COMMENTARY

Individual control over reproduction: an underestimated element in the maintenance of sex?

N. K. MICHIELS,* L. W. BEUKEBOOM,† J. M. GREEFF* & A. J. PEMBERTON‡

*Max-Planck Institute for Behavioural Physiology Seewiesen, PO Box 1564, D-82305 Starnberg, Germany
†Institute of Evolutionary and Ecological Sciences, University of Leiden, PO Box 9516, NL-2300 RA Leiden, The Netherlands
‡Marine Biological Association of the UK, The Laboratory, Citadel Hill, Plymouth PL1 2PB, UK

Introduction

West *et al.* (1999) convincingly argue that combining traditional hypotheses on the maintenance of sex into a pluralistic framework provides a more plausible explanation for the enigmatic success of sexuality. By merging (1) more efficient elimination of deleterious mutations with (2) better tracking of environmental changes (often parasites) and allowing for synergism between both, West *et al.* (1999) show that sexuality becomes much more robust against invasion by asexuality. Their approach abandons traditional attempts to find a single and sufficient explanation for sex. However, once accepting that a mixture of ingredients may be the best recipe to explain sex, we strongly suggest adding at least one more component. Here, we argue that the pluralistic approach (West *et al.*, 1999) could be further strengthened by not concentrating solely on population-level processes, but by encompassing the important role that individuals may play.

We focus on two assumptions that population geneticists often make and that are inherent to the hypotheses within the pluralistic framework, namely that offspring are produced (1) randomly and (2) without paternal care. Under these assumptions, offspring produced by a sexual female can be represented as a quality array of randomly produced progeny in a 1:1 sex ratio (Fig. 1, bold lines). For asexuals, this distribution is compressed to a single all-female class with some small variance due to mutation (not shown). Sex is favoured when the advantage of producing few, better adapted and less mutation-loaded offspring outweighs the cost of producing males plus the cost of producing low-fitness offspring. Under random mating, high-quality sexual individuals lose most as they are likely to have relatively poorer mates, whereas low-quality individuals will benefit as they are likely to have better mates. This equalizing effect limits the benefits of sex.

Correspondence: Professor N. Michiels. Present address: Institute for Animal Evolution and Ecology,
Huefferstraße 1, D-48149 Münster, Germany.
Tel: +49 251 83 24661; fax: +49 251 83 24668;
e-mail: michiels@uni-muenster.de

However sex is not usually random. Sexual individuals can actively influence the quality of their progeny (Fig. 1, dashed lines) and data from behavioural ecology suggest that they do this specifically in an attempt to capitalize on the benefits and reduce the costs of recombination. This results in a net advantage of
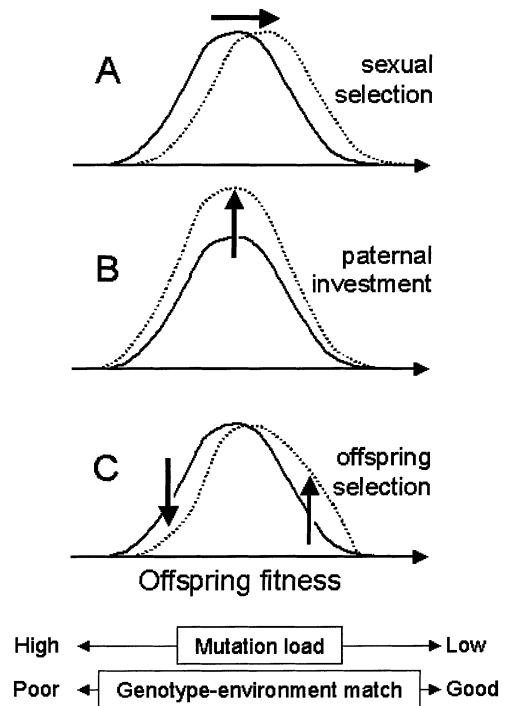
Fig. 1 Distribution of offspring quality that a sexual individual can expect to produce. Bold curve: sample distribution under random mating (shape arbitrarily chosen). (A–C) Three ways in which individuals can improve offspring fitness (dashed lines): (A) by selecting better mates, (B) by obtaining help in raising offspring and (C) by differential treatment of offspring. Option A is the only one that can move the upper range of the distribution. Options B and C can improve the shape of the distribution, but only within the set range. Options B and C are also available to asexuals, but since they start out with a very narrow distribution (not shown), the scope for improvement is accordingly limited.

sexuality at the population level. We suggest four processes sexual individuals have at their disposal to improve offspring quality and quantity. The first two (sexual selection and paternal care) are inherently linked since their strengths are inversely correlated (Trivers, 1972). The third one is specific for animals that can reduce the cost of producing males. The fourth summarizes post-zygotic effects.

Better offspring through sexual selection (Fig. 1A)

Differential selection on individuals for access to mates through mate choice and competition for mates is known to be particularly strong on traits that indicate parasite resistance and/or mutation load (often measured as developmental instability) (Hamilton *et al.*, 1990; Møller & Swaddle, 1997). Nonrandom mating in relation to these traits gives parents the possibility of actively choosing the appropriate mate to obtain offspring with fewer mutations and/or a better genome–environment match than under random mating (Fig. 1A; A. J. Pemberton, in preparation). For example, mammals actively choose mates of dissimilar MHC genotype and thereby increase the genetic variation in the immune response system of their offspring (Jordan & Bruford, 1998). Choosiness is particularly important for high-quality individuals in order to prevent their offspring from sliding back to a given population mean. The resultant assortative mating within this 'upper class' may force low-quality individuals to accept mates of a lower than average quality. At the population level, sexual selection has the potential to flatten the quality distribution of the progeny produced, thereby increasing exposure of deleterious mutations and enhancing the genotype–environment match in the next generation. If parent quality affects offspring number in addition to quality, sexual selection may also result in a larger skew towards fewer low-quality and more high-quality progeny.

Sexual selection is ubiquitous (Anderson & Iwasa, 1996) and takes place at all conceivable levels: from precopulatory interactions between individuals (Andersson, 1994) down to post-copulatory selection through sperm and pollen competition (Birkhead & Møller, 1998) or cryptic female choice (Eberhard, 1995). Parents may be able to select their own gametes. In mice, mutant spermatocytes appear to be selectively eliminated during spermatogenesis (Walter *et al.*, 1998). There is also growing evidence for nonrandom fusion of oocytes with sperm. For example, the combination of MHC alleles in eggs and sperm affects the fertilization efficiency (Wedekind *et al.*, 1996; Rulicke *et al.*, 1998). In addition to MHC-dependent mate choice (review by Jordan & Bruford, 1998) all this indicates active control over disease resistance in progeny, resulting in a better genome–environment match.

More young through paternal care (Fig. 1B)

Males are the main cost of sex, since the production of sons reduces a sexual population's intrinsic growth rate by a factor two (Maynard Smith, 1978). However, this only applies when males are mere sperm donors. When they also provide resources, they can increase the number of progeny a female produces, up to the point at which they may cancel out this two-fold cost. Although both low- and high-quality offspring may benefit equally from paternal care (Fig. 1B), it is the increased number of high-quality offspring that matters most, as it is this category that potentially enhances the spread of fitter gene combinations. In addition to support from males, a female can also receive help from her own offspring. Since helpers are not exposed to sexual selection while helping, they may be of use even if unfit for reproduction themselves, thus reducing the cost of producing low-quality offspring through sexuality.

Although asexual females may cash in on male assistance in sexual populations as well, males will be under strong selection to recognize asexuals or their (all-female) broods (Loyning & Kirkendall, 1996). Refined assessment of females may already be in place since males that invest heavily in offspring will be under selection to distinguish cheating from faithful sexual females (see below).

Reduced cost of males through skewed sex allocation (not illustrated)

Individual control over sex allocation is well developed in haplodiploid organisms (Wrensch & Ebbert, 1993). Here the sex ratio is typically skewed towards females to reduce local mate competition between brothers (Hamilton, 1967). As a consequence, the cost of males is also reduced. A female-biased sex allocation is also known from many hermaphrodites (e.g. Petersen & Fischer, 1996), and is expected when matings are rare (Greeff & Michiels, 1999) or when sexuality includes some selfing (Charlesworth & Charlesworth 1981). Alternatively, asexuals arisen from hermaphroditic ancestors may not have shut down their male function completely, and still pay the cost of male allocation (Weinzierl *et al.*, 1998). All these mechanisms will reduce the cost of reproduction in sexuals relative to that in asexuals, making asexual modes of reproduction less likely.

Progeny screening (Fig. 1C)

Parents also have post-zygotic mechanisms at their disposal to improve average offspring quality. First, by cutting investment in poor offspring, there are more resources for fitter progeny, skewing the distribution of offspring quality in favour of the fittest (Fig. 1C).

Selective abortion is a first mechanism to achieve this. There is actually evidence for higher abortion rates in humans among MHC-similar couples (Alberts & Ober, 1993), suggesting that investment in offspring with a less variable (presumably less flexible) immune system is avoided. Second, young may be fed differentially after birth. Third, parents may put an upper limit on overall investment and induce competition among their progeny, which again results in quality-dependent allocation of resources. Even when parents are completely ignorant about offspring quality within a brood, less fit young may serve as food for fitter sibs. Finally, a female that reproduces repeatedly may make maternal investment in each single brood dependent on the quality of the likely father of that brood, thus economizing resource allocation over her lifetime (Møller & Thornhill, 1998).

Discussion

There appear to be many ways in which individual behaviour can increase the benefits of sex beyond the random mating expectation. Empirical data from behavioural ecology indicate that nonrandomness is strong and widespread in sexual species. Trivers (1972) pointed out that the strength of sexual selection (our first point) is inversely related to the extent of paternal care (our second point). This is because males that do not offer paternal care, and can afford to spend more on attracting mates or fighting off rivals. It means that from our first two sets of mechanisms, at least one is likely to apply to any given system. It can therefore be no surprise that the mechanisms listed above have now been documented in many and diverse species groups, and are manifested at very basic levels, such as spermatogenesis or sperm–egg interactions. Moreover, they represent such direct advantages for the individual that employs them, that there must be strong selection in favour of them. The mere fact that individuals appear to base mating decisions on environmental adaptation and mutation load actually supports the pluralistic paradigm proposed by West *et al.* (1999). Looking at what individuals do may therefore offer an alternative approach to quantify the relative importance of mutations vs. genotype–environment matches.

'Individual quality-control' should be seen as a magnifying glass that exposes small defects in potential sexual partners. Importantly, it anticipates natural selection by parasites or mutations by stressing and unveiling unfavourable genotypes before they enter the next generation. As a result, sexual populations may actually be much fitter (and evolving faster) than expected under random mating, and the advantage asexuals need to compete with sexuals should accordingly be higher. For all these reasons, we think that 'individual quality-control' deserves a central place in a pluralistic theory of sex.

One cautionary note should be added at this point. Sexual reproduction results in a number of conflicts during reproduction because the parties, parents and offspring, are not genetically identical. These conflicts may reduce the efficiency of the mechanisms we propose. For instance, offspring may attempt to deceive their parents into believing that they are actually the best or most needy of food. But here selection for honest signals in progeny could reduce the risk. Similarly, the work on conflicts between males and females regarding certainty of paternity and paternal care (Harada & Iwasa, 1996) takes on a new light in this context. In these cases of conflict, the extent to which females can deceive males will be directly related to the degree to which sexual selection and paternal care act in unison. However, when males win, as is the case with paternally imprinted genes (Haig, 1993), the cost of sex can actually be higher.

Note that co-operative behaviour and offspring selection are two mechanisms that asexuals also have at their disposal to improve their reproductive success (Chao & Levin, 1981; Lively & Johnson, 1994). Yet, the potential advantage may be much smaller. First, in co-operative asexuals resources are primarily needed to produce own eggs and helping will be reduced to providing access to common resources or sharing common tasks such as alertness or defence. A helping male, on the other hand, does not invest in eggs, and will be able to provide his female not only with services, but also with resources that may allow her to produce more offspring. Second, genetic variance among asexual offspring is so limited that the maximum benefit of offspring screening is accordingly narrow for an asexual female.

Although most theoretical studies of the evolution of sexuality have actually acknowledged that nonrandom mating or parental care may influence the outcome of their models, the importance of these phenomena has always been minimized. We hope that pluralism at the population level will also lead to open-mindedness about the constituent individuals.

References

Alberts, S.C. & Ober, C. 1993. Genetic variability of the MHC: a review of nonpathogen-mediated selective mechanisms. *Yb. Phys. Antropol.* **36**: 71–89.

Andersson, M. 1994. *Sexual Selection.* Princeton University Press, Princeton.

Andersson, M. & lwasa, Y. 1996. Sexual selection. *Trends Ecol. Evol.* **11**: 53–58.

Birkhead, T.R. & Møller, A.P. 1998. *Sperm Competition and Sexual Selection*. Academic Press, San Diego.

Chao, L. & Levin, B.R. 1981. Structured habitats and the evolution of anticompetitor toxins in bacteria. *Proc. Natl. Acad. Sci. USA* **78**: 6324–6328.

Charlesworth, D. & Charlesworth, B. 1981. Allocation of resources to male and female functions in hermaphrodites. *Biol. J. Linn. Soc.* **15**: 57–74.

Eberhard, W.G. 1996. *Female Control: Sexual Selection by Cryptic Female Choice*. Princeton University Press, Princeton.

Greeff, J.M. & Michiels, N.K. 1999. Sperm digestion and reciprocal sperm transfer can drive hermaphrodite sex allocation to equality. *Am. Nat.* **153**: 421–430.

Haig, D. 1993. Genetic conflicts in human pregnancy. *Quart. Rev. Biol.* **68**: 495–532.

Hamilton, W.D. 1967. Extraordinary sex ratios; a sex-ratio theory for sex linkage and inbreeding has new implications in cytogenetics and entomology. *Science* **156**: 477–488.

Hamilton, W.D., Axelrod, R. & Tanese, R. 1990. Sexual reproduction as an adaption to resist parasites (A review). *Proc. Natl. Acad. Sci. USA.* **87**: 3566–3573.

Harada, Y. & Iwasa, Y. 1996. Female mate preferences to maximize paternal care: a two-step game. *Am. Nat.* **147**: 996–1027.

Jordan, W.C. & Bruford, M.W. 1998. New perspectives on mate choice and the MHC. *Heredity* **81**: 239–245.

Lively, C.M. & Johnson, S.G. 1994. Brooding and the evolution of parthenogenesis: strategy models and evidence from aquatic invertebrates. *Proc. R. Soc. Lond. B* **256**: 89–95.

Loyning, M.K. & Kirkendall, L.R. 1996. Mate discrimination in a pseudogamous bark beetle (Coleoptera: Scolytidae): male *Ips acuminatus* prefer sexual to clonal females. *Oikos* **77**: 336–344.

Maynard Smith, J. 1978. *The Evolution of Sex.* Cambridge University Press, Cambridge.

Møller, A.P. & Swaddle, J.P. 1997. *Asymmetry, Developmental Stability and Evolution. Oxford Series in Ecology and Evolution.* Oxford University Press, Oxford.

Møller, A.P. & Thornhill, R. 1998. Male parental care, differential partential investment and sexual selection. *Anim. Behav.* **55**: 1507–1515.

Petersen, C.W. & Fischer, E.A. 1996. Intraspecific variation in sex allocation in a simultaneous hermaphrodite – the effect of individual size. *Evolution* **50**: 636–645.

Rulicke, T., Chapuisat, M., Homberger, F.R., Macas, E. & Wedekind, C. 1998. MHC-genotype of progeny influenced by parental infection. *Proc. R. Soc. Lond. B* **265**: 711–716.

Trivers, R.L. 1972. Parental investment and sexual selection. In: *Sexual Selection and the Descent of Man, 1871–1971* (B. Campbell, ed.), pp. 136–179. Heinemann, London.

Walter, C.A., Intano, G.W., Mccarrey, J.R., Mcmahan, C.A. & Walter, R.B. 1998. Mutation frequency declines during spermatogenesis in young mice but increases in old mice. *Proc. Natl. Acad. Sci. USA.* **95**: 10015–10019.

Wedekind, C., Chapuisat, M., Macas, E. & Rulicke, T. 1996. Non-random fertilization in mice correlates with the MHC and something else. *Heredity* **77**: 400–409.

Weinzierl, R.P., Berthold, K., Beukeboom, L.W. & Michiels, N.K. 1998. Reduced male allocation in a parthenogenetic hermaphrodite (*Dugesia polychroa,* Tricladida, Platyhelminthes). *Evolution* **52**: 109–115.

West, S.A., Lively, C.M. & Read, A.F. 1999. A pluralist approach to sex and recombination. *J. Evol. Biol.* **12**: 1003–1012.

Wrensch, D.L. & Ebbert, M.A. 1993. *Evolution and Diversity of Sex Ratio: Insects and Mites.* Chapman and Hall, New York.