COMMENTARY

Explanation and prediction and the maintenance of sexual reproduction

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Evolutionary biology has often sat rather uneasily with fundamental principles of scientific explanation. Hempel (1965) has pointed out that, in science, there is an equivalence of explanation and prediction. In other words, a theory and/or a set of observations A can be said to explain a set of observations B if, and only if, B is predictable from knowledge of A. This criterion, widely accepted in epistemological philosophy, is often not satisfied in adaptive explanations of the phenotype. Frequently, an evolutionary explanation for a phenotypic trait is postulated, but one in which the trait is logically predicted by the explanatory idea only if quantitative values of unobserved parameters fall in a certain range. Measurement of the parameters can be dauntingly difficult. What, regrettably often, happens instead, is that the explanatory idea is accepted as correct on the basis of its intuitive reasonableness or appeal. Once this has happened, then the conjunction of the trait to be explained and the explanatory hypothesis are seen as jointly constituting evidence that the unobserved parameters fall in the required range.

The explanations of the continued persistence of sexual reproduction in the face of a theoretical two-fold advantage for apomictic parthenogens form a good example of an incomplete logical coupling between explanation and prediction. The fundamental problem has been expressed by Maynard Smith (1978) as being that, if the number of surviving offspring produced by a female is independent of the sex of these offspring, and whether or not they are produced sexually or asexually, then a dominant mutation generating apomictic parthenogenesis would be expected to have a two-fold fitness advantage relative to its allele in a wild population. Given that such apomictic mutations are possible, why are sexual species not replaced by their apomict descendants?

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Very large numbers of hypotheses have been suggested to try to account for the persistence of sex, in the face of this expected two-fold advantage. These hypotheses postulate mechanisms whereby sexually produced offspring have a higher Darwinian fitness than their asexual competitors. Such models create a short-term advantage to sex, such that the sexual subset of the population will be able to resist invasion from apomictic mutations. Clearly, there is another suite of explanations for sex which invoke a duration of asexual species which is short in palaeontological time, although long enough for an asexual mutation to have time to selectively replace its sexual progenitor.

West *et al.* (1999) argue that traditional ways of looking at the advantage of sex may be falsely unitary, in that they tend to contrast different models and look for data sets which will convincingly resolve amongst them. In particular, considering the two most strongly supported theories of forces giving advantages to sexual organisms, that of environmental fluctuation and the Red Queen, and the Mutational Deterministic hypothesis of synergistic deleterious mutations, they believe that the ubiquity of parasites and of deleterious mutations predicts that realistic models of sex must combine these two processes.

I believe that the set of models for sexual reproduction, taken either singly or together, do not, at present, meet the criterion for being a true explanation. They are still insufficient in their details for us to be able to predict the widespread occurrence of sexual reproduction if we did not know of this independently of our modelling efforts. Indeed, what has been motivating the search for a single unitary explanation for the persistence of sex has been the conviction that precisely one of the models will,

when the parameters relevant to the model have been accurately empirically determined, turn out to be a much more powerful explanation than it currently appears.

It is impossible to deny the realism of mixed models of sexual advantage since it would seem highly likely that a series of processes favouring sex might well be acting simultaneously. Thus, it seems appropriate to multiply their relative contributions to the fitness of sexuals in the hope that the product passes the magical threshold of two, such that the suite of processes jointly explain the phenomenon. However, for all its reasonableness, there are also dangers inherent in this approach. It should be remembered that processes which lead to an advantage for sexual reproduction have been searched for and enumerated, simply because the mystery to be explained consists of the persistence of sexual types when their two-fold competitive disadvantage should, all else being equal, preclude this. There has been no correspondingly systematic logical or empirical search for mechanisms which might favour asexual genotypes, giving them fitness benefits in excess, even, of the expected two-fold advantage. Thus, the true cocktail of selective forces operating on a competition between sexual and apomictic forms would be expected to also include various selective mechanisms favouring asexuality, such as the preservation of epistatic selectively favoured genotypes by apomixis, which have not been rigorously considered or investigated. Simply taking the apparently interesting side of the balance sheet, focusing on the forces favouring sex, may result in the two-fold advantage required, but may not be a realistic description.

The authors make a plea for the empirical testing of the models being postulated, and no scientist could object to empiricism. However, I fear that a major empirical programme to put values to the relevant parameters might well be more difficult that these authors anticipate. The authors cite the work of Kelley (1994) who showed a 1.55 fitness advantage of sexually produced progeny over apomicts in a grass, due to the impact of pathogens. Since this was done after one generation of apomixis, too soon for the Mutational Deterministic process to have its effects, they could conclude that the required fitness advantage of two could be attained if synergistic deleterious mutations were arising at a mutation rate of 0.51 per genome, rather than the 1.39 required if they alone were to produce the entire two-fold advantage required.

There are two important questions to be considered in making the decision to pursue this type of experimentation more generally.

The first question is whether we require the ubiquity of sex to be matched by a ubiquity of sexual advantage. In other words, do we postulate that all sexual populations are such that a mutation which would create an apomictic clone would be incapable of spreading in that population? One of the remarkable features of the model for the spread of apomictic parthenogens is the remarkable rapidity of the process. A new mutation with a two-fold reproductive advantage has around an 80% chance of spreading to fixation in a population, and, if it does so, will complete its spread to fixation in a few tens of generations (given population sizes of up to a few millions). For almost all types of organisms, it is impossible to imagine a fixation process occurring in this time throughout a geographically widespread species, and the rate of fixation will, realistically, be limited by restricted migration between populations. In order to prevent the apomictic mutation from spreading to fixation in the species, it only has to encounter a single sexual subpopulation in an environmental situation yielding a two-fold reproductive advantage to sexuals. When it does, it will be unable to invade this subpopulation, and the species will persist with sexual and asexual subpopulations. (Whether we expect to find this situation in a typical species will depend upon the mutation rate to viable and fertile apomictic parthenogens, and this rate might be quite low, so we should not be surprised that most species consist of entirely sexual subpopulations.) Once sexual and asexual subpopulations exist, the long-term advantages of sexuality will come to the fore, and it is likely that, ultimately, only the sexual subpopulation will leave descendants. Indeed, once population subdivision is considered, the distinction between long- and short-term mechanisms for the maintenance of sexuality may be less distinct than models normally suppose. (While, here, I have envisaged a geographical subdivision of subpopulations, analogous arguments operate on an ecological scale with niche partitioning generating frequency-dependent selection (Maynard Smith, 1998).)

The point of this discussion is that sexuality may be capable of persistence at the level of the species despite its invasibility by apomictic clones in the majority of the subpopulations. Thus, in the case of Kelley's (1994) data, there is no necessary reason to suppose that the rate of deleterious mutation in this particular population was sufficiently high that, when combined with the measured fitness advantage, it would produce an overall advantage for sexuals of over two. This is quite apart from the problem that synergism is required between the deleterious mutations, for which evidence is lacking (Barton & Charlesworth, 1998).

The second and related concern about measurement of the fitness advantage of sexual progeny relative to apomicts is the extremely low repeatability expected for this measurement from species to species, population to population and perhaps even from year to year. The advantage depends on the particular spectrum of pathogens or parasite genotypes infecting the population, and there is no reason to expect it to remotely resemble a biological constant. If one is taking the approach of identifying the genome-wide deleterious mutation rate, U, as that required to generate a two-fold advantage overall, the estimates of this quantity will oscillate wildly depending upon the particular estimate of the

environmental advantage of sexuals. Had Kelley (1994) found 1.2 as the relative fitness of sexuals, the *U* required would be over one, yet if he had found 1.9, the *U* required would be 10 times less.

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