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Multiple mating, sperm competition and meiotic drive

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Abstract

Most discussions of 'sperm competition' have ignored the potential for competition among the different sperm genotypes present in the ejaculate of a single male. Rivalry within ejaculates may limit cooperation among the members of an ejaculate when they compete with sperm produced by other males. A gene that gains an advantage in competition within an ejaculate (a segregation distorter) may increase in frequency even if it is associated with significant costs to organismal fitness. Therefore, selection will favor genes expressed in males that suppress competition within ejaculates. This may explain why sperm function is largely controlled by the diploid genotype of the male progenitor, rather than by the genotypes of individual haploid sperm. Females who mate with multiple males reduce the relative advantage of a segregation distorter whenever the distorter impairs the competitive effectiveness of the ejaculates in which it occurs. If the distorter is associated with costs to organismal fitness, selection will favor female mating behavior that reduces the distorter's equilibrium frequency. Competition within ejaculates may thus be one reason why females choose to mate with multiple males.

'Sperm competition' has been defined as competition within a single female between the sperm from two or more males for the fertilization of her ova (Parker, 1970; Birkhead and Møller, 1992). In effect, sperm competition is seen as a continuation of intermale conflict within the female reproductive tract. This definition ignores the possibility of competition among the haploid sperm produced by a single diploid male. If a sperm's phenotype were determined by the diploid genotype of its male

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progenitor, without influence from the particular genes that the sperm itself received, there could be no competition within an ejaculate and natural selection would act on the behavior of entire ejaculates to increase the reproductive success of the diploid male. An ejaculate would behave as if it were an 'organ' of the male body, with the possibility of a complex division of labor among its constituent parts. If, on the other hand, a sperm's fate were determined by its own genetic constitution, natural selection could favor traits that conferred a competitive advantage on certain sperm while reducing the competitive effectiveness of the ejaculate as a whole. An ejaculate might behave as if it were a stampeding herd of spermatozoa, each sperm for itself in the race for an egg. Cooperation within an ejaculate would still be possible, as among any group of relatives, but this would not be an automatic assumption.

Differences in competitive ability among the haploid genotypes present in the ejaculate of a single male are known as 'meiotic drive' or 'segregation distortion'. Unambiguous examples of meiotic drive prove that a sperm's fate is not always independent of its own genetic constitution. The segregation distortion (*SD*) system of *Drosophila melanogaster* (Temin et al., 1991; Lyttle, 1993) and the *t*-haplotype system of mice (Silver, 1985, 1993) are examples of autosomal meiotic drive in which most + sperm fail to function in the ejaculates of $SD/+$ or $t/+$ males. The Sex Ratio (*SR*) system of *D. pseudoobscura* is an example of X-linked drive in which most offspring of *SR* males inherit the *SR*-bearing X chromosome from their father, with a corresponding distortion in the sex ratio (Wu and Hammer, 1991). Whereas most discussions of 'sperm competition' have ignored competition within ejaculates (Parker and Begon (1993) and Manning and Chamberlain (1994) are recent exceptions), discussions of meiotic drive have largely ignored competition between ejaculates (Wu, 1983a is a notable exception). The present paper addresses the interaction between these two levels of selection.

For lack of a more suitable collective noun, an 'ejaculate' will refer to a group of sperm – produced by a single male – before, as well as after, the sperm are transferred to a female and whether or not the sperm are ejaculated or transferred by some other means. Multiple mating will refer to copulation with multiple males, not repeated copulations with a single male. Repeated copulations involving a single pair will be considered to increase the size of his 'ejaculate' within her reproductive tract, but not to constitute different ejaculates. Meiotic drive during oogenesis will not be considered.

Competition within and between ejaculates

For simplicity, our model will assume that all viable sperm in a mixture of sperm have an equal probability of fertilizing an egg. This means that the competitive ability of a class of sperm can be measured by its relative concentration in the mixture. Suppose that the population of fertile males consists of two genotypes, *DD* and *Dd*, and that the relative concentration of viable sperm in the ejaculate of a *Dd* male is $(1-s)$ of which a fraction k are *d* sperm. In this scenario, k measures the

strength of meiotic drive within the ejaculate of a *Dd* male, whereas s measures the competitive disadvantage of a *Dd* male's ejaculate relative to a *DD* male's ejaculate. (DD males produce ejaculates with a concentration of viable sperm defined as unity.) The assumption of only two male genotypes is justifiable if *d* is rare or if *dd* males are lethal or sterile (as is the case for *SD* homozygotes in *Drosophila* and *t* homozygotes in mice).

A female's mating behavior can influence the probability (p) that an egg will be fertilized by a *d* sperm, even if the female chooses her partners independently of their genotype at the *D* locus. This is because her pattern of partners influences the relative importance of competition beween ejaculates and competition within ejaculates. If *Dd* ejaculates are less competitive than *DD* ejaculates ($s > 0$), increased competition between ejaculates will disadvantage *d* sperm. The extreme possibilities are single mating and sperm pooling. Under single mating, a female mates once with a randomly chosen male and there is no competition between ejaculates. If the proportion of *Dd* males in the mating population is y, then the probability p that a female will choose a *d* sperm is:

$$p = ky \tag{1}$$

By contrast, under sperm pooling, all males in the population ejaculate an equal volume of semen into a well-mixed puddle and the female chooses a sperm at random. In this case, competition among ejaculates is maximal, and

$$p = \frac{k(1-s)y}{1-sy} \tag{2}$$

If $s > 0$, fewer eggs are fertilized by *d* sperm under sperm pooling than under single mating, whereas the opposite is true if $s < 0$. The probability of being fertilized by a *d* sperm is independent of the mating system for $s = 0$.

Females that mate with multiple males will occupy a position somewhere on the continuum between single mating and sperm pooling. The greater the number of males sampled, and the more equal the contribution of each male, the closer the mating system approaches the limit of sperm pooling and the greater the importance of competition between ejaculates. In other words, a female favors sperm from competitive ejaculates by mating with multiple males. These arguments are presented more formally in Appendix 1. Genetic models are presented in Appendix 2 for a recessive lethal driving allele in the limiting cases of single mating and sperm pooling. If $s > 0$, the effect of sperm pooling is to reduce the equilibrium frequency of *d* and to make the conditions that are necessary for *d* to persist in the population more restrictive. The opposite is true if $s < 0$.

Meiotic drive and female behavior

The analysis of the previous section shows that a female's mating behavior can influence the probability that an egg will be fertilized by a meiotically-driven sperm, even when the female is unable to distinguish driving from non-driving males,

provided that the two types of males differ in the competitive ability of their ejaculates. This conclusion has two corollaries:

(1) The mating behavior of females can influence the equilibrium frequency of a driving allele or haplotype. If the aggregate behavior of females causes fewer eggs to be fertilized by *d* sperm, then the equilibrium frequency of *d* will decrease. For example, if *Dd* males are disadvantaged in competition between ejaculates, an increased incidence of multiple mating will reduce the frequency of *d*, assuming of course that the relative viabilities of the genotypes are unchanged.

(2) Genes that drive during spermatogenesis can select for changes in female mating patterns. If a polymorphic equilibrium exists, *d* must be associated with an average selective disadvantage to balance the segregation distortion in its favor in the ejaculates of *Dd* males, otherwise *d* would have spread to fixation. Therefore, genes in females will benefit from mating behaviors that increase their chance of being associated with a paternal *D* haplotype in the next generation. In the absence of other selective forces, female behavior will evolve to minimize the probability that eggs are fertilized by *d* sperm.

Whether multiple mating by females favors or disfavors a drive agent will depend on the mechanism of segregation distortion. Meiotic drive could result from an absolute improvement in the competitive abilities of *d* sperm after ejaculation. In this case ($s < 0$), the driving haplotype would be favored by mating systems that increased the opportunities for competition between ejaculates, because a *d* sperm would enjoy a competitive edge over all *D* sperm it encountered, whether these were members of its own ejaculate or the ejaculates of other males. Alternatively, the *d* halplotype could sabotage *D* sperm before ejaculation. In this case, *d* sperm would gain no advantage over *D* sperm in the ejaculates of other males. Multiple mating by females would favor the *D* haplotype if *Dd* males were less successful in competition between ejaculates because they produced fewer functional sperm (i.e., if $s > 0$). The *t* haplotype, *SD* and *SR* systems of meiotic drive all fall into the category of pre-ejaculatory sabotage.

The *t*-haplotype system provides the clearest example of a driving allele that gains an advantage in competition within ejaculates at the cost of reduced competitiveness of the ejaculate as a whole. Although a $t/+$ male's ejaculate contains equal numbers of *t* and $+$ sperm, the $+$ sperm are ineffective at fertilization (Hammerberg and Klein, 1975; Silver and Olds-Clarke, 1984; Brown et al., 1989). Therefore, if a $+/+$ female mates with a $t/+$ male, most of her progeny will be $t/+$. By contrast, if the female is inseminated by an equal-parts mixture of $t/+$ and $+/+$ semen, the $+/+$ component fathers most of her offspring (Olds-Clarke and Peitz, 1985; the best estimate of *s* from their data is 0.70). Similarly, $+$ sperm produced by $+/+$ spermatocytes are fully functional, whereas $+$ sperm produced by $t/+$ spermatocytes are non-functional, in the ejaculates of chimeric males formed by the aggregation of $t/+$ and $+/+$ embryos (Seitz and Bennett, 1985). Mating with multiple males will clearly reduce the expected proportion of a female's eggs that will be fertilized by *t*-bearing sperm.

In the sex ratio (SR) and segregation distorter (SD) systems of Drosophila, the driving chromosome eliminates sperm with the non-driving chromosome before

ejaculation. Mature sperm bundles from the testes of *SD* and *SR* males contain half the number of spermatids of bundles from the testes of standard males (*SR*: Ellison and Policansky, 1970; *SD*: Tokuyasu et al., 1977; Hausechteck-Jungen and Hartl, 1982). The *SR* chromosome is transmitted to most offspring when an *SR* male is mated to a virgin female but *SR* males suffer reduced fertility (relative to standard males) when mated to nonvirgin females (Wu, 1983b). The equilibrium frequency of *SR* in laboratory populations of *D. pseudoobscura* was about 40% when females were prevented from remating but fell to 15% when females were allowed to remate (Beckenbach, 1983). Wu (1983b) hypothesized that *SR* males are less effective at displacing the sperm of a previous male because they transfer a smaller volume of ejaculate. Curtsinger and Feldman (1980) however did not find significant virility differences between genotypes in their laboratory populations.

The fertility of *SD/+* males of *D. melanogaster* is initially similar to *+/+* males, but *SD/+* males remain fertile for fewer days. This suggests that the early ejaculates of *SD/+* males contain similar numbers of sperm to the ejaculates of *+/+* males, but that the sperm reserves of *SD/+* males are depleted sooner (Hartl et al., 1967). Mature males of both genotypes transfer fewer sperm in successive matings but this decline is faster for *SD/+* males (Peacock and Erickson, 1965; data do not control for genetic background). Under some circumstances, the faster decline in fertility with multiple mating could result in an overall competitive disadvantage for the ejaculates of *SD/+* males, but conclusive evidence is lacking.

Most mechanisms of pre-ejaculatory sabotage will handicap a driving male's ejaculate in competition with the ejaculates of non-driving males. Therefore, a female who mates with multiple males will reduce the probability that her eggs will be fertilized by a sperm carrying a driving chromosome. The genes that determine her behavior benefit because they will, on average, be present in fitter diploid genotypes in the next generation. Thus, the avoidance of meiotic drive is one reason why females might choose to mate with multiple males. There is no shortage of other possible reasons (see the lists of Halliday and Arnold, 1987; Keller and Reeve, in press) and comparative studies are needed to resolve the relative importance of the different possible factors. If the avoidance of meiotic drive is a significant determinant of female mating behavior, one would predict higher levels of multiple mating in populations where meiotic drive agents have been long established. An association with multiple mating is not predicted however for newly-arisen, relatively unsophisticated systems of drive because such drive agents would be less likely to invade a population in which females regularly mate with multiple males. As always, selection on males to prevent females from remating may act as a constraint on the options available to females.

Progeny tests of wild-caught females reveal high rates of multiple insemination in *D. pseudoobscura* (Cobbs, 1977; Levine et al., 1980) and *D. melanogaster* (Milkman and Zeitler, 1974). Comparable field data are not available for mice, but multiple paternity of litters has been reported from captive populations (Levine, 1958; Oakeshott, 1974). These are all species in which meiotic drive in males has probably been an important selective force. One of the major obstacles to a comparative test is to find a credible control group that lacks meiotic drive. The discovery of meiotic

drive usually requires detailed genetic knowledge of a species, and absence of evidence is not conclusive evidence of absence.

Meiotic drive and the "sexy sperm"

Knowlton and Greenwell (1984) suggested that multiple mating might be favored because "by mixing the sperm of several males, females could insure that their eggs were fertilized by the most competitively successful sperm, increasing the likelihood that their sons would also have competitive sperm" (also see Harvey and Bennett, 1985). Curtsinger (1991) labelled this idea the "sexy-sperm" hypothesis, but argued that the proposed payoff was illusory because selection for multiple mating disappears once heritable variation in sperm quality is exhausted. He presented a formal two-locus model in which one locus determined the competitive ability of a male's ejaculate and a second locus determined a female's propensity to mate multiple times. An allele for multiple mating was able to hitch-hike to intermediate frequency as a selectively-favored allele at the other locus swept to fixation. If there was a cost to multiple mating, the allele for multiple mating would then be eliminated as linkage disequilibrium between the loci decayed. Keller and Reeve (in press) have defended the sexy-sperm hypothesis by suggesting mechanisms (recurrent mutation, cyclical selection) that could maintain heritable variation in sperm quality.

The meiotic drive hypothesis posits the existence of a selectively maintained polymorphism, in which the segregation advantage of the driving allele is balanced by a viability advantage of the non-driving allele. Under this hypothesis, alleles (at other loci) that favor multiple mating become preferentially associated with the more-competitive (non-driving) allele, as occurs in sexy-sperm models. Alleles for multiple mating can become fixed in the population because, unlike Curtsinger's (1991) model, heritable differences in sperm competitive ability persist at equilibrium. The principal advantage of multiple mating is the superior viability of non-driving genotypes rather than the greater competitive ability of their sperm. Thus, the meiotic drive hypothesis predicts that multiply-mated females will have offspring with higher average viability than singly-mated females (cf. Madsen et al., 1992). This is not a prediction of the sexy-sperm hypothesis.

Genetic control of sperm function

A gene gains no segregation advantage from meiotic drive at an unlinked locus but suffers all of the viability and fertility costs associated with the driving chromosome. Therefore, the genome has a collective interest in suppressing competition within ejaculates because most genes of the genome will be unlinked to any given locus (assuming multiple chromosomes). This argument can be expressed in terms of direct benefits to individual genes because an unlinked modifier that suppresses drive will be less likely to be associated with a driving chromosome in

the next generation, and will thereby avoid the fitness costs associated with the chromosome (Eshel, 1985; Crow, 1991; Haig and Grafen, 1991).

A simple way to suppress competition within an ejaculate would be to vest genetic control of sperm function in the diploid genome of the male progenitor, and to organize meiosis in such a way that genes in the diploid have no way of determining which sperm receive copies of any particular gene. If a gene is unable to favor some sperm over others on the basis of their haploid genotypes, the best that the gene can do is to maximize the reproductive success of the diploid collective and take a chance on the fair segregation of meiosis (for similar ideas in political science see Harsanyi, 1953, 1955). From this perspective, the randomizing processes of meiosis function as a "veil of ignorance" (Rawls, 1970) behind which genes make decisions concerning the allocation of reproductive opportunities among sperm.

Spermatogenesis and sperm function are indeed under predominantly diploid control in the two best-studied species, *Drosophila melanogaster* and the laboratory mouse. In both species, sperm with gross deficiencies of genetic material are able to fertilize eggs. This is most clearly demonstrated in crosses between translocation heterozygotes. Complementary nondisjunction in spermatogenesis and oogenesis can result in a balanced zygote that receives both copies of a chromosome segment from one gamete and no copies from the other. The recovery of the balanced zygote proves that the chromosome segment was not necessary for the function of the nullisomic gamete. Such crosses have shown that no part of the *Drosophila* genome need be present in a sperm nucleus for that sperm to function (Muller and Settles, 1927; Lindsley and Grell, 1969). The coverage of the mouse genome is less complete, but the zygotic products of a nullisomic sperm and a disomic egg have been recovered for most chromosome regions (Cattanach and Beechey, 1990).

Diploid control of sperm function may have evolved because of the genome's collective interest in the suppression of competition within ejaculates. The crucial distinction is whether or not a gene is able to favor some sperm genotypes relative to others rather than whether a gene is expressed before or after meiosis. Mammalian spermatogenesis takes place within a syncytium, with cytoplasmic bridges linking clusters of spermatocytes before meiosis and clusters of spermatids after meiosis. These cytoplasmic connections are not severed until the final stages of sperm differentiation (Fawcett et al., 1959; Handel, 1987). Several genes are actively transcribed during the haploid syncytial stage (Hecht, 1986; Erickson, 1990), yet the evidence from complementary nondisjunction suggests that few, if any, genes need actually be present in a sperm nucleus for that sperm to function. Haploid transcription is compatible with diploid control of sperm phenotype, provided that the products of genes expressed in haploid nuclei are shared equally among the members of the syncytium (see Willison et al. (1988) and Braun et al. (1989) for evidence that haploid products are indeed shared among spermatids).

Spermatogenesis is syncytial in *Drosophila* but, even so, transcription ceases before meiosis and the postmeiotic differentiation of spermatids utilizes stored transcripts (Lindsley and Tokuyasu, 1980; Hackstein, 1987). Spermatogenesis may be particularly vulnerable to meiotic drive in *Drosophila* because meiosis is achiasmate and the first division is reductional at all loci. Therefore, a gene that simply

sabotaged sister cells after meiosis I could be an effective drive agent (see Haig and Grafen, 1991; Haig, 1993). The absence of haploid gene expression could be interpreted as an additional safeguard against this increased risk of meiotic drive. If so, haploid expression should be absent in other insects with achiasmate meiosis but present in species with chiasmate meiosis. Data to test this hypothesis are lacking.

Whereas sperm function of animals is largely determined by the diploid male genome, the postmeiotic behavior of pollen grains is largely determined by their own haploid genome (Ottaviano and Mulcahy, 1989; Mascarhenas, 1992). Cytoplasmic channels exist between sporocytes before meiosis, but, in most angiosperms, these are severed during meiosis (Heslop-Harrison, 1966) so that the transcripts of a haploid nucleus are localized to a single pollen grain. Meiotic segregation therefore can result in morphological and biochemical differences between the pollen grains of a single anther (e.g., Mangelsdorf, 1932; Twell et al., 1990). Haploid effects may be tolerated in pollen because postmeiotic competition between males is relatively more important in plants than in animals (Sivinski, 1979). Pollen grains are usually dispersed individually or in small groups and an individual stigma may receive pollen from several pollen donors. If most of a pollen grain's competitors are non-relatives rather than close kin, then genes expressed in pollen have little to gain by sabotaging other pollen grains within their own anther.

Members of the Orchidaceae display a wide range of pollen dispersal modes. Some species disperse their pollen as individual grains (monads); other species disperse their pollen as meiotic tetrads; and yet other species disperse their pollen in large clusters. The larger the number of pollen grains in the dispersal unit, the greater the risk of maladaptive competition among the members of the unit if pollen development is under haploid control. Significantly, pollen development is synchronous for members of the same dispersal unit but asynchronous for different dispersal units formed within an anther (Barber, 1942). Synchronous development suggests that there is common genetic control (i.e., effectively diploid control) for all haploid members of the dispersal unit. This conclusion is supported by the observation that development of chromosomally-deficient pollen grains proceeds normally in synchronous groups of pollen whereas deficient grains abort early in species with monad pollen (Barber, 1942; Heslop-Harrison, 1953). Synchronous development and genetic complementation of deficient pollen grains appear to be consequences of the postmeiotic persistence of cytoplasmic connections between the members of a dispersal unit (Heslop-Harrison, 1968).

Divisions of labor within ejaculates

If there is competition for fertilization between the ejaculates of different males, a male might gain an advantage if some of the members of his ejaculate forsook the scramble for an egg to block the sperm of other males. Such "helper" sperm would sacrifice their own chances of leaving descendants to increase the chances of their "egg-seeking" comrades (Silberglied et al., 1984; Baker and Bellis, 1988). A stable division of labor among the haploid members of an ejaculate would be unproblem-

atic if roles were assigned at random with respect to sperm genotype and were enforceable by the diploid genome. Otherwise, haploid genomes that shirked their responsibilities in competition between ejaculates would be overrepresented among the successful sperm of a male's ejaculate. Cooperation within an ejaculate could still evolve, despite haploid genetic influences, if competition among the ejaculates of different males were sufficiently intense.

One way that the diploid collective could ensure an evolutionarily stable division of labor between egg-seeking and helper sperm would be to disable helper sperm so that they could not produce viable offspring even if they were to fertilize an egg. The best option available for genes expressed in helper sperm would then be to assist other members of the ejaculate. The sterile sperm morphs of prosobranch molluscs (Gall, 1961), butterflies (Silberglied et al., 1984) and pentatomid bugs (Schrader, 1960) all lack nuclei or possess aneuploid genomes, possibly for this reason. The "short" and "long" sperm morphs of *Drosophila pseudoobscura* provide a counterexample. Both sperm classes possess euploid nuclei, but only the long sperm are found in fertilized eggs (Snook et al. 1994).

The problem of compliance does not arise in the ejaculates of genetically-haploid males (i.e. males of haplodiploid species or of diplodiploid species that eliminate paternal chromosomes from functional sperm) because all sperm produced by a haploid male are genetically identical. Therefore, a comparative study of sperm behavior and morphology in diploid and haplodiploid species should be a good test of whether competition within ejaculates has been a significant obstacle to sperm specialization within ejaculates.

Conclusions

A gene that gains an advantage in competition among the haploid products of a single diploid male may increase in frequency even if it has substantial negative effects on the fitness of most other genes with which it is associated. The parliament of genes (i.e., the genome acting as a collective entity) is therefore predicted to promote institutions that limit the opportunities for successful meiotic drive (Leigh, 1971). That is, most of the genome is unlinked to any particular driving locus and modifiers at unlinked loci (expressed in the diploid phase) are selected to reduce the intensity of drive (Eshel, 1985; Crow, 1991). From this perspective, the diploid control of sperm function can be interpreted as an institution that suppresses competition within ejaculates for the greater benefit of the ejaculate as a whole. Similarly, if meiotic drive reduces the overall success of an ejaculate in competition with the ejaculates of other males, then genes expressed in females that influence the probability of multiple mating are, in effect, modifiers of meiotic drive and will be selected to reduce the advantage enjoyed by the driving allele.

The possibility of genetic conflicts within an ejaculate has two important implications for discussions of 'sperm competition'. First, competition among the members of an ejaculate for access to ova may limit the degree of cooperation within the ejaculate in conflicts with the sperm of other males. (Conversely, competition

among the ejaculates of different males may limit the expression of conflict within ejaculates.) Second, meiotic drive in males provides yet another reason why females might choose to mate with multiple males – to reduce the probability that their eggs are fertilized by sperms carrying driving chromosomes.

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Appendix 1

Suppose that a female chooses a sperm at random from a sperm puddle, of which a fraction x (by volume) is contributed by *Dd* males and a fraction $(1-x)$ by *DD* males. If the relative concentration of viable sperm in the ejaculate of a *Dd* male is $(1-s)$ of which a fraction k are *d* sperm, then the probability (p) that the female will choose a *d* sperm is

$$p = k(1-s)\frac{x}{1-sx} \tag{A1}$$

If females choose the contributors to the puddle (and the relative contribution of each contributor) without regard to male genotypes at the *D* locus, the expected value of x is simply the proportion of *Dd* males in the population. The variance of x among individual females is determined by the mating system. It is maximized when each female mates with a single male, and minimized when every male in the population contributes equally to the sperm puddle from which a female chooses sperm. The greater the number of males that contribute to the puddle, and the more equal is each male's contribution, the smaller the variance in x. The probability that a female will select a *d* sperm is a function of the variance. This can be seen by taking the first and second derivatives of (A1) with respect to x.

$$\frac{\partial p}{\partial x} = k(1-s)\left[\frac{sx}{(1-sx)^2} + \frac{1}{1-xs}\right] \tag{A2}$$

$$\frac{\partial^2 p}{\partial x^2} = k(1-s)\left[\frac{2s^2x}{(1-xs)^3} + \frac{2s}{(1-xs)^2}\right] \tag{A3}$$

The first derivative is positive for all biologically-meaningful values of s (i.e., $s < 1$). As expected, the proportion of eggs fertilized by *d* sperm increases with the relative contribution of *Dd* males to the sperm puddle. The second derivative has the same sign as s. If *Dd* males produce a lower concentration of viable sperm than *DD* males ($0 < s < 1$), the second derivative is positive for all x in [0, 1]. Therefore, deviations in x above the mean (which increase p) have greater effect on the expectation of p than deviations in x below the mean (which decrease p). Multiple mating, which reduces the variance in x, reduces the expected value of p. On the other hand, if *Dd* males produce a higher concentration of viable sperm than *DD* males ($s < 0$), the second derivative is negative for all x in [0, 1]. In this case, multiple mating would increase the probability that a female's eggs are fertilized by a *d* sperm. For the special case of $s = 0$, a female's mating behavior has no effect on p.

Appendix 2: genetic equilibria

If a *d* allele is able to invade a population of *D* alleles, its equilibrium frequency will depend on the relative viability and fertility of *dd* homozygotes. We will consider the special case where *dd* is a zygotic lethal. Let y be the frequency of *Dd*

individuals in the mating population of generation g, let Δy be the change in the frequency of *Dd* between generations g and $g+1$, and let w be the viability of a *Dd* heterozygote relative to a viability of 1 for a *DD* homozygote. Then

$$\Delta y = \frac{w[(1-p_e)p_s + p_e(1-p_s)]}{w[(1-p_e)p_s + p_e(1-p_s)] + (1-p_e)(1-p_s)} - y \tag{B1}$$

where p_e and p_s represent the frequencies of d among the eggs and sperm that produce the zygotes of generation $g+1$. The gametic frequencies are functions of y: p_s depends on the mating behavior of females, whereas p_e is independent of female behavior.

Changes in mating behavior that reduce the frequency of d alleles among successful sperm reduce the proportion of *Dd* individuals in the next generation. Suppose that two populations are established with the same initial frequency of genotypes, but that the populations differ in the aggregate mating behavior of females such that $p_s = p_1$ for the first population and $p_s = p_2$ for the second population. The effect of the difference in mating behavior on the frequency of genotypes in the next generation is given by

$$\Delta y_1 - \Delta y_2 = \frac{w[(1-p_e)p_1 + p_e(1-p_1)]}{W_1} - \frac{w[(1-p_e)p_2 + p_e(1-p_2)]}{W_2} \tag{B2}$$

where $W_i = w[(1-p_e)p_i + p_e(1-p_i)] + (1-p_e)(1-p_i)$, which simplifies to

$$\Delta y_1 - \Delta y_2 = \frac{w(p_1 - p_2)(1-p_e)^2}{W_1 W_2} \tag{B3}$$

Equation (B3) has the same sign as $p_1 - p_2$. The frequency of *Dd* individuals in the next generation will be greater in whichever population has the higher value of p_s. As a corollary, if $p_1 > p_2$ and $\Delta y_1 = 0$, $\Delta y_2 < 0$. That is, changes in mating behavior that reduce p_s (for a given frequency of *Dd* males) reduce the frequency of *Dd* individuals at polymorphic equilibria.

Single-mating and sperm-pooling lie at the extremes of a continuum with respect to the effect of female behavior on the success of the d allele (if females choose their mates independently of male genotypes at the D locus). These two limiting cases are analysed in detail below. The parameter s does not appear in the single-mating model and, when $s = 0$, the sperm-pooling model reduces to this simpler case. The single-mating model is equivalent to a model analyzed by Lewontin (1968) when driving homozygotes are lethal.

Case 1: single mating

Rather than analyse the roots of Δy directly we will analyse the roots of $W\Delta y$ where

$$\begin{aligned} W\Delta y &= w[(1-p_e)p_s + p_e(1-p_s)] - wy[(1-p_e)p_s + p_e(1-p_s)] \\ &\quad - y(1-p_e)(1-p_s) \\ W &= w[(1-p_e)p_s + p_e(1-p_s)] + (1-p_e)(1-p_s) \end{aligned} \tag{B4}$$

$W\Delta y$ has the same sign as Δy for all y in the unit interval [0, 1] because (i) $W\Delta y = \Delta y = 0$ when $y = 0$ and (ii) $W > 0$ for $0 < y \leq 1$ (since this implies, $0 < p_e \leq 1$). Therefore, the roots of $W\Delta y$ are the same as the roots of Δy within this interval. Moreover, the first derivatives of $W\Delta y$ and Δy will have the same sign in the neighborhood of these roots. This is helpful because $W\Delta y$ is easier to handle mathematically than is Δy.

If females mate with a single male, $p_e = y/2$ and $p_s = ky$. Substituting for p_e and p_s in (B4) gives

$$W\Delta y = \tfrac{1}{2}[(2kw + w - 2)y + (2k - 4kw - w + 1)y^2 + k(2w - 1)y^3] \quad \text{(B5)}$$

When $W\Delta y = 0$, equation (B5) becomes a cubic with roots α, β, γ

$$\alpha = 1 + \frac{(w-1) - \sqrt{4k(1-k)(2w-1) + (w-1)^2}}{2k(2w-1)}$$

$$\beta = 1 + \frac{(w-1) + \sqrt{4k(1-k)(2w-1) + (w-1)^2}}{2k(2w-1)}$$

$$\gamma = 0 \quad \text{(B6)}$$

The necessary and sufficient condition for the driving allele d to be able to invade a population fixed for D can be obtained by analysing (B5) at $y = 0$, namely

when $y = 0$,

$$W\Delta y = 0, \quad \frac{\partial W\Delta y}{\partial y} = \frac{2kw + w - 2}{2} \quad \text{(B7)}$$

therefore, d can invade if

$$\frac{2kw + w}{2} > 1 \quad \text{(B8)}$$

The qualitative behavior of $W\Delta y$ can be further deduced by analysing (B5) at $y = 1$

when $y = 1$,

$$W\Delta y = \frac{k-1}{2}, \quad \frac{\partial W\Delta y}{\partial y} = \frac{k-w}{2} \quad \text{(B9)}$$

Sperm lethality ($k = 0$), incomplete drive ($k < 1$) and complete drive ($k = 1$) will be treated separately. For the special case of sperm lethality ($k = 0$), equation (B5) becomes a quadratic. Allele d can invade if $w > 2$, in which case there is a polymorphic equilibrium at $y = (w - 2)/(w - 1)$. This corresponds to the unlikely scenario where d is a haploid lethal in sperm and a recessive lethal in diploids, but persists because it more than doubles the fitness of female heterozygotes. For $k = 0$ and $w \leq 2$, the root at $y = 0$ has nonpositive slope and is the only stable state of the population.

For incomplete drive ($0 < k < 1$) and d able to invade, $W\Delta y$ is zero with positive slope at $y = 0$, is less than zero at $y = 1$, and approaches infinity for large positive

values of y (for all values of w at which d can invade). Therefore, there must be a root to the cubic at $y = 0$, a root between $y = 0$ and $y = 1$, and a root for $y > 1$. These roots correspond (in order) to γ, α, β from (B6). The second root (α) defines a unique stable equilibrium at which *Dd* and *DD* individuals are both present in the mating population. An exhaustive search of the parameter space (not shown) reveals that γ is the only root in [0, 1] for all cases of incomplete drive ($0 < k < 1$) with d unable to invade.

For complete drive ($k = 1$), there is an equilibrium at $y = 1$. The qualitative behavior of $W\Delta y$ (and hence Δy) depends on w. Two threshold values of w are important: $w = 2/3$ divides cases where d is unable to invade ($w \leq 2/3$) from cases where d is able to invade ($w > 2/3$); $w = 1$ divides cases where the equilibrium at $y = 1$ is unstable ($w < 1$) from cases where the equilibrium is stable ($w \geq 1$). These thresholds define three kinds of dynamic behavior:

(1) For $w \leq 2/3$, $\alpha \leq 0$, $\beta = 1$, $\gamma = 0$. The root at $y = 0$ has nonpositive slope. Therefore, d is unable to invade. The root at $y = 1$ has negative slope and is therefore unstable. This equilibrium corresponds to a population in which all surviving individuals are *Dd* heterozygotes, produced from *D* eggs and *d* sperm. The equilibrium is unstable to the introduction of small numbers of viable *D* sperm. The only stable state of the system is a population of *DD* individuals ($y = 0$). For the special case of $w = 1/2$, equation (B5) becomes a quadratic but the system retains the same qualitative behavior: a stable equilibrium at $y = 0$, with an unstable equilibrium at $y = 1$.

(2) For $2/3 < w < 1$, $0 < \alpha < 1$, $\beta = 1$, $\gamma = 0$. The root at $y = 0$ has positive slope. Therefore, d is able to invade. The system has a stable polymorphic equilibrium (α) with *DD* and *Dd* individuals both present. As in the previous case, the root at $y = 1$ is unstable to the introduction of small numbers of viable *D* sperm.

(3) For $w \geq 1$, $\alpha = 1$, $\beta \geq 1$, $\gamma = 0$. In this case, the root at $y = 1$ is the only attractor in [0, 1]. The stable state of the system is a population in which all surviving individuals are *Dd* heterozygotes, produced from *D* eggs and *d* sperm.

Case 2: sperm pooling

If females select a sperm at random from a well-mixed puddle of ejaculates contributed by all the males in a large population, $p_e = y/2$ and $p_s = k(1 - s)y/1 - sy$. Substituting for p_e and p_s in (B4) gives

$$W\Delta y = \frac{1}{2(1 - sy)}[(2kw - 2ksw + w - 2)y + (1 - 4kw + 4ksw - sw - w + 2s + 2k - 2ks)y^2 + (2kw - 2ksw - s - k + ks + sw)y^3] \quad \text{(B10)}$$

$W\Delta y$ has the same sign as Δy for all y in the unit interval [0, 1] because (i) $W\Delta y = \Delta y = 0$ when $y = 0$, (ii) $W > 0$ for $0 < y \leq 1$. Moreover, $s < 1$ (because *Dd* males are assumed to produce some viable sperm). Therefore, Δy has the same sign

as the sum of the terms within the square brackets for all y in [0, 1]. Setting this sum equals to zero gives a cubic that has the same roots as Δy within [0, 1]. The roots of the cubic are

$$\alpha = 1 + \frac{(w - sw - 1) - \sqrt{4(1-k)(1-s)C_3 + (w - sw - 1)^2}}{2C_3}$$

$$\beta = 1 + \frac{(w - sw - 1) + \sqrt{4(1-k)(1-s)C_3 + (w - sw - 1)^2}}{2C_3}$$

$$\gamma = 0 \tag{B11}$$

where

$$C_3 = 2kw - 2ksw - s - k + ks + sw$$

The necessary and sufficient condition for the driving allele d to be able to invade a population fixed for D is found by analysing (B10) at $y = 0$,

when $y = 0$,

$$W\Delta y = 0, \quad \frac{\partial W\Delta y}{\partial y} = \frac{2kw - 2ksw + w - 2}{2} \tag{B12}$$

therefore, d can invade if

$$\frac{2kw(1-s) + w}{2} > 1 \tag{B13}$$

The invasion criterion for sperm pooling (B13) is more rigorous than the invasion criterion for single mating (B8) when $s > 0$, but less rigorous when $s < 0$. The qualitative behavior of $W\Delta y$ can be further deduced by analysing (B10) at $y = 1$

when $y = 1$,

$$W\Delta y = \frac{k-1}{2}, \quad \frac{\partial W\Delta y}{\partial y} = \frac{k - w + sw}{2(1-s)} \tag{B14}$$

For incomplete drive ($k < 1$) with d able to invade, $W\Delta y$ is zero with positive slope at $y = 0$ and is less than zero at $y = 1$. Therefore, there must be a root to the cubic at $y = 0$ with positive slope and a root between $y = 0$ and $y = 1$ with negative slope. Given these constraints, there is no way to draw a cubic with a third root in the interval [0, 1]. The second root defines a stable polymorphic equilibrium at which *Dd* and *DD* individuals are both present in the mating population. For a given value of w, the frequency of *Dd* individuals at this equilibrium is lower under sperm pooling than under single mating if *Dd* males are disadvantaged in competition between ejaculates ($0 < s < 1$) and the opposite if *Dd* ejaculates are competitively superior to *DD* ejaculates ($s < 0$). These conclusions about the equilibrium frequency of the driving allele are consequences of the corollary to equation (B3) discussed in the opening section of this Appendix.

For complete drive ($k = 1$), the qualitative behavior of $W\Delta y$ depends on w and s. The conclusions are essentially the same as for single mating except that the invasion criterion is $w > 2/(3 - 2s)$ and the equilibrium at $y = 1$ becomes stable for $w \geq 1/(1 - s)$. When there are no competitive differences between male ejaculates ($s = 0$), the threshold values of w are the same as for the single-mating case, namely $w > 2/3$ (invasion criterion) and $w \geq 1$ (stability of equilibrium at $y = 1$).