2. WHY ARE THERE SO MANY TINY SPERM? SPERM COMPETITION AND THE MAINTENANCE OF TWO SEXES

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ABSTRACT

It is suggested that sperm competition (competition between the sperm from two or more males over the fertilization of ova) may account for the fact that sperm are so small and so numerous. In the entire absence of sperm competition, selection may favour an increase in sperm size so that the sperm contributes nutriment to the subsequent viability and success of the zygote. However, an extremely low incidence of sperm competition is adequate to prevent sperm size increasing. Vertebrate sperm should remain at minimal size provided that double matings (one female mated by two males) occur more often than about 4 times the ratio of sperm size:ovum size. The classical theory that sperm are small simply because of the difficulties of ensuring that ova do get fertilized may also explain sperm size, and both effects (sperm competition and ensuring fertilization) are likely to contribute to the stability of anisogamy. Large numbers of sperm can be produced because sperm are tiny and the optimal allocation of reproductive reserves to ejaculates is not trivially small even when double matings are rather rare. It is suggested that of its total mating effort, a male vertebrate should spend a fraction on sperm that is roughly equivalent to a quarter of the probability of double mating.

1. INTRODUCTION

Despite the fact that anisogamy is the rule in multicellullar animals and plants, biologists have devoted rather little attention to an interpretation of why evolution has produced and maintained males and females. Why not, say, five sexes, each producing its

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own characteristic gamete? Early theories for the evolution of anisogamy (Kalmus, 1932; Kalmus & Smith, 1960; Scudo, 1967) assumed that selection would act to favour efficiency in fertilization at the species or group level, and considered only isogamy versus anisogamy. Recently, Parker, Baker & Smith (1972) proposed that anisogamy might result from disruptive selection acting on a continuous range of variants, each variant producing gametes of a characteristic size. The assumptions of disruptive selective theory are as follows.

(i) A large population of adults releases its gametes into an external medium (e.g. sea water) so that gametes fuse randomly, independent of size.
(ii) There is a fixed energy budget per parent, so that if an adult produces gametes of size m, the relative number of gametes produced is proportional to m^{-1}
(iii) The viability (or other components of fitness) of a zygote increases with its size. Thus a zygote produced by the fusion of two large gametes (each with high provisioning) survives better than one resulting from the fusion of two intermediate-sized gametes, or one from a large and small gamete.

Provided that in (iii) the size of the zygote exerts an important enough effect on its survival, the evolutionarily stable strategy (ESS; Maynard Smith, 1974) is anisogamy, i.e. a population consisting of males (microgamete producers) and females (megagamete producers) will be stable. This result (originally obtained by computer simulation) has been confirmed analytically by a number of authors (Bell, 1978; Charlesworth, 1978; Maynard Smith, 1978; Hoekstra, 1980). Some empirical support (Knowlton, 1974; Bell, 1978) is available from the fact that in various groups of algae, a trend towards anisogamy (from isogamy) is associated with a trend towards multicellularity (from unicellularity). During the evolution of anisogamy, selection is likely to favour sperm that fuse disassortatively (with ova), and probably ova that fuse disassortatively (with sperm); see Parker (1978). Fisher's principle (1930) explains why the sex ratio stabilizes at unity.

Although the disruptive selection theory forms a basis for the origin and maintenance of the two sexes, assumption (i) above will be adequate as an approximation only for many plants, and certain animals with external fertilization (e.g. a large population of sessile external fertilizers). Animals with internal fertilization seem perhaps most disparate from the concept of the original model. The aim of the present paper is to consider reasons why anisogamy remains stable even when the reproductive pattern changes from external fertilization to internal fertilization. I argue that it is essentially sperm competition that is responsible for maintaining anisogamy. Sperm or ejaculate competition is competition between the sperm of different males over the fertilization of the ova (Parker, 1970*a*). In sessile animals with external fertilization, there will be a high degree of sperm competition if spawning tends to be synchronous. In species with internal fertilization sperm competition may be much reduced, but probably never entirely absent. Without sperm competition, anisogamy may be unstable, because it would pay males to increase the provisioning in each sperm so as to contribute to the survivorship of the zygote.

There appear to be two central questions. Firstly, what keeps sperm small and devoid of any provisioning for the zygote? Secondly, why are so many sperm produced?

2. ANISOGAMY IN SMALL GROUPS OF SYNCHRONOUS EXTERNAL FERTILIZERS

I first investigate the robustness of the anisogamy ESS to the effects of group size. Suppose that anisogamy and disassortative fusion have evolved in a population of synchronous external fertilizers. What happens when there are just n males in each spawning group?

Here and elsewhere we seek conditions under which selection will act against small increases in the size of the sperm. Sperm become reduced to the least size because this allows so many of them to be produced; high productivity yields an advantage through sperm competition. We can assume that ovum size must be stabilized at a unique optimum if sperm contribute nothing to the zygote. The anisogamy ESS must conform to a Nash equilibrium in which it will not pay the male to supply provisioning in the sperm, nor will it pay the female to deviate from her unique optimum specified by zero sperm contribution to the zygote. We can test the robustness of the anisogamy ESS by testing whether a mutation will spread that contributes some investment to the zygote.

Let us assume that the provisioning from the ovum contributes an amount F to the survival prospects of the zygote. At the anisogamy ESS, the male contributes nothing via the sperm to zygote survival. Thus investment in each ovum is optimized at $m_{f\text{opt}}$ while investment in each sperm is set at an arbitrary minimum level $m_{\min}$. Suppose a mutant male could invest $m > m_{\min}$ in each sperm and thereby raise the survival prospects of the zygote by an amount $b(m)$. By so doing, the mutant produces less total sperm than a normal male. Normal males produce (relatively) $m_{\min}^{-1}$ sperm, whereas the mutant produces m^{-1} sperm. The mutant will therefore obtain less fertilizations than a normal male, but produces zygotes that survive better.

With n males and f females in each group, the expected fitness of a normal male will be:

$$\frac{fF}{n}.$$

A mutant male playing $m > m_{\min}$ will obtain fitness:

$$\underbrace{f\left[F+b(m)\right]}_{\text{viability of zygote}} \quad \underbrace{\frac{m^{-1}}{(n-1)m_{\min}^{-1}+m^{-1}}}_{\text{proportion of fertilizations obtained by mutant}} = \frac{\text{mutant's sperm number}}{\text{total number of sperm}}.$$

Thus for $m_{\min}$ to be an ESS against m requires that

$$\frac{F}{n} > [F+b(m)]\frac{m_{\min}}{(n-1)m+m_{\min}} \tag{1}$$

and is clearly independent of sex ratio.

The $m_{\min}$ strategy will be locally stable (i.e. resistant to small increases in the amount of provisioning in sperm) if

$$\frac{\mathrm{d}}{\mathrm{d}m}\left\{[F+b(m)]\frac{m_{\min}}{(n-1)m+m_{\min}}\right\}\Bigg|_{m_{\min}} < 0\,.$$

The logic behind this assertion is explained in Fig. 1. Differentiation gives the result that result that $m_{\min}$ is stable if

$$b'(m_{\min}) < \frac{F(n-1)}{nm_{\min}}. \tag{2}$$

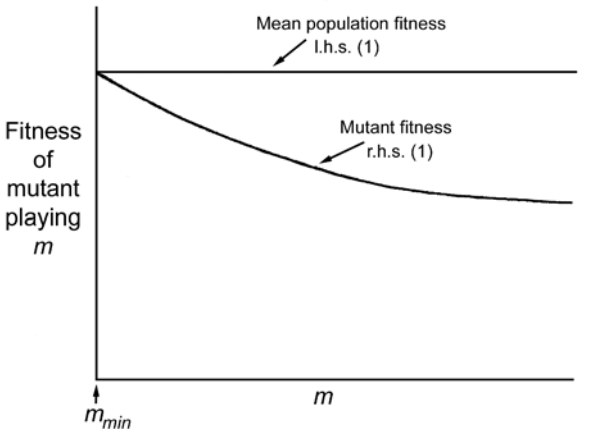

FIG. 1. The fitness of a mutant that produces sperm of size m, in a population where all other males play $m_{\min}$ (minimal sized sperm with no provisioning for zygote) is given by the right hand side of equation (1). In the case shown here, $m_{\min}$ would be locally stable since all mutants with $m > m_{\min}$ have lower fitness than the rest of the population. The condition for $m_{\min}$ to be locally stable is therefore that the differential coefficient with respect to m of the right hand side of (1), evaluated at $m_{\min}$, is negative.

We can proceed little further until we know more about $b'(m_{\min})$, which is the rate at which provisioning via sperm would contribute to zygote viability. Consider as follows. If the male parent supplies no investment to the zygote, the ESS investment in each ovum for the female parent, $m_{f\mathrm{opt}}$, is given by the tangent method (Smith & Fretwell, 1974) as shown in Fig. 2. Thus if we plot zygote viability b against the provisioning m_f supplied

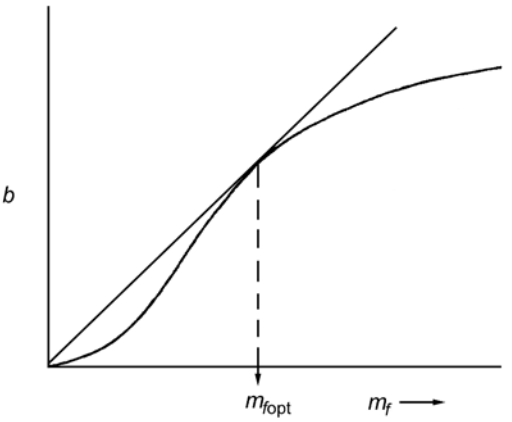

FIG. 2. Optimal provisioning $m_{f\text{opt}}$ for the female to supply the ovum, assuming that the male will supply nothing to the zygote. The optimum is given by the tangent to $b'(m_f)$ drawn from the origin (see Smith & Fretwell, 1974). This gives the maximum number of surviving offspring by maximizing the gain rate obtainable from limited reserves. Obviously, at $m_{f\text{opt}}$, $b'(m_f) = b(m_f)/m_f$ as in equation (3).

by the female to the ovum, assuming zero sperm provisioning, we expect that at $m_{f\text{opt}}$ the gradient of the tangent equals the gradient of $b(m_f)$. So we can write

$$b'\left(m_{f\text{opt}}\right) = \frac{b\left(m_{f\text{opt}}\right)}{m_{f\text{opt}}} = \frac{F}{m_{f\text{opt}}}. \tag{3}$$

Assuming that provisioning via sperm and via ova would affect zygote survival equivalently, then it is easy to see that $b'(m_{f\text{opt}}) = b'(m_{\min})$ because females will supply $m_{f\text{opt}}$ if males supply $m_{\min}$ to the zygote. We can therefore substitute (3) into (2) to give the condition that

$$\frac{m_{f\text{opt}}}{m_{\min}} > \frac{n}{n-1} \tag{4}$$

for $m_{\min}$ to be locally stable.

Rule (4) states that in order for zero provisioning from sperm to be stable, we need roughly the "anisogamy ratio" (ratio of ovum size to sperm size) to be greater than the number n of males in each spawning group divided by $(n - 1)$. Obviously, for large groups, the sperm size can almost equal the ovum size before a mutant with extra provisioning will spread. Even when usually only two males compete for fertilizations, sperm size should not increase from $m_{\min}$ unless the anisogamy ratio is less than 2. This result is interesting because it implies that once a state has been attained in which there is a disassortative fusion and where the ovum supplies all the zygotic reserves, it is unlikely to pay males to provision sperm unless there is no sperm competition ($n \rightarrow 1$). Then it will always be favourable to increase zygotic reserves by sperm provisioning.

3. SPERM SIZE WITH INTERNAL FERTILIZATION

I have established that for synchronous external fertilizers (on which the original anisogamy model of Parker *et al*. was based), sperm competition is essential to maintain anisogamy with zero sperm provisioning. Internal fertilization must reduce dramatically the number of occasions on which sperm competition occurs. How will this affect the stability of the anisogamy ESS?

We retain all the features of the model outlined in section 2, except that sperm competition arises on only proportion p of occasions. Thus with frequency p two males mate with the same female, with frequency $(1 - p)$ the female is mated by just one male. When two males mate with the same female, the success of male i in competition with male j is taken as before as

$$\frac{\text{number of } i \text{ sperm}}{\text{total sperm } i + j}.$$

Support for this model as an approximation for vertebrates comes from the work of Martin *et al.* (1974) on chickens and Lanier *et al.* (1979) on rats. For the $m_{\min}$ strategy to be stable against a mutant male that invests $m > m_{\min}$ in each sperm requires that

$$(1-p)F + p\frac{F}{2} > (1-p)[F + b(m)] + p[F + b(m)]\frac{m^{-1}}{m^{-1} + m_{\min}^{-1}} \tag{5}$$

$$\therefore\ F\left(1 - \frac{p}{2}\right) > [F + b(m)]\left[(1-p) + p\left(\frac{m_{\min}}{m + m_{\min}}\right)\right]$$

which is directly equivalent to equation (1). By the same technique used in section 1, we can see that $m_{\min}$ will be locally stable if the differential coefficient with respect to m of the RHS of (5) is negative when evaluated at $m_{\min}$. This gives the condition that

$$b'(m_{\min}) < \frac{pF}{m_{\min}(4 - 2p)} \tag{6}$$

for m to be stable. Remembering again that if the male plays $m_{\min}$, the female must play $m_{f\text{opt}}$ and $b'(m_{\min}) = F/m_{f\text{opt}}$ (Fig. 2; equation (3)), we can substitute into (6) to obtain

$$\frac{m_{f\text{opt}}}{m_{\min}} > \frac{4 - 2p}{p} \tag{7}$$

for $m_{\min}$ to be stable. As expected, if $p = 1$, the condition is the same as for equation (2) with $n = 2$. If p is small, we obtain the approximation that

$$p > 4\frac{m_{\min}}{m_{f\text{opt}}}$$

to retain the m_{min} ESS. At high anisogamy ratios, sperm competition can be extremely rare and yet will still be entirely adequate to prevent invasion by mutants with sperm that contain provisioning for the zygote. All that is required is roughly that double matings are more frequent than 4 divided by the anisogamy ratio.

This model appears equally applicable to mobile external fertilizers such as certain fish, in which many spawnings involve a single male and female, but some spawnings involve a "sneak" male as well as the primary male.

In most vertebrates, the ovum is vastly larger than the sperm and the anisogamy ratio commonly exceeds 10^6. Suppose sperm were so large as to be equivalent to one thousandth the size of an ovum; then anisogamy would be stable provided that double mating occurs for at least 0.4% of litters. Thus provided that mobility and internal fertilization arose at a stage after a disassortative fusion and high anisogamy ratio had evolved, there is no reason to suspect that the reduced potential for sperm competition should lead to a change in the minimal investment characteristic of the sperm. Anisogamy is a remarkably robust ESS.

Essentially, the reason it does not pay to increase sperm provisioning is that a unit increase in investment in each sperm causes significant cost, but insignificant benefit. For example, doubling the sperm size halves the sperm number, which causes significant losses when there is sperm competition. But doubling the sperm size would effect a virtually insignificant increase in the viability of the zygote.

Of course, selection will favour mechanisms in the female to consume what are, for her, excess sperm. Considerable phagocytosis of sperm appears to take place in the female genital tract in vertebrates; the female may therefore profit by male ejaculate expenditure. To the extent that the offspring may benefit from the products of the phagocytosis, the male may also benefit indirectly if the affected offspring are his own. Alternatively, the male may benefit even more directly by adopting various forms of parental care. But it will not pay him to increase his provisioning of the zygote by increasing the amount of reserves bound up in each sperm.

For some groups with internal fertilization, double mating may not lead to approximately equal chances for each male, even when they both transfer equal amounts of sperm. For instance, in insects it appears quite common that the last male to mate displaces much of the previously-stored sperm from the female's sperm stores, and replaces it with his own (e.g. Lefevre & Jonsson, 1962; Parker, 1970*a*,*b*; Waage, 1979). It is obvious that provided sperm displacement is not total, some sperm competition still occurs. Suppose that the last male displaces proportion z of the previous ejaculate on a volumetric basis (some evidence for this comes from Lefevre & Jonsson, 1962; Parker, 1970*b*). Then if sperm are small, there will be relatively more of them left in the $(1 - z)$ volume remaining undisplaced, than if sperm are large. If we apply exactly the same model as for vertebrates, and allow that the mutant male with $m > m_{min}$ can mate first or last with equal probability, we need that:

$$F\left(1-\frac{p}{2}\right) > (1-p)\left[F+b(m)\right]+\frac{p}{2}\left[F+b(m)\right] \times\left[\frac{zm_{\min}}{(1-z)m+zm_{\min}}+\frac{(1-z)m_{\min}}{zm+(1-z)m_{\min}}\right]$$

if $m_{\min}$ is to be an ESS.

Applying the usual technique, we find that to retain minimal sperm provisioning requires that:

$$\frac{m_{f\text{opt}}}{m_{\min}} > \frac{2-p}{pz(1-z)} \tag{8}$$

or if p is small, then approximately

$$p > \frac{m_{\min}}{m_{f\text{opt}}} > \frac{2}{z(1-z)}.$$

It is easiest to satisfy (8) when a second male displaces half of the first male's sperm z $(1-z)$ is maximized when $z = ½$). At this level, we need only that double matings are more frequent than 8 divided by the anisogamy ratio in order to be stable. This is admittedly less easy to satisfy than for vertebrates, but not such that anisogamy will be threatened. However, if displacement is very high (or alternatively, very low) then the product z $(1-z)$ becomes very small, and condition (8) progressively less easy to satisfy.

The highest degree of priority achieved by the last male to mate that has so far been recorded for an insect is 0.997 for the bug *Abedus herberti* (Smith, 1979). Even at this exceptional level of sperm displacement, the $m_{\min}$ strategy would be relatively safe, since the ratio of sperm size/ovum size is several orders of magnitude greater than 0.003, the product z $(1-z)$.

In short, variations in the exact pattern of sperm competition are unlikely to affect our general conclusion. Provided that even occasionally the sperm from more than one male compete over fertilizations, anisogamy is likely to be stable and sperm should not contain provisioning for the zygote. They should have minimal size.

4. AN ALTERNATIVE HYPOTHESIS

The classical interpretation of small sperm size is that the best chances of ensuring that an ovum gets fertilized occur when there are as many sperm as possible. By making sperm tiny, a maximum number can be produced; this maximizes the chances that one of them will find the egg.

As Cohen (1973) has plausibly argued, it is not easy to accept this solution for vertebrates since ejaculates can often be diluted vastly (for artificial insemination) without loss in fertility. It is also difficult to accept for insects because fertility usually decreases only when the sperm supply becomes very depleted and normally the female would either be dead, or would have remated before this stage is reached (e.g. Parker, 1970*b*). However, no model appears to have been devised to examine the classical

proposition that sperm are small simply to provide enough of them to ensure a high probability of fertilization. Note that this does differ from the sperm competition theory for the maintenance of small sperm size. The sperm competition model assumes that the probability of fertilization is independent of sperm numbers over a very wide range, and argues that sperm are small to produce high numbers to outcompete other ejaculates. The classical model ignores sperm competition and suggests that high sperm numbers are necessary for fertilization.

Suppose that the probability g of successful fertilization increases with increasing sperm numbers up to an asymptotic value of 1.0 (see Fig. 3). The maximum number of sperm that can be contained in an ejaculate is $m_{\min}^{-1}$ this gives the highest attainable probability of fertilization with a single mating. We again seek the condition under which the $m_{\min}$ strategy will be an ESS, and again assume that increasing the size of each sperm (by decreasing sperm numbers) can increase the survivorship prospects of the zygote.

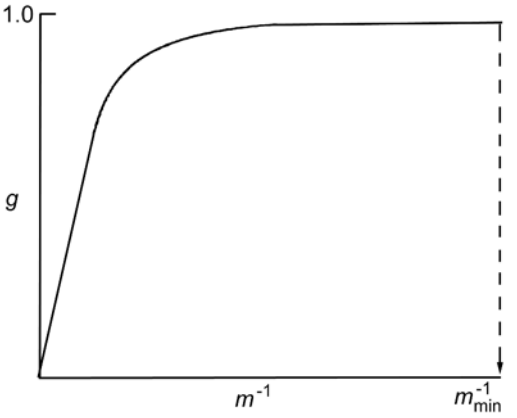

FIG. 3. Probability that an ovum is fertilized in relation to increasing sperm numbers in an ejaculate. There is no competition between ejaculates and the maximum number of sperm is proportional to $m_{\min}^{-1}$

Following earlier arguments, $m_{\min}$ will be stable if

$$g\left(m_{\min}^{-1}\right)F > g(m^{-1})\left[F + b(m)\right]$$

in which strategy m is again a rare mutant with $m > m_{\min}$. By the usual technique, stability of $m_{\min}$ occurs if

$$\frac{m_{f\text{opt}}}{m_{\min}} > \frac{g\left(m_{\min}^{-1}\right)}{g'\left(m_{\min}^{-1}\right)\cdot m_{\min}^{-1}}. \tag{9}$$

If we take the probability of fertilization to be about 1 at sperm number $m_{\min}^{-1}$ (for many species this seems to be a reasonable approximation), then in order for the classical theory to explain the maintenance of small sperm, we need the following rule to hold.

The anisogamy ratio (ovum size/sperm size) must exceed the reciprocal of the product of sperm number ($m_{\min}^{-1}$) and the gradient, $g'(m_{\min}^{-1})$. This gradient is the rate at which sperm number contributes to the probability of the fertilization, when sperm have minimal size $m_{\min}$. The fact that sperm dilution has little effect on the probability of fertilization suggests that $g'(m_{\min}^{-1})$ is very small.

We can see from the case of cattle that condition (9) could possibly account for the maintenance of anisogamy. The number of sperm ejaculated by a bull is $5–15 \times 10^9$ (Polge, 1972; Bishop, 1961). The sperm is one twenty thousandth the size of the bovine egg (Bishop & Walton, 1960), giving an anisogamy ratio of 2×10^4. The maximum probability of conception from a normal insemination appears to be around 0.75.

Substituting into (9), we need

$$g'\left(m_{\min}^{-1}\right) > \frac{0.75}{(5\times10^9)\times(2\times10^4)}$$

$$g'\left(m_{\min}^{-1}\right) > 0.75\times10^{-14}$$

for the classical theory to account for the maintenance of anisogamy. Data are available (from artificial insemination studies) about the way in which the probability of conception declines with increasing dilution of the ejaculate. Very roughly, there appears to be only a 1% drop in probability of conception as the number of sperm drops from 17×10^6 to 7×10^6 (Salisbury & Van Denmark, 1961). Thus $g'(m^{-1})$ over this range must be less than $0.01/10^7 = 10^{-9}$. We would therefore except that with a normal ejaculate, containing some thousand times more sperm, the gradient $g'(m_{\min}^{-1})$ must be well below 10^{-9}. Until further data are available which justify the fitting of an explicit form to $g(m^{-1})$, we are unable to make a firm conclusion as to whether the classical theory is robust enough to explain the extremely small size of the sperm. However, it seems likely that at least part of the reason for having tiny sperm relates to increasing the probability of conception.

5. WHY ARE THERE SO MANY SPERM?

Both sperm competition and the problem of increasing the chalice of conception will act to keep sperm size to a minimum. It is therefore easy to see why sperm does not contribute to the reserves necessary for zygote survival; i.e. why the male sex persists even when there is internal fertilization. Sperm stay tiny because it will pay, on a fixed resource budget, to produce as many of them as possible. The models described above tell us why the sperm-producing strategy persists, but tell us rather little about the actual number, or amount of sperm that should be produced. Why do males produce so many sperm?

Cohen (1969, and elsewhere) has proposed an ingenious and startling answer to this question. He suggests that males produce so many sperm because most sperm are defective. Defective sperm might arise from errors in meiosis; such errors could be so prevalent that only a tiny fraction of the sperm in each ejaculate are suitable for

fertilization. As evidence, Cohen found a highly significant correlation between mean chiasmata frequency and what he termed "sperm redundancy" (= number of sperm ejaculated/number actually used in fertilization).

However, Cohen's theory does not really answer the question "why so many tiny sperm?". Ova are also products of meiosis, and therefore should suffer the same risk of defectiveness. Cohen argues that this might indeed be the case, and suggests that the high prevalence of oocyte atrysia may be due to removal of these defective female gametes. Thus although he may be correct that gametes become increasingly defective with increasing chiasmata frequency, this is a side issue to the central problem of whether gametes should be tiny and unprovisioned, or large and highly provisioned. The assumption appears to be that because the ova are costly, it pays the female to sort out suitable ones before they are provisioned; sperm on the other hand are not expensive and so it need not pay the male to eliminate defective ones. This therefore prejudges the issue of why sperm are small and numerous.

We can best answer the question "why so many sperm?" by considering how much of his reproductive resources a male should allocate to an ejaculate. Given that each sperm will be tiny because of sperm competition, then if we find a male should invest a significant proportion of reproductive resources on an ejaculate, we can explain why vast numbers of sperm are produced.

In some sessile animals with internal fertilization, there may be no alternative reproductive strategy to profligate gamete production. Thus where males compete only by sperm competition, they will spend all their efforts on sperm and so that the total investment per male on sperm may approximate to that invested per female in ova; gametic masses should be roughly similar. Hence at high anisogamy ratios, sperm numbers will be high relative to ovum numbers.

The equal gametic expenditure rule breaks down if we allow alternative reproductive strategies for males, such as enhanced mobility for mate searching, etc. It also breaks down if sperm competition is not prevalent.

Consider the vertebrate model in which there is internal fertilization, and sperm competition occurs perhaps only rarely when the same female mates with two males with frequency p. We must trade off expenditure on sperm against expenditure on an alternative reproductive strategy such as mobility. Let us assume that if a male expends heavily on sperm, he does so at a cost in terms of the number of new females he is likely to encounter, because he has less resources left for mate-searching. Thus a mutant spending proportion k of his total reproductivity resources on sperm will obtain:

$$\frac{1-k}{1-k_*}$$

matings relative to each obtained by a normal male that expends k_* on sperm. In other words, the relative number of females encountered is directly proportional to the distance a male moves relative to other males. (This model may not be accurate if males obtain females by fights, though it may for various reasons serve as a reasonable approximation even then.) Once again, we assume that when two ejaculates compete, success is based

on the "raffle" principle, i.e. chances of fertilization are equivalent to (self's sperm number)/(total sperm number).

At the ESS expenditure k_* we require that

$$\underbrace{(1-p)+\frac{p}{2}}_{\text{mean fitness of population strategy } k_*} > \underbrace{(1-p)\left(\frac{1-k}{1-k_*}\right)}_{\text{mutant fitness through single matings}} + \underbrace{\frac{pk}{k_*+k}\left(\frac{1-k}{1-k_*}\right)}_{\text{mutant fitness through double matings}} .$$

For all $k \neq k_*$, the mutant fitness should be less than mean population fitness. Thus if we plot mutant fitness against k, the result should be a peak at $k = k_*$. Hence if k_* is an ESS then

$$\frac{\mathrm{d}}{\mathrm{d}k}\left[(1-p)\left(\frac{1-k}{1-k_*}\right)+\frac{pk}{k_*+k}\left(\frac{1-k}{1-k_*}\right)\right]\Bigg|_{k_*} = 0, \text{for } k = k_*.$$

and the second derivative of the left hand side should be negative, indicating that this is indeed a maximum rather than a minimum.

Differentiating, we obtain

$$k_* = \frac{p}{4-p} \tag{10}$$

or, for small p,

$$k_* \approx \frac{p}{4}$$

and the second derivative is negative, as required. This result suggests that a male should expend, of his total mating effort, a proportion k_* that is roughly equivalent to a quarter of the probability of double mating.

Consider red deer. Suppose that hinds are mated by two males as infrequently as once in a hundred occasions, which seems a very conservative estimate. Then a stag should spend a quarter of a percent of his total mating effort on sperm. Bearing in mind his immense energetic expenditure on antlers, roaring and fighting, large body size, and mate-searching, the estimate of a quarter percent gametic expenditure seems not to be at all excessive. Thus sperm competition may well account for why there are so many sperm, as well as accounting for the related question of why sperm are so tiny. Even though internal fertilization and reduced sperm competition may usually be associated with a reduction in male gametic expenditure, infrequent double matings can lead to non-trivial expenditures on sperm.

It would be interesting to investigate the degree to which sperm numbers correlated with increased sperm competition in internal fertilizers. Data on the incidence of double

matings in nature are not readily available. From Cohen's (1969) work, it is clear that insects produce far fewer sperm than mammals. Sperm storage organs in the female insect are highly developed compared to those in mammals. There is therefore little purpose in introducing vastly more sperm than can adequately fill these stores, unless extra sperm are needed to achieve "sperm flushing" during sperm displacement. In mammals, the situation is quite different; there is a vast genital tract in which sperm survive for (usually) a relatively short time. Here the "raffle principle" is likely to apply; i.e. the more sperm ejaculated, the better the chance of success when another male also mates with the same female.

ACKNOWLEDGEMENTS

Much of this work was done during a recent Dahlem Conference on Animal Mind–Human Mind; I hope it may in some way compensate for my singularly undistinguished performance there. I am indebted to Miss Jane Farrel for typing.

NOTE

This manuscript incorporates changes published in an Erratum (1982). *Journal of Theoretical Biology,* **98**, Issue 4, 707.

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