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COMMENTARY

ON THE MODELLING OF SEXUAL SELECTION*

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A review of GENETIC MODELS OF SEXUAL SELECTION, by *Peter O'Donald; Cambridge University Press, Cambridge and New York;* $32.50. xii + 250 p; ill; index. 1980.

INTRODUCTION

THE FIRST chapter of this detailed treatment of the conceptually difficult topic of sexual selection is an admirable account of the history of thought on the subject from Darwin to the present. The second is a thoughtful review of the natural history of sexual competition and mate choice. We can heartily recommend these chapters to those with even a casual interest in sexual selection. The rest of the book will be understood only by those willing to pay really close attention to both verbal and mathematical representations. Our efforts were not as rewarding as we had hoped they would be.

THE ASSUMPTION OF DISCRETE PHENOTYPIC CLASSES

Chapter Three deals analytically with purely phenotypic models of sexual selection and introduces assumptions that we believe to have unfortunate consequences for later genetic models. Basic to this modelling is the postulate that a population is made up of discrete phenotypic classes, each with specified mating advantages or mate-choice inclinations. This is convenient for genetic analysis, because discrete phenotypes can be assigned to postulated single-locus genotypes, but some justification besides convenience should have been provided. The reader should be given some idea of whether such simplified assumptions are realistic summaries of information or are chosen purely for convenience in the absence of information. If the author's convenience is the main factor, he should explain why more realistic approaches are not feasible. If he believes that his assumptions are good approximations to reality, he should review some evidence in justification of this position.

We will illustrate this point with what might be regarded as an approach more realistic than O'Donald's for problems con-

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sidered in Chapters 4 through 6. The most basic requirement is that females show some variation in receptiveness to males of different phenotypes, indexed perhaps by one variable, say d for darkness. We might observe a large number of encounters of females with males of different d-values, some of which result in mating. For each d we may be able to calculate a probability $P(d)$ that an encounter with a female will result in a mating. Could a distribution of $P(d)$ (e.g., Fig. 1) form the basis for analytical modelling? Unfortunately not. Such a distribution would not enable us by itself to deduce male genotype frequencies at equilibrium, even if we had complete understanding of the genetics of character d. The $P(d)$ curve may be the average of many such curves, one for each female in the population. The collection of component curves might carry adequate information, but an observed average would not. Put in another way, we need to know how the $P(d)$ curve is composed.

One simple possibility is that the curve is composed of two nearly separate distributions, darker and lighter, which could be adequately approximated by assuming discrete phenotypic classes (Fig. 2). The distribution of mating success could be envisioned as arising in two ways. Perhaps half the females accept either phenotype, with a probability of ½ per encounter, and half accept only dark, again with a probability of ½ per encounter (Fig. 3a). Or perhaps half accept only light, with a probability ½ per encounter, and half accept only dark, with a probability 1 per encounter (Fig. 3b). It is clear that the equilibrium phenotype frequencies will be different in the two cases: In

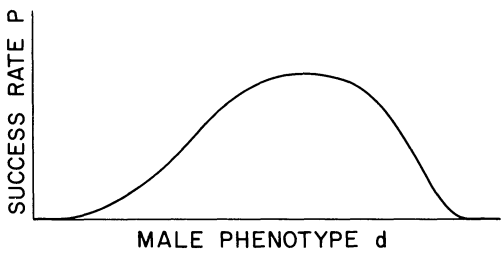

FIG. 1. A POSSIBLY REALISTIC RELATIONSIP OF MALE MATING SUCCESS TO PHENOTYPE

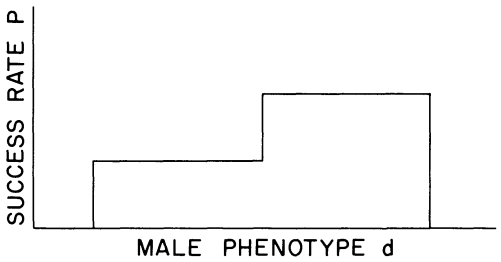

FIG. 2. ANALTYICALLY CONVENIENT APPROXIMATION TO FIG. 1

3a there must be a surplus of dark, but in 3b dark and light will be of equal frequency.

Thus to build a model to predict genotype frequencies we need to specify the composition of the $P(d)$ distribution curve. One possibility is to follow O'Donald and suppose that α, γ and $1 - \alpha - \gamma$ of the females mate only with dark, only with light, and at random, respectively, and then choose α and γ to get a best fit to observed data (Fig. 4). This gives O'Donald's model 4.1. Parameters such as α and γ may be artificial, but an analysis along the above lines indicates why we may nevertheless have to put up with them. It also gives a clearer picture of how they may be related to data that may be gathered.

O'Donald makes no attempt to justify his parameters on the basis of biological observations. It is not enough to use statistical methods to find best values for the parameters. One needs to look for patterns of behavior that may actually create the different kinds of females postulated. Cooke's massive Snow Goose study (Cooke, 1978; Cooke, Finney, and Rockwell, 1976) provides the best information to date on assortative mating. It is unfortunate that O'Donald did not refer to any of this work. It is ideally suited to his assortative mating models of Chapter 9, because the white-blue color dimorphism quite likely depends on two alleles at a single locus. Cooke found that, as a rule, birds with white parents choose white mates, those with blue parents blue mates, and birds from mixed parentage choose either color. Such observations could provide an excellent opportunity to generate *a priori* values for O'Donald's assortative mating parameters a and c, which could then

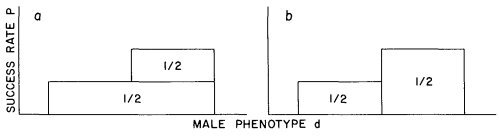

FIG. 3. TWO POSSIBLE INTERPRETATIONS OF THE FEMALE PREFERENCE BEHAVIOR MODELED IN FIG. 2

be tested against data. This would be more meaningful than his *a posteriori* statistical techniques.

Reliance on one-locus polymorphisms as evolutionary explanations for the phenomena of sexual selection leads O'Donald to some unfortunate positions in treatments of frequency-dependent mating success. In the original and best-known examples, males of a given strain in mating competition with another strain do best when they are in the minority. Such rare-male advantages can even accrue (occasionally) to reduced-viability mutant forms in competition with normals. O'Donald understandably sees such preferential mating with mutants as maladaptive. He also implies that any generally prevalent mate-choice mechanisms that would lead to this kind of experimental result must also be maladaptive. Evidently he would deny that a normal mate-choice mechanism can be tricked into giving maladaptive results with a highly unusual male phenotype in the special environment of an experimental vessel. Yet he sees nothing implausible in assigning a consistent preference for a low-fitness male to a substantial minority of a population of females. In his models, a reduced frequency of a type of male is a reduced supply in relation to some prevalent level of demand by females who prefer that type.

To us it seems unlikely that all the diverse phenomena of mate choice can be successfully modeled as single-locus polymorphisms. Frequency dependence is sometimes positive, with minority phenotypes at a special disadvantage, and it is often absent or experimentally undetectable. Minority types are especially liable to rejection when there are evolutionary reasons to expect rejection, as when a male is from a subspecies to which the females' subspecies has evolved the beginnings of ethological isolation (Ehrman, 1970; 1972).

Close conformity of experimental data to one-locus Mendelian models will of course be unlikely if phenotypes are strongly affected by environmental effects, such as learning. Mating behavior of many animals can be influenced by prior experience (e.g., the Snow Goose observations cited above). This is best established for Drosophila in the work of Pruzan, Ehrman, Perelle, and Probber (1979), unfortunately not available to O'Donald when he wrote the manuscript. These observations make Ehrman's original proposal of individual habituation, as an explanation for rare-male advantage, more believable. O'Donald has his greatest difficulty when the data show male types to have maximum advantage at intermediate frequencies. He suggests that such observations must be attributed to sampling variation among tested groups of females. This is refuted by the well-replicated work of Ehrman, Anderson, and Blate (1977), which shows mating success for males with a special amylase allele to be consistently higher at a frequency of about 0.25 than at either higher or lower frequencies.

A GENERAL EQUILIBRIUM CONDITION

In Chapters 4 through 7 O'Donald presents a variety of mathematical models which track the changing frequencies of a pair of alleles a and A at a single locus under various assumptions of female mating preference for the three male genotypes. Both polygynous and monogamous mating systems are considered; mating preferences are fixed or frequency (of the a-A genotypes) dependent; and the a-A genotypes are

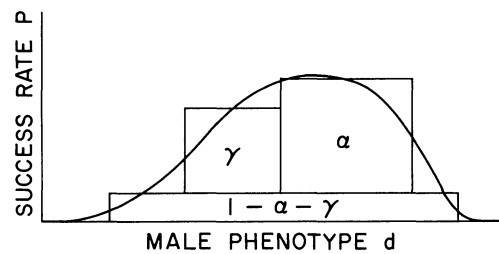

FIG. 4. ASSUMPTION UNDERLYING O'DONALD'S MODELLING

assumed to have or not to have different (natural) selection advantages in males or in both sexes. A prevalent and important assumption underlying these four chapters is what O'Donald calls *random* preferential mating: there is no correlation between the preference a female exercises and her a–A genotype. Thus the mating preferences of females are not genetic and not heritable. O'Donald later departs from this assumption. In Chapter 8, mating preferences are made genetic, and computer simulations are used to track the evolution of mating preferences. Not surprisingly, at equilibrium, there is some assortment between preference alleles and the genotypes which they prefer. In Chapter 9, models of preference with some assortment are examined.

Although O'Donald, in Section 1.5, makes clear the distinction between random and assortative preferential mating, there may be some confusion in a reader's mind in Chapter 7, in which models of random preferential mating in monogamous populations are discussed. Confusion arises because models are fitted to Arctic Skua data in which there is strong evidence for assortative mating of the intermediate phenotype. Indeed, O'Donald uses this evidence to check his estimate of the mating preference parameters obtained from data of breeding times. With such assortative mating, how can his random preferential mating models apply? In particular, the formula for p_* in Section 7.4 surely cannot apply to the Arctic Skua. It would seem that Chapter 9 is the place to discuss these data. [In fact, more recent data (O'Donald, 1980) have cast doubt on this evidence for assortative mating.]

The calculations required to work through the various models in Chapters 4 through 7 are often tedious and, we feel, repetitious. The reader may suspect there to be a few simple general findings which would save some work and expose more clearly what the different models have in common. A book that presents a systematic account of a number of related models has a duty to identify any such general findings, not only to help the reader to reach an immediate understanding but to be of a service to those who wish to further refine the models. Such general results also help to lay bare the extent to which conclusions depend specifically on the assumptions used.

In each model in these four chapters, changes in genotype frequencies are determined by male and female genotype fitnesses. These fitnesses, which are in general the net result of the action of natural selection and mating preference, are sex- and frequency-dependent. In this case one needs a modification of the standard selection result (e. g., Crow and Kimura, 1970, p. 179–80). We will assume, as does O'Donald, that the fitnessess have been normalized so that, at each set of genotype frequencies, the average genotype fitness is unity in each sex.

Assume that alleles $A_1, A_2, \ldots A_n$ are segregating at a single locus in a diploid sexual population. Let P_{ii} be the frequency of the homozygote A_iA_i and $2P_{ij}$ the frequency of the heterozygote A_iA_j enumerated at the zygote stage. Let the genotype fitnesses be $1 + s_{ij}$ in females and $1 + t_{ij}$ in males where s_{ij} and t_{ij} may depend on the frequencies; always $s = \Sigma P_{ij}s_{ij} = \bar{t} = \Sigma P_{ij}t_{ij} = 0$. Let $P_i = \Sigma P_{ij}$ be the current frequency of A_i. Then, at mating, the frequency of A_i in the female and male gamete pools will be

$$\begin{aligned} f_i &= \Sigma_j P_{ij}(1 + s_{ij}) = P_i(1 + s_i) \\ m_i &= \Sigma_j P_{ij}(1 + t_{ij}) = P_i(1 + t_i) \end{aligned} \qquad (1)$$

where $s_i = \Sigma_j P_{ij} s_{ij}/P_i$ and $t_i = \Sigma_j P_{ij} t_{ij}/P_i$ are the average fitness excesses of A_i in the two sexes. The new frequency of A_i in the next zygote generation is then

$$P'_i = (f_i + m_i)/2 = P_i + P_i(s_i + t_i)/2. \qquad (2)$$

The change in gene frequency is

$$\Delta P_i = P_i(s_i + t_i)/2 \qquad (3)$$

and the equilibrium condition is that, for each i, either $P_i = 0$ or $s_i + t_i = 0$.

O'Donald's many formulae for Δp, of which 4.1.3 is the first, can all be derived from Equation (3) by simply working out the fitness excesses s_i and t_i. His method using mating tables and changes in genotype frequencies is more cumbersome and less desirable because it depends on an assumption of random mating between male and female gamete pools (which is what *random* mating preferences imply) whereas the derivation of (3) requires no assumption on the mating structure.

As an example, we use (3) to derive his

4.1.3. In this first model of complete mating preference $A = A_1$ is dominant over $a = A_2$, and proportions α and γ of the females prefer the dominant and recessive phenotypes as mates; the remaining mate at random. If genotypes AA, Aa, and aa have zygote frequencies u, v, and w then one easily derives the mating frequencies P_T of 4.1.1 for different male genotypes. For example,

$$P_T(AA) = \alpha u/(1 - w) + u(1 - \alpha - \gamma).$$

Setting this equal to $u(1 + t_{11})$ we get

$$t_{11} = [w(\alpha + \gamma) - \gamma]/(1 - w).$$

Since A is dominant, $t_1 = t_{11} = t_{12}$ and all $s_{ij} = 0$. Letting p be the frequency of A, we get 4.1.3 immediately from (3):

$$\Delta p = \tfrac{1}{2} p t_1 = \tfrac{1}{2} p [w(\alpha + \gamma) - \gamma]/(1 - w). \quad (4)$$

If we wish to know something about genotype rather than gene frequencies we have to make some assumtions about the mating structure. If we assume random mating between male and female gamete pools then the next generation zygote genotype frequencies are found from

$$P_{ij}' = (m_i f_j + m_j f_i)/2. \quad (5)$$

We observe that even with random mating these are not in Hardy-Weinberg proportions unless $m_i = f_i$. We will have Hardy-Weinberg equilibrium frequencies if $s_i = t_i = 0$ for all alleles with positive frequency. In particular, if one sex, say female, has genotype-independent fitness, then all $s_i = 0$ and at equilibrium t_i must also vanish and we get Hardy-Weinberg frequencies. This is true for all of O'Donald's models except for those with natural selection acting in both sexes. We believe that our equation (3) provides more immediate and generally applicable understanding than O'Donald's many separate treatments.

The assumption of random mating between gamete pools does not hold in O'Donald's Chapter 8, in which female preferences are determined at a second locus. Even if there is a free recombination between the loci, there will be some nonrandom assortment between genotypes at the two loci and a female's preference genotype will correlate somewhat with her a-A genotype. O'Donald's method of obtaining his formulae in Chapters 4 through 6 leave him no way of knowing which actually apply in Chapter 8 and which are invalid, and in fact he makes some errors. The various formulae he obtains for Δp all follow from (3) and so apply to the models of Chapter 8. But the formulae he obtains for equilibrium gene frequencies usually depend on Hardy-Weinberg equilibrium and do not apply. For example, 4.1.4, $w_* = \gamma/(\alpha + \gamma)$, follows from 4.1.3 and therefore applies to Chapter 8, but 4.1.5, $q_* = \sqrt{\gamma/(\alpha + \gamma)}$, does not. O'Donald is aware of the genotype assortment in Chapter 8, but does not seem to realize that it invalidates any application of his formulae for p_*, for on pages 177 and 183 he uses these formulae to predict the equilibrium frequency of A. In fact, because of the assortment, levels of homozygosity will be increased above those predicted by Hardy-Weinberg. Thus, in the models of Chapter 8, 4.1.5 will overestimate q_* and hence underestimate $p_* = 1 - q_*$. O'Donald observes (p. 179) that this formula underestimates the calculated value, but ascribes the discrepancy to a slow approach to equilibrium.

This confusion on gene and genotype frequencies pervades Chapter 8 and may confuse the unwary reader. For example, page 207 states:

> . . . .[I]f A starts at a high frequency, it may sometimes be lost completely. When this occurs, the equilibrium frequency predicted by the mating preferences that have evolved is always less than the actual frequency. The actual frequency therefore follows the predicted frequency down to zero.

The point is that current mating preferences predict not *gene* frequencies but *genotype* frequencies. It would be entirely possible to have the frequency of A greater than the p_* predicted by Chapter 4 or 5, and yet have the frequency of AA so small that the frequency of A would increase rather than decrease.

THE NATURE OF THE EQUILIBRIA IN CHAPTER 8

In Chapter 8, O'Donald has female mating preferences genetically determined and, using various preference models, he tracks the coevolution of genes for the preferred character at the A locus and the mating preferences at the B locus. He always has two al-

leles *a* and *A* at the character locus and two or three alleles at the preference locus. Sometimes the *A* locus goes to fixation, but in the more interesting cases a nontrivial equilibrium configuration is attained, the frequencies of the various *B* alleles being just right to hold the *A* alleles in balance. Even with free recombination between the loci, there is usually linkage disequilibrium at such points.

The gene frequency equilibria have special properties and need careful discussion. They are certainly not stable in the usual mathematical sense. In fact, any time there is a nontrivial equilibrium in such a situation, it is always a point on a one (at least) dimensional curve of possible equilibrium points. Interior equilibria are never isolated. The curve is stable but points on it are not. If the system is perturbed it will return to the curve, but not likely to the same point. It is especially noteworthy that there is nothing to stop the system from wandering along the curve, and no doubt it will do so in response to other sorts of selection or to genetic drift. Equilibrium curves of this type arise routinely in the modelling of genetically determined mate preference and their evolutionary stability has been studied by Lande (in press) and Kirkpatrick (in press).

If the system starts away from the equilibrium curve, it will move to a point on the curve determined by the starting position. O'Donald points out the importance of initial frequencies in determining the final equilibrium, but he comments on page 184 that sometimes the initial frequencies make little difference. In mathematical terms he is saying that a small piece of the equilibrium curve may have a large "basin" of attraction, but he sums up by saying that a range of initial frequencies give rise to the same equilibrium. This is misleading, for the probability is always zero that two initial frequencies give rise to exactly the same equilibrium. One must look carefully at the structure of the set of trajectories along which the system can move.

As a simple example, suppose there are two alleles, *b* and *B*, at the preference locus, and suppose *A* and *B* are completely dominant. Suppose *BB* and *Bb* females mate only with *AA* and *Aa* males, and *bb* females mate only with *aa* males. This is a "C" model in O'Donald's symbolism, rather simpler than those which he considers. Assume also a free recombination between the two loci. There are then 9 possible genotypes in the population, and in each generation the system is specified by a point in an 8-dimensional simplex (with 9 vertices) giving the zygote genotype frequencies (a 2-dimensional simplex is a triangle). We have analyzed the structure of the set of equilibrium points by computer. They all lie on a one-dimensional curve connecting the vertex at which the frequencies of *A* and *B* are both one to the vertex at which both these frequencies are zero. At each point on this curve the frequencies of *A* and *B* are identical, as might be expected by the symmetry of the situation. However, the amount of assortment between the alleles *A* and *B* generally decreases as the frequencies of these alleles increase along the curve. To generate such values we started a simulated population in Hardy-Weinberg and linkage-equilibrium at equal frequencies of *A* and *B*, and tracked the population for about 100 generations. Gene frequencies did not change by more than 10^{-10}, but the genotype frequencies changed markedly. These seemed to settle down after 100 generations, although, as O'Donald points out, it is difficult, using only the computer, to be sure you are close to equilibrium because ultimate rates of change of frequency are very slow but will persist for a long time.

For each point on the equilibrium curve there is a 7-dimensional surface in the simplex consisting of all points from which the system will move to that equilibrium. Each trajectory is contained in one of these 7-dimensional surfaces. As an example, in Table 1 we tabulate 4 starting points (p_A, p_B) taken at Hardy-Weinberg and linkage-equilibrium and indicate the value of p_*, the equilibrium proportion of *A* and *B*, for each. Clearly the chance that two nearby points in the simplex are on the same 7-dimensional surface is negligible, although as O'Donald observes, these surfaces sometimes "open out" (like flowers) and attract a large portion of the simplex towards a small portion of the equilibrium curve.

In Section 8.7, O'Donald introduces the action of natural selection in males at the *A*

TABLE 1

Dependence of equilibrium frequency of A and B on starting frequencies

B-females choose A-males and b-females choose a-males.

Starting frequencies		Equilibrium frequency of both A and B
p_A	p_B	p_*
0.5	0.1	0.043
0.1	0.5	0.610
0.05	0.2	0.275
0.2	0.05	0.027

locus. This is quite an interesting situation because we now have the possibility of an equilibrium at which a (natural) selective disadvantage is balanced by a favorable mating preference. Indeed, using his "C" models, he obtains equilibrium polymorphisms corresponding to a wide range of selective values. It is important to observe that these polymorphisms have the same "quasi-stable" character as those obtained in the absence of natural selection. Each equilibrium polymorphism is a point on an entire curve of equilibrium points, and only the curve is stable.

O'Donald does not make this clear. In fact in Fig. 8.13 he indicates one equilibrium point for each value of the selective coefficient of A without mentioning that the same s, with different starting configurations, can lead to different points. In fact, he does not say what starting point he used to obtain the equilibrium points of Fig. 8.13.

To see what the equilibrium curve looks like for a fixed value of s we have programmed O'Donald's "C" model with A and B dominant. We assume BB and Bb females mate only with AA and Aa males, and bb females mate at random. We assume a viability penalty of $s = 0.2$ against the AA or Aa male genotype. We started with a variety of values of p_A and p_B in Hardy-Weinberg and linkage-equilibrium and ran the population for 100 generations with free recombination between the loci. In Fig. 5 we mark the final a-A genotype frequency of the point. The equilibrium curve (at least its projection in the a-A triangle) follows quite closely the Hardy-Weinberg curve from the point $p_*(A) = 0$ to $p_*(A) = 1$. The equilibrium frequency of B lags behind that of A, and the percentage assortment decreases as $p_*(A)$ increases.

THE BALANCE BETWEEN NATURAL SELECTION AND MATE PREFERENCE

The possibility of a balance between natural and sexual selection is one of the more controversial ideas in the theory of sexual selection. In simplest genetic terms we have the possibility of an equilibrium polymorphism with two alleles, one favored by natural selection, the other by sexual selection through mate preference. There are many examples of variation in some male character, the more extreme forms apparently favored by the female, but almost certainly of some natural disadvantage to the owner. An interesting question is whether one can plausibly model a situation in which some of the variation can be heritable in an equilibrium configuration. Certainly in such a model it must be true that two alternative male phenotypes have the same net fitness; otherwise, because of the heritability, we could not be at equilibrium.

In Section 8.7 O'Donald constructs several such models and shows that equilibrium polymorphisms can result in a wide range of

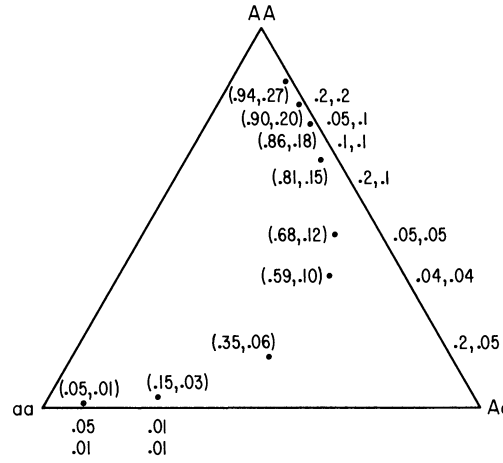

FIG. 5. PROJECTION ON THE a-A TRIANGLE OF THE EQUILIBRIUM CURVE FOR $s = 0.2$

Next to each point is marked the equilibrium values of $p_*(A)$ and $p_*(B)$. Beneath or to the right of each are recorded the starting values of p_A and p_B.

circumstances. As we discussed in the last section, there are some difficulties with the stability of these polymorphisms, but nevertheless, the models are important and interesting. O'Donald restricts himself to one mode of natural selection, what we will call *viability selection*. There is another mode, what we will call *fecundity selection*, which also needs to be discussed in any general treatment of sexual selection. This is important because the two modes of selection have different properties with regard to possible types of equilibria, but they are often confused with one another in general discussions.

To be simple and precise, suppose we have two male phenotypes, R for reserved and E for extravagant. E males are better at attracting females but suffer some selective disadvantage. This disadvantage is a *viability* penalty if selection acts on males before mating so that an E male zygote has a smaller probability of reaching maturity than an R male zygote, but females who mate with E males get as many offspring as those who have R mates. The disadvantage is a *fecundity* penality if selection acts after mating so that E and R males have the same chance of reaching maturity, but a female who chooses an E male gets fewer offspring than one who chooses an R male. Fecundity penalties could result if E males were less fertile than R males, but in practice the penalty is more likely to be related to the nature of E-ness. Some examples of fecundity selection might be:

(1) Dominant and satellite males at a lek. E males are those who win positions in the center, and R males are satellites. If it is more risky for a female to get to the center of the lek and obtain an E male than accept a quick and early insemination from a satellite male, then any such disadvantage (possibility of injury, or poorer nest-site, or other late-breeding penalty) can be considered fecundity selection against the E male phenotype.

(2) Territoriality, resource competition, and the sexy son. The E males attract more females, which therefore have more crowded territories, reduced resource availability, and lower nesting success. Weatherhead and Robertson (1979) have observed this situation in blackbirds and suggest that the economic penalty suffered by these females is offset by the advantage of having E males as sons.

(3) Territorial defense and offspring genotypes. Bartholomew (1970), in a study of pinniped polygyny, has suggested that higher pup mortality (incidental to male combat) for females breeding in overcrowded central territories is offset by the superior genetic contribution of a dominant mate.

(4) Male parental care. Grant (1980) studied reproductive success and mate choice in the johnny darter and found variation in the agressiveness with which males at their nests respond to other males, as measured, for example, by the distance of pursuit. The E males are overly aggressive and suffer higher levels of egg predation and nest loss, but appear to attract more females to spawn.

If the E-R dichotomy has a heritable component, it is difficult to see how there could be an equilibrium polymorphism representing a balance between fecundity selection and any genetically determined mate choice. If, at such an equilibrium, the E and R males have the same fitness, so that the E male's increased mating success is exactly offset by the decreased number of offspring per mate, then the female who chooses E has a lower fitness than one who choose R and the mate preference "locus" cannot be at equilibrium.

Taylor and Williams (unpub.) have constructed a general model in which a "dynamic" balance between fecundity selection and sexual selection is maintained by a constant genetic force which transmutes E-ness into R-ness. For example, E males may be those with fewer deleterious mutations at any of N loci, and the force is genetic mutation. Or E males are those with favorable gene combinations across loci and the force is recombination. Such models seem capable of balancing fecundity disadvantages of 4 to 5 per cent.

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