Sperm competition games: sperm size and number under gametic control

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SUMMARY

This paper examines sperm competition games in which both size and number of gametes in an ejaculate can be varied strategically, but under the control of the haploid sperm. It is a companion to the previous paper (Parker (Proc. R. Soc. Lond. B 253, 245 (1993))) for the case where the strategic control is by the diploid parent. Under haploid control, the optimal balance between size and number of sperm in an ejaculate becomes quite different from that predicted for diploid control; there is a conflict between parental and gametic interests over sperm size and number. This occurs because both intra-ejaculate and inter-ejaculate sperm competition are present with haploid control, whereas intra-ejaculate competition is absent under diploid control. The magnitude of this conflict is affected by the risk of inter-ejaculate sperm competition, and by the fact that gametic interests depend on the nature of available mutations.

If ejaculate expenditure trades off against numbers of matings achieved, with haploid control there is escalation towards maximal expenditure on the ejaculate. If the ejaculate expenditure is fixed by the diploid parent, but there is a size-number trade-off under haploid control, solutions depend on the mode of action of the mutation affecting sperm size or number. If mutant-bearing sperm deviate entirely at the expense of non-mutant sperm, any increase in size or number will spread. If mutant sperm deviate entirely at their own expense, the evolutionarily stable strategy (ess) is the same as for diploid control; there is no conflict. If mutant and non-mutant sperm compete for limited resources, the ess depends on the type of mutation: (i) mutations affecting sperm size give very large sperm, decreasing as inter-ejaculate sperm competition increases; (ii) mutations affecting sperm number give very small sperm, increasing as inter-ejaculate sperm competition increases. Conflict between diploid and haploid expression is reduced by inter-ejaculate sperm competition, but is not lost, even under maximum competition. We suggest that size and other morphological variation of sperm may sometimes reflect the result of conflict between different mutational types under haploid expression, and their conflict with diploid expression.

1. INTRODUCTION

This paper is a companion to the preceding paper in this volume (Parker 1993), which gives a more detailed discussion of the sperm size problem and of the assumptions of the models. Here we examine ejaculation strategies when fertilization success depends both on the size and number of sperm in competition, and where these characters are determined by haploid genes. We must distinguish immediately (see also Boorman & Parker 1976) between 'intra-ejaculate sperm competition' (equivalent to 'gamete competition', a term sometimes used by geneticists), which refers to competition for fertilizations between different sperm genotypes within an ejaculate, and 'inter-ejaculate sperm competition' which refers to competition between ejaculates (Parker 1970). The unqualified term 'sperm competition' ubiquitously refers to inter-ejaculate competition. We propose it should continue to be used in that sense, unless, as in the present paper, there is a need to differentiate inter-ejaculate competition from a haploid expressed, intra-ejaculate component.

Geneticists usually favour the notion that phenotypic characteristics of the gamete are established by the diploid parent, as assumed in the preceding paper. However, although there is much evidence in favour of this view (see, for example, Beatty 1971), it is clear that meiotic drive and other forms of haploid expression do exist in which a sperm's phenotype is determined by its haploid genotype (see, for example, Moore 1971; Hecht et al. 1986). Under these circumstances, the optimal balance between size and number of sperm in an ejaculate becomes quite different from those predicted for diploid control; there is a conflict between parental and gametic interests over sperm size.

In mammalian spermatogenesis there is a phase of growth of the spermatogonium to form the (diploid) primary spermatocyte, which then divides by meiosis to form two (haploid) secondary spermatocytes. These then undergo the second meiotic division to form four spermatids, which undergo further differentiation to produce the sperm. Maturing sperm cluster around the Sertoli cells from which they are thought to obtain materials required for differentiation.

Throughout this paper we consider a mutation
arising in a diploid parent, which is then heterozygous at that locus. The mutation causes sperm bearing it to be either larger or more numerous than those differentiated from spermatics which do not bear the mutation. A mutation could affect sperm size either: (i) by allowing a secondary spermatocyte bearing it to gain a disproportionate amount of the cytoplasmic reserves of the diploid primary spermatocyte (rather as in the formation of polar bodies in oogenesis); or (ii) by causing sperm bearing it to gain more resources from the Sertoli cells. Such a mutation would plausibly only affect sperm size and not sperm numbers if the number of divisions after the first meiotic division remains constant. However, a mutation might affect sperm numbers rather than size if, say, those meiotic products bearing the mutation continue to divide but can nevertheless achieve the same size despite their extra division.

Equations (1)–(9) of the present paper are analogous to (1)–(9) of Parker (1993) for diploid control.

2. ANALYSIS

First consider the case where ejaculate expenditure trades off against the number of matings that a male can achieve. Following previous papers (Parker 1990a, b, 1993), assume that a locally stable evolutionarily stable strategy (ESS; Maynard Smith 1982) exists, and that it can be solved by the usual technique of differentiation of the fitness function, $W(J, I)$, which is the fitness of a JI individual in an II population. Specifically, $W(J, I)$ is the total mutant progeny of a mutant male whose J-bearing sperm deviate from the ESS strategy I, in a population playing I. J is rare, and hence all males carrying J are heterozygous (JI). $W(J, I)$ is the product of $n$, the number of matings the mutant male achieves, and $v$, the expected number of mutant progeny gained at each mating:

$$W(J, I) = n(J, I) \cdot v(J, I).$$

The two strategic parameters are the number of sperm, $s$, and the size or mass, $m$, of each one. We again seek an ESS consisting of the pair of strategies $s^*, m^*$. Mutation J causes either $s \neq s^*$, or $m \neq m^*$, i.e. unilateral deviations in one or other parameter, but only in sperm that bear the mutation. Thus the size or number of a particular sperm type in the ejaculate is determined by haploid genotype: half the spermatics bear the mutation, and half do not. This contrasts with the analysis of the preceding paper (Parker 1993) where the diploid genotype determines the size or number of sperm, and where all sperm from the same male are similar.

The condition for local stability of $s^*$ is that

$$-n(s, I)/n'(s, I) = v(s, I)/v'(s, I),$$

evaluated at $s = s^*$, (2a) and that for local stability of $m^*$:

$$-n(m, I)/n'(m, I) = v(m, I)/v'(m, I),$$

evaluated at $m = m^*$. (2b)

The primes denote the first derivative with respect to $s$ or $m$. The second derivative of equation (1) must be negative, so that equations (2a) and (2b) yield maxima.

Consider $n(J, I)$, the number of matings. Following the companion paper, each male has a fixed total energy budget, $R$, and the cost of obtaining a mating in the ESS population is $C$. The number of matings per male is $R/(C + s^*m^*)$, whereas a mutant-bearing male will have an altered number of matings as half its secondary spermatocytes result in a different reproductive expenditure from the other half. If the mutation changes sperm number so that a secondary spermatocyte bearing it produces $s \neq s^*$ sperm, the number of matings gained becomes $R/[C + 0.5(s^* + s^*m^*)]$; if the mutation changes sperm size to $m \neq m^*$, the male gets $R/[C + 0.5(s^*m^* + s^*m^*)]$ matings. The number of matings gained by the mutant, relative to the mutant average, standardizing $(C + s^*m^*) = 1$, is

$$n(s, I) = 1/[1 - 0.5s^*(s - s^*)],$$

(3a)

for deviations in $s$, the sperm numbers, and

$$n(m, I) = 1/[1 - 0.5s^*(m - m^*)],$$

(3b)

for deviations in mass, $m$, of each sperm.

Assume that sperm competition between two males occurs in proportion, $p$, of all matings. To calculate $v$, the expected number of mutant progeny gained per mating, there are now both gamete competition and sperm competition components to consider. Let $G(J, I)$ be the probability of success of mutant J-bearing sperm when competition occurs against an opponent male playing the ESS, I. $G(J, I)$ includes both intra- and inter-ejaculate sperm competition. On $(1-p)$ occasions there is no inter-ejaculate sperm competition; there is only intra-ejaculate competition between J- and I-bearing sperm within the mutant male’s ejaculate. Let $F(J, I)$ be the probability of success of mutant J-bearing sperm against I-bearing sperm from the same ejaculate. For a mutation affecting sperm numbers

$$v(s, I) = [(1-p)F(s, I) + pG(s, I)]$$

(4a)

and for a mutation affecting sperm size

$$v(m, I) = [(1-p)F(m, I) + pG(m, I)].$$

(4b)

Remember that when the ‘mutant’ J actually plays the ESS ($s = s^*, m = m^*$), $F^* = 0.5$ (there is only intra-ejaculate competition: mutant sperm have equal chances against the non-mutant sperm from the same ejaculate), and $G^* = 0.25$ (there is both inter- and intra-ejaculate competition: mutant sperm have equal chances, but represent only half of one ejaculate out of two). By using equations (3a) and (4a) we can solve equation (2a), and similarly with equations (3b) and (4b) we can solve equation (2b). At the ESS, if one exists,

$$m^* = 4[(1-p)F^*(s^*, I) + pG^*(s^*, I)]/(1-p/2),$$

(5a)

$$s^* = 4[(1-p)F^*(m^*, I) + pG^*(m^*, I)]/(1-p/2),$$

(5b)

Thus

$$s^*/m^* = [(1-p)F^*(m^*, I) + pG^*(m^*, I)]/[F^*(s^*, I) + pG^*(s^*, I)],$$

(6)

which can be given a similar interpretation to the
solution for diploid control: around the ess, the ratio (sperm number/sperm size) must equal the ratio (marginal gain from sperm size/marginal gain from sperm number). For the present case, the marginal gains are now the weighted average effects under the two conditions of ejaculate competition.

3. LOADED RAFFLES

In the loaded raffle model, the probability of success of a given sperm is proportional to its competitive weight, \( r \), divided by the summed competitive weights of all sperm in competition. If sperm are equal the raffle is 'fair' and each has equal chances; if competitive weights differ, the raffle is 'loaded'. Increasing a sperm's size increases its competitive weight and hence its probability of being drawn in the fertilization raffle. For J-bearing sperm, the intra-ejaculate competition function is

\[ F_j = r_j s_j / (r_i s_i + r_j s_j), \]  

(7a)

and the 'inter-plus-intra-ejaculate' competition function is

\[ G_j = r_j s_j / (3 r_i s_i + r_j s_j), \]  

(7b)

where subscripts \( i \) and \( j \) refer to the sperm ess and mutant sperm, respectively.

Assuming that the competitive weight of a sperm increases with its size (mass), \( m \), we now seek an ess, \( s^* \), \( m^* \). Setting \( r_i s_i = r(m^*) s^* \), \( r_j s_j = r(m^*) s^* \), gives

\[ F'(m^*, I) = r'(m^*) / 4 r(m^*), \]  

(8a)

and setting \( r_j s_j = r(m^*) s^* \), \( r_i s_i = r(m^*) s^* \), gives

\[ G'(s^*, I) = 3 r'(m^*) / 16 r(m^*), \]  

(8b)

Substituting equations (8a) and (8b) into equations (5a) and (5b) and manipulating shows that an ess ejaculate mass (as a proportion of reproductive effort per mating) requires

\[ s^* m^* = (4 - p) / (4 - 2p). \]  

(9)

This is clearly impossible: for any sperm competition risk \( 0 < p < 1 \), it requires the proportion of expenditure per mating on the ejaculate to exceed 1. Equation (9) suggests that gametic control will drive ejaculate to ever larger sizes until (hypothetically) all reproductive effort is spent on the ejaculate.

Indeed, sperm numbers will always increase ejaculates to maximum expenditure. Consider a mutation which causes J-bearing sperm to increase in number from \( s \) to \( s + \delta \), where \( \delta \) represents an arbitrary tiny increase in number. This 'marginal' mutation spreads to maximum expenditure. Consider a mutation which causes J-bearing sperm to increase in number from \( s \) to \( s + \delta \), where \( \delta \) represents an arbitrary tiny increase in number. This 'marginal' mutation spreads to maximum expenditure.

A mutant for marginal increase in sperm size from \( m \) to \( m + \delta \) spreads only if the left-hand side of the above equation exceeds the right-hand side. This can apply to a range of values of sperm number, \( s \), for some monotonic increasing forms of \( r(m) \) with decreasing gradient. For instance, if \( r(m) \) is proportional to \( m^* \) (0 < \( x < 1 \)), the ejaculate expenditure is

\[ s, m^* = x(4 - p) / (4 - 2p), \]

which is below 1.0 for all \( p \) if \( x < \frac{1}{2} \). This gives a positive relation between \( s, m^* \) and \( p \), varying across the limits from \( x \) to 1.5\( x \). However, this is an ess only if sperm number is fixed. 'Number' mutations will always invade if they increase \( s \), although these will tend to reduce sperm size (\( m^* \) decreases as \( s \) increases if \( r(m) \) is monotonic increasing with decreasing gradient).

This prediction – that if sperm size or number is controlled by haploid expression, there will be escalation to maximal ejaculate expenditure – contrasts markedly with diploid control. Under haploid control, escalation occurs because a 'marginal' number mutation's competitive advantage is always felt through intra-ejaculate sperm competition, and outweighs the losses in numbers of matings. Under diploid control, there is no intra-ejaculate competition; all sperm are equal. The ess ejaculate expenditure per mating is then \( p(4 - 2p) \) (Parker 1993).

So, if ejaculates trade off against mating effort, a conflict clearly exists between genes expressed in the diploid phase to control ejaculate expenditure, and those expressed in the haploid.

4. SIZE-NUMBER TRADE-OFFS

Under diploid control, the ess sperm size is given directly by the marginal value theorem as

\[ m^* = r(m^*) / r'(m^*), \]  

(10)

whether: (i) ejaculate mass remains constant, and sperm size trades off against sperm number; or (ii) sperm size or number can increase unilaterally as a trade-off against number of matings.

What applies under haploid control if ejaculate expenditure is constant (e.g. controlled by the parent), and sperm size trades off directly against sperm number? Let ejaculate expenditure remain constant as \( M = sm \) (sperm size times number). For haploid control, fitness (replication of mutant J gene) is maximized by maximizing \( v = (1 - p) F_i + p G_j \), as the number of matings, \( n \), is now independent of \( s \) or \( m \). Functions \( F_i \) and \( G_j \) now depend critically on the assumptions one makes about the biology of the mutations. We examine three cases; each has two types of mutant to consider (size or number). Expressions for \( F_i \) and \( G_i \) are given in Appendix 1.
Figure 1. Marginal value solutions for ess sperm size, $m^*$, under the size-number trade-off model with haploid control, where mutant and non-mutant sperm compete for limited resources (case 3; see text). For comparison, the adult (diploid) control solution is shown by the shaded tangent to $r(m)$ which intersects the origin. (a) Solutions for $m^*$ when two males compete at all matings ($p = 1.0$). Where mutations affect sperm size, the ess is given by a tangent line to $r(m)$ which intersects the abscissa at two $m^*$ units to the left of the origin. Decreasing inter-ejaculate sperm competition risk, $p$, causes sperm size to increase: the tangent line must then intersect $m$ at more than two $m^*$ units to the left of the origin. Where mutations affect sperm number, the ess is given by a tangent line which intersects the abscissa at $|m^*|$ units to the right of the origin (so that $|m^*|$ units separate the intersect and $m^*$). Decreasing sperm competition risk, $p$, causes sperm size to decrease: the tangent line must then intersect $m$ at more than a $m^*$ units to the right of the origin. (b) Solutions for $m^*$ when vast numbers of males ($n \to \infty$) compete at each mating. Where mutations affect sperm size, the ess is given by a tangent line to $r(m)$ which intersects the abscissa at one $m^*$ unit to the left of the origin. Where mutations affect sperm number, the ess is given by a tangent line which intersects the abscissa at $\frac{1}{2}m^*$ units to the right of the origin. This gives esss closer to, but not coincident with, the diploid ess.

(a) Case 1. Mutant sperm deviate entirely at the expense of non-mutant sperm

Suppose that J-bearing sperm take more resources, entirely at the expense of non-J sperm. J always spreads, however much it reduces the size or number of non-mutant sperm.

Consider a size mutation that causes J-sperm to have a size of $(m + \epsilon)$, at the expense of non-mutant sperm, which therefore have a size $(m - \epsilon)$. There are 2 of each sperm type, because numbers are not affected. Such a mutation could be one that causes secondary spermatocytes to take more than half of the cytoplasm during meiosis, at the expense of non-J sisters, after which both products undergo the same number of divisions. It is easy to show that any mutation causing an arbitrarily small change, $\epsilon$, in size will spread if $er'(m)(2 - p) > 0$;

i.e. all that is required is that size is increased ($\epsilon$ is
positive) and that increasing size increases competitive weight \((r'(m) \text{ is positive})\).

Now consider a sperm number mutation causing spermatids to produce 0.5\((s + e)\) sperm, at the expense of non-J sisters, which then represent 0.5\((s - e)\) of the total. Sperm size is unaffected, remaining as \(m^*\). Such a mutation could be one that again causes secondary spermatocytes to take more than half of the cytoplasm during meiosis, at the expense of non-J sisters, after which the products are genetically programmed to undergo division until size \(m^*\) is achieved. This spreads if

\[ e(2-p) > 0; \]

i.e. it always spreads if numbers are increased \((e \text{ is positive})\).

Such mutations as these constitute a form of meiotic drive; they can always invade and are likely to fixate if no cost is associated with homozygosity for J: for example, it seems plausible that a JJ male might produce less ejaculate than an II male, because of the conflict between J sister products at meiosis. Should this occur, polymorphism for I and J seems possible.

(b) Case 2. Mutant sperm deviate entirely at their own expense

Suppose that mutation J causes some rearrangement of the size–number allocation after the first meiotic division: an IJ male produces equal-sized secondary spermatocytes (of mass \(M/2\)), but the I and J products then differentiate differently, within the constraint that \(ms/2 = M/2\). Thus, in contrast to case 1, deviation by J is now paid for entirely at its own expense; it does not affect size or number of I products. By the usual techniques, it is easy to show that the eSS for this case is the same as that for diploid control: the optimal-sized sperm (for both size and number mutation) is given by equation (10). There is no conflict between parental and gametic interests.

(c) Case 3. Mutant and non-mutant sperm compete for limited resources

In case 1, J profits entirely at the expense of I; in case 2, J deviates entirely at its own expense. Both these extremes seem plausible biologically, depending on the expression of the mutation. However, a further possibility is that J-bearing products compete with I-products for fixed resources, thus altering the size or number of all sperm.

This is best explained by two examples. The first is a sperm size mutation, which has consequences for sperm number. Suppose that all products undergo equal numbers of divisions, and are hence equal in number, but J-bearing spermatids are more effective, in competition with I-bearers, at gaining resources from Sertoli cells. They hence become larger but, because of the fixity of resources, this increased burden of demand reduces the total number of sperm.

The second example is a sperm number mutation and has consequences for sperm size. Suppose that J-bearing products gain more from the Sertoli cells and differentiate more; they are hence more numerous, but achieve the same size as I-bearers. However, size is reduced for all sperm because there are more of them in competition for the fixed resources.

As with diploid control and case 2 above, eSS solutions can now exist for suitable \(r(m)\) relations. But unlike these cases, solutions now differ for the sperm number mutation and the sperm size mutation.

Consider the mutation that changes the size of all haploid products carrying it, to \(m \neq m^*\). Because I and J sperm compete for the \(M\) units of resource, the total number of sperm becomes \(M/[0.5(m + m^*)]\), half of which are J-bearers. By the usual method, at the eSS

\[
\begin{align*}
(4-p)/p m^* &= r'(m^*)/r(m^*),
\end{align*}
\]

which clearly differs from the diploid control case shown in equation (10). If there is no sperm competition \((p = 0)\), equation (11) suggests that sperm should be of maximum size.

Now consider the sperm number mutation. A mutation that changes the number of all haploid products carrying J to \(s \neq s^*\) changes the size of each sperm (whatever their genotype) to \(M/[0.5(s + s^*)]\), half of which are J-bearers. We then get the result that

\[
\begin{align*}
[p/(4-p)] m^* &= r'(m^*)/r(m^*),
\end{align*}
\]

which differs both from the diploid control (equation (10)) and from the haploid control ‘size mutant’ (equation (11)). If there is no sperm competition \((p = 0)\), equation (12) suggests that sperm should be of minimum size.

It is clearly impossible to satisfy equations (11) and (12) simultaneously. Figure 1a shows the marginal value solutions for the two optima for the (hypothetical) case where there is maximum sperm competition with \(p = 1.0\). This generates haploid optima that are closest to the diploid optimum, but in essence either drive for increased sperm size or increased sperm number can occur, depending on the nature of available mutations and their expression. As inter-ejaculate sperm competition risk, \(p,\) gets smaller, the optima diverge towards vast numbers of tiny sperm or few huge sperm. For mutations affecting sperm size, increased sperm competition risk causes sperm size to decrease (equation (11)); for mutations affecting sperm number, increased risk causes sperm size to increase (equation (12)).

Can even higher levels of inter-ejaculate sperm competition (higher than the present model allows) cause the two mutational optima to converge to the diploid optimum? We conclude that they cannot, even though they will further reduce the disparity between the three optima. Suppose that mating typically involves \(n\) males in a spawning group, where \(n \gg 1\). Maximum sperm competition occurs when the spawning group size is vast \((n \to \infty)\). All matings now involve both inter- and intra-ejaculate competition, and hence the optimum consists of maximizing function \(G;\) function \(F\) (for intra-ejaculate competition alone) is lost. Expressions for the two types of mutation, \(G(m, 1)\)
and $G(s,1)$, are given in Appendix 2. For size mutations we obtain

$$\frac{(2n-1)/(n-1)}{m^*} = r'(m^*)/r(m^*), \quad (13)$$

and for number mutations:

$$\frac{(n-1)/(2n-1)}{m^*} = r'(m^*)/r(m^*). \quad (14)$$

If $n = 2$, equations (13) and (14) are equivalent to equations (11) and (12) with $p = 1.0$; just two males compete at every mating. However, equations (13) and (14) confirm the view that increasing inter-ejaculate sperm competition causes the two optima to become closer: when $n$ approaches infinity, the optima are closest, but are still unequal (see figure 1b). Sperm (inter-ejaculate) competition constrains, but does not eliminate, gametic selfishness arising from haploid expression.

5. DISCUSSION

Sperm in many animal groups are highly variable. Previous interpretations of this variation have been that it represents: (i) 'maladaptive noise'; presumably the result of genetic drift of near-neutral mutations affecting sperm phenotype; or (ii) adaptive specialization to sperm competition by 'warfare': different sperm morphs having different functions to increase the fertilization probability of the ejaculate as whole (the 'kamikaze sperm hypothesis' (Baker & Bellis 1988; see also Silberglied et al. 1984)). We here propose a third hypothesis, that: (iii) sperm variation may sometimes represent the result of conflict between different mutational types under haploid expression, and their conflicts with diploid expression. Hypothesis (i) seems unlikely, especially as most aspects of sperm morphology must be highly fitness related; its best prospects perhaps occur in systems with little inter-ejaculate sperm competition and full diploid control. Hypothesis (ii) seems plausible as a solution for clearly established sperm polymorphisms such as the eupyrene-apyrene dichotomy in Lepidoptera (see, for example, Silberglied et al. 1984), and for other discontinuous variation (e.g. biflagellate, bicephalous, macrocephalous, etc. sperm in mammals) (Baker & Bellis 1988). It could also have a role in more subtle variation (R. R. Baker, personal communication). Hypotheses (ii) and (iii) are not mutually exclusive.

It is clear from the present analysis that mutations expressed in the haploid sperm can increase sperm number and sperm size above their respective optima for diploid expression, even if this leads to extinction of the local population, as in certain forms of meiotic drive (e.g. for sex ratio (Hamilton 1967)). Any suppressor that can restore the balance back towards drive (e.g. for sex ratio (Hamilton 1967)); Any for diploid expression, even if this leads to extinction of variation (R. R. Baker, personal communication). Beilis 1988). It could also have a role in more subtle sometimes represent the result of conflict between sperm competition by 'warfare' different number and sperm size above their respective optima expressed in the haploid sperm can increase sperm macrocephalous, etc. sperm in mammals) (Baker & Bellis 1988). It could also have a role in more subtle variation (R. R. Baker, personal communication). Hypotheses (ii) and (iii) are not mutually exclusive.

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oogonia, and for the evolution of polar bodies, which are typically seen as inevitable results of meiosis, despite their lack of any parallel in spermatogenesis. It is interesting to note that in both forms of gametogenesis, most of the growth occurs under diploid control, i.e. in the primary spermatocyte and primary oocyte stage, and hence before meiosis. Highly speculative possibilities are that degeneration of oogonia relates to resolution of conflict between cytoplasmic and nuclear elements, and that polar bodies relate to resolution of conflicts between haploid and diploid expression. The relatively large size of each ovum makes the potential conflicts here much greater.

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APPENDIX 1. Expressions for $F_j, G_j$, in the size–number trade-offs

(a) Case 1. Mutant sperm deviate entirely at the expense of non-mutant sperm

(i) Deviations in sperm size

$$F_j = \frac{r(m) s}{r(m) s + r^* s^*},$$

$$G_j = \frac{r(m) s}{r(m) s + r^* s^* + 2r^* s^*}$$

(ii) Deviations in sperm number

$$F_j = \frac{r^* 0.5(s + e)}{r^* 0.5(s + e) + r^* 0.5(s - e)},$$

$$G_j = \frac{r^* 0.5 s}{r^* 0.5(s + e) + r^* 0.5(s - e) + r^* 0.5 s^*}.$$

(b) Case 2. Mutant sperm deviate entirely at their own expense

(i) Deviations in sperm size

$$F_j = \frac{r(m) s}{r(m) s + r^* s^*} + \frac{r^* 0.5 s + r^* 0.5 s^*}{r^* 0.5 s + r^* 0.5 s^*} - \frac{r^* 0.5 s}{r^* 0.5 s + r^* 0.5 s^*}$$

$$G_j = \frac{r(m) s}{r(m) s + r^* s^* + 2r^* s^*}$$

(ii) Deviations in sperm number

$$F_j = \frac{r(m) 0.5 s}{r(m) 0.5 s + r^* 0.5 s^*} + \frac{r^* 0.5 s + r^* 0.5 s^*}{r^* 0.5 s + r^* 0.5 s^*} - \frac{r^* 0.5 s}{r^* 0.5 s + r^* 0.5 s^*}$$

$$G_j = \frac{r(m) 0.5 s}{r(m) 0.5 s + 1.5r^* s^*}$$

(c) Case 3. Mutant and non-mutant sperm compete for limited resources

(i) Deviations in sperm size

$$F_j = \frac{r(m) s}{r(m) s + r^* s^*},$$

$$G_j = \frac{r(m) s}{r(m) s + r^* s^* + 2r^* s^*}$$

where $s_0 = \frac{0.5M}{m^*}$, $s^* = \frac{0.5M}{m^*}$.

(ii) Deviations in sperm number

$$F_j = \frac{r(m) s}{r(m) s + r^* s^*},$$

$$G_j = \frac{r(m) s}{r(m) s + r^* s^* + 2r^* s^*}$$

where $m_0 = \frac{M}{0.5(s + s^*)}$.

APPENDIX 2. Expressions for $G_j$ in size–number trade-offs where $n$ males compete

These are parallel to case 3 in Appendix 1, where mutant and non-mutant sperm compete for limited resources.

(i) Deviations in sperm size

$$F_j = \frac{r(m) s}{r(m) s + r^* s^* + 2r^* s^*},$$

$$G_j = \frac{r(m) s}{r(m) s + r^* s^* + 2r^* s^*}$$

where $s_0 = \frac{0.5M}{m^*}$, $s^* = \frac{0.5M}{m^*}$.

(ii) Deviations in sperm number

$$F_j = \frac{r(m) s}{r(m) s + r^* s^* + 2r^* s^*},$$

$$G_j = \frac{r(m) s}{r(m) s + r^* s^* + 2r^* s^*}$$

where $m_0 = \frac{M}{0.5(s + s^*)}$.

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