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Parent–offspring correlation in fitness under fluctuating selection

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Cyclical selection that does not result in gene fixation may maintain a parent–offspring (P–O) correlation in fitness that is, most of the time, not far below one-half. This tends to happen when the following conditions are fulfilled: (i) slow cycling in allele frequencies occurs at many loci independently; (ii) most alleles make only small differences to fitness; (iii) dominance in allelic fitness contribution is rare and overdominance rarer still. In contrast, drastic cyclical selection (dependent on large fitness differences without overdominance), because direction reverses so frequently, may give P–O correlations in fitness that are near zero or even negative. Also, if much fitness variance is maintained by heterozygote advantage, or is caused environmentally, the correlation is reduced in proportion to the ratio of relevant standard deviations.

P–O correlation in fitness that is almost permanently positive may help to explain mate choice in sexually promiscuous species. The most likely source of the multiple independent cyclical systems of selection required is host–parasite relations. To cause evolution of mate choice, and the subsequent manifestations of sexual selection, intermediate rather than extremely long cycles are most effective. A chooser's achievement for the offspring depends on the fitness variance in the sex chosen; in extremely long cycles this variance is on average close to zero.

1. INTRODUCTION

A crucial, almost paradoxical, problem haunting Darwinism from its very beginning is the source and maintenance of heritable variance of fitness. Natural selection, by definition, operates to destroy the unfit. In spite of this, plenty of non-environmental variation exists, and some of it certainly affects fitness. Knowledge of genes and the genetic structure of populations has provided a fairly satisfactory answer to a part of the seeming paradox. It has obviated Darwin's worry about blending inheritance, and it has helped to explain the maintenance of variability of fitness in natural populations at equilibrium. Populations at equilibrium are, of course, not going anywhere under natural selection – but, does this matter? Perhaps they only move once in a while when a beneficial mutation occurs. Unfortunately, even if this is acceptable it leaves a major unresolved puzzle about *heritable* variation in fitness and about sexual selection. Traditional genetic models

of population at equilibrium predict either no heritability of fitness or (with epistasis, recombination and mutation brought in) a very small one (for example, Taylor & Williams 1982; for alternative possibilities, see Mayr 1976; Lande 1975). Thus theory suggests that careful mate choice can achieve little, if anything at all, towards improving the fitness of offspring. A pessimism towards a strictly Darwinian view of sexual selection naturally arises from this point and seems reflected in many current writings: selecting females are assumed to be pursuing resources controlled by males rather than qualities of the males themselves (for example, Emlem & Oring 1977; Halliday 1978).

Assume, for example, a diploid one-locus n-allele model of viability selection with random mating. Let w_{ij} be the viability of an individual of type $A_i A_j$ $(i,j = 1,\ldots,n)$. One can readily show that, in equilibrium, the frequencies $\hat{p}_1,\ldots,\hat{p}_n$ of the alleles $A_1,\ldots,A_n$, satisfy the set of equations:

$$\sum_{i=1}^{n} \hat{p}_i w_{ij} = \overline{W} \quad (j = 1,\ldots,n), \tag{1.1}$$

where $\overline{W}$ is the average fitness in the entire population. Hence, a random female associating her genes with a specific gene A_j, has a chance p_i to pass the allele A_i to her offspring $(i = 1,\ldots,n)$ and thus to provide it with viability w_{ij}. The average viability of such an offspring will be

$$\sum_{i=1}^{n} p_i w_{ij},$$

which at equilibrium $(p_i = \hat{p}_i)$ is equal, as we see, to $\overline{W}$, independently of j. Thus 'choice' of any particular A_j-bearing male gains her nothing.

In other words, despite differences in fitness among individuals, there are no 'good genes' and 'bad genes' at equilibrium. The most fit will have offspring which are not more fit, on the average, than those of other individuals. Hence, a female cannot increase the fitness of her offspring by choosing an appropriate mate.

The theoretical finding is not surprising if we consider the meaning of equilibrium. Indeed, if offspring carrying a specific gene were better off on the average than others, then the frequency of this gene would increase instead of staying constant. Yet theory to the effect that females ought not to care about intrinsic quality in their mates is in disagreement with the apparent strong sexual preference oberved in many natural populations and with the total of energy put at one stage or another into the adornment and performances of sexual selection (Taylor & Williams 1982).

Actually any theory of sexual selection that at all resembles Darwin's original has, whether explicitly expressed or not, a necessity for the assumption of a positive heritability of fitness. Exceptions to this would be the theories of immediate advantage where the preference focuses qualities or attributes that are of significance for brood rearing. For example, selection for health in a mate purely so as to increase the chance that a mate survives through brood rearing and can participate fully in the parental care, would be excepted. So also would be selection of a mate purely for the good territory it holds, promising abundant food for the young. But these exceptions are neither Darwinian (Arnold 1983) nor

are they general enough to account for all cases of sexual selection. Theories resembling Darwin's emphasize *genetic* endowment to offspring, and it is from this that a requirement of positive heritability follows. In some versions the endowment sought is imagined to be a general above-average fitness (Zahavi 1975): fitness, that is, in its everyday sense. In other versions a sexual selection episode starts with an advantage like this or else with some special utilitarian character that is under rapid promotion by natural selection. Later, through the polygyny which the preference has created, extra advantages to the preferred character are added (Fisher 1930; O'Donald 1980; Lande 1981). All such models, which typically assume a constant environment (for example, Maynard Smith 1978; Davis & O'Donald 1976), have in common the difficulty that the variance on which they depend either dies away completely or at least exhausts its heritable component as the population approaches equilibrium. The basis of effectiveness of mate choice is thus eroded and whatever choice persists when it is gone (or reaches the low level that persistent mutation can justify) achieves, if anything at all, only a lowering of overall fitness (O'Donald 1980, p. 32; Kirkpatrick 1982). These results, already seemingly so unsatisfactory for the welfare of the species as a whole, are likely to be exacerbated by yet another cost that lies in the maintenance of the choosiness itself. A terminal state of generally lowered fitness along with a residual variation that has very low heritability seems to us inevitable in any genetic model that confines attention to sexual selection in a constant environment. Following such models species in which strong sexual selection had developed for any reason should show signs of having diminished their abundance. Perhaps many have; we do not discount the likelihood or the dysgenic power of the processes that are intrinsic in these models (for example, O'Donald 1980, p. 32). However, the ubiquity of observations indicating strong sexual selection in stable abundant populations, and the fact that some animal groups have undergone considerable speciation with a mating system involving high sexual selection apparently intact throughout (for example, bower birds), convinces us that a mistake must lie in the basic assumptions of the models. Changing this assumption, although it changes the model, by no means invalidates the previously important ideas (the 'runaway process' of Fisher; Zahavi's 'handicap principle'; how sexual selection may still operate under monogamy; and so on), but it does offer what seems to us a generally deeper and more plausible view.

While it is now widely accepted that environmental change of some sort is necessary to the evolution of sex (for example, Williams 1975; Maynard Smith 1978), we believe that it is also a crucial factor in the evolution of sexual preference. More specifically, redirecting old hints from papers by Haldane (1949) and later writers towards still older problems, Hamilton (1980, see also 1982 and references therein for parallel work) suggested that fluctuations in selection forces owing to host–parasite cycling may be the major basis of the evolution and maintenance of sex. Under the assumptions that lead to this view, a situation may arise where many simultaneous host–parasite cycles affect many gene loci more or less independently. In this study we will show that when such simultaneous cycles occur and are mostly rather slow they can provide a *permanent* source of *heritable variance* of fitness in the population. Thus the cycles, via the heritability

they establish, can serve as a basis for the evolution of sexual preference. This preference in turn, in this model, must be a relatively beneficial influence on health, although whether it pays its costs in this regard is a question we will not address.

As a first step we introduce some notation and analyse the advantage obtained by a choosing female (to take the predominant case) as a function of the heritability and the variance of fitness in the population.

If a correlate of fitness in a potential partner is being assessed during mate choice, it is far more likely to be, at the outset, a correlate of viability than of fertility. Direct correlates of fertility of a male would often be hard or even impossible to assess. Therefore, we assume for present purposes that viability *is* fitness. Assume that such fitness of a given individual at a given time is a function only of its genotype as expressed at the time in question. We are ignoring for the present the possibility of environmental variance in fitness. Assume, for simplicity, that this function is independent of sex. Let X_t be a random variable, proportional to the viability of a random adult individual in generation t ($t = 1, 2, 3, \ldots$). Y_t is the viability of a random newborn offspring in the same generation. By $P(Y_{t+1} | X_t)$ we denote the conditional distribution of the viability of an offspring, born in generation $t+1$ to a parent (say, father) whose viability is X_t.

The regression coefficient of Y_{t+1} over X_t is given by

$$B(Y_{t+1}, X_t) = \frac{\operatorname{cov}(X_t, Y_{t+1})}{\operatorname{var} X_t} = R(X_t, Y_{t+1}) \sqrt{\left(\frac{\operatorname{var} Y_{t+1}}{\operatorname{var} X_t}\right)}, \tag{1.2}$$

where $R(X_t, Y_{t+1})$ is the correlation between the fitness of a father and that of its offspring. Such a correlation is often referred to as the heritability, in the broad sense (Falconer 1960), of fitness in generation t. This value depends on the genetic set-up of the population in generation t, but also on the forces of selection in both generation t and $t+1$.

From (1.2) it follows that a female who succeeds in obtaining a mate of fitness Δ standard deviations above the mean increases the expected fitness of her offspring, on the average, by

$$G(\Delta) = \Delta \sqrt{(\operatorname{var} X_t)}\, B(Y_{t+1}, X_t) = \Delta \sqrt{(\operatorname{var} Y_{t+1})}\, R(X_t, Y_{t+1}). \tag{1.3}$$

Sexual preference of fit mates can therefore be selected for, only under conditions that guarantee the preservation of both substantial heritability and variance of fitness in the population. In the following sections we show that these two conditions are met when the population is simultaneously exposed to many, more or less independent, slow, environmental cyclings, each interacting with a different locus. Such cycling may be typical of a system composed of a host and many parasites (Hamilton & Zuk 1982).

We start by studying one locus effect of fluctuating environment on the heritability of fitness.

2. Heritability of fitness owing to one locus effect in a fluctuating environment. A host–parasite cycle

Let us assume first a simple model of one locus with two alleles A and a in a host population that is afflicted by a parasite (or perhaps two). Assume that owing to some co-evolutionary cycle arising from the parasitism the selection coefficients for genotypes at the A locus are fluctuating. As already mentioned host–parasite systems very readily go into cycles. The cycles could be of a demographic kind first postulated by Volterra, in which case perhaps one allele could be adaptive for low host densities and the other for high. Or the cycles could involve some genotype matching between host and parasite, in which case one allele tends to confer resistance to one parasite or parasite genotype and the other allele to another (Eshel & Akin 1983); or there could be some combination of demographic and resistance cycling. The details need not concern us. We postulate merely that fluctuations in coefficients of selection occur and that in the consequent cycles changes in gene frequency are not so extreme that fixation will occur. Notice, however, that our focus here on the host–parasite relation as a source of fluctuating selection is only due to our belief in its being the most likely source; this is by no means a necessary assumption in the development of the model.

Let the viability of the three genotypes AA, Aa, and aa at generation t be proportional to $1+\alpha_t$, $1+\beta_t$, and $1+\gamma_y$ respectively. Being interested in *relative values* we can assume, without loss of generality that $\gamma_t = -\alpha_t$ (a normalizing factor can always be chosen so that the arithmetic mean of the two homozygote fitnesses will be 1). β_t is then the deviation of the fitness of the heterozygote from the average of those of the homozygote fitnesses. Let $p_1 = p_1^{(t)}$, $p_2 = p_2^{(t)}$, and $p_3 = p_3^{(t)}$ be the relative frequencies of the three genotypes after selection. Being interested in the initial advantage of female preference, we first assume random mating of males and females. The frequencies of the three genotypes AA, Aa, and as among newborn offspring in the next generation will be p^2, $2pq$, and q^2 respectively when

$$p = p^{(t)} = p_1 + \tfrac{1}{2}p_2 = 1-q. \tag{2.1}$$

Let us now calculate the distribution of the offspring fitness Y_{t+1} as a function of the father's fitness X_t. Note that for any father, there is a probability p that an allele passed by the (random) mother is A and a probability q that this allele is a. Hence, if $X_t = 1+\alpha_t$, that is, if the father is of type AA, then there is a probability p that the offspring will be of type AA in which case $Y_{t+1} = 1+\alpha_{t+1}$, and a probability q that the offspring will be of type Aa in which case $Y_{t+1} = 1+\beta_{t+1}$.

In other words:

$$\left.\begin{aligned} P(Y_{t+1} = 1+\alpha_{t+1} \mid X_t = 1+\alpha_t) &= p, \\ P(Y_{t+1} = 1+\beta_{t+1} \mid X_t = 1+\alpha_t) &= q, \\ P(Y_{t+1} = 1-\alpha_{t+1} \mid X_t = 1+\alpha_t) &= 0. \end{aligned}\right\} \tag{2.2}$$

But, $P(X_t = 1+\alpha_t) = p$, hence

$$\begin{aligned} &P(X_t = 1+\alpha_t;\, Y_{t+1} = 1+\alpha_t) \\ &\quad = P(X_t = 1+\alpha_t)\, P(Y_{t+1} = 1+\alpha_{t+1} \mid X_t = 1+\alpha_t) = p_1 p \end{aligned}$$

and in the same way

$$\left.\begin{aligned} P(X_t = 1+\alpha_t;\ Y_{t+1} = 1+\beta_t) &= p_1 q, \\ P(X_t = 1+\alpha_t;\ Y_{t+1} = 1-\alpha_t) &= 0. \end{aligned}\right\} \tag{2.3}$$

Dealing with the other two father types similarly, we obtain the combined distribution of X_t, Y_{t+1} as given by table 1.

TABLE 1. COMBINED DISTRIBUTION OF X_t AND Y_{t+1}

$X_t \backslash Y_{t+1}$	AA $1+\alpha_{t+1}$	Aa $1+\beta_{t+1}$	aa $1-\alpha_{t-1}$	total
AA $1+\alpha_t$	$p_1 p$	$p_1 q$	0	p_1
Aa $1+\beta_t$	$(p_2 p)/2$	$p_2/2$	$(p_2 q)/2$	p_2
aa $1-\alpha_t$	0	$p_3 p$	$p_3 q$	p_3
total	p^2	$2pq$	q^2	1

By straightforward calculation we get the covariance:

$$\begin{aligned} \operatorname{cov}(X_t, Y_{t+1}) = (p_1 q + p_3 p)\,[\alpha_{t+1} + (q-p)\,\beta_{t+1}]\,\alpha_t \\ + [(\tfrac{1}{2} - 2pq)\,\beta_{t+1} - \tfrac{1}{2}(p-q)\,\alpha_{t+1}]\,p_2 \beta_t. \end{aligned} \tag{2.4}$$

As a special case, if fluctuations of the environment affect mainly the homozygotes, leaving the viability of the heterozygote close to the average, we get $\beta_t \approx 0$ and (2.4) becomes

$$\operatorname{cov}(X_t, Y_{t+1}) = (p_1 q + p_3 p)\,\alpha_t \alpha_{t+1} + O(\beta_t, \beta_{t+1}). \tag{2.5}$$

In this case we also have:

$$\operatorname{var} X_t = [p_1 + p_3 - (p_1 - p_3)^2]\,\alpha_t^2 + O(\beta_t), \tag{2.6}$$

$$\operatorname{var} Y_{t+1} = 2pq\alpha_{t+1}^2 + O(\beta_{t+1}). \tag{2.7}$$

Denote by $x = p^{(t-1)}$ the frequency of the allele A among adults in generation $t-1$. We know that the frequencies of genotypes among adults in generation t are:

$$\left.\begin{aligned} p_1 = p_1^{(t)} &= \frac{1+\alpha_t}{W} x^2, \\ p_2 = p_2^{(t)} &= 2\frac{1+\beta_t}{W} x(1-x), \\ p_3 = p_3^{(t)} &= \frac{1-\alpha_t}{W}(1-x)^2, \end{aligned}\right\} \tag{2.8}$$

where

$$W = 1 + (2x-1)\,\alpha_t + 2x(1-x)\,\beta_t. \tag{2.9}$$

Hence

$$\left.\begin{aligned} p &= \frac{x}{W}[1 + \alpha_t x + \beta_t (1-x)], \\ q &= \frac{1-x}{W}[1 - \alpha_t (1-x) + \beta_t x]. \end{aligned}\right\} \tag{2.10}$$

Inserting (2.8) to (2.10) into (2.5)–(2.7), we obtain:

$$R(x_t, Y_{t+1}) = \frac{1}{2}\left[1-\frac{x(2-x)\alpha_t^2}{(1-\alpha_t x)(1-\alpha_t+\alpha_t x)}\right]^{-\frac{1}{2}} \frac{\alpha_t\alpha_{t+1}}{|\alpha_t\alpha_{t+1}|}+O(\beta_t,\beta_{t+1})$$

$$= \pm\frac{1}{2}\left[1-\frac{x(2-x)\alpha_t^2}{(1+\alpha_t x)(1-\alpha_t+\alpha_t x)}\right]^{-\frac{1}{2}}+O(\beta_t,\beta_{t+1}), \tag{2.11}$$

the sign being positive unless the selection forces alter their direction in the generation step in question. Hence, if the cycle is short, the heritability of fitness may be negative during a substantial part of the time. If, on the other hand, cycles are long and the selection forces are weak the term involving α_t^2 becomes negligible and (2.11) may then be written as:

$$R(X_t, Y_{t+1}) = \pm\tfrac{1}{2}+O(\alpha_t^2)+O(\beta_t,\beta_{t+1}), \tag{2.12}$$

the sign being positive most of the time. Moreover, for any $-1 \leqslant \alpha_t \leqslant 1$ and $0 \leqslant x \leqslant 1$ one can readily verify the inequality

$$\frac{x(2-x)}{(1+\alpha_t x)(1-\alpha_t+\alpha_t x)} \leqslant \frac{1}{1-\alpha_t^2}, \tag{2.13}$$

while for selection forces not too drastically high (say $|\alpha_t| < \frac{1}{2}$)

$$\left[1-\frac{\alpha_t^2}{1-\alpha_t^2}\right]^{\frac{1}{2}} \geqslant 1-\alpha_t^2. \tag{2.14}$$

By using (2.13) and (2.14), (2.11) becomes:

$$O \leqslant \tfrac{1}{2}-|R(X_t, Y_{t+1})| \leqslant \alpha_t^2/2+O(\beta_t,\beta_{t+1}). \tag{2.15}$$

Thus, even for such substantial selection forces as when AA is 50 % more viable than aa (implying $\alpha_t = 0.2$), R lies between 0.5 and 0.48.

We conclude that if cycling is slow and if the selection forces, determined by one locus without dominance, are not too drastically high, then the heritability of fitness, as determined solely by that locus, stays close to $\frac{1}{2}$ most of the time.

3. The effect of dominance and overdominance on the heritability of fitness owing to one locus effect

To study the general one-locus effect on fitness-heritability let us return to (2.4) and use the notation

$$\beta_t = \theta_t\alpha_t. \tag{3.1}$$

Here θ_t is the degree of dominance at generation t. $\theta_t = 0$ or $\theta_t = 1$ is the case of full dominance, $0 \leqslant \theta_t \leqslant 1$ is the case of subdominance. As a special case, if $\theta_t = \theta$ is fixed over time (2.4) becomes

$$\frac{1}{\alpha_t\alpha_{t+1}}\operatorname{cov}(X_t, Y_{t+1})$$

$$= p_1q+p_3p+[p_1q+p_3p+(p_2/2)](q-p)\theta+(p_2/2)(q-p)^2\theta^2 \tag{3.2}$$

and by a direct calculation from table 1 we have

$$\frac{1}{\alpha_t^2}\operatorname{var} X_t = [p_1+p_3-(p_1-p_3)^2]-2(p_1-p_3)p_2\theta+p_2(1-p_2)\theta^2, \tag{3.3}$$

$$\frac{1}{\alpha_{t+1}^2}\operatorname{var} Y_{t+1} = 2pq+4(q-p)pq\theta+2pq(1-2pq)\theta^2. \tag{3.4}$$

If the selection forces operating on the locus in question are not so drastic as to shift the genotype frequencies much from the Hardy–Weinberg distribution we have

$$p_1 = p^2[1+O(\alpha_t)], \quad p_2 = 2pq[1+O(\alpha_t)], \quad p_3 = q^2[1+O(\alpha_t)].$$

Therefore

$$\frac{1}{\alpha_t\alpha_{t+1}}\operatorname{Cov}(X_t, Y_{t+1}) = [pq+2pq(q-p)\theta+pq(q-p)^2\theta^2][1+O(\alpha_t)] \tag{3.5}$$

and

$$\frac{1}{\alpha_t^2}\operatorname{Var} X_t = \frac{1}{\alpha_{t+1}^2}\operatorname{Var} Y_t[1+O(\alpha_t)]. \tag{3.6}$$

Hence, from (3.4), (3.5) and (3.6):

$$\begin{aligned} R(X_t, Y_{t+1}) &= \frac{1}{2}\frac{1+2\theta(1-2p)+\theta^2(1-4pq)}{1+2\theta(1-2p)+\theta^2(1-2pq)}\frac{\alpha_t\alpha_{t+1}}{|\alpha_t\alpha_{t+1}|}+O(\alpha_t)+O(\alpha_{t+1}) \\ &= \pm\frac{1}{2}\left[1-\frac{2pq\theta^2}{[1+(q-p)\theta]^2+2pq\theta^2}\right]+O(\alpha_t)+O(\alpha_{t+1}) \end{aligned} \tag{3.7}$$

the sign, as before, being positive as long as sign α_t = sign α_{t+1}.

In the case $0 < \theta < 1$ of subdominance one can use the fact that the expression

$$\frac{2pq\theta_t^2}{[1+\theta(q-p)]^2+2pq\theta^2}$$

obtains its maximum when

$$p = \frac{\theta^2}{2-\theta^2}. \tag{3.8}$$

It then equals $(1+\theta)/2$ and (3.8) can, therefore be written as

$$\tfrac{1}{2} \geqslant |R(X_t, Y_{t+1})| \geqslant \frac{1}{2}\left(1-\frac{\theta^2}{2-\theta^2}\right)+O(\alpha_t)+O(\alpha_{t+1}). \tag{3.9}$$

Moreover, the right side of (3.9) is indeed a lower bound of $|R(X_t, Y_{t+1})|$, obtained for the special frequency (3.8). However, in the case of subdominance, if the cycle is long, the value $2pq$ is likely to be very small most of the time and as follows from (3.7), $R(X_t, Y_{t+1})$ is likely to stay even closer to $\frac{1}{2}$.

The situation is different in the case of overdominance, say $\theta > 1$ where $\alpha_t > 0$ (or $\theta < -1$, where $\alpha_t < 0$). In this case (3.7) still guarantees a non-negative value of $R(X_t, Y_{t+1})$ as long as the selection does not alter its direction. Yet for different

values of p it may obtain any value between zero and a half. The value $R(X_t, Y_{t+1}) = 0$ is obtained for:

$$p = \frac{\theta - 1}{2\theta} = \hat{p}(\theta), \quad \text{say.} \tag{3.10}$$

Values close to $\frac{1}{2}$ are obtained for p close to 0 or to 1. Note, however, that in the case of constant environment $\alpha_t = \alpha$, $\theta = \theta$, $(t = 1, 2, 3, \ldots)$ the frequency $p = \hat{p}(\theta)$ of the allele A is globally stable. Hence when the cycle is long, as assumed before, and the selection forces are changed slowly, p is likely to stay near $\hat{p}(\theta)$.

We find, therefore, sharply divergent expectations according to whether A does or does not imply overdominance. If the cycle is long, then the heritability of fitness owing to a subdominant locus is likely to stay close to $\frac{1}{2}$ most of the time. On the other hand the heritability of fitness owing to an overdominant locus is likely to stay close to zero most of the time.

4. The effect of multicycling: preservation of both heritability and variance of fitness

Until now we have restricted our study to the effect of one locus on the heritability of fitness. Denoting by X_t and Y_t the contribution of this locus to the general fitness of a random adult and a random newborn offspring respectively, we have seen that the fitness heritability $R(X_t, Y_{t+1})$ attains values close to a maximum of half most of the time when the selection forces operating on this locus are moderate, when they are subject to the effect of a slow cycling, and when a polymorophism is maintained without overdominance. Unfortunately one must admit that such a locus can hardly be much of a source for a substantial variance of fitness in the population. Indeed, either the selection forces operating on this locus are very small or one allele must be close to fixation during most of each half of the cycle. In both cases the advantage $G(\Delta) = \Delta R(X_t, Y_{t+1}) \sqrt{(\text{Var } Y_{t+1})}$ of a female attempting to choose a mate Δ standard deviations fitter then the average cannot be large (see (1.3)). The situation appears to be different when we regard, more realistically, the combined effect of many non-synchronized host–parasite cyclings, each having a moderate or minor effect on a different, non-epistatic, locus.

Denote by $X_t^{(i)}$ and $Y_t^{(i)}$ the contribution of the locus i to the general fitness of a random adult and random newborn offspring respectively,

$$X_t = \sum_{i=1}^{n} X_t^{(i)}, \quad Y_t = \sum_{i=1}^{n} Y_t^{(i)}. \tag{4.1}$$

With independent effects of the various loci

$$\text{var } Y_{t+1} = \sum_{i=1}^{n} \text{var } Y_{t+1}^{(i)}. \tag{4.2}$$

With large enough n, this overall fitness variance in the population may not be small even if the component variances owing to individual loci are so. Moreover, with the law of large numbers, the overall variance may stay more or less constant

even when each of the values var $Y_{t+1}^{(i)}$ is changed from one generation to the next. To estimate the multilocus fitness-heritability $R(X_t, Y_{t+1})$, we employ the assumption that the cycles are non-synchronized and loci are non-epistatic; therefore, for $i \neq j$

$$R(X_t^{(i)}, Y_{t+1}^{(j)}) = 0 \tag{4.3}$$

that is, the effect of locus i on the father's fitness, is not correlated with the effect of locus j on the fitness of its offspring. Hence

$$\begin{aligned}\operatorname{cov}(x_t, Y_{t+1}) &= E(\sum_{ij} X_t^{(i)} Y_{t+1}^{(j)}) - \sum_{ij} EX_t^{(i)} Y_{t+1}^{(i)} \\ &= \sum_{ij} \operatorname{cov}(X_t^{(i)}, Y_{t+1}^{(j)}) = \sum_{i} \operatorname{cov}(X_t^{(i)}, Y_{t+1}^{(i)}) \\ &= \sum_{i} \sqrt{(\operatorname{var} X_t^{(i)} \operatorname{var} Y_{t+1}^{(i)})}\, R(X_t^{(i)} Y_{t+1}^{(i)})\end{aligned}$$

and

$$\begin{aligned}R(X_t, Y_{t+1}) &= \frac{\sum_i \sqrt{(\operatorname{var} X_t^{(i)} \operatorname{var} Y_{t+1}^{(i)})}\, R(X_t^{(i)}, Y_{t+1}^{(i)})}{\sqrt{(\sum_i \operatorname{var} X_t^{(i)} \sum_i \operatorname{var} Y_{t+1}^{(i)})}} \\ &= C \frac{\sum_i \sqrt{(\operatorname{var} X_t^{(i)} \operatorname{var} Y_{t+1}^{(i)})}\, R(X_t^{(i)}, Y_{t+1}^{(i)})}{\sum_i \sqrt{(\operatorname{var} X_t^{(i)} \operatorname{var} Y_{t+1}^{(i)})}}\end{aligned} \tag{4.4}$$

where

$$C = \frac{\sum_i \sqrt{(\operatorname{var} X_t^{(i)} \operatorname{var} Y_{t+1}^{(i)})}}{\sqrt{(\sum_i \operatorname{var} X_t^{(i)} \sum_i \operatorname{var} Y_{t+1}^{(i)})}} \leqslant 1. \tag{4.5}$$

Thus, the fitness-heritability $R(X_t, Y_{t+1})$ is equal to the weighted average of the one-locus values $R(X_t^{(i)}, Y_{t+1}^{(i)})$, multiplied by the constant C, the weights of averaging being the geometric means $\sqrt{(\operatorname{var} X_t^{(i)} \operatorname{var} Y_{t+1}^{(i)})}$ of fitness variances in the parent and in the offspring populations.

From §3 and (4.4) it therefore follows that if overdominant loci (even under cycling effect) are the main source of fitness-variance, then overall fitness heritability $R(X_t, Y_{t+1})$ must be close to zero.

If cycles are mostly short, then some of the values $R(X_t^{(i)}, Y_{t+1}^{(i)})$ are small, and some are negative and $R(X_t, Y_{t+1})$ must, again, be small (or even negative!).

However, if the selection forces operating on each locus separately are weak and if cycles are long so that they are changed slowly, one may safely use the approximation

$$\operatorname{var} X_t^{(i)} \approx \operatorname{var} Y_{t+1}^{(i)}. \tag{4.6}$$

In this case (4.5) becomes $C \approx 1$ and from (4.4) we get:

$$R(X_t, Y_{t+1}) \approx \frac{\sum_i \text{var } Y_{t+1}^{(i)} R(X_t^{(i)} Y_{t+1}^{(i)})}{\sum_i \text{var } Y_{t+1}^{(i)}} \tag{4.7}$$

(the left side being always a little bit smaller than the right side owing to inequality (4.5)).

If, in addition, we assume no overdominance, we know that $|R(X_t^{(i)}, Y_{t+1}^{(i)})| \approx \frac{1}{2}$, the sign being positive most of the time, and it follows from (4.7) that

$$R(X_t, Y_{t+1}) \approx \tfrac{1}{2} \tag{4.8}$$

the approximation being also an upper bound.

5. Discussion

The present work concentrates on the limited question of the maintenance of a heritable variance sufficient for the evolution of sexual preference, treating specially the case where preference is based on a polygenic assessment. It has been shown above that contrary to intuition, it is possible to have a selection process that does not exhaust the additive variance that it feeds on. With periodic reversals of the selection at many different loci, and with the timing of reversals uncorrelated, variance in fitness can be maintained at an almost steady value. In this situation the parent–offspring correlation in fitness is steady to the same degree at a value that may approach but does not exceed one half. How closely the parent–offspring correlation approaches $\frac{1}{2}$ depends on, firstly, the average dominance of fitness effects at the loci, with overdominance specially tending to reduce correlations towards zero, and, secondly, the average length of cycles, with long periods making for closer approach to the limit of $\frac{1}{2}$ except in the case where overdominance is common.

It was argued earlier (1.3) that $\Delta\sqrt{(\text{var } Y)}\, R(X, Y)$ shows the expected increment to offspring fitness for a female who obtains a male of fitness which is Δ standard deviations about the mean. Actually a female cannot be expected to gain as much as this for a given amount of effort (represented in Δ), because the phenotypic quality she selects always includes non-heritable effects from the environment. Assume that the female is choosing as a phenotypic quality $Z = Y + \xi$ where ξ is the environmental effect, assumed independent of genotype, and Δ is now in units of standard deviations of Z. Then it can be shown that the gain to a choosing female is $G(\Delta) = \Delta\sqrt{(\text{var } Y)}\, R(X, Y)\ (\sqrt{\text{var } X}/\sqrt{\text{var } Z})$. Since the correcting quotient factor is less than one and the rest of the regression is unchanged, a given amount of effort now achieves less, as would be expected. In other words environmental variation in the perceptible characters that correlate with fitness makes choosiness for those characters and consequent phenomena of sexual selection less likely to evolve.

With this point made, the way is cleared to appreciate a more interesting

diminution of the advantage of choosing. Since, excluding the case of common overdominance, $R(X, Y)$ rises toward $\frac{1}{2}$ as cycles lengthen, it might seem that the power of sexual selection would always be greater for longer mean periods of cycling. However, this neglects the fact that if the cycles are long, gene frequencies almost inevitably go very extreme with consequent reduction of the variances. In fact, in reasonable models $G(\Delta)$ must tend to zero as the mean cycle becomes very long, and this, combined with the fact that if all cycles are of period 2, $G(\Delta)$ must be negative, implies that this function has a maximum for some intermediate median period. With the mixture of cycles occurring in real life, there is therefore some mean periodicity that maximizes the advantage that females can get for their offspring by a given effort of mate selection.

The problem of characterizing cycle mixtures that would maximally promote sexual selection in the above sense will not be pursued here. Such a study would require more detailed specification of a model with attention paid to the nonlinear dynamics that fixes the nature of the cycles, whereas here we have only been concerned with very general assumptions about cycle length.

Although parasite coevolution was suggested earlier to clarify the underlying idea, our result does not require that parasites be the cause of cycling. Other kinds of biotic interaction might cause cycling. So also might fluctuations of the physical environment. Physical examples here would be dry to wet cycles occurring on a time scale of a few to many generations, hot and cool periods similarly, or changes owing to the sun spot cycle. However, such physical fluctuations do not seem to be likely to be as important as the biotic factors for two reasons. First, they do not slacken their pressure when alleles approach fixation. Consequently, it is relatively hard to maintain polymorphisms under regimes of this kind (Hamilton *et al.* 1981). Second, even if non-biotic 'cycles' occur and are buffered against fixation in some way that does not involve overdominance to the degree that destroys the parent–offspring correlation (see Felsenstein (1976) for possibilities), it remains hard to believe that there can be many such cycles operating at one time. Our result involving a parent–offspring correlation that is both steady and substantial requires many cycles co-occurring – in effect, polygenic cycling. If species were subject to just a few drastic cycles it is likely, firstly, that such cycles would have been noticed, and, secondly, that sexual selection based on them would show dramatic switches in preferred traits at particular points of the cycle: again, a phenomenon never observed. In contrast to the potentially fluctuating physical factors, parasites, on the other hand, are very numerous. Every species of substantial body size (for example, any bird or mammal) has many parasites, and a high proportion of them are quite host specific. The characteristics of parasites relevant to co-evolutionary genetical cycling have been reviewed elsewhere (Hamilton 1982).

While any numerous large free-living animal such as a bird or mammal is likely to have a numerous set of parasites, the same becomes less likely to apply as we review primary parasites themselves as potential hosts of hyperparasites and so on down. The very fact of being a parasite tends to ensure a life that is somewhat secluded biotically, although some exception must be made for ectoparasites and species with complex life cycles and free-living stages. Altogether parasites are

likely to experience fewer more drastic cycles or changes of selection that are dependent on genotypes of their hosts. If the parasite's life cycle is fairly long, as usual in macroparasites, this may well select for maintenance of sex, but it will not be conducive to sexual selection occurring on the lines discussed here. These predictions seem to fit roughly with the facts of parasite sexuality (for a recent discussion see Bell (1982)).

Returning to hosts in general, another prediction is that we expect sexual selection to be most highly developed in the part of the host range where host-specific parasites are most numerous. This would normally be the centre of the range. Examples of this are easily found. Unfortunately they are also open to explanations not involving parasitism (Eshel 1979). Far more testing of the ideas in this paper would be a direct search for genetical evidence of polygenic cycling.

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