5.32 gives sexual females a *LGMF* > 2, compared to a *LGMF* = 1 for each asexual clone, when environments alternate (Fig.1). This corresponds to a fecundity of 6. With longer cycles lower *F* values are necessary, and if cycles are longer than four generations *F* values less than 5, corresponding to a fecundity of 5, allow a *LGMF* > 2. If selection before the time of mating is high, fecundity has to be raised to compensate.

Additional loci for viability can enhance this effect (Fig. 1). Hardy-Weinberg gene frequencies were used to compute the *LGMF* values. With two loci fitness values necessary for compensation are $F = 3.23$ when environments alternate, for three loci $F = 2.61$. This means that fecundity must be raised, to 11 with two loci and to 18 with three loci. With tight linkage, $r = 0.1$ (where r represents the recombination fraction, 0 indicating no crossing over to 0.5), these fecundity values can be lowered to 7 and 8 respectively. With longer environmental period and three loci, fitness values smaller than 2 are possible.

Where each locus has its own specific set of environments determining gene viability, high recombination rates are favourable if cycles are of different length or stochastic.

Diploid Populations

In the diploid strain the viability locus shows complete dominance. The dominant genotype *A-* confers a fitness of F in environment one and F^{-1} in environment two, and the homozygous recessive genotype *aa* a fitness of F^{-1} in environment one and F in environment two. The frequencies of *A*, *P*, and *a*, *Q*, are expressed as ratios of the more common allele when rare to allow the analysis. For females, subscripts h and r signify whether an *a* allele is from the heterozygote or the homozygote recessive.

If *A* is rare the common allele $a = (PQ + Q^2) / (PQ + Q^2) = 1$ and $A = (P^2 + PQ) / (PQ + Q^2) = R$. To avoid including another variable, $a_h = PQ / (PQ + Q^2) = 1_h$ and $a_r = Q^2 / (PQ + Q^2) = 1_r$ are used, giving $1_h + 1_r = 1$. To simplify, $F \times 1_h = F_h$ and $F \times 1_r = F_r$, and likewise for F^{-1} (Table 2).

TABLE 2. Diploid strain zygote values from matings when the dominant allele is rare

	Environment one		Environment two	
♀ \ ♂	R (*A*)	1 (*a*)	R (*A*)	1 (*a*)
RF (*A*)	R^2F (*AA*)	RF (*Aa*)	RF^{-1} (*A*)	R^2F^{-1} (*AA*)	RF^{-1} (*Aa*)
F_h (*a*)	RF_h (*Aa*)	F_h (*aa*)	F_h^{-1} (*a*)	RF_h^{-1} (*Aa*)	F_h^{-1} (*aa*)
F_r^{-1} (*a*)	RF_r^{-1} (*Aa*)	F_r^{-1} (*aa*)	F_r (*a*)	RF_r (*Aa*)	F_r (*aa*)

The expected ratio R_1 of the frequencies of the allele A and the allele a after the first generation in environment one is:

$$R_1 = \frac{R_0^2F + \frac{1}{2}R_0F + \frac{1}{2}R_0F_{h0} + \frac{1}{2}R_0F_{r0}^{-1}}{\frac{1}{2}R_0F + \frac{1}{2}R_0F_{h0} + \frac{1}{2}R_0F_{r0}^{-1} + F_{h0} + F_{r0}^{-1}} = R_0\frac{R_02F + F + F_{h0} + F_{r0}^{-1}}{R_0(F + F_{h0} + F_{r0}^{-1}) + 2F_{h0} + 2F_{r0}^{-1}} \qquad (10)$$

$$1_{h1} = R_0(F + F_{h0} + F_{r0}^{-1}) \;/\; [R_0(F + F_{h0} + F_{r0}^{-1}) + 2F_{h0} + 2F_{r0}^{-1}] \qquad (10a)$$

$$1_{r1} = (2F_{h0} + 2F_{r0}^{-1}) \;/\; [R_0(F + F_{h0} + F_{r0}^{-1}) + 2F_{h0} + 2F_{r0}^{-1}] \qquad (10b)$$

in which R_0 is the ratio of the frequency of A to a in the initial generation. This recursion is carried forward through $0.5n$ generations in environment one. In environment two F is replaced by F^{-1} and F^{-1} by F in equation (10). The ratio at the beginning of the next n-generation cycle after the nth generation is:

$$R_0' = R_n = R_{n-1}\frac{R_{n-1}2F^{-1} + F^{-1} + F_{hn-1}^{-1} + F_{rn-1}}{R_{n-1}(F^{-1} + F_{hn-1}^{-1} + F_{rn-1}) + 2F_{hn-1}^{-1} + 2F_{rn-1}} \qquad (11)$$

Since most a alleles are in the homozygote recessive, F_r approximates to F and F_r^{-1} to F^{-1}, and for sufficiently low initial frequencies of A (low R_0) the frequency ratio after a full cycle is approximately:

$$R_0' = R_0\left(\frac{F + F^{-1}}{2F^{-1}}\right)^{0.5n}\left(\frac{F^{-1} + F}{2F}\right)^{0.5n} \qquad (12)$$

This corresponds to the haploid case, equation (3), the recursion indicating that the allele A increases in frequency when rare, equation (4).

The *LGMF* in equation (13) is for the sexual population as a whole, $\bar{W}$. R_0 is the ratio of A to a in the mating formula for the initial generation, environment one, and R_{n-1} is the ratio of A to a in the mating formula for the final generation, environment two, in the complete n generation cycle. The new R value is obtained each generation by using the frequency ratio from the previous generation with either equation (10) or (11), depending on which environment is active. Where there are more than two generations in a cycle, the intervening mating formulae with the relevant frequency ratio and fitness values are inserted into (13). Male values can be cancelled out, but have been left in the mating formulae for completeness.

$$\bar{W} = \sqrt[n]{\frac{(R_0F + F_{h0} + F_{r0}^{-1})(R_0 + 1)}{(R_0 + 1)(R_0 + 1)} \cdots \frac{(R_{n-1}F^{-1} + F_{hn-1}^{-1} + F_{rn-1})(R_{n-1} + 1)}{(R_{n-1} + 1)(R_{n-1} + 1)}} \qquad (13)$$

If a is rare (Table 3), $a_h = PQ / (P^2 + PQ) = R_h$ and $a_r = Q^2 / (P^2 + PQ) = R_r$, giving $R_h + R_r = R$ and $A = P / P = 1$:

TABLE 3. Diploid strain zygote values from matings when the recessive allele is rare

Environment one			Environment two		
♀ \ ♂	1 (A)	R (a)	♀ \ ♂	1 (A)	R (a)
F (A)	F (AA)	RF (Aa)	F^{-1} (A)	F^{-1} (AA)	RF^{-1} (Aa)
R_hF (a)	R_hF (Aa)	RR_hF (aa)	R_hF^{-1} (a)	R_hF^{-1} (Aa)	RR_hF^{-1} (aa)
R_rF^{-1} (a)	R_rF^{-1} (Aa)	RR_rF^{-1} (aa)	R_rF (a)	R_rF (Aa)	RR_rF (aa)

The expected ratio R_1 of the frequencies of the allele a and the allele A after the first generation in environment one is:

$$R_1 = \frac{\frac{1}{2}R_0F + R_0R_{h0}F + R_0R_{r0}F^{-1} + \frac{1}{2}R_{h0}F + \frac{1}{2}R_{r0}F^{-1}}{F + \frac{1}{2}R_0F + \frac{1}{2}R_{h0}F + \frac{1}{2}R_{r0}F^{-1}}$$

$$= \frac{R_0(F + R_{h0}2F + R_{r0}2F^{-1}) + R_{h0}F + R_{r0}F^{-1}}{2F + R_0F + R_{h0}F + R_{r0}F^{-1}} \qquad (14)$$

$$R_{h1} = (R_0F + R_{h0}F + R_{r0}F^{-1}) \;/\; (2F + R_0F + R_{h0}F + R_{r0}F^{-1}) \qquad (14a)$$

$$R_{r1} = R_0(R_{h0}2F + R_{r0}2F^{-1}) \;/\; (2F + R_0F + R_{h0}F + R_{r0}F^{-1}) \qquad (14b)$$

in which R_0 is the ratio of the frequency of a to A in the initial generation. The ratio at the beginning of the next n-generation cycle is:

$$R_0' = R_n = \frac{R_{n-1}(F^{-1} + R_{hn-1}2F^{-1} + R_{rn-1}2F) + R_{hn-1}F^{-1} + R_{rn-1}F}{2F^{-1} + R_{n-1}F^{-1} + R_{hn-1}F^{-1} + R_{rn-1}F} \qquad (15)$$

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

$$R_0' = R_0\left(\frac{2F}{2F}\right)^{0.5n}\left(\frac{2F^{-1}}{2F^{-1}}\right)^{0.5n} \qquad (16)$$

It is clear from equation (16) that the recessive allele a if rare will be slow to increase in frequency in a large population when cycles are long, and this is so if its frequency is below 10^{-6}. Under stochastic conditions its increase is rapid.

Sexual Reproduction

Although the recessive allele is slow to increase in frequency when very rare, the system is still protected. Fitness values need not be symmetrical nor the reduced fitness value the reciprocal of F, for the maintenance of polymorphism. Initial allele frequencies are immaterial, and polymorphism is maintained at the viability locus without recurrent mutation. The same results apply when the environmental period is irregular or conditions are stochastic.

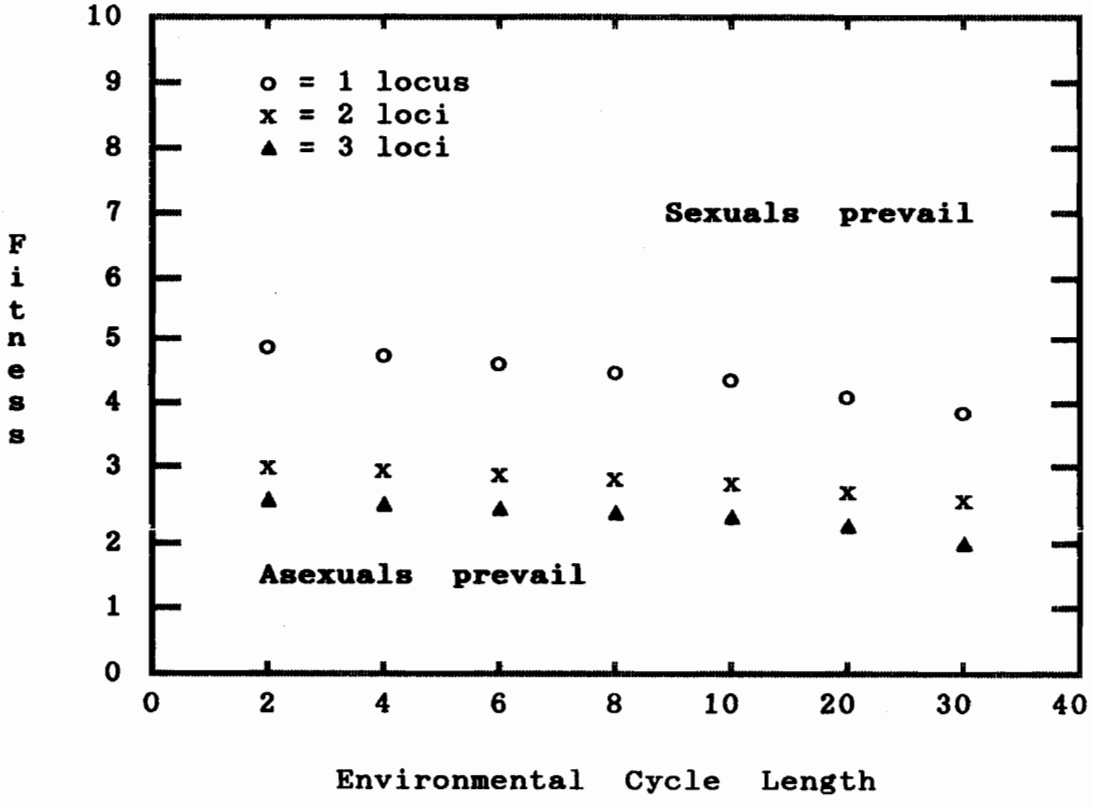

FIG. 2. Fitness values necessary for the diploid sexual strain to overcome the asexual strain. Environmental cycle length is in generations. The loci are not linked.

The *LGMF* for the sexual population can be calculated in the same way as for the dominant allele when rare, using equations (14) and (15) for R values, the same result being obtained:

$$\bar{W} = \sqrt[n]{\frac{(F + R_{h0}F + R_{r0}F^{-1})(1 + R_0)}{(1 + R_0)(1 + R_0)} \cdots \frac{(F_{n-1}^{-1} + R_{hn-1}F^{-1} + R_{rn-1}F)(1 + R_{n-1})}{(1 + R_{n-1})(1 + R_{n-1})}} \quad (17)$$

The same Hardy-Weinberg *LGMF* equation corresponds to both (13) and (17):

$$\bar{W} = \sqrt[n]{(P_0F + Q_{h0}F + Q_{r0}F^{-1})(P_0 + Q_0) \cdots (P_{n-1}F^{-1} + Q_{hn-1}F^{-1} + Q_{rn-1}F)(P_{n-1} + Q_{n-1})} \quad (18)$$

The new allele frequencies in environment one are obtained from:

$$P_1 = P_0 \frac{2P_0F + Q_0F + Q_{h0}F + Q_{r0}F^{-1}}{2(P_0F + Q_{h0}F + Q_{r0}F^{-1})(P_0 + Q_0)} \quad (19a)$$

$$Q_{h1} = P_0 \frac{Q_0F + Q_{h0}F + Q_{r0}F^{-1}}{2(P_0F + Q_{h0}F + Q_{r0}F^{-1})(P_0 + Q_0)} \quad (19b)$$

$$Q_{r1} = Q_0 \frac{2Q_{h0}F + 2Q_{r0}F^{-1}}{2(P_0F + Q_{h0}F + Q_{r0}F^{-1})(P_0 + Q_0)} \quad (19c)$$

From (19b) and (19c), $Q_1 = Q_{h1} + Q_{r1}$. In environment two F and F^{-1} values are interchanged. If there are more than two generations in a cycle, the intervening mating formulae with the relevant allele frequencies and fitness values are inserted into (18).

Similar results to those for the haploid are obtained, but necessary fitness values are lower due to the extra buffering effect exerted by the heterozygous female. From initial gene frequencies of 10^{-4} the diploid strain usually drives the asexual strain to extinction within 200 generations under cycling conditions, and prevails most of the time under stochastic ones.

The value $F = 4.84$ gives sexual females a $LGMF > 2$ when environments alternate (Fig. 2). If cycles are longer than 18 generations F values slightly less than 4 allow a $LGMF > 2$. The lowest fecundity to compensate for the full cost of meiosis is 5 when cycles are shorter than 18 generations, 4 when cycles are longer than 17 generations.

With two loci for viability, fitness values necessary for compensation under alternating environments are $F = 2.90$, for three loci $F = 2.45$ (Fig. 2). This means that fecundity must be raised, to 9 for two loci and to 15 for three loci. With tight linkage, $r = 0.1$, fecundity can be lowered to 8 and 9 respectively if dominant genes are not favoured under the same environment, and to 6 and 7 respectively if dominant genes are favoured under the same environment. With longer environmental period and three loci fitness values smaller than 2 are possible.

As in the haploid case, where each locus has its own specific environments determining gene viability, high recombination rates are favourable if cycles are of different length or stochastic.

Conclusion

Under fluctuating selection, a currently less fit gene inherited by the young from the male is sheltered from direct selection if the female expresses the currently more fit gene while the young is dependent on her, e.g. while *in utero*. This

buffering effect allows the sexual population to recover from changed conditions more rapidly than an asexual population.

Temporal variation in selective intensities was first identified by Haldane and Jayakar[4] to favour polymorphism in the diploid case in the absence of overdominance of the heterozygote. That temporal variation in selective intensities can also act in the haploid case to maintain polymorphism was shown in part I[1].

An equivalent of spatial variation in selective intensities[5] is also present. Haldane[6], and later Owen[7] and Li[8], noted that a stable polymorphism was possible if the same gene substitution affected the fitness of the two sexes in opposite directions. The difference in the models is that the two sexes of the same genotype see the same immediate fitness; it is their fitness in terms of progeny successfully raised that differs. This two-sex effect on its own cannot maintain polymorphism here.

Environmental cycle length has a relatively mild effect in the models when compared to the effect it has in the models for heritability of progeny number[1]. Fluctuations of the physical environment might cause cycling where genes for viability are involved, but it is hard to imagine many such cycles acting at one time. Although one locus with two alleles is sufficient for the maintenance of sex in a two state fluctuating environment in both haploid and diploid populations, fitness differences have to be relatively large. Additional viability loci enhance the effect of meiosis, and a more likely situation would be polygenic cycling since smaller fitness differences would be necessary.

The biotic environment may provide the required fluctuation to drive these polygenic cycles. The Red Queen[9] theory entailing the gene-for-gene interaction between pathogens[10,11] and parasites[12,13] and their hosts is a likely candidate. Certainly pregnant and lactating females have to be very "fit" to resist parasite build-up and pathogens.

May and Anderson[14] contend that antagonistic coevolution favours sex only when parasites have severe effects on host fitness. Roughgarden's[15] one locus diploid model requires very high fitness values to compensate for the full cost of meiosis. Howard and Lively[16] recently presented haploid models with two loci requiring low fitness values, but interaction with Muller's ratchet[17] was necessary to confer evolutionary stability to sexual reproduction. Sex would only be stable in the longterm if clones were eliminated by this process at least as fast as they were generated by mutation.

Although relatively large fitness differences are necessary in the one locus models presented here, when compared to those in Howard and Lively's models[16], corresponding fecundity is low, 6 or less. It is to be borne in mind that a mild direct effect on female viability can have a drastic indirect effect on the young, e.g. fever can cause abortion and anaemia reduce a female's

ability to nourish or protect her young. Vulnerability of the young to changes in their environment and to predation could greatly amplify the female fitness effect.

Additional loci for viability lower fitness values necessary for meiosis to prevail, and although relatively higher fecundity is entailed, this remains low, below 20 when three loci are involved. Linkage can lower this requisite to below 10 if environmental cycles are the same length. This fecundity requisite is probably also involved in Roughgarden's[15] two locus diploid model, but linkage appears to be ineffectual.

The cost of evolution in terms of fall in population size with changing environment is low in the one locus models compared to other one locus models [1,18], zero for the haploid strain when environmental cycles are shorter than ten generations, and for the diploid strain when cycles are shorter than six generations. Additional loci for viability, with their own specific environmental cycles, allow a reduction in the cost of evolution where cycles are long if recombination levels are high.

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