Polymorphism in Genes That Influence Sperm Displacement

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ABSTRACT

Paternity of offspring of multiply inseminated females is in many organisms highly skewed, with an advantage generally going to the male that most recently mated. Variation in sperm competitive ability can result in strong natural selection, and one expects that a gene that offers an advantage in sperm displacement would, all else being equal, be rapidly fixed, leaving low equilibrium levels of variability in sperm competition. However, empirical studies have demonstrated genetic variation in sperm displacement, begging the question of how this variation can be maintained. Here we develop a population genetic model to find conditions that maintain polymorphism in alleles that influence sperm displacement. We consider a one-locus model in which allelic variants have pleiotropic effects on fecundity and mating ability in addition to sperm displacement. This model can admit more than one stable polymorphism, and we find conditions for protected polymorphism. Induced overdominance is not necessary for stable polymorphism. These results have direct bearing on the observed variation in the ability of resident sperm to defend against displacement.

DARWIN identified sexual selection as a special form of differential reproductive success that includes strict competition among males resulting in no benefit to the species as a whole. Sperm displacement appears to be such a case, wherein male gametes compete for successful fertilization in multiply inseminated females, first noted by Aristotle (PAYNE and KAHRS 1961). Based on empirical literature, models of sperm displacement consider the displacing ability as a property of the diploid genotypes of the males (PROUT and BUNDGAARD 1977). Here we extend the models with a particular focus on mechanisms that allow maintenance of variation in sperm displacement.

This study was prompted by the findings of variation in two components of sperm displacement among lines of *Drosophila melanogaster* that were homozygous for different extracted chromosomes from natural populations (CLARK *et al.* 1995). In this survey, the 152 extracted-chromosome lines were tested for "offense" sperm displacement, defined as their ability to displace sperm previously deposited by males bearing the recessive markers *cn; bw*. The same extracted lines were also tested for their ability to resist displacement by a subsequent mating by a *cn; bw* male (the "defense" component). The statistic *P2*, defined as the fraction of offspring sired by the second male (BOORMAN and PARKER 1976), was used as the metric for the offense component. The distribution of *P2* has a mode at nearly 1 and a tail of less fit lines with lower *P2* values (Figure 1). This is a pattern one would expect under mutation-selection equilibrium, with selection driving *P2* to its maximal value, and rare mutations at a small number of loci resulting in deleterious, low *P2* values. CLARK *et al.* (1995) used *P1*, the fraction of offspring sired by the first male in defense tests, as a statistic to quantify the defense component. The distribution of *P1* among extracted lines cannot be easily explained in terms of mutation-selection balance, because it appears to have a mode at the lowest fitness, and a tail of higher fitness (higher *P1*) values. To explain the observed pattern of defense values by mutation-selection equilibrium, one would have to propose a very large number of loci capable of mutating to deleterious alleles. A more likely explanation for polymorphism is some form of balancing selection possibly mediated by pleiotropic effects on other components of fitness.

The phenomenology of sperm displacement in Drosophila suggests several types of pleiotropic effects that may be associated with differences in sperm displacement. By identifying some of the molecules that play a role in these processes, the nature of pleiotropic effects can be very clearly seen. A class of small peptides known as male accessory gland proteins (Acps) are present in seminal fluid and are transmitted to the female during mating. One of the Acps, known as sex peptide, has been implicated in more than one aspect of Drosophila reproduction, including remating behavior (CHEN 1984) and egg laying behavior (CHEN 1984; HERNDON *et al.* 1995). Unspecified components of seminal fluid, possibly Acps, also have an influence on female longevity (CHAPMAN *et al.* 1995). The antagonistic effect of seminal fluid components on male and female life histories motivates a population genetic model of the phenomenon.

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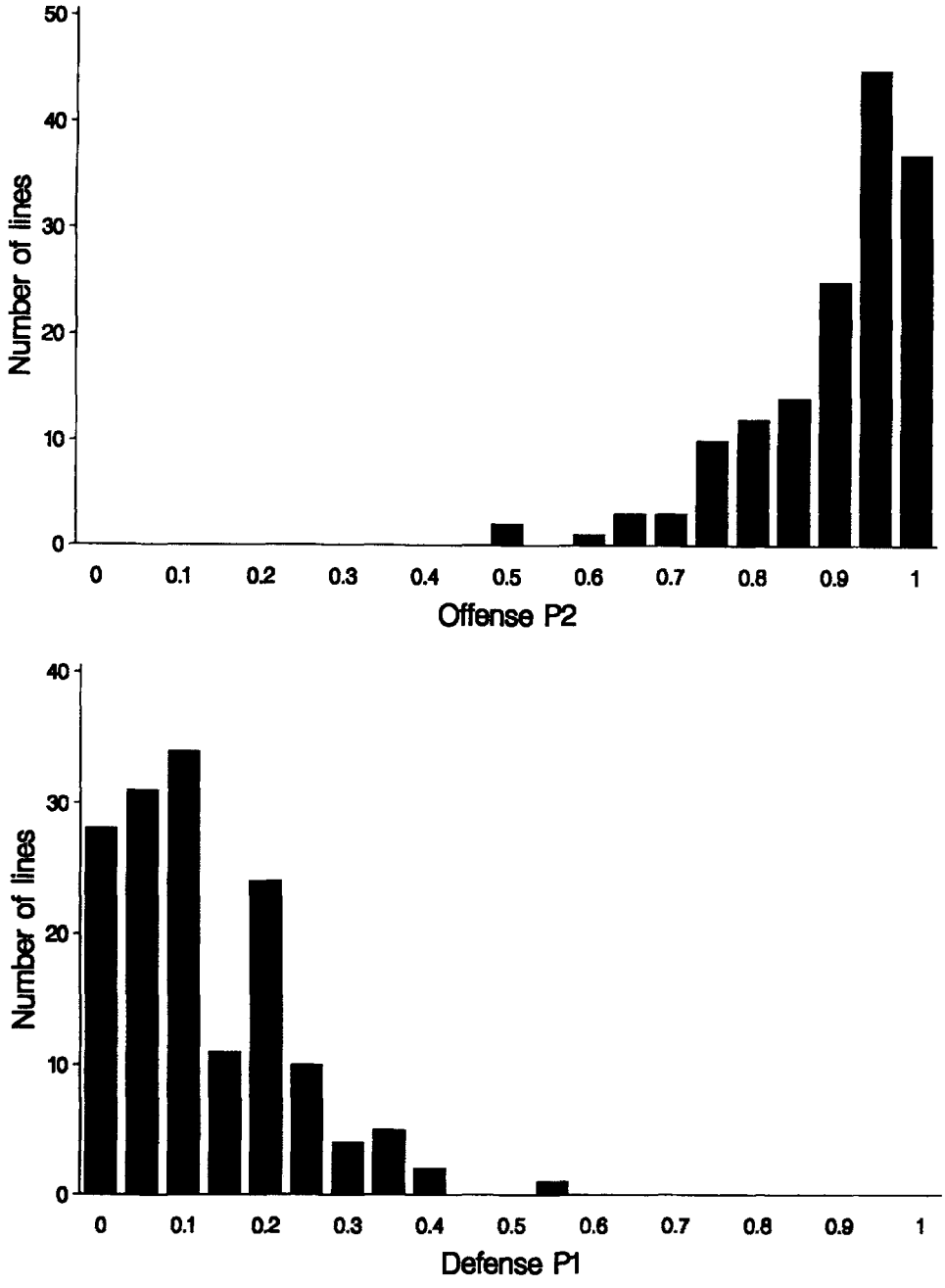


FIGURE 1.—Distributions of observed sperm displacement parameters among 152 chromosome-extracted lines of *D. melanogaster* (from CLARK *et al.* 1995). Defense (*P1*) and offense (*P2*) parameters are defined in the text.

While the effects of accessory gland proteins on mating and oviposition behavior have been clearly demonstrated, the role that Acps play in sperm displacement is less clear. CLARK *et al.* (1995) implicated Acps in the phenomenon of sperm displacement by scoring DNA variation in genes encoding seven male accessory gland proteins in the lines whose sperm displacement was quantified. The *Acp* genes were all found to exhibit polymorphism, but more importantly, the variation exhibited allele-specific differences in the defense component of sperm displacement (but not the offense component). Lines of Drosophila selected on the basis of life history traits also exhibited differences in defense components of sperm competition (SERVICE and FALES 1993). The most likely mechanism whereby Acps influence sperm displacement is by alteration of sperm retention and storage. One of the models for sperm competition in Drosophila suggests that there is inefficient storage of sperm immediately after mating (NEWPORT and GROMKO 1984), and the phenomenon of sperm precedence involves factors that determine when and how sperm are stored.

Males may gain an advantage over other males in sperm competition by inducing a female to avoid remating. Clear evidence supports the claim that Acps are sufficient to cause transiently reduced receptivity of females following mating (SMITH 1956; CHEN 1984; KALB *et al.* 1993). Remating behavior is somewhat more complicated and includes factors such as sperm depletion (MANNING 1962, 1967; GROMKO and PYLE 1978; FUKUI and GROMKO 1989; GROMKO and MARKOW 1993), depletion of some other substance in seminal fluid,

time of male-female interaction, temperature and, in laboratory studies, the use of anesthesia (GROMKO *et al.* 1984). Note that it is the male–male competition that gives those males who gain control over the female's reproduction an advantage.

CLARK *et al.* (1995) observed no correlation between offense and defense components of sperm displacement. If the phenomenon were entirely explained by volumetric displacement, this result is unexpected. However, we know that females retain only about $^1/_5$ of the sperm transmitted during mating. This implies that each mating can fill the sperm storage capacity of a female, and hence variation in defense cannot be purely volumetric. The excess of sperm transfer suggests that the significant associations of Acps with defense (and not offense) implies that Acps mediate sperm competition by differential sperm storage. The lack of an effect on offense components makes it clear why no correlation between the two sperm displacement components is expected.

The complexity of sperm competition necessitates that only a few key aspects be considered in a model. Based on the observations of the consequences of multiple mating in Drosophila, we have chosen to focus on three pleiotropic attributes: sperm displacement, mating success, and induced fecundity. There may be several physiological mechanisms whereby variation in sperm displacement is mediated, including amount of sperm transferred, sperm size, or sperm storage ability (PARKER 1993). When a male Drosophila mates several times in succession, he becomes less successful at displacing resident sperm, suggesting a negative relation between frequency of mating (mating success) and sperm displacing ability (GROMKO *et al.* 1984). Observations of sex peptide's effects demonstrate that seminal fluid proteins may influence a female's subsequent fecundity (CHEN *et al.* 1988), so this factor is incorporated into the model. A key result of this model is that polymorphism in sperm displacing ability is made much more likely by allowing these pleiotropic effects.

MATERIALS AND METHODS

Definitions and assumptions: We assume that females carry sperm from at most two males. This is consistent with several allozyme studies of multiple mating in the field that have given very low estimated frequencies of triple matings (COBBS 1977). d_{ij} is the fraction of offspring sired by genotype ij when a female mates twice and the second mated male has genotype ij. d_{ij} is therefore the offense component for genotype ij, and $^1/_2 \le d_{ij} \le 1$. Empirically, we have found that defense components of sperm displacement are also important, and the model can be parameterized instead in terms of defense parameters $\sim d_{ij}$, indicating the fraction of offspring sired by the first male. Both parameterizations produce precisely the same recursion for the model. The reason is any offspring not fathered by the second male must be fathered by the first male, so offense is 1 − defense. This encompasses complex processes such as antagonistic pleiotropy between offense and defense, which as noted above, CLARK *et al.* (1995) did not find. It should be emphasized that to allow independent variation in offense and defense requires a structurally different model such that the female's fecundity depends on the sperm displacement of both males. The same genetic locus may also have pleiotropic effects on mating success and on the induced fecundity of the mated female. Mating success of male genotypes *AA*, *Aa*, and *aa* are m_{AA}, m_{Aa}, and m_{aa}, respectively, and the respective induced fecundities are f_{AA}, f_{Aa}, and f_{aa}.

The recursion: The model has one locus with two alleles, *A* and *a*. Let x be the frequency of *A* in female gametes and y be the frequency of *A* in male gametes. The frequencies of genotypes *AA*, *Aa*, and *aa* (denoted p, h, and q) after random union of gametes are xy, $x + y - 2xy$, and $1 - x - y + xy$, respectively. Assume that there is no selection on female gametes, so that the array of alleles present in the female's gametes is determined by the genotype frequencies of females. The frequency of gametes contributed by each male depends on the parameters f_{ij} and d_{ij}, as depicted in Table 1. Combining the male and female gametes, we get the recursion

$$\begin{aligned}
\bar{w}\tilde{p} &= p^2 m_{AA}^2 f_{AA} + phm_{AA}m_{Aa}[f_{AA}d_{AA} + f_{Aa}(1 - d_{Aa})] \\
&\quad + pqm_{AA}m_{aa}[f_{AA}d_{AA} + f_{aa}(1 - d_{aa})], \\
\bar{w}\tilde{h} &= h^2 m_{Aa}^2 f_{Aa} + phm_{AA}m_{Aa}[f_{Aa}d_{Aa} + f_{AA}(1 - d_{AA})] \\
&\quad + hqm_{Aa}m_{aa}[f_{Aa}d_{Aa} + f_{aa}(1 - d_{aa})], \\
\bar{w}\tilde{q} &= q^2 m_{aa}^2 f_{aa} + pqm_{AA}m_{aa}[f_{aa}d_{aa} + f_{AA}(1 - d_{AA})] \\
&\quad + hqm_{Aa}m_{aa}[f_{aa}d_{aa} + f_{Aa}(1 - d_{Aa})], \qquad (1)
\end{aligned}$$

where $\bar{w}$ is the sum of the right sides. The above gives the recursion from zygotes to gametes. To complete the generation, we have random union of gametes from males and females

$$\begin{aligned}
p' &= (\tilde{p} + \tilde{h}/2)\,(p + h/2), \\
h' &= (\tilde{p} + \tilde{h}/2) + (p + h/2) - 2(\tilde{p} + \tilde{h}/2)\,(p + h/2), \\
q' &= 1 - p' - h'. \qquad (2)
\end{aligned}$$

Additive case: Let the sperm displacement parameters d_{AA}, d_{Aa}, and d_{aa} be $^3/_4 + s$, $^3/_4$, and $^3/_4 - s$, where $0 \le s \le {}^1/_4$. Also let the respective mating successes and fecundities be 1, $(1 + m)/2$, m, and 1, $(1 + f)/2$, f. This additive pattern produces directional selection in each component and allows the system to be described with just three parameters.

Dominance case: Because induced overdominance can retain a polymorphism in pleiotropic systems, we need to consider the case of complete dominance in the same direction for all components. Let the sperm displacement parameters d_{AA}, d_{Aa}, and d_{aa} be D, D, and d. Also let the respective mating successes and fecundities be m, m, 1, and f, f, 1. Substituting into the general recursion gives

$$\begin{aligned}
\bar{w}\tilde{p} &= pm[mf(p + h) + q(1 + k)] \\
\bar{w}\tilde{h} &= hm[mf(p + h) + q(1 + k)] \\
\bar{w}\tilde{q} &= q[q + m(f - k)(1 - q)], \qquad (3)
\end{aligned}$$

where $k = Df - d$ and $\bar{w}$ is the sum of the right sides. The generation is then completed by uniting male and female gametes as in the above cases.

Numerical simulations: To explore the opportunity for multiple equilibria and to verify the algebraic results, numerical simulations were performed. For each case that was tested, 10,000 random parameter values were generated, and for each, 100 random starting conditions were used. Each was run until the change in allele frequency across generations was $<10^{-12}$, and numerical behavior was compared to that predicted from the analytical expressions evaluated for each numerical case.

TABLE 1

Offspring from doubly mated females

First male	Second male	Frequency	Male gamete contribution		
			AA	*Aa*	*aa*
AA	*AA*	$p^2 m_{AA} m_{AA} F_{AA}$	*1*	*0*	*0*
	Aa	$phm_{AA} m_{Aa} f_{Aa}$	*1-*d_{Aa}	d_{Aa}	*0*
	aa	$pqm_{Aa} m_{aa} f_{aa}$	*1-*d_{aa}	*0*	d_{aa}
Aa	*AA*	$hpm_{Aa} m_{AA} f_{AA}$	d_{AA}	*1-*d_{AA}	*0*
	Aa	$h^2 m_{Aa} m_{Aa} f_{Aa}$	*0*	*1*	*0*
	aa	$hqm_{Aa} m_{aa} f_{aa}$	*0*	*1-*d_{aa}	d_{aa}
aa	*AA*	$pqm_{aa} m_{AA} f_{AA}$	d_{AA}	*0*	*1-*d_{AA}
	Aa	$hpm_{aa} m_{Aa} f_{Aa}$	*0*	d_{Aa}	*1-*d_{Aa}
	aa	$q^2 m_{aa} m_{aa} f_{aa}$	*0*	*0*	*1*

RESULTS

General case: The general case presents algebra that is too formidable to solve all interior equilibria and determine their stability, so instead we will begin by determining the conditions under which the two boundaries are guaranteed to be unstable. Such a condition is also known as a protected polymorphism. We find these conditions by determining the Jacobian stability matrix for the linearized recursion and evaluating the eigenvalues of this matrix at the fixations. The conditions for instability of the $p = 1$ fixation is that the leading eigenvalue of this matrix is >1. In a like fashion, the condition for instability of the $q = 1$ fixation is obtained. Taken together, these conditions for invasion of A when rare and for invasion of a when rare provide expressions for protected polymorphism

$$\frac{f_{aa} m_{aa} + [(1 - d_{aa})\ f_{aa} + d_{Aa} f_{Aa}]\, m_{Aa}}{2\, f_{aa} m_{aa}} > 1 \quad (4)$$

and

$$\frac{f_{AA} m_{AA} + [(1 - d_{AA})\ f_{AA} + d_{Aa} f_{Aa}]\, m_{Aa}}{2\, f_{AA} m_{AA}} > 1. \quad (5)$$

These conditions are useful for illustrating a few properties of the model. For example, if all $f_{ij} = 1$ and all $m_{ij} = 1$, the only selection component that affects the polymorphism is determined by the sperm displacement parameters d_{ij}. In this case, overdominance in d_{ij} is necessary and sufficient for polymorphism. In the case with all $d_{ij} = {}^1/_2$, so no sperm displacement occurs, the fitness of each genotype is simply determined by the product of the male mating success and fecundity. As expected, the condition for protected polymorphism is simple overdominance in net fitness: $f_{Aa} m_{Aa} > f_{aa} m_{aa}$ and $f_{Aa} m_{Aa} > f_{AA} m_{AA}$. Finally, if there is sperm displacement, but the second male fathers ${}^3/_4$ of the offspring regardless of genotype ($d_{ij} = 0.75$ for all ij) and all f_{ij} equal, overdominance in m_{ij} is sufficient to protected polymorphism.

Additive case: The additive model can sustain a stable polymorphism with just three free parameters by having some degree of opposition among the selection components (antagonistic pleiotropy). Solving the eigenvalues of the stability matrix gives the same results as direct substitution into the formulae for the eigenvalues of the general case. A polymorphism is protected in the additive model if

$$\frac{(4m + 4)\, s + (f + 3)\, m + f + 11}{16} > 1,$$

$$\frac{(-4fm - 4f)\, s + (11f + 1)\, m + 3f + 1}{16fm} > 1. \quad (6)$$

If m and f are both less than one, and $s > 0$ or if $m > 1$, $f > 1$ and $s > 0$, then these conditions are not both satisfied and fixation is guaranteed. Some degree of antagonistic pleiotropy is required to stabilize the polymorphism. On the other hand, existence of selection in opposing directions in different components does not guarantee a polymorphism.

Dominance case: When we do a standard stability analysis, calculating the Jacobian stability matrix, we find that the characteristic polynomial, evaluated at the fixation $(p, h, q) = (1, 0, 0)$ is $(1 - \lambda)\lambda^2$, so the linearized system does not give any information about the stability. The other fixation gives an eigenvalue of $[(Df - d + 1)\, m + 1]/2$. Letting $k = Df - d$, the condition for instability of this fixation is $(k + 1)\, m > 1$.

To determine stability of the fixation of allele A, we infer the recursion from the ratio of the dominant and recessive phenotypes before and after selection. The phenotype frequencies are as follows:

$$\bar{w}(\tilde{p} + \tilde{h}) = (1 - q)\, m[mf - q(mf - (1 + k))],$$

$$\bar{w}\tilde{q} = q[m(f - k) + q(1 - m(f - k)]. \quad (7)$$

Defining $s = \tilde{q}/(\tilde{p} + \tilde{h})$, we get

$$s' = \frac{s^2 + (f - k)\, ms}{(k + 1)\, ms + fm^2}. \quad (8)$$

Evaluate at $s = 0$ and obtain the eigenvalue $(f - k)/$

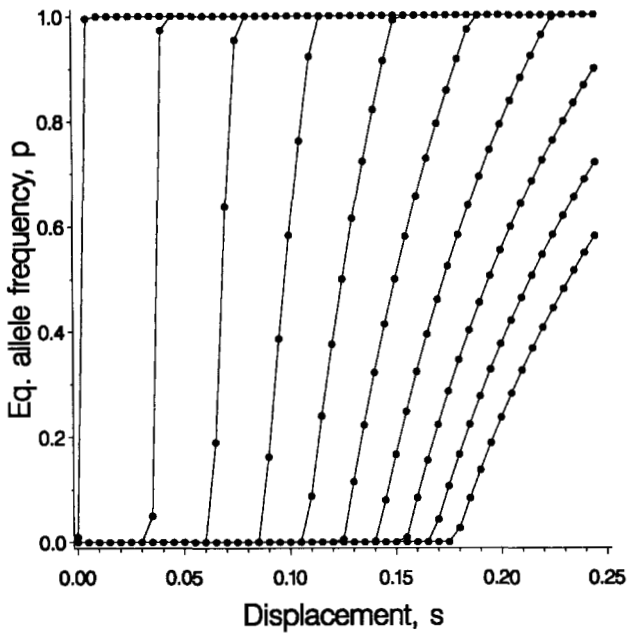


FIGURE 2.—Stability of the polymorphism is global in the additive model. Each dot represents the value of sperm displacement parameter s and the equilibrium allele frequency. Values connected by lines have the same value of induced fecundity, which had values 1, 1.1, 1.2, . . . , 1.9 (moving left to right). Male mating success was the same in all runs ($m_{if} = 1$).

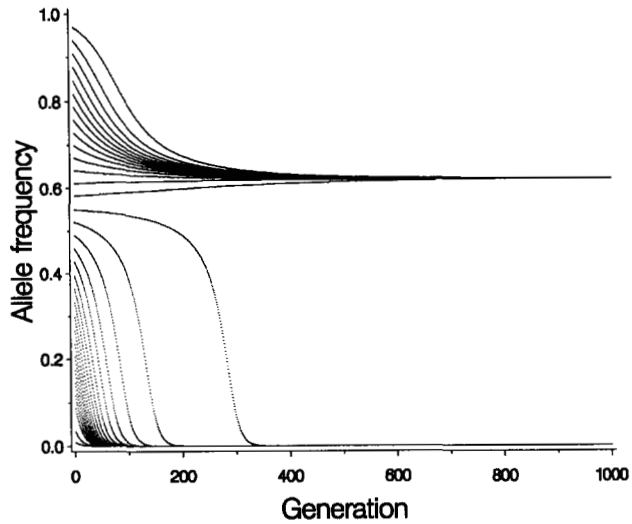


FIGURE 3.—Stability is not always global. With parameters $d_{ij} = 0.64$, 0.95, and 0.18; $f_{ij} = 0.70$, 0.87, and 0.55; and $m_{ij} = 0.22$, 0.15, and 0.43 for genotypes *AA*, *Aa*, and *aa*, respectively, both the fixation at $p = 0$ and a polymorphism with allele frequency 0.619 are locally stable.

fm. The point $s = 0$ is the same as $(p, h, q) = (1, 0, 0)$, so this fixation is unstable if $(f - k)/fm > 1$. The dominant case will sustain a protected polymorphism if

$$k > (1 - m)/m \quad \text{and} \quad k < f(1 - m). \qquad (9)$$

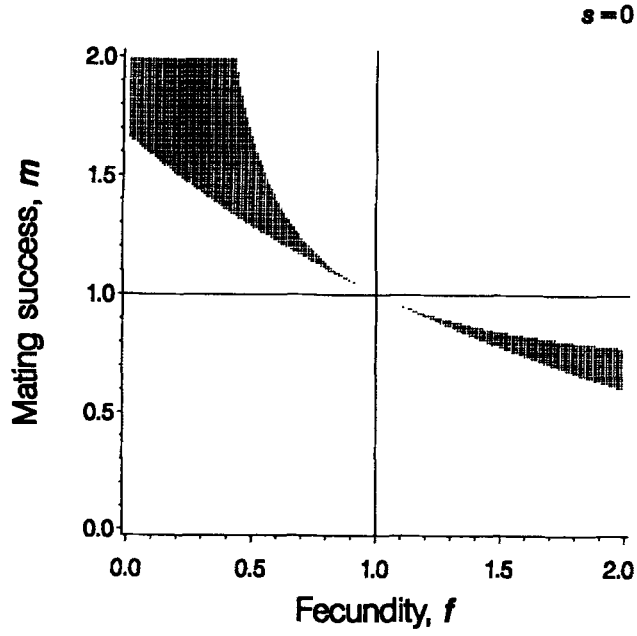
Numerical simulations: Numerical results suggest that in both the additive and dominance cases whenever the conditions for protected polymorphism were met, there was in fact global stability of a unique interior equilibrium. The allele frequency at the unique equilibrium is determined by the three parameters as shown in Figure 2. Global stability was not a property of the general model, and in fact numerical cases were found having two simultaneously stable equilibria with an unstable equilibrium at ~0.57 (Figure 3).

The model is capable of retaining polymorphism over a large range of parameter values. Of the 10,000 random parameter sets, 3171 maintained a stable polymorphism in the general case, while 377 cases had a stable polymorphism in the additive case, and 109 of the 10,000 cases with a dominant pattern maintained a stable polymorphism. Figure 4 illustrates part of the parameter space that admits a stable polymorphism and shows that in the case of the additive model the opportunity for polymorphism is reduced by sperm displacement.

Figures 5 and 6 depict the parameter space for a stable equilibrium in the dominance case. Figure 5 shows specific values of f and m and the application of the conditions on k that imply that $1 < m < 1/f$ or $1 > m > 1/f$. Figure 6 gives the whole f, m space that is necessary for stable equilibrium. This demonstrates that antagonistic pleiotropy is necessary but not sufficient, since additional limits are required. It can be shown that there are always combinations of D and d inside $k = Df - d$ that can be included in any point in the space. These include cases where $d = D$ so that $k = d(f - 1)$. Since the conditions cannot be met when $k = f - 1$ (see Figure 5), D must be <1, and the biologically determined lower limit is $D = {}^1/_2$. The fact that $D = {}^1/_2$ can be included shows that an equilibrium can occur due to antagonistic pleiotropy between f and m when there is simple sperm mixing.

DISCUSSION

CLARK *et al.* (1995) observed extensive variation in both offense and defense components of sperm displacement, and the results were consistent with effects being mediated by the diploid male genotype. In more recent work, K. HUGHES (unpublished data) found significant levels of dominance variance in sperm displacement, consistent with segregating genetic variation for this fitness component. As noted in the Introduction, the distribution of defense *P1* values is difficult to reconcile with a simple model of mutation-selection balance, suggesting that there are pleiotropic effects associated with variation in defense. The theoretical results presented here propose a plausible mechanism whereby this variation in fitness could be maintained in a natural population. We emphasize that these results apply only to induced fecundity effects after either the first or second mating, and that a model with independent fecundity effects over two or more matings is more complicated and is not considered here. PROUT and BUNDGAARD (1977) presented the population genetics



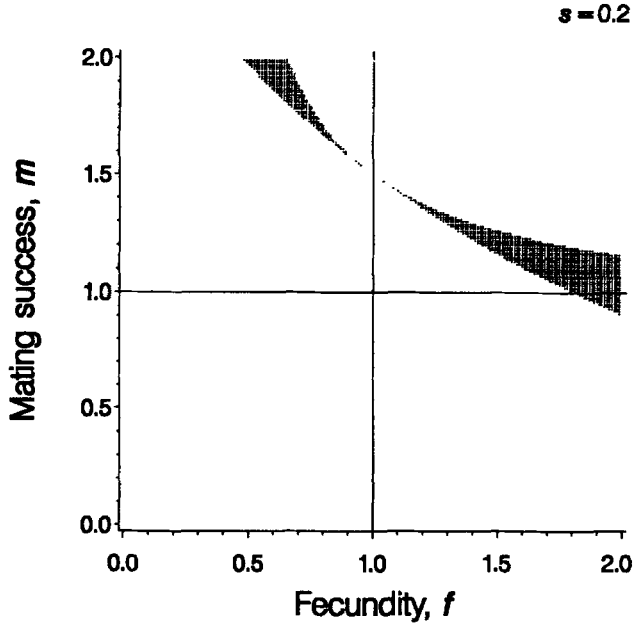
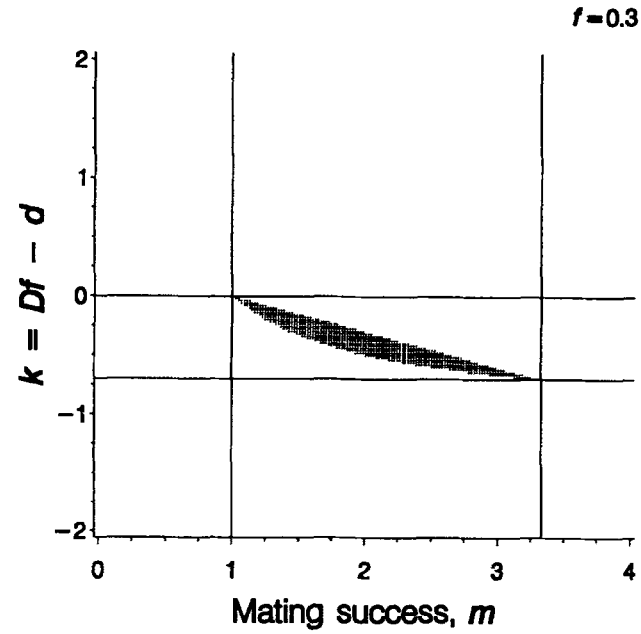


FIGURE 4.—Parameter space that admits polymorphism in the additive model. In the top figure, $s = 0$, so there is no sperm displacement, while in the bottom figure, $s = 0.2$.



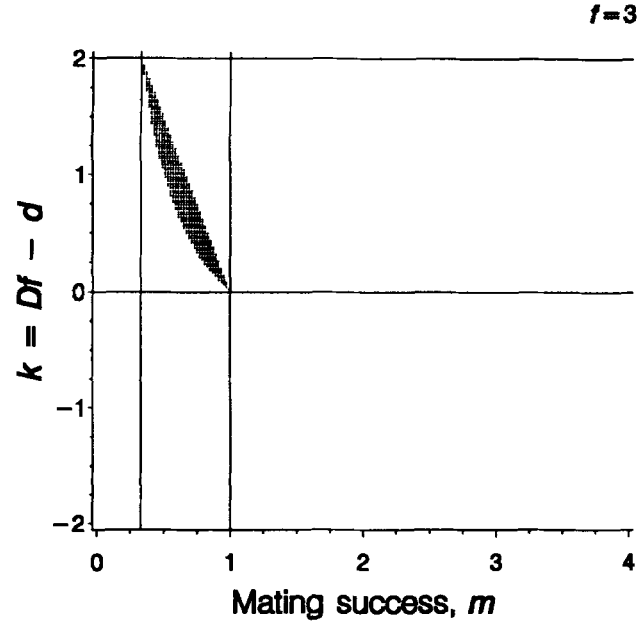


FIGURE 5.—Two numerical examples showing the proportion of the parameter space (shaded) admitting polymorphism in the dominance case. Note that the condition is $(1 - m)/m < k < f(1 - m)$, where $k = Df - d$. It follows that, as shown in the figure, the region corresponding to $m > 1$ (top) admits polymorphism only if $m < 1/f$ ($m < 1/0.3 = 3.33$), and the region with $m < 1$ (bottom) admits polymorphism only if $m > 1/f$ ($m > {}^1/_3 = 0.33$). Note that when $m = 1/f$, $k = f - 1$, as shown by the reference lines at $k = -0.7$ (top) and $k = 2$ (bottom).

theory for the case of sperm displacement determined by a single gene, and they found that a stable polymorphism in a sperm-displacement locus can be maintained if the sperm displacement parameters are either overdominant or nontransitive. In particular, for the three genotypes at one diallelic locus, the displacement parameters are nontransitive if *AA* outcompetes *Aa*, *Aa* outcompetes *aa*, but *aa* outcompetes *AA*. In our model with pleiotropic effects on fecundity and on mating success, heterozygote advantage can also yield a stable polymorphism, but in this case induced overdominance is neither necessary nor sufficient to guarantee poly-

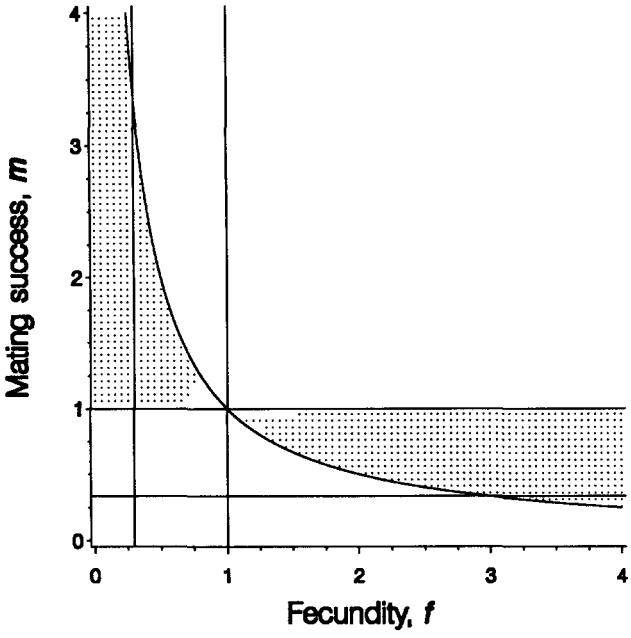


FIGURE 6.—The shaded area shows the general equilibrium conditions for f and m, and includes the numerical examples from Figure 5. Reference lines are drawn for $m = {}^1/_3$ (corresponding to $f = 3$ and $k = 2$) and for $m = 3.33$ (corresponding to $f = 0.3$ and $k = -0.7$).

morphism. The "reason" antagonistic pleiotropy can maintain variation, apart from induced overdominance, is the unusual frequency-dependent properties of this system, arising from the interactions of two male genotypes. Because the outcome of this interaction depends on the relative frequencies of the pairs of genotypes, some parameters give the system a rare-type advantage, leading to a stable polymorphism.

Although the details of the molecular mechanism by which sperm displacement is accomplished remain unclear, it is useful to consider pleiotropic consequences of alternative possible mechanisms. Seminal proteins may influence sperm displacement either by directly altering the capacity of sperm to affect fertilization, or by altering the female's ability to store the sperm. In the first case, the proteins of the first and second male interact directly with the sperm to result in loss of motility, incapacitation, or death. This would require that the proteins somehow act on the other male's sperm more than one's own sperm, a possibility that exists because Drosophila transfer seminal fluid before sperm transmission (FOWLER 1973). Evidence that seminal proteins incapacitate sperm that are resident in the female's storage organs came from experiments by HARSHMAN and PROUT (1994) demonstrating reduced fecundity of mated females by subsequent mating with spermless males that transmit seminal proteins. There is also suggestive evidence that seminal proteins affect the female's reproductive tract in such a way as to influence her ability to store sperm from one or the other male. For example, sex peptide influences a female's remating latency and egg laying rate (CHEN *et al.* 1988). Seminal proteins may increase a male's gametic success if they cause females to store sperm in a way that resists displacement by subsequent matings.

The suggestion that accessory gland proteins may be involved in the process of sperm displacement (CLARK *et al.* 1995) raises the possibility of using molecular evolutionary approaches to determine how sperm displacement evolution occurs. Seminal proteins exhibit high levels of polymorphism (COULHART and SINGH 1988) and of interspecific divergence (THOMAS and SINGH 1992), indicating that either natural selection is driving the divergence of these genes, or that they are unusually mutable. Results of AGUADÉ *et al.* (1992) show that the pattern of polymorphism and divergence is not consistent with neutrality, supporting the idea that natural selection drives the divergence and/or polymorphism. Further analysis of the molecular mechanisms for action of genes encoding seminal proteins is likely to clarify the role of pleiotropic effects on different components of reproduction and may provide an explanation for the patterns of variation seen at the population level.

Sperm competition occurs only when females multiply mate, and a long standing debate in evolutionary biology centers on the issue of what is the advantage to females who mate multiply? HARVEY and BENNETT (1985) suggested that by mating with many males, females provide an opportunity for sperm competition to occur, thereby producing offspring bearing genes of the male with more competitive sperm. It has been proposed that this system is analogous to Fisher's runaway sexual selection as developed by KIRKPATRICK and others (KIRKPATRICK 1982, 1985). CURTSINGER (1991) did a two-locus model of this situation, with one locus influencing female remating behavior and a second locus influencing sperm displacement. He found that the sperm displacement locus evolves very quickly to fixation (or equilibrium), and after it attains equilibrium there is no longer any advantage to females mating multiply. Thus, the opportunity for sperm competition to drive the evolution of female remating behavior appears to be limited. It should be emphasized that this situation is different from classical runaway sexual selection theory, because in the case of sperm displacement, the trait will evolve independently in the absence of female choice. It is possible that, in the past, female choice could have played some role, but it is certainly not necessary.

KELLER and REEVE (1995) try to revive female choice as an explanation for multiple mating, explaining that all that is necessary is a means of retaining polymorphism in sperm displacement. They argue that if polymorphism could be retained and linkage disequilibrium with female behavior genes were present, then sperm competition could continue to drive female behavior. The question we consider here is what maintains the residual polymorphism. For this, KELLER and REEVE (1995) propose that either mutation-selection balance or female choice is operating in a displacement cycling

system, which (they argue) would occur if the three genotypes were nontransitive in their displacement effects, as in the rock-scissors-paper game. However, as noted above, PROUT and BUNDGAARD (1977) analyzed a diploid sperm displacement model that showed that nontransitivity in the three diploid genotypes can result in a stable equilibrium that does not cycle, even as it approaches the equilibrium point, although we have found that the three allele model can exhibit cycles. In the two-allele model proposed here, where the stable polymorphism could be due to antagonistic pleiotropy, there is no sign of cycling behavior, and because it goes rapidly to equilibrium, we would expect that adding a second female-behavior locus would not result in invasion and fixation of a female remating allele.

Results from the dominance model demonstrate that an equilibrium due to antagonistic pleiotropy can only be obtained if there is at least some sperm mixing. We conjecture that this principle should apply to models with more complex genetics. Our model is clearly testable, and experiments are underway to quantify pleiotropic effects on mating success, induced fecundity and other aspects of our extracted chromosome lines.

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