doi:10.1111/j.1558-5646.2012.01641.x

GENETIC CONFLICTS, INTRINSIC MALE FERTILITY, AND EJACULATE INVESTMENT

Leif Engqvist[1,2,3]

[1]*Centre for Ecological and Evolutionary Studies, Rijksuniversiteit Groningen, The Netherlands*

[2]*Evolutionary Biology, University of Bielefeld, Germany*

[3]*E-mail: leif.engqvist@uni-bielefeld.de*

Received June 15, 2011
Accepted March 3, 2012

Few aspects of biology are linked to so many evolutionary conflicts as sperm production and fertilization. Segregation distortion and maternal inheritance of cytoplasmic genes, causing maladapted males, are common sources of variation in the competitive ability of sperm, leading males to vary in their intrinsic fertility. Here, I theoretically analyze the effect of such variation in male intrinsic fertility on ejaculate investment. The model reveals that with increasing variation in male fertility, males should overall spend less resources on their ejaculates. Furthermore, if males differing in intrinsic fertility are able to invest differently in sperm production, there are two contrasting outcomes. Typically, less fertile males should invest more. However, if female mating frequency is relatively low and differences between males relatively large, the most common male genotype should invest more. These results have important consequences both for the understanding of sperm competition strategies as well as for the evolution of female polyandry and female mating preferences.

KEY WORDS: **Fertility, individual-based simulations, mother's curse, segregation distortion, sexual selection, sperm competition.**

When females mate with more than one male and sperm competition occurs (cf. Parker 1970), male reproductive success will not only be affected by males' access to female mating partners, but also by the success of their sperm in the subsequent competition for fertilizations. Sperm competition is thus a strong selective force that has considerable effects on many aspects of male reproductive biology (Parker 1970; Birkhead and Møller 1998; Simmons 2001; Birkhead et al. 2009). One interesting aspect of sperm competition that has received much theoretical and empirical attention regards what amount of their reproductive resources males should allocate to sperm production and how these sperm should be allocated to subsequent matings (Wedell et al. 2002; Parker and Pizzari 2010).

Many theoretical analyses on sperm competition have assumed that all males within a population are equivalent (Parker and Pizzari 2010), but this is most often not the case. Males might for instance differ in the competitive roles they occupy (Parker 1990a,b). However, males might also differ in other aspects, such as attractiveness, condition, or sperm quality and this is likely to have an effect on how they should invest their reproductive resources. In fact, Tazzyman et al. (2009) showed that males that differ in the cost of achieving matings (i.e., attractiveness) are predicted to differ in resource allocation to sperm competition: attractive males should invest less (see also Engqvist 2011). However, according to the study by Tazzyman et al. (2009), the amount of reproductive resources available should not produce selection for differing ejaculate investment strategies. Still, another study (Engqvist and Reinhold 2007) showed that male variation in sperm competitiveness (sperm reserves) has a strong effect on optimal sperm allocation. Here, I will analyze the common situation where there are differences in male intrinsic fertility affecting the sperm competitiveness of males.

A widespread origin of intermale variation in sperm competition is caused by genetic conflicts (Burt and Trivers 2006). Segregation distorters are "selfish genes" that are able to increase their transmission to future generations by killing or incapacitating the sperm that do not carry them, resulting in meiotic drive. Males heterozygous for segregation distorters

EVOLUTION
INTERNATIONAL JOURNAL OF ORGANIC EVOLUTION

consequently have reduced fertility (Burt and Trivers 2006; Price et al. 2008a; Price and Wedell 2008). With no associated costs, segregation distorters are expected to rapidly reach fixation within a population. However, if there are inherent costs for individuals carrying the segregation distorter, a polymorphism will be expected (Pomiankowski 1999; Weissing and van Boven 2001; Burt and Trivers 2006), with variation in male fertility as a result. Furthermore, cytoplasmic genetic elements, such as mitochondria, are typically exclusively maternally transmitted to the zygote, so mitochondrial function is selected only in females and not in males (but see Unckless and Herren 2009; Wade and Brandvain 2009). Nevertheless, there is ample evidence that sperm viability and motility is affected by mitochondria (Ruiz-Pesini et al. 2000; May-Panloup et al. 2003; Froman and Kirby 2005; Dowling et al. 2007b; Smith et al. 2010), which should have effects on the competition success of sperm (but see Dowling et al. 2007a; Friberg and Dowling 2008). Thus, male fertility might be reduced by suboptimal mitochondrial genes (Frank and Hurst 1996; Gemmell et al. 2004; Zeh 2004; Zeh and Zeh 2005; Dowling et al. 2008). If selection in females is weak or absent, a relatively large proportion of males are expected to carry these suboptimal mitochondrial genes (Frank and Hurst 1996; Innocenti et al. 2011), a phenomenon that has been referred to as mother's curse (Gemmell et al. 2004). Optimal resource allocation patterns might differ for such males compared to intrinsically more fertile males. For instance, should males that are intrinsically less fertile compensate and invest more in sperm traits, or instead concentrate investment on traits affecting mating success?

In this manuscript, I will analyze the effect of variation in male fertility driven by genetic conflicts on the evolution of sperm competition strategies. I will analyze two different scenarios. In a first basic model, males are not able to make their allocation strategy dependent on their "fertility genes." Thus, males do not "know" which genotype they belong to and will follow an unconditional strategy. Still, the population frequency of selfish genes (and overall fertility) can have an effect on the evolutionarily stable male ejaculate allocation. In the second model, it is assumed that males, which differ in intrinsic fertility, have conditional strategies. Thus, they have information about which genotype they belong to and can allocate their resources accordingly. This scenario would be particularly biologically relevant, if one assumes that epispastic interactions have evolved (i.e., genes coding for a resource allocation strategy are differently expressed depending on genetic background). In relation to selfish genetic elements, conditional strategies have been demonstrated for instance in mice, where individuals carrying the *t*-complex in many respects (e.g., aggression, mate choice) behave differently from wild-type mice (Lenington 1991; Lenington et al. 1996; Carroll et al. 2004).

Methods

BASIC MODEL STRUCTURE

In sperm competition, the best ejaculate strategy for a particular male will depend on the strategies used by his competitors. An evolutionarily stable strategy (ESS) approach (Maynard Smith 1982) is therefore often appropriate to analyze these problems (Parker 1998; Parker and Pizzari 2010). The focus here is on what quantity of his resources a male should allocate to the ejaculate in each mating. We denote an ejaculation strategy by $\hat{s}$ if it used by almost all males in the population (resident strategy). The reproductive success (w) of a male will be the product of the number of matings (n) and the expected value (fertilization success, v) of each one. For a male with a mutant ejaculate strategy s in a resident population, this amounts to: $w(s,\hat{s}) = n(s,\hat{s}) \cdot v(s,\hat{s})$. In line with many other sperm competition models (e.g., Parker 1998; Tazzyman et al. 2009), I will assume that there is a trade-off between investment in winning fertilizations (i.e., ejaculate investment) and obtaining matings. This trade-off is expressed as $R = n(c + Ds)$, where s is ejaculate size, R is the total amount of male reproductive resources, c is the cost of each mating, and D the cost of each ejaculate unit. If we instead measure R and c in units of D, this simplifies to $R = n(c + s)$. The relative number of matings of a mutant male with ejaculate size s in a resident population with ejaculate size $\hat{s}$ will thus be equal to

$$\tilde{n}(s,\hat{s}) = \frac{R/(c+s)}{R/(c+\hat{s})} = \frac{c+\hat{s}}{c+s}. \quad (1)$$

In all models, the success in sperm competition follows the principle of a raffle. However, it is assumed that a certain proportion of all males are carrying genes that affect their fertility. Thus although such males, here called type-2 males, invest the same amount of energy in sperm, they will have a fertilization disadvantage (or advantage) relative to type-1 males. This fertility disadvantage is represented by the parameter r ($r \geq 0$). To illustrate this, the fertilization success of a type-1 male with ejaculate investment s in competition with N other males, of which i are type-2 and $N - i$ are type-1 males, all with ejaculate investment $\hat{s}$, will be equal to

$$v_1(s,\hat{s}|\,N,i) = \frac{s}{s + i \cdot r\hat{s} + (N-i)\hat{s}}. \quad (2)$$

The corresponding fertilization success of a type-2 male will be

$$v_2(s,\hat{s}|\,N,i) = \frac{rs}{rs + i \cdot r\hat{s} + (N-i)\hat{s}}. \quad (3)$$

It is indirectly assumed here that populations are large enough in relation to female mating frequency that the probability of a mutant male mating more than once with the same female is small. The biological interpretation of the parameter r is the relative number (in relation to type-1 males) of sperm that will be present near the egg cell at the time of fertilization, hence, the relative

number of sperm that can potentially fertilize the ova. In case of segregation distortion, it will be a measure of how many male sperm are killed or incapacitated and therefore will not be able to fertilize the egg cell (i.e., the level of drive). In case of mtDNA, it will be a measure of how strongly the motility of the sperm is affected. Also note that a value of $r < 1$ corresponds to a disadvantage (subfertility) and $r > 1$ an advantage (superfertility) in relation to type-1 males. In all models it is assumed that a certain proportion of the population (q) will be type-2 males whereas the proportion of type-1 males will be $1 - q$. It is also assumed that this frequency is not affected by the evolutionarily stable ejaculation strategy; hence, it is assumed as a parameter in the models. Finally, it should be noted that a population consisting of a fraction q of type-2 males with intrinsic fertility r compared to type-1 males is mathematically equivalent to the situation where there is a fraction $1 - q$ type-2 males with relative fertility r^{-1}. It will thus be sufficient to analyze and present results for the case where type-2 males are subfertile ($r < 1$). Corresponding solutions for superfertile type-2 males ($\tilde{r} = r^{-1} > 1$) can easily be found.

NUMERICAL SOLUTIONS AND INDIVIDUAL-BASED SIMULATIONS

It is only possible to find analytical solutions of evolutionary equilibria, and thus potential ESSs, using very restrictive assumptions on female mating frequency (see Appendices A and B). Therefore, solutions were calculated numerically. In those situations where female mating frequency was assumed to follow a Poisson distribution, it was not possible to calculate exact numerical solutions. There, the upper tail of the distribution had to be disregarded for computational reasons. This refers to a very small fraction of females (cutoff at 10^{-9}) with exceptionally high mating frequency. Subsequently, invasion and convergence stability criteria (see Eshel 1983; Dieckmann and Law 1996; Geritz et al. 1998; Leimar 2009) were evaluated numerically. In all cases, it was verified that equilibria were both evolutionarily and convergence stable.

Additionally, individual-based simulations were performed to complement the mathematical results. The simulations relax the assumptions used in the analytical approach, that is, that mutations have small effect and only occur in resident populations in dynamical equilibrium. Details of the simulations are given in Appendix C.

Analyses and Results

MODEL 1: UNCONDITIONAL EJACULATE ALLOCATION

Here it is assumed that males have no information (cf. Parker 1990a) on which fertility genes they carry. We therefore search for a common unconditional ESS for all males given the frequency q of type-2 males in the population. The reproductive success of a mutant male in a resident population will be equal to

$$w(s, \hat{s}|q) = \tilde{n}(s, \hat{s}) \cdot \left[\sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N; i \mid q) \cdot ((1-q) \cdot v_1(s, \hat{s}|N, i) + q \cdot v_2(s, \hat{s}|N, i)) \right]. \quad (4)$$

Here, P_N is the probability that a given male mating will result in competition with the sperm from N other males (see also Engqvist and Reinhold 2006; Fromhage et al. 2008; Tazzyman et al. 2009), and $Bin(N; i \mid q)$ represents the binomial probability that a female mating with N other males will mate with exactly i type-2 males, given the population frequency q of such males. Note that males do not have any information on their own status and the probability that they will be type-1 males is $(1 - q)$, and q is the probability that they are type-2 males. In each case the male will have the fertilization success v_1 and v_2 (see eqs. 2 and 3), respectively.

We can find the ESS (s^*) by solving $\frac{\partial w(s,\hat{s})}{\partial s}|_{s=\hat{s}=s^*} = 0$. However, the solution is neither very pleasing to the eye nor very informative (see Appendix A). Nevertheless, it can be shown that $\frac{\partial s^*}{\partial q}|_{q=0} < 0$ and $\frac{\partial s^*}{\partial q}|_{q=1} > 0$ for any level of sperm competition P_N. This implies that deviations from a monomorphic population, where all males are equal, will always result in a decrease in ejaculate investment. Similarly, it can be shown (see Appendix A) that $\frac{\partial s^*}{\partial r}|_{r=1} = 0$ and $\frac{\partial^2 s^*}{\partial r^2}|_{r=1} < 0$. Thus, deviations in r away from $r = 1$ will result in a decrease in the ESS ejaculate investment. Importantly, the fact that there is a decrease is independent of whether variation is caused by sub- or superfertile type-2 males (i.e., whether $r < 1$ or $r > 1$). Furthermore, the deviation will increase with increasing variation in male intrinsic fertility ($\sigma^2 = q(1 - q)$), and increasing differences in fertility between males (increasing deviations from $r = 1$) (see Fig. 1 and Appendix A). Quantitative numerical solutions and individual-based simulations confirm these analytical conclusions (see Fig. 1). Using individual-based simulations, the conclusion that increased variation in intrinsic male fertility will lead to a decrease in ejaculate investment can also be made for the more general case, where there is a continuous fertility variation (see Fig. 2). This is a more realistic scenario in the case where fertility variation is caused by variation in mitochondrial function.

MODEL 2: CONDITIONAL EJACULATE ALLOCATION

Here it is assumed that the ejaculate strategy of type-1 males can be different from the ejaculate strategy of type-2 males. We are searching for an evolutionary equilibrium describing the joint ESSs for both types of males—s_1^* for type-1 males and s_2^* for type-2 males. The derivation of the fitness equations is quite

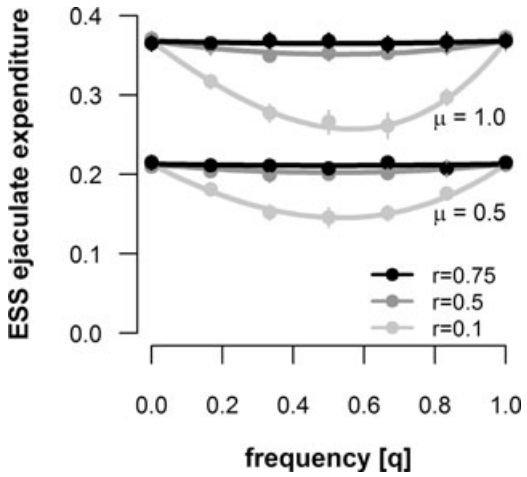

Figure 1. **ESS ejaculate investment for the unconditional model. The level of sperm competition is modeled using a full-range approach, where it is assumed that females mate at least once and the number of female additional matings follows a Poisson distribution with an expected number of μ additional mates. Lines depict analytical solutions and points and error bars show the mean ± SD outcome of the individual-based simulations ($n = 10$). Ejaculate expenditure is expressed relative to the total expenditure on a given mating ($s^*/c + s^*$). The upper set of lines show the result for $\mu = 1.0$ and the lower for $\mu = 0.5$. All results are calculated assuming $c = 1$. Qualitatively similar results can be obtained for other female mating frequency distributions. Here only results for subfertile ($r < 1$) type-2 males are shown. Corresponding results for superfertile ($r > 1$) type-2 males can be found by setting $\tilde{r} = r^{-1}$ and $\tilde{q} = 1 - q$.**

straightforward. First we assume that most type-1 males in the population follow the strategy $\hat{s}_1$, and most type-2 males the strategy $\hat{s}_2$. The fitness of a type-1 male following a mutant strategy s_1 in this population is given by

$$w_1(s_1, \hat{s}_1 \mid \hat{s}_2, q) = n_1(s_1, \hat{s}_1 \mid \hat{s}_2) \cdot \left(\sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N; i \mid \hat{q}') \cdot \frac{\mathrm{s}_1}{\mathrm{s}_1 + \mathrm{i} \cdot \mathrm{r}\hat{\mathrm{s}}_2 + (\mathrm{N} - \mathrm{i}) \cdot \hat{\mathrm{s}}_1} \right). \quad (5)$$

Correspondingly, the fitness of a type-2 male following a mutant strategy s_2 will be

$$w_2(s_2, \hat{s}_2 \mid \hat{s}_1, q) = n_2(s_2, \hat{s}_2 \mid \hat{s}_1) \cdot \left(\sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N; i \mid \hat{q}') \cdot \frac{\mathrm{rs}_2}{\mathrm{rs}_2 + \mathrm{i} \cdot \mathrm{r}\hat{\mathrm{s}}_2 + (\mathrm{N} - \mathrm{i}) \cdot \hat{\mathrm{s}}_1} \right). \quad (6)$$

Here $\mathrm{Bin}(N; i \mid q')$ represents the probability that a female mating with N other males will mate with exactly i type-2 males, given the probability $\hat{q}'$ that females will mate with such males. However, unless $\hat{s}_1 = \hat{s}_2$, $\hat{q}'$ will not equal q, the population frequency of type-2 males, because if different types of males invest differently in matings, their mating success will differ and the probability to compete against different types of males will change. We can represent the mating success of type-1 males by $\hat{n}_1 = R/(c + \hat{s}_1)$, and that of type-2 males by $\hat{n}_2 = R/(c + \hat{s}_2)$. Assuming that males with mutant ejaculate strategies are rare, the proportion of female matings that involve type-2 males will be given by $\hat{q}' = \frac{q\hat{n}_2}{q\hat{n}_2 + (1-q)\hat{n}_1}$.

We can find the ESSs by solving the following equation system

$$\begin{cases} \dfrac{\partial}{\partial s_1} w_1(s_1, \hat{s}_1 \mid \hat{s}_2, q)\Big|_{s_1 = \hat{s}_1 = s_1^*} = 0 \\ \dfrac{\partial}{\partial s_2} w_2(s_2, \hat{s}_2 \mid \hat{s}_1, q)\Big|_{s_2 = \hat{s}_2 = s_2^*} = 0 \end{cases}. \quad (7)$$

It is not possible to find analytical solutions to this. Still, it is straightforward to find numerical solutions for given values of r and q. It is also possible to gain some more general insight (see Appendix B), using perturbation analysis (Hinsch 1991). It can be shown that the outcome depends crucially on the level of sperm competition and the magnitude of the fertility difference between males. For small values of expected female mating frequencies (Figs. 3A–C, 4), males belonging to the less fertile genotype are expected to invest more in sperm except for relatively large deviations from $r = 1$. Then the most common genotype should invest more. For larger values of expected female mating frequency the resulting pattern changes (Figs. 3D–F, 4). Independent of their frequency, less fertile males are always predicted to produce larger ejaculates (Figs. 3D–F, 4).

Discussion

It was shown here that intrinsic differences in male fertility will affect overall male sperm investment. Variation in male fertility caused by for instance mtDNA mutations or selfish segregation distorters will have as an effect that males should overall spend less resources on ejaculate investment (Figs. 1, 2). The reason for this is that as intermale fertility differences become larger, fertilization success will be less influenced by ejaculate investment; instead intrinsic male fertility will dictate sperm competition success. In contrast, when all males are equal, ejaculate investment is the only influencing factor, and males are expected to invest more. If males can allocate resources to sperm investment dependent on their own fertility, it was predicted that less fertile males should partly compensate their disadvantage by investing more in sperm competition, unless differences between males are very large. Then the most common genotype should invest more. This can be related to a study by Engqvist and Reinhold (2007), where it was found that males should maximize investment against similar competitors. The reason for this result is that against identically strong competitors, the outcome of sperm competition will be

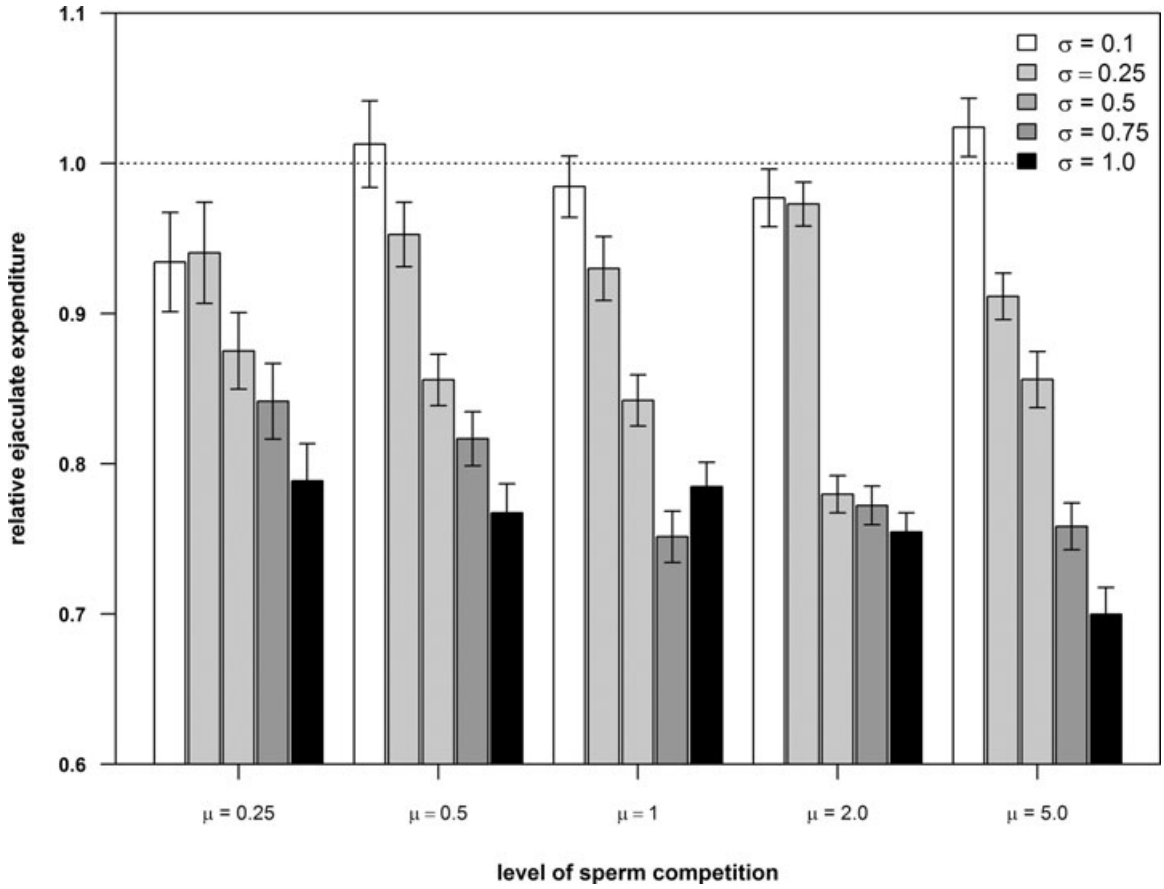

Figure 2. ESS ejaculate investment for the unconditional model when intrinsic male fertility is continuously distributed around a mean of $r = 1$. The value σ gives the standard deviation of the truncated Gaussian distribution (only positive values of r are possible). The value μ corresponds to the expected number of female additional mates (Poisson distributed). Results are given as the mean ± SE ejaculate strategy from individual-based simulations ($n = 20$ for each parameter combination). Values are given in relation to expected values given no variation in fertility (dotted line). The decrease in ejaculate expenditure with increasing variation in fertility was statistically highly significant for all values of μ (all $F_{1,98} > 19.5$, all $P < 0.0001$). The magnitude of this effect was stronger at higher levels of sperm competition (variation × female mating frequency: $F_{1,496} = 12.8$, $P < 0.001$).

determined by sperm investment only. In contrast, the outcome of a raffle between very dissimilar competitors will to a large extent be affected by the difference in intrinsic sperm competitiveness, and not so much by the actual investment in that particular mating (Engqvist and Reinhold 2007). If a male belongs to a common genotype, he will often face competition against similar competitors. Consequently, such males should invest more than rare genotypes in sperm competition. For rare males, which face sperm competition against dissimilar competitors, ejaculate investment will only weakly influence the outcome of sperm competition. Because they are rare, subfertile males will lose and superfertile males will win irrespective of their investment. As rare subfertile males will have such a low gain from their investment, they should instead strive to gain more matings and so increase their chances of fertilization from those matings not involving sperm competition. However, for inferior competitors the chance of gaining paternity without competition will become negligible as the degree of sperm competition increases. This is the reason why the pattern that the most common type should invest the most vanishes with increasing female mating frequency (see Figs. 3, 4). Rare subfertile males will then benefit by investing more in ejaculates, as this is the only way to gain any paternity at all. Superior competitors, however, will have a lower return from sperm investment as they will gain a large share of paternity nonetheless, and increasing investment will only weakly affect fertilization success. Thus, when sperm competition is common, subfertile males will always benefit more from sperm investment than superfertile males.

The situation analyzed here bears resemblance, both with respect to the competition structure and results, to the nonrandom loaded raffle with favored and disfavored roles (Parker 1990a). The rationale for that model was to analyze situations in which the first or the second male to mate may be favored. In the present analyses this would correspond to whether males are favored in

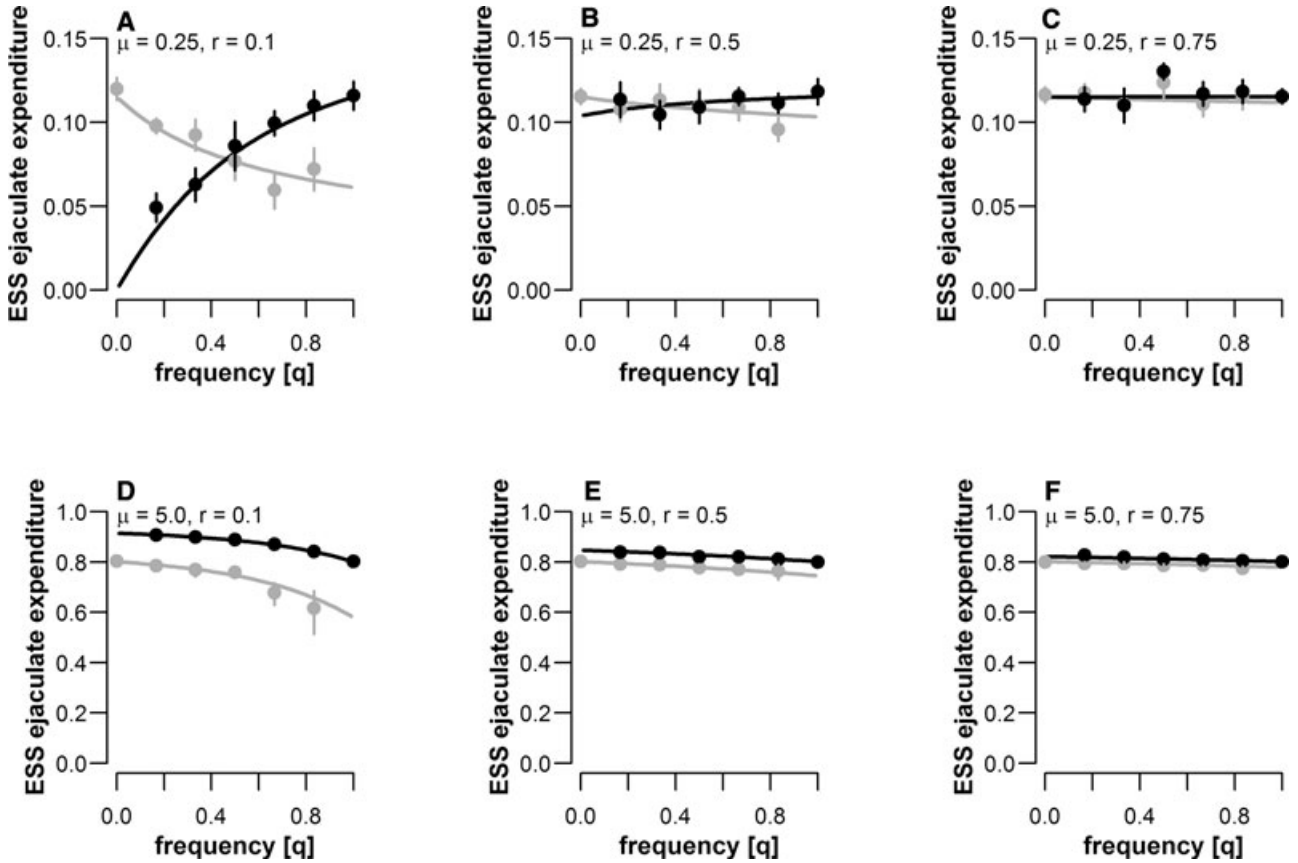

Figure 3. ESS ejaculate investment for type-1 males (gray) and type-2 (subfertile) males (black) in the conditional model. The level of sperm competition is modeled using a full-range approach, where it is assumed that females mate at least once and the number of female additional matings follows a Poisson distribution with an expected number of μ additional mates. Lines depict analytical solutions and points and error bars show the mean ± SD outcome of the individual-based simulations ($n = 10$). Ejaculate expenditure is expressed relative to the total expenditure on a given mating ($s^*/c + s^*$). Type-2 strategies at $q = 0$ and type-1 strategies for $q = 1$ are not shown, as they are not expressed in any individual, and hence there is no selection on these values. All results are calculated assuming $c = 1$.

sperm competition because of superior intrinsic fertility. However, this model cannot be directly applied to the present situation, as in a loaded raffle there is always competition between exactly one disfavored and one favored male. This is fundamentally different from the present situation where we have variation in male fertility. Here sperm competition can occur between any number of fertile and subfertile males, and even continuous variation in intrinsic fertility is possible. Nevertheless, two important predictions that are related to the present analyses can be inferred from the loaded raffle model (Parker 1990a, 1998; Parker and Pizzari 2010): (1) overall ejaculate investment should decrease with increasing "unfairness" of the game (see also Williams et al. 2005; Engqvist and Reinhold 2006; Fromhage et al. 2008), and (2) if males have information about their role, males in the disfavored role should invest more. It is interesting that in the different scenario analyzed here, both these predictions are reinforced, but with both extensions and modifications. Here it is shown that ejaculate investment should decrease with increasing differences (r) between male competitiveness (corresponding to unfairness). However, the effect of fertility variation, one of the central issues here, cannot be resolved from the loaded raffle model. The results presented here demonstrate that ejaculate investment is predicted to be lowest at intermediate frequencies of subfertile males (see Fig. 1) corresponding to the largest variation in male sperm competitiveness. In addition, this prediction could also be extended to the much more general case where there are not only two male types, but continuous variation in intrinsic male fertility (Fig. 2). The second prediction from the loaded raffle model that males in the disfavored role should invest more (Parker 1990a) was reflected in the present study with the prediction that, typically, less fertile males should invest more. Nevertheless, it is also shown here that this prediction is not general. When differences between males are large and sperm competition risk is low, the common male type should invest more irrespective of its disadvantage/advantage. The main reason behind the prediction that common males should invest more is that common males will more often compete against a competitor of similar strength. However, loaded raffles are always staged between a favored and

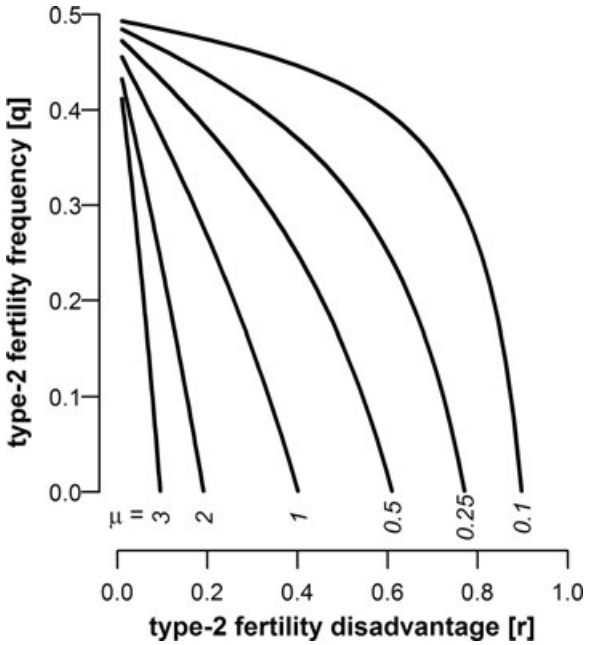

Figure 4. Lines showing, for any value of subfertility disadvantage, the frequency at which subfertile type-2 and type-1 males are expected to invest equally in ejaculates. The different lines indicate different levels of sperm competition. The value μ corresponds to the expected number of female additional mates (Poisson distributed). Above the lines subfertile type-2 males are expected to invest more, whereas below the lines superfertile type-1 males are expected to invest more. For *q*-values larger than 0.5, subfertile type-2 males are under all circumstances expected to invest more—they will both be less fertile and belong to the most common genotype.

disfavored male. Thus, competition between similar males never occurs and this provides a likely explanation for the differences in predictions.

In all analyses presented here, the population frequency of different genotypes was assumed to be fixed. Thus, changes in male sperm allocation strategies did not change this frequency. This situation most closely resembles the mother's curse situation with suboptimal mtDNA genotypes. In this situation intrinsic fertility will not be heritable, and thus not subject to evolutionary change due to selection (Frank and Hurst 1996; Gemmell et al. 2004; Zeh and Zeh 2005; Innocenti et al. 2011). However, the assumption that male sperm allocation strategies do not change the frequency of male fertility genotypes will be violated in the segregation distortion scenario. Here, the frequency of drive will be influenced by the ejaculate investment of different genotypes. Therefore, to completely understand the association between the occurrence of meiotic drive and male ejaculate investment, we need to develop more dynamic models, taking changes in the frequency of segregation distortion into account. Nevertheless, the present analyses do already give some insights and predictions for the relationship between meiotic drive and male ejaculate allocation. One would, for instance, expect that populations with high incidence of meiotic drive should invest less in sperm competition traits, and this effect should be more pronounced in systems with stronger drive. Furthermore, if males can allocate resources dependent on their own status, there are two contrasting scenarios. If females mate with many males and segregation distortion is relatively common in the population, we would expect less fertile males to invest more. However, with low risk of sperm competition and low frequency of segregation distortion, the opposite pattern is expected. There is to my knowledge no study explicitly demonstrating effects on sperm production and ejaculate investment in relation to segregation distortion (see review by Price and Wedell 2008). However, there are some indications that segregation distortion does indeed affect male reproductive investment. In mice, the *t*-complex is a segregation distorter that causes reduction in male fertility (Lyon 1987). Yet, male *t*-genotypes are more dominant resulting in better survival and mating success than wild-type males (Lenington et al. 1996; but see Carroll et al. 2004). Possibly, this is because these genotypes invest less in sperm competition traits and therefore have more resources available to secure matings. This would fit well with the predictions presented here, as the *t*-haplotype usually occurs at relatively low frequencies (Ardlie and Silver 1998) in a species with relatively low levels of sperm competition (Ardlie and Silver 1998; Dean et al. 2006). Furthermore, in *Drosophila* there is evidence that the fertility disadvantage of males carrying segregation distorters is more pronounced in multiple mating situations than for virgin males (Jaenike 1996; Atlan et al. 2004), indicating effects on sperm production.

The expectations for the situations when male fertility differences are caused by suboptimal mtDNA (i.e., mother's curse) are similar to the ones described for segregation distortion. However, in contrast to segregation distortion, polymorphism of mitochondrial genes affecting male fertility will be facilitated by random genetic drift processes. Fixation of neutral or near neutral alleles due to genetic drift will be less pronounced in large populations (Crow and Kimura 1970), and allelic variation is therefore expected to be larger in those populations. An interesting and novel prediction that can easily be tested would therefore be that males in larger populations invest less in sperm competition. However, size differences of populations must have a relatively long history, as fertility variation and accompanying ejaculation strategies must have time to evolve.

As in many sperm competition models (but see Williams et al. 2005; Fromhage et al. 2008), the effects of strategic allocation on female mating frequency are largely ignored in the present study. However, many of the processes analyzed here will certainly affect female mating behavior. Female polyandry might be expected to evolve as a response to decreased fertility caused

by for instance segregation distortion (Haig and Bergstrom 1995; Price et al. 2008b; Manser et al. 2011). If male fertility variation is associated with a reduction in male ejaculate investment, this effect will be amplified and females would benefit even more from multiple mating and an increased female mating rate is expected as a response to male strategic ejaculation. However, male strategic allocation might not only affect female mating rate but also preferences. In some situations, especially when female mating rate is low, we expect less fertile males to reduce investment in sperm competition and instead concentrate investment on traits affecting mating success, such as attractive ornaments. On the other hand females are expected to prefer fertile males (Keller and Reeve 1995; Arnqvist and Nilsson 2000; Jennions and Petrie 2000) and males lacking segregation distorters (Reinhold et al. 1999). Thus, male compensatory investment will be in conflict with the evolutionary interests of females, with interesting implications for the coevolutionary dynamics between male reproductive resource allocation patterns and female mate preferences. Possibly genetic conflicts might trigger the fast evolution of new female mating preferences (see also Wiens 2001; van Doorn and Weissing 2006), and such evolving preferences are likely to affect male sperm strategies (Tazzyman et al. 2012). Nevertheless, the present study was a first effort to investigate male ejaculate allocation in relation to genetic conflicts causing a reduction in male fertility. To deepen our understanding of the evolutionary causes and consequences of sperm competition, future studies should also aim to take the coevolutionary dynamics affecting male and female reproductive behavior into account.

ACKNOWLEDGMENTS

I would like to thank F. Weissing, A. Manser, P. Stockley, and two anonymous reviewers for helpful criticism on an earlier version of this manuscript.

LITERATURE CITED

Ardlie, K. G., and L. M. Silver. 1998. Low frequency of *t* haplotypes in natural populations of house mice (*Mus musculus domesticus*). Evolution 52:1185–1196.

Arnqvist, G., and T. Nilsson. 2000. The evolution of polyandry: multiple mating and female fitness in insects. Anim. Behav. 60:145–164.

Atlan, A., D. Joly, C. Capillon, and C. Montchamp-Moreau. 2004. Sex-ratio distorter of *Drosophila simulans* reduces male productivity and sperm competition ability. J. Evol. Biol. 17:744–751.

Birkhead, T. B., D. J. Hosken, and S. Pitnick, eds. 2009. Sperm biology: an evolutionary perspective. Academic Press, San Diego, CA.

Birkhead, T. R., and A. P. Møller, eds. 1998. Sperm competition and sexual selection. Academic Press, San Diego, CA.

Burt, A., and R. L. Trivers. 2006. Genes in conflict: the biology of selfish genetic elements. Harvard Univ. Press, Cambridge, MA.

Carroll, L. S., S. Meagher, L. Morrison, D. J. Penn, and W. K. Potts. 2004. Fitness effects of a selfish gene (the *Mus t* complex) are revealed in an ecological context. Evolution 58:1318–1328.

Crow, J. F., and M. Kimura. 1970. An introduction to population genetics theory. Harper and Row, New York.

Dean, M. D., K. G. Ardlie, and M. W. Nachman. 2006. The frequency of multiple paternity suggests that sperm competition is common in house mice (*Mus domesticus*). Mol. Ecol. 15:4141–4151.

Dieckmann, U., and R. Law. 1996. The dynamical theory of coevolution: a derivation from stochastic ecological processes. J. Math. Biol. 34:579–612.

Dowling, D. K., U. Friberg, and G. Arnqvist. 2007a. A comparison of nuclear and cytoplasmic genetic effects on sperm competitiveness and female remating in a seed beetle. J. Evol. Biol. 20:2113–2125.

Dowling, D. K., A. L. Nowostawski, and G. Arnqvist. 2007b. Effects of cytoplasmic genes on sperm viability and sperm morphology in a seed beetle: implications for sperm competition theory? J. Evol. Biol. 20:358–368.

Dowling, D. K., U. Friberg, and J. Lindell. 2008. Evolutionary implications of non-neutral mitochondrial genetic variation. Trends Ecol. Evol. 23:546–554.

Engqvist, L. 2011. Male attractiveness is negatively genetically associated with investment in copulations. Behav. Ecol. 22:345–349.

Engqvist, L., and K. Reinhold. 2006. Theoretical influence of female mating status and remating propensity on male sperm allocation patterns. J. Evol. Biol. 19:1448–1458.

———. 2007. Sperm competition games: optimal sperm allocation in response to the size of competing ejaculates. Proc. R. Soc. Lond. B 274:209–217.

Eshel, I. 1983. Evolutionary and continuous stability. J. Theor. Biol. 103:99–111.

Frank, S. A., and L. D. Hurst. 1996. Mitochondria and male disease. Nature 383:224.

Friberg, U., and D. K. Dowling. 2008. No evidence of mitochondrial genetic variation for sperm competition within a population of *Drosophila melanogaster*. J. Evol. Biol. 21:1798–1807.

Froman, D. P., and J. D. Kirby. 2005. Sperm mobility: phenotype in roosters (*Gallus domesticus*) determined by mitochondrial function. Biol. Reprod. 72:562–567.

Fromhage, L., J. M. McNamara, and A. I. Houston. 2008. Sperm allocation strategies and female resistance: a unifying perspective. Am. Nat. 172:25–33.

Gemmell, N. J., V. J. Metcalf, and F. W. Allendorf. 2004. Mother's curse: the effect of mtDNA on individual fitness and population viability. Trends Ecol. Evol. 19:238–244.

Geritz, S. A. H., E. Kisdi, G. Meszena, and J. A. J. Metz. 1998. Evolutionarily singular strategies and the adaptive growth and branching of the evolutionary tree. Evol. Ecol. 12:35–57.

Haig, D., and C. T. Bergstrom. 1995. Multiple mating, sperm competition and meiotic drive. J. Evol. Biol. 8:265–282.

Hinsch, E. J. 1991. Perturbation methods. Cambridge Univ. Press, Cambridge, U.K.

Innocenti, P., E. H. Morrow, and D. K. Dowling. 2011. Experimental evidence supports a sex-specific selective sieve in mitochondrial genome evolution. Science 332:845–848.

Jaenike, J. 1996. Sex-ratio meiotic drive in the *Drosophila quinaria* group. Am. Nat. 148:237–254.

Jennions, M. D., and M. Petrie. 2000. Why do females mate multiply? A review of the genetic benefits. Biol. Rev. 75:21–64.

Keller, L., and H. K. Reeve. 1995. Why do females mate with multiple males? The sexually selected sperm hypothesis. Adv. Study Behav. 24:291–315.

Leimar, O. 2009. Multidimensional convergence stability. Evol. Ecol. Res. 11:191–208.

Lenington, S. 1991. The t-complex: a story of genes, behavior, and populations. Adv. Study Behav. 20:51–86.

Lenington, S., L. C. Drickamer, A. S. Robinson, and M. Erhart. 1996. Genetic basis for male aggression and survivorship in wild house mice (*Mus domesticus*). Aggressive Behav. 22:135–145.

Lyon, M. F. 1987. Distorter genes of the mouse *t*-complex impair male fertility when heterozygous. Genet. Res. 49:57–60.

Manser, A., A. K. Lindholm, B. König, and B. C. Bagheri. 2011. Polyandry and the decrease of a selfish genetic element in a wild house mouse population. Evolution 65:2435–2447.

May-Panloup, P., M. F. Chrétien, F. Savagner, C. Vasseur, M. Jean, Y. Malthièry, and P. Reynier. 2003. Increased sperm mitochondrial DNA content in male infertility. Hum. Reprod. 18:550–556.

Maynard Smith, J. 1982. Evolution and the theory of games. Cambridge Univ. Press, Cambridge, U.K.

Parker, G. A. 1970. Sperm competition and its evolutionary consequences in the insects. Biol. Rev. 45:525–567.

———. 1990a. Sperm competition games: raffles and roles. Proc. R. Soc. Lond. B 242:120–126.

———. 1990b. Sperm competition games: sneaks and extra-pair copulations. Proc. R. Soc. Lond. B 242:127–133.

———. 1998. Sperm competition and the evolution of ejaculates: towards a theory base. Pp. 3-54 *in* T. R. Birkhead and A. P. Møller, eds. Sperm competition and sexual selection. Academic Press, San Diego, CA.

Parker, G. A., and T. Pizzari. 2010. Sperm competition and ejaculate economics. Biol. Rev. 85:897–934.

Parker, G. A., M. A. Ball, P. Stockley, and M. J. G. Gage. 1996. Sperm competition games: individual assessment of sperm competition intensity by group spawners. Proc. R. Soc. Lond. B 263:1291–1297.

———. 1997. Sperm competition games: a prospective analysis of risk assessment. Proc. R. Soc. Lond. B 264:1793–1802.

Pomiankowski, A. 1999. Intragenomic conflict. Pp. 121–152 *in* L. Keller, ed. Levels of selection in evolution. Princeton Univ. Press, Princeton, NJ.

Price, T. A. R., and N. Wedell. 2008. Selfish genetic elements and sexual selection: their impact on male fertility. Genetica 132:295–307.

Price, T. A. R., A. J. Bretman, T. D. Avent, R. R. Snook, G. D. D. Hurst, and N. Wedell. 2008a. Sex ratio distorter reduces sperm competitive ability in an insect. Evolution 62:1644–1652.

Price, T. A. R., D. J. Hodgson, Z. Lewis, G. D. D. Hurst, and N. Wedell. 2008b. Selfish genetic elements promote polyandry in a fly. Science 322:1241–1243.

Reinhold, K., L. Engqvist, B. Misof, and J. Kurtz. 1999. Meiotic drive and evolution of female choice. Proc. R. Soc. Lond. B 266:1341–1345.

Ruiz-Pesini, E., A. C. Lapeña, C. Díez-Sánchez, A. Pérez-Martos, J. Montoya, E. Alvarez, M. Díaz, A. Urriés, L. Montoro, M. J. López-Pérez, et al. 2000. Human mtDNA haplogroups associated with high or reduced spermatozoa motility. Am. J. Hum. Genet. 67:682–696.

Simmons, L. W. 2001. Sperm competition and its evolutionary consequences in the insects. Princeton Univ. Press, Princeton, NJ.

Smith, S., C. Turbill, and F. Suchentrunk. 2010. Introducing mother's curse: low male fertility associated with an imported mtDNA haplotype in a captive colony of brown hares. Mol. Ecol. 19:36–43.

Tazzyman, S. J., T. Pizzari, R. M. Seymour, and A. Pomiankowski. 2009. The evolution of continuous variation in ejaculate expenditure strategy. Am. Nat. 174:E71–E82.

Tazzyman, S. J., R. M. Seymour, and A. Pomiankowski. 2012. Fixed and dilutable benefits: female choice for good genes or fertility. Proc. R. Soc. Lond. B 279:334–340.

Unckless, R. L., and J. K. Herren. 2009. Population genetics of sexually antagonistic mitochondrial mutants under inbreeding. J. Theor. Biol. 260:132–136.

van Doorn, G. S., and F. J. Weissing. 2006. Sexual conflict and the evolution of female preferences for indicators of male quality. Am. Nat. 168:742–757.

Wade, M. J., and Y. Brandvain. 2009. Reversing mother's curse: selection on male mitochondrial fitness effects. Evolution 63:1084–1089.

Wedell, N., M. J. G. Gage, and G. A. Parker. 2002. Sperm competition, male prudence and sperm-limited females. Trends Ecol. Evol. 17:313–320.

Weissing, F. J., and M. van Boven. 2001. Selection and segregation distortion in a sex-differentiated population. Theor. Popul. Biol. 60:327–341.

Wiens, J. J. 2001. Widespread loss of sexually selected traits: how the peacock lost its spots. Trends Ecol. Evol. 16:517–523.

Williams, P. D., T. Day, and E. Cameron. 2005. The evolution of sperm-allocation strategies and the degree of sperm competition. Evolution 59:492–499.

Zeh, J. A. 2004. Sexy sons: a dead end for cytoplasmic genes. Proc. R. Soc. Lond. B 271:S306–S309.

Zeh, J. A., and D. W. Zeh. 2005. Maternal inheritance, sexual conflict and the maladapted male. Trends Genet. 21:281–286.

Associate Editor: P. Stockley

Appendix A

For the unconditional model, the fitness function is given by

$$w(s,\hat{s}|q) = \tilde{n}(s,\hat{s}) \cdot \left[\sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N;i\,|\,q) \cdot \left((1-q)\frac{s}{s+ir\hat{s}+(N-i)\hat{s}} + q\frac{rs}{rs+ir\hat{s}+(N-i)\hat{s}} \right) \right].$$

We can find the ESS (s^*) by setting $\frac{\partial w(s,\hat{s})}{\partial s}|_{s=\hat{s}=s^*} = 0$, which gives

$$\begin{aligned} &\partial \frac{\tilde{n}(s,\hat{s})}{\partial s}\bigg|_{s=\hat{s}=s^*} \cdot \left[\sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N;i\,|\,q) \right. \\ &\left. \cdot \left((1-q)\frac{s}{s+ir\hat{s}+(N-i)\hat{s}} + q\frac{rs}{rs+ir\hat{s}+(N-i)\hat{s}} \right) \right]_{s=\hat{s}=s^*} \\ &+ \tilde{n}(s,\hat{s})|_{s=\hat{s}=s^*} \cdot \left[\sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N;i\,|\,q) \right. \\ &\cdot \left((1-q)\frac{\partial}{\partial s}\left(\frac{s}{s+ir\hat{s}+(N-i)\hat{s}} \right) \right. \\ &\left. \left. + q\frac{\partial}{\partial s}\left(\frac{rs}{rs+ir\hat{s}+(N-i)\hat{s}} \right) \right) \right]_{s=\hat{s}=s^*} = 0. \end{aligned}$$

The first set of summations (without differentials) can be simplified to $\sum_{N=0}^{\infty} P_N (N+1)^{-1}$. After inserting $\tilde{n}(s,\hat{s}) = (c+\hat{s})/(c+s)$ (see eq. 1), $\partial\tilde{n}/\partial s = -(c+\hat{s})/(c+s)^2$, and differentiating and evaluating the remainder of the expression, we

can solve for s^*, which gives

$$s^* = c\frac{\displaystyle\sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N;i\,|\,q)\left(\frac{(1-q)\,(ir+N-i)}{(1+ir+(N-i))^2}+\frac{qr\,(ir+N-i)}{(r+ir+(N-i))^2}\right)}{\displaystyle\sum_{N=0}^{\infty} P_N \frac{1}{N+1} - \sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N;i\,|\,q)\left(\frac{(1-q)\,(ir+N-i)}{(1+ir+(N-i))^2}+\frac{qr\,(ir+N-i)}{(r+ir+(N-i))^2}\right)}. \tag{A.1}$$

Without specific knowledge of P_N, which characterizes the level of sperm competition and is affected by female mating frequency, it is obviously not possible to make any quantitative predictions. However, we can still gain some general qualitative insight into the ESS described in equation (A.1). If we define

$$f(q,r\,|\,N) = \sum_{i=0}^{N} \mathrm{Bin}(N;i\,|\,q)\left(\frac{(1-q)(ir+N-i)}{(1+ir+(N-i))^2} + \frac{qr(ir+N-i)}{(r+ir+(N-i))^2}\right),$$

the ESS can be written as

$$s^* = c\frac{\displaystyle\sum_{N=0}^{\infty} P_N \cdot f\,(q,r\,|\,N)}{\displaystyle a - \sum_{N=0}^{\infty} P_N \cdot f\,(q,r\,|\,N)} \text{ where } a = \sum_{N=0}^{\infty} P_N \frac{1}{N+1} > 0.$$

The main interest here is on the effect of male fertility differences, hence, on the effect of the parameters r and q. We can therefore next analyze how the ESS will change with the frequency type-2 males, by taking the partial differential with respect to q:

$$\frac{\partial s^*}{\partial q} = c\frac{\displaystyle a\sum_{N=0}^{\infty} P_N \cdot \frac{\partial}{\partial q}(f(q,r\,|\,N))}{\left[\displaystyle a - \sum_{N=0}^{\infty} P_N \cdot f(q,r\,|\,N)\right]^2}.$$

It is obvious that the denominator will always be positive, thus the qualitative behavior will be determined by the numerator alone. It can be shown that

$$\frac{\partial}{\partial q}\left(f\,(q,r\,|\,N)\right)\Big|_{q=0} = -\frac{N}{N+1}\cdot\left(\frac{r-1}{r+N}\right)^2,$$

which is negative irrespective of r and N $(N>0)$, hence $\frac{\partial s^*}{\partial q}|_{q=0} < 0$. Similarly, it can be shown that

$$\frac{\partial}{\partial q}\left(f\,(q,r\,|\,N)\right)\Big|_{q=1} = \frac{N}{N+1}\cdot\left(\frac{r-1}{Nr+1}\right)^2,$$

which is positive irrespective of r, from which follows that $\frac{\partial s^*}{\partial q}|_{q=1} > 0$. The implication of this is that deviations from a monomorphic homogenous population, where all males are equal, will always result in a decrease in ejaculate investment. This decrease is independent from whether variation is caused by sub- or superfertile type-2 males.

An alternative way to reach similar, yet even more far-reaching, conclusions is to take the partial derivatives of the ESS s^* with respect to r around $r = 1$:

$$\left.\frac{\partial s^*}{\partial r}\right|_{r=1} = c\frac{\displaystyle a\sum_{N=0}^{\infty} P_N \cdot \frac{\partial}{\partial r}(f(q,r\,|\,N))\Big|_{r=1}}{\left[\displaystyle a - \sum_{N=0}^{\infty} P_N \cdot f(q,r\,|\,N)\Big|_{r=1}\right]^2}.$$

It can be shown that $\frac{\partial}{\partial r}(f(q,r\,|\,N))|_{r=1} = 0$, and hence $\frac{\partial s^*}{\partial r}|_{r=1} = 0$. Thus very small differences in fertility between males will have a very small effect on the ESS. The second derivative can be simplified to

$$\left.\frac{\partial^2 s^*}{\partial r^2}\right|_{r=1} = c\frac{\displaystyle a\sum_{N=0}^{\infty} P_N \cdot \frac{\partial^2}{\partial r^2}(f(q,r\,|\,N))\Big|_{r=1}}{\left[\displaystyle a - \sum_{N=0}^{\infty} P_N \cdot f(q,r\,|\,N)\Big|_{r=1}\right]^2}$$

(where the simplification is facilitated due to the fact that $\frac{\partial}{\partial r}(f(q,r\,|\,N))|_{r=1} = 0$). Again it thus suffices to consider the numerator. It is straightforward to show that $\frac{\partial^2}{\partial r^2}(f(q,r\,|\,N))|_{r=1} = -\frac{2Nq(1-q)}{(1+N)^3}$, which is always negative, and hence $\frac{\partial^2 s^*}{\partial r^2}|_{r=1} < 0$.

Appendix B

For the conditional model, the fitness functions for type-1 and type-2 males are given by

$$w_1\,(s_1,\hat{s}_1|\hat{s}_2,q) = n_1\,(s_1,\hat{s}_1|\hat{s}_2) \cdot\left(\sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N;i|\hat{q}')\cdot\frac{s_1}{s_1+ir\hat{s}_2+(N-i)\hat{s}_1}\right) \tag{B.1}$$

and

$$w_2(s_2,\hat{s}_2|\hat{s}_1,q) = n_2(s_2,\hat{s}_2|\hat{s}_1) \cdot\left(\sum_{N=0}^{\infty} P_N \sum_{i=0}^{N} \mathrm{Bin}(N;i|\hat{q}')\cdot\frac{rs_2}{rs_2+ir\hat{s}_2+(N-i)\hat{s}_1}\right). \tag{B.2}$$

An evolutionary equilibrium corresponding to a potential ESS must satisfy

$$\left.\frac{\partial w_i}{\partial s_i}\right|_{s_i=\hat{s}_i=s_i^*} = 0 \quad \text{for} \quad i = \{1, 2\}. \tag{B.3}$$

The solution to this equation is more straightforward to interpret and also easier to obtain if we assume that there is a value m with the following property $m = \frac{s_2^*}{s_1^*}$, where $m \geq 0$. This value thus gives the relative sperm investment of type-2 males compared to type-1 males.

However, to proceed in this model we must start by defining P_N, which describes the level of sperm competition in equations (B.1) and (B.2). Dependent on mathematical tractability, this has been represented by risk models, where females mate once or twice, by intensity models where all females mate N times, and by so-called full-range models, where female mating frequency can effectively follow any distribution (Parker and Pizzari 2010). The qualitative outcome here depends on P_N, and I will therefore show analyses of all these scenarios and start with sperm competition risk.

In the sperm competition risk scenario, there is a probability (risk) p that a female will mate or has mated with another male. On average, females will thus mate $1 + p$ times. From a male perspective, a fraction $2p$ of these will result in competition with the sperm from one other male, whereas the rest $(1 - p)$ will not result in any sperm competition. Thus, in this case $P_0 = (1 - p)/(1 + p)$ and $P_1 = 2p/(1 + p)$ (see also Parker et al. 1997). If we insert this into equations (B.1) and (B.2), we can obtain a closed-form expression of equation (B.3), but this is unwieldy. Furthermore, closed-form solutions could not be obtained. Nevertheless, we can find an exact solution for the special case where $r = 1$. Thus, to attain some more general insight, we can find approximate solutions around this value by means of perturbation analysis (Hinsch 1991). For $r = 1$, all males are equal and the model is equivalent with a fair raffle model (Parker 1990a), and gives the solutions $m = 1$ and $s_1^* = cp/(2 - p)$. The latter is equivalent with the solution given by Parker et al. (1997), but there expressed in a different unit. The first two terms in the approximation will be $s_1^* \approx c\frac{p}{2-p} - (1 - r)c\frac{p^2q}{(2-p)}$ and $m \approx 1 + (1 - r)p/2$. The last expression gives the relative sperm investment. Thus, we can conclude that subfertile type-2 males with an $r < 1$ should invest more than type-1 males in sperm production and superfertile type-2 males with an $r > 1$ should invest less, at least as long as the difference in fertility is small. Hence, less fertile males are predicted to partly compensate their disadvantage by producing larger ejaculates. However, with increasing deviations from $r = 1$, we may need to take further terms of the approximation into account. The second order of m can be calculated as

$$m_2 = \frac{1}{16}(p + 2) \cdot (2p^2q - p^2 - 6pq + 5p + 4q - 2).$$

This term can indeed be both positive and negative and larger in magnitude than the first-order term. Thus, it is possible that $m < 1$ although $r < 1$ (and $m > 1$ although $r > 1$). One can show that for $0 \leq p \leq 1$ and $0 \leq q \leq 1$, $\frac{\partial m_2}{\partial q} > 0$ and $\frac{\partial m_2}{\partial p} > 0$. Thus, m, the relative investment of type-2 males will be lowest (and possibly <1) for small values of p and q (i.e., when they are rare). In words this means that when sperm competition risk is low enough and differences in fertility is sufficiently high, males belonging to the most common genotype should invest more.

If females typically mate with more than one male, the risk model will only poorly describe sperm competition. In the intensity model, the assumption is made that females mate with exactly $\tilde{N}$ other males. In that case the term P_N will be $P_N = \begin{cases} 1 \text{ for } N = \tilde{N} \\ 0 \text{ for } N \neq \tilde{N} \end{cases}$ For this model, it is also possible to find approximate analytical solutions by perturbation analysis. For $r = 1$ the model corresponds to the conventional intensity model (Parker et al. 1996), and the solution with these values, $s_1^* = \tilde{N}c$ (and $m = 1$), is equivalent with the solution given there. The first-order approximations are given by $s_1^* \approx \tilde{N}c - \frac{\tilde{N}qc}{2}(1 - r)$ and $m \approx 1 + \frac{1}{2}(1 - r)$. Thus, m will be relatively independent of the frequency q. In fact, q appears for the first time in the third-order term. Thus, in contrast to the results from the risk model, $m > 1$ for $r < 1$, and $m < 1$ for $r > 1$, even for large deviations from $r = 1$. Hence, independent of their frequency, less fertile males are predicted to compensate their disadvantage by producing more sperm.

For the full-range approach, one can assume that females mate at least once and the number of female additional matings follows a Poisson distribution with an expected number of μ. Thus on average females will perform $1 + \mu$ matings. From a male perspective, a fraction $(1 + N) \cdot \text{Pois}(N; \mu)$ of those will result in sperm competition with exactly N other males ($N = \{0, 1, 2, \ldots\}$), where $\text{Pois}(N; \mu) = \frac{e^{-\mu} \cdot \mu^N}{N!}$ is the probability that females will mate with $1 + N$ males. In that case the term P_N will be described by $P_N = \frac{(N+1) \cdot \text{Pois}(N;\mu)}{\mu+1}$. Here it is not possible to find an analytical solution even when $r = 1$. Therefore, we cannot perturb the solution. Nevertheless, we can examine the numerical solutions for different parameters. These analyses confirm the previous analytical results, but reveal no further major insights. For small values of expected female mating frequencies, the system behaves like a risk model. Males belonging to the less fertile genotype are expected to invest more in sperm except for relatively large deviations from $r = 1$. Then the most common genotype should invest more. For larger values of expected female mating frequency μ, the solutions become more and more

similar to the intensity solution. Independent of their frequency, less fertile males are predicted to produce larger ejaculates.

Appendix C

All simulations were made in discrete nonoverlapping generations. Populations had an equal sex ratio consisting of 10,000 individuals. At the beginning of each generation a fraction $1 - q$ of all individuals were assigned to be type-1 and the rest type-2. This was done to avoid random genetic drift during the simulations. The genes for sperm investment were coded as numbers representing the amount of reproductive resources spent on sperm. Mutation rate was set to 0.001 per gene locus and generation, and the standard deviation of the normally distributed mutation step was set to 0.01. If mutations rendered negative values, these were altered to zero.

Each generation consisted of a single reproductive bout. To begin with, the precise identities of all male mating partners were generated for all females. First, the actual number of male mating partners was determined as a random integer drawn from the probability function describing female mating frequency. Subsequently, the identity of each male was assigned by sampling from the available males. The probability that a given male was selected as a mating partner was proportional to the male's expected mating success (determined by his sperm allocation strategy—see eq. 1) in relation to the expected mating success of all males. (Thus, it can happen that males mate twice with the same female.) Each female produced exactly two offspring (to keep population size constant) and the probability that an offspring was sired by each specific male was determined as described in equations 2 and 3.

In the conditional model, males carried the genes for both strategies. However, only one was expressed depending on which fertility-type males belonged to. The recombination rate was set to 0.5. In the first generation of each simulation, the males were assigned a random allele. In the unconditional model, simulations ran for 1000 generations, after which the strategy values were evaluated. The models with two conditional strategies took somewhat longer to reach equilibria and simulations therefore ran for 5000 generations.