Females aren't perfect: maintaining genetic variation and the lek paradox

The evolution of lek mating systems and female choice for 'good genes' has received unparalleled inquiry on both theoretical and empirical fronts in evolutionary ecology over the past decade. On a lek, males typically congregate and display exaggerated sexual traits (for example the elaborate trains of male peacocks) to choosy females, which receive only sperm (genes) from their mate. Females generally prefer males with the most exaggerated trait, presumably because these males have the highest quality genes. Population genetic theory predicts that unanimous female choice for these 'popular' males will, however, lead to rapid loss of variation in male genetic quality and, subsequently, females will gain nothing from their choice. This conundrum has been termed the 'paradox of the lek'. To date, the most convincing resolution of the paradox has assumed that exaggerated sexual traits are controlled by relatively large numbers of loci and, consequently, high mutational input maintains the genetic variation. Empirical evidence shows that sexual traits do, in fact, exhibit greater genetic variation than comparable nonsexual characters. Thus, females are choosing for good genes (or avoiding bad genes) in their mates. An alternative to mutational input, the increased genetic variation in sexual traits could be maintained by weaker selective loss; for example, if females occasionally made mistakes when choosing their mate. However, this simple alternative solution to the lek paradox has received little attention. That is, until now.

James Randerson, Francis Jiggins and Laurence Hurst¹ model the evolution of a choice gene that allows discrimination between good and bad genes in potential mates. They also show that the stable maintenance of both genetic variation and the choice gene is possible, as long as the choosing individuals make occasional mistakes when discriminating between mates. They use the intriguing system of the butterfly Acraea encedon, which, owing to a malekilling bacterium, has sex-role-reversed leks (females display to choosy males). In their model, the bad gene is represented by the bacterial infection, which is 'inherited' from mother to offspring through the egg. They then introduce a mutant choice gene that enables males to differentiate between infected and uninfected females and show that it can successfully invade into the population. Interestingly, if male discrimination is perfect then the infection is always ousted from the population. However, if there is even a small amount of error males occasionally mate with infected females or occasionally reject uninfected females then both infected and uninfected individuals, as well as the choice gene, are retained in the population. Generally, as the error rate increases so does the frequency of infection.

Thus, error-prone mate choice provides a novel solution to the lek paradox.

How likely is it that the choosy sex (typically females) makes mistakes when selecting their mate? Given that there is a cost to evaluating mate quality, the optimal accuracy of assessment will probably fall below perfect. Quantifying the actual rate of these errors in natural systems will be important to determine the relative contributions that imperfect mate choice and mutational input make to the observed genetic variation that underlies sexual traits. Interestingly, if natural selection is less error-prone in its assessment of genetic quality, as compared with individuals during mate choice, then the model by Randerson et al.1 could also explain the observed difference in genetic variation between sexually and naturally selected traits. Their model could also be extended to incorporate quantitative traits and to examine the effects of variance in assessment accuracy among individuals. For example, higher quality individuals might make fewer mistakes. In other recent studies, it has also been shown that error-prone mate choice can influence male mating tactics and lek size. It is becoming clear that imperfect mate choice is an instrumental force in the evolution of lek mating systems and more generally mate choice.

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Reference

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Parasite manipulation: how extensive is the extended phenotype?

The many ways in which the genes of one organism can have an extended phenotypic influence on the living body of another organism is no better illustrated than in the subtle and varied ways in which parasite genes find phenotypic expression in the behaviour of their hosts. For over 30 years, biologists have been fascinated by the adaptive manipulation of host behaviour by parasites, and the debate over its extent and magnitude shows no sign of diminishing, as illustrated by two recent papers.

Although the existence of the manipulation of behaviour by parasites is not in any doubt,

Robert Poulin¹ argues that the magnitude of its effect might have been overestimated by biologists, owing to publication biases that were prevalent in the early years of this developing paradigm. In support of his assertion, he shows that the magnitude of parasite-induced changes in behaviour has declined significantly since the first papers on the subject were published in the 1970s and early 1980s.

Meanwhile, William Eberhard² has added an important contribution to the debate, with a report in Nature on a fascinating discovery made in oil palm plantations in Costa Rica. He reports on an ichneumonid parasitoid (Hymenoepimecis sp.) that manipulates the behaviour of an orbweaving spider (Plesiometa argyra) to increase its own survival chances during rainstorms. The wasp larva spends the first two weeks of its life feeding on the blood of its host, while the spider continues with its normal web-building activities. Then, on the night that it will finally kill its host, the larva induces the spider to build a web that is totally different from any web it has built previously. Instead of a typical orb web, the new construction comprises a dense central hub anchored by between two and eight robust guys or drag lines, making the web appear more like a canopy or awning. Once the spider has built this 'cocoon web', the wasp larva moults, kills and eats the spider, then spins the pupal cocoon in which it will hang by a line beneath the new web. Eberhard argues that the modifications to the spider's web make it much stronger and more resistant to the impact of heavy rains, which are a significant mortality factor in a related wasp species.

This instance of parasite manipulation of behaviour is notable for two reasons. First, unlike many of the examples analysed by Poulin, there is no question that the change in behaviour is due to the influence of the parasite and is detrimental to the host (although the relative advantage of the cocoon web over the orb web for wasp survival has yet to be explicitly demonstrated). Second, it might be possible to determine the precise mechanisms that are generating the behavioural change; a feat that has rarely, if ever, been achieved for such a complex manipulation. Already, it is clear that the behavioural change is mediated by a rapid-acting, long-lasting biochemical injected into the spider by the wasp. Thus, it should be possible to isolate and characterize the active substances. If this could be done, then light would be shed not only on the evolution of spider web-making behaviour, but also on the tools used by wasp genes to extend their phenotype within their spider hosts.

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References

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- 2 Eberhard, W.G. (2000) Spider manipulation by a wasp larva. *Nature* 406, 255–256