The evolution of polyandry: intrinsic sire effects contribute to embryo viability

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Abstract

Females typically mate with more than one male despite the costs incurred, thus questioning Bateman's principle. A series of genetic benefits have been proposed to account for the evolution of polyandry, including the acquisition of viability genes for offspring. The 'intrinsic male quality' hypothesis suggests that polyandry increases the probability that females produce offspring sired by males that bestow high viability on their offspring. Heritable variation in viability is the basic requirement for the occurrence of this genetic benefit. By using a half-sib breeding design with a species of cricket in which polyandry is known to increase hatching success, we present clear experimental evidence that intrinsic male quality contributes to embryo viability. Despite recent support for the evolution of polyandry based on compatibility of genotypes between males and females, we show that hatching success is not determined by an interaction between paternal and maternal genotypes but rather that sons inherit paternal genes that influence the viability of eggs laid by their mates. Moreover, our data implicate a potential role for indirect genetic effects of male accessory gland products on embryo viability. Additive genetic contributions to embryo viability may be an important factor underlying the frequently observed benefits of polyandrous behaviour.

Introduction

Sexual dimorphism in gamete size is thought to have created sexual conflict over reproduction. Typically, males increase their fitness by mating with many mates while females are expected to maximize their reproductive success with only one or a few matings (Bateman, 1948; Trivers, 1972). Consequently females have traditionally been viewed as coy and reluctant to accept multiple partners (Knight, 2002). Evidence that mating can incur costs for females has further supported this argument (Daly, 1978; Chapman *et al.*, 1995; Rolff & Siva-Jothy, 2002). Nevertheless, the females of many species mate with multiple partners (Smith, 1984; Birkhead & Møller, 1998; Simmons, 2001b). Explaining

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this discrepancy between observation and theory is currently receiving considerable attention.

Procurement of material benefits from males at copulation has been shown to contribute to the fitness of polyandrous females (Ridley, 1988; Arnqvist & Nilsson, 2000). However, material benefits cannot explain polyandry in the majority of species where females do not receive direct benefits from mating. Thus, a series of genetic benefits have been proposed for the evolution of polyandry, including the 'intrinsic male quality' and 'genetic incompatibility' hypotheses (Curtsinger, 1991; Watson, 1991; Keller & Reeve, 1995; Zeh & Zeh, 1996, 1997; Yasui, 1997, 1998; Jennions & Petrie, 2000; Fox & Rauter, 2003).

The intrinsic male quality hypothesis proposes that polyandrous females can increase the probability that their eggs are fertilized by males of superior genetic quality. The hypothesis includes both pre- and post-copulatory mechanisms of female choice. Traditional precopulatory female choice could allow females to

'trade-up' when a male is deemed of superior genetic quality to a female's previous mate/s as indicated by the expression of his secondary sexual trait (Kempenaers *et al.*, 1992; Jennions & Petrie, 2000). In this paper we focus on the potential postcopulatory mechanism. That is, females could potentially increase the genetic quality of their offspring, even in the absence of indicator mechanisms, by facilitating sperm competition and/or cryptic female choice (Birkhead *et al.*, 1993; Keller & Reeve, 1995; Yasui, 1997). For example if sperm successful in sperm competition were also more effective in producing viable offspring, then polyandry would increase the average viability of offspring. Alternatively, in the absence of a link between sperm competitiveness and heritable viability, polyandrous females could increase the probability that some of their offspring were sired by males with high viability and thereby increase the average viability of their offspring (Yasui, 1998; Fox & Rauter, 2003). Both mechanisms require heritable variation in offspring viability.

A second genetic benefit hypothesis for the evolution of polyandry is the incompatibility avoidance hypothesis, which argues that offspring viability depends on an interaction between parental genotypes. Genetic benefits could thus be obtained from polyandry if the sperm of males with genetic elements that are compatible with a female are more likely to fertilize her eggs (Olsson *et al.*, 1996; Zeh & Zeh, 1996, 1997; Tregenza & Wedell, 1998, 2000, 2002; Newcomer *et al.*, 1999).

Both correlational (Madsen *et al.*, 1992; Kempenaers *et al.*, 1999) and experimental studies (Tregenza & Wedell, 1998; Newcomer *et al.*, 1999; Simmons, 2001a; Fedorka & Mousseau, 2002) have found evidence that polyandrous females obtain benefits of increased egg or embryo viability. The results from these studies are increasingly being taken as support for the genetic incompatibility hypothesis although in many cases genetic compatibility represents the null hypothesis for explaining elevated hatching success where indirect tests for intrinsic male differences have failed. Nevertheless, there is evidence from studies of precopulatory female choice that intrinsic genetic differences between males can contribute to offspring fitness (Petrie & Williams, 1993; Møller & Alatalo, 1999; Jennions & Petrie, 2000), and comparisons of naturally 'cross-fostered' maternal half-siblings have demonstrated that there can be strong paternal genetic effects on characters related to fitness (Sheldon *et al.*, 1997). Yet no study has directly examined whether intrinsic differences between males can account for embryo viability within a quantitative genetic framework.

The females of several species of gryllid cricket have been found to accrue fitness benefits from polyandry in the form of increased hatching success (Tregenza & Wedell, 1998; Fedorka & Mousseau, 2002). Consistent with these studies, the proportion of eggs hatching is greater for polyandrous than for monandrous females of the Australian field cricket *Teleogryllus oceanicus* (Le Guillou) (Simmons, 2001a). Eggs fail to hatch not because they are unfertilized but because embryos die during development (Simmons, 2001a). We have conducted an experiment to examine whether increased embryo viability in this species stems from heritable differences between males or from the avoidance of reproductive failure due to genetic incompatibilities.

Materials and methods

Breeding design, matings and hatching success

We used a half-sib breeding design to test for additive genetic variance in a male's ability to induce high hatching success in his mates. Experimental crickets were obtained from an outbred laboratory stock that were the fourth generation from 120 adult females collected in Carnarvon, North Western Australia.

Crickets were bred in plastic containers, fed with cat chow *ad libitum* and supplied with a Petri dish containing a pad of moist cotton wool for oviposition. Sexes were separated before the penultimate instar and 30 males and 120 females were collected at random from the stock populations. Each male was provided with four females and housed in a 1 L container for 7 days to ensure matings. Females were then housed individually and allowed to oviposit for 21 days. Resultant nymphs were kept in 5 L containers and again the sexes were separated before the penultimate instar. We originally generated 22 sire families and 66 dam families distributed in groups of three per sire.

We used sons from each dam family in mating trials to assess the hatching success of females with which they were paired. Moreover, to partition variance in hatching success between potential genetic effects of intrinsic male quality (sire effects) and potential genome wide genetic incompatibilities between males and their mates, the sons from each dam family were mated to one of two groups of females that were either relatively similar or more variable in their genetic backgrounds. Thus, from each dam family two sons were selected at random. One son was mated twice to each of three full sibling females while the other was mated twice to each of three unrelated females, subject to the criteria that the source families for both related and unrelated sets of females were not related to the male being tested. Matings were carried out in small plastic boxes (7 cm × 7 cm × 5 cm). Pairs were observed closely to ensure matings occurred. After mating the males were left to guard the females for 40 min thus preventing females from removing the spermatophore. To standardize the volume of ejaculate and numbers of sperm received by each female, spermatophores were experimentally removed after 40 min and the pair left until they mated a second time (in crickets sperm and seminal fluid are transferred simultaneously, Khalifa, 1949; Simmons, 1986). The second

spermatophore was also removed after 40 min. For any given duration of spermatophore attachment there is no significant among male variation in the number of sperm transferred (Simmons *et al.*, 2003). After mating males were left to recover for a period of 10 days before being frozen. They were later thawed, weighed to the nearest 0.01 mg before being dissected to determine the wet weight of the accessory gland, and the width of their pronotums measured to the nearest 0.01 mm using digital calipers. After their matings, females were provided with a Petri dish containing damp sand and allowed to oviposit for 10 days before being frozen. A total of 396 females were mated twice to 132 different sons.

Eggs were rinsed from the sand and a random sample placed onto moist filter paper and incubated at 25 °C. The mean number of eggs screened per female was 292.8 ± 5.6, range 23–735 (only 4.5% of females had egg counts lower than 100). Hatching was checked every other day until two weeks after the last nymph emerged. The proportion of eggs hatching was arcsine transformed for statistical analysis and all means are presented with ±1SE. Data were analysed using a mixed model nested analysis of variance with female relatedness group as a within subject factor.

Sperm transfer and viability

We detected a high incidence of hatching success equal to zero: 30.5% (121 of 396) of females did not produce any viable eggs. To control for sperm transfer failures or male azoospermia we dissected these females and checked for sperm in the spermatheca. Cases in which mating had taken place without sperm transfer accounted for only 14% of females for whom hatching success was zero. Two sire families consistently presented sons incapable of transferring sperm to the female and these families were excluded from the analysis, as were eight other cases that did not involve sperm transfer that were distributed randomly across different sire families. The final sample size in our analysis was thus reduced to 352 females mated twice to 120 different sons, distributed across 60 dams nested within 20 sire families.

For 15 sire families we examined the viability of sperm produced by four brothers of the males in the hatching success experiment. To assess sperm viability, males were paired with a random female and observed to ensure that spermatophore transfer took place. Males were then left undisturbed for two hours to produce a second spermatophore. This spermatophore was removed and ruptured in 20 μL of Beadle saline. After establishing the optimal amounts of live/dead stain reagents required (Damiens *et al.*, 2002), 5 μL of sperm were mixed with an equal volume of 1 : 50 diluted 1 mM SYBR-14 (Live/Dead sperm viability assay, Molecular Probes) and left in the dark for 10 min before adding 2 μL of 2.4 mM Propidium Iodide. The sample was incubated in the dark for 10 min and then observed under a fluorescence microscope

(blue excitation filter at $\lambda = 490$ nm). 500 sperm per sample were scored to obtain proportions of live (green stained) and dead (red stained) sperm.

Results

Mean hatching success across all females ($n = 352$ females) was 17 ± 1.2% with a wide range of variation (0–87.8%). There was a significant effect of sire ($F_{19,40} = 1.98$, $P < 0.05$, Cohen's standardized effect size (Cohen, 1988), $d = 0.484$) on the proportion of eggs that hatched, suggesting the presence of significant genetic variance for embryo viability (Fig. 1). The dam effects were not significant ($F_{40,40} = 0.58$, n.s., $d = 0.381$). The genetic incompatibility hypothesis predicts that embryo viability depends on an interaction between sire and dam genotypes. Thus, hatching success should vary less across a male's mates when they are from a similar genetic background than when they have relatively greater genetic variability. Thus, the within subject variance in hatching success for sons mated to three unrelated full sibling females should be lower than for their brothers mated to three unrelated females. The within subject effect of female relatedness group was not significant ($F_{1,40} = 1.55$, n.s., $d = 0.098$). Critically for distinguishing between the intrinsic male quality and the incompatibility hypotheses, the sire by female relatedness group interaction was not significant ($F_{19,40} = 0.78$, n.s., $d = 0.304$). We also calculated the standard deviation of the hatching success of males mated to females in each relatedness group. The genetic incompatibility hypothesis predicts that the standard deviation in hatching success should be significantly lower for males mated to groups
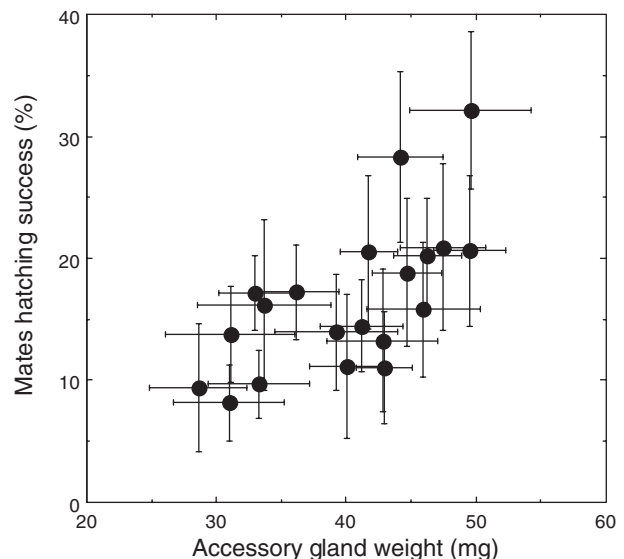
Fig. 1 Genetic covariance between a male's accessory gland weight and the hatching success of eggs laid by his mates. Data are the sire family means (±1SE).

of females who are sisters compared with males mated to groups of unrelated females, because the later should have greater genome wide variability. Again, there was no significant difference in standard deviation between female relatedness groups (the mean ± SE standard deviation across sisters was 17.37 ± 1.79 and across unrelated females was 18.03 ± 1.79; $F_{1,40} = 0.07$, n.s., $d = 0.024$), and again the interaction term between sire and female relatedness group was not significant ($F_{19,40} = 0.74$, n.s., $d = 0.343$).

Patterns of genetic variance in hatching success were assessed from the average hatching success attributable to each son across his different mates, regardless of female relationship group. The genetic analysis was thus fully balanced with two sons per dam family, and three dams per sire. Variance components were extracted from a nested analysis of variance with sire as the main effect and dams nested within sires as a random factor (Becker, 1984). As in our full analysis, there was a significant effect of sire ($F_{19,40} = 2.015$, $P < 0.05$) and no significant effect of dam ($F_{40,60} = 0.613$, n.s.) on the average hatching success across a male's mates. The narrow sense heritability of hatching success was 0.46 ± 0.29. Coefficients of variation were calculated following the method proposed by Houle (1992). There were high levels of additive genetic variation ($CV_A = 21.10$), residual variation ($CV_R = 58.86$), and total phenotypic variation ($CV_P = 62.53$). Sperm viability could not account for the observed variability in hatching success. Sperm viability was high ($84.4 \pm 0.5\%$ live sperm, range 61–96%) and sire family mean sperm viability did not predict sire family mean hatching success ($F_{1,13} = 0.02$, n.s.). However, accessory gland products could influence the observed patterns in hatching success. There was a strong and significant sire effect on male investment into the accessory glands ($F_{19,37} = 2.16$, $P < 0.05$, $d = 0.655$), the narrow sense heritability being 0.85 ± 0.55 ($CV_A = 11.08$, $CV_R = 21.30$, $CV_P = 24.01$). Moreover, there was significant sire covariance between hatching success and accessory gland weight (nested ANCOVA, $F_{19,43} = 4.18$, $P < 0.0001$) and a significant correlation between the sire family means of these traits (+0.662, $n = 20$, $P < 0.01$; see Fig. 1). The sire family mean correlation provides a conservative estimate of the underlying genetic correlation (Lynch & Walsh, 1998). Using the ANCOVA method (Becker, 1984) the estimate of the genetic correlation was 0.793 ± 0.452. There was no genetic variance due to sire for either soma weight (calculated as body weight minus the sum of the weights of the testes and accessory glands) or testes weight (soma weight: $F_{19,37} = 0.89$, n.s.; testes weight: $F_{19,37} = 1.06$, n.s.), in agreement with previous results (Simmons, 2003). Moreover, the sire effect on male investment into the accessory glands ($F_{19,37} = 2.49$, $P < 0.01$, $d = 0.696$) and the significant covariance between hatching success and gland weight ($F_{19,42} = 3.19$, $P < 0.001$) persisted when soma weight was entered into the analyses as a covariate.

Discussion

Our experiments provide a direct test of two hypotheses for the evolution of polyandry: the hypothesis that polyandrous females can ensure their offspring are sired by males with intrinsically high genetic quality, and the hypothesis that polyandrous females enhance offspring viability by avoiding reproductive failure resulting from incompatible parental genomes. The data show that intrinsic differences between males do contribute to embryo viability, and thus hatching success, while the evidence that embryo viability depends on an interaction between male and female genotypes is weak.

A necessary condition for the intrinsic male quality hypothesis is additive genetic variance for male effects on offspring fitness. Our experimental design has shown that embryo viability has a substantial sire component. There were high levels of additive genetic variation, residual variation, and total phenotypic variation in hatching success. These patterns are consistent with the general observation that fitness traits such as fecundity and longevity show high CV_As and CV_Rs and a corresponding high evolvability (Houle, 1992) because of their greater mutational variability (Houle et al., 1996). Potential direct benefits of polyandry arising from avoidance of infertility (Sheldon, 1994; Arnqvist & Nilsson, 2000) cannot contribute to the variation in hatching success observed here because we excluded from our analysis cases in which there was no sperm present in the spermatheca at post-mortem. Furthermore, sperm viability did not predict hatching success for 15 sire families examined. This is in agreement with the results shown elsewhere that hatching failure arises primarily from embryo failure and not because eggs are unfertilized (Simmons, 2001a).

The average hatching success across all females was just 17%. The females in this study were mated monandrously and the low average hatching success is consistent with previous studies of this (Simmons, 2001a) and other species (Tregenza & Wedell, 1998; Fedorka & Mousseau, 2002) that show low embryo viability for monandrous females. Our data also show that some males can induce hatching success as low as 0% and as high as 88% so that by mating polyandrously a female will increase the chance that at least some of her mates will be capable of inducing high embryo viability. The average hatching success of polyandrous females would therefore be expected to be higher than that of the monandrous females reported here. Indeed, previous studies of *T. oceanicus* have shown that polyandrous females have a higher hatching success than monandrous females (Simmons, 2001a).

Although our results suggest that the variation in hatching success due to sires is additive genetic in nature, the precise mechanism underlying the sire effect must remain equivocal. First, there may be linkage between genes that determine fertilization success and offspring viability, the so-called good sperm process (Yasui, 1997;

Hosken *et al.*, 2003). This seems unlikely because a previous study of *T. oceanicus* failed to find an association between a male's competitive fertilization success and the hatching success induced in his mates (Simmons, 2001a). Second, additive genetic variation in hatching success due to sires could arise because of genes that control development of the embryo *per se*, a pure 'good genes' mechanism. Females could then mate with different males to reduce variance in embryo viability, and in this way increase long term average fitness (Yasui, 1998; Fox & Rauter, 2003). However, an alternative, although not mutually exclusive explanation, is that the sire effect indirectly influences embryo survival via effects on maternal contributions to eggs (see also Simmons, 2001a). One possible candidate for such an effect could be the accessory gland products produced by males. Accessory gland products (Acps) have been identified in *Teleogryllus*. Proteins secreted by the accessory gland not only contribute to the construction of the spermatophore (Loher & Edson, 1973) but they are incorporated into the ejaculate, together with prostaglandin synthetase from the testes, and converted in the female to prostaglandin that stimulates vitellogenesis and oviposition (Stanley-Samuelson & Loher, 1983, 1986; Stanley-Samuelson *et al.*, 1986, 1987). Consistent with previous studies (Simmons, 2003), we found considerable genetic variation in accessory gland weight. Recent work with seed beetles has revealed genetic variation for ejaculate effects on egg production by females (Czesak & Fox, 2003). In addition to increasing the quantity of eggs produced by the female, it is possible that the quantity and/or quality of Acps produced in the glands of *T. oceanicus* might increase the quality of eggs and thereby enhance the viability of embryos developing within them. The strong genetic correlation between hatching success and male accessory gland weight found in *T. oceanicus* is consistent with such an explanation and suggests that part of the sire effect observed here could represent a paternal indirect genetic effect on offspring viability. Indirect genetic effects hold important implications for evolution because traits that have little or no direct genetic basis can change across generations if the environment that contributes to such traits has a genetic basis (Mousseau & Fox, 1998; Wolf *et al.*, 1998; Qvarnstrom & Price, 2001). Further studies of the effect of accessory gland weight on the production of Acps and their effects in *T. oceanicus* will be required to distinguish between direct and indirect sire effects on elevated hatching success.

Recently it has been shown that female blue tits, *Parus caeruleus*, increase offspring heterozygosity and fitness through extra-pair matings (Foerster *et al.*, 2003), supporting the idea that polyandry has arisen to allow females to obtain compatible genes for their offspring. However, support for the intrinsic male quality hypothesis in blue tits comes from the same population of birds since females also pursue extra-pair copulations with older and larger close neighbours that do not lead to increased offspring heterozygosity but are nevertheless associated with enhanced offspring fitness (Foerster *et al.*, 2003). It seems likely that mate choice for genetic compatibility and good genes work together, as has recently been shown in mice (Roberts & Gosling, 2003). While our results show a clear sire effect on viability benefits for the offspring of polyandrous crickets, we are cautious in rejecting the possibility that an interaction between parental genotypes might also contribute to offspring viability. Our test for genetic incompatibility was based on the use of sisters as individuals with a relatively similar genetic background in contrast to unrelated females who differed in their genetic background (see also Wilson *et al.*, 1997). If genetic incompatibility were determined by a single locus with two alleles, rather than by multiple loci with many alleles, our protocol would be less effective in detecting a male × female interaction in determining hatching success. Nevertheless, in a previous study of *T. oceanicus*, hatching success was shown not to be related to paternity skew in double mated females; if females increase hatching success through incompatibility avoidance, paternity should have been skewed toward the male with a compatible genotype (Simmons, 2001a).

In conclusion, our data show that there are intrinsic differences between males in their ability to sire offspring with high embryo viability. These sire effects may be directly and/or indirectly genetic in nature and polyandrous females can acquire these effects to elevate the viability of their offspring (Simmons, 2001a). While we would not reject the possibility that incompatibility avoidance could also contribute to hatching success, our data show that sire effects on hatching success are larger in magnitude than potential incompatibility effects, and moreover that they are not dependent upon variation in female genotype. The intrinsic male quality hypothesis could explain polyandrous behaviour in many taxa, including socially monogamous species and those for whom reliable indicators of intrinsic quality are lacking.

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