

# 1 *The problem*

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This chapter should be read as a guide to the rest of the book. In it, I outline what seem to me to be the central problems to be solved, I indicate briefly the theories and kinds of evidence which may be relevant to their solution, and, when it seems helpful, direct the reader to the chapters and sections in which each matter is discussed further.

The central question is as follows: what selective forces maintain sexual reproduction and genetic recombination in nature? It may help to classify the various theories; first, according to the time scale on which selection is supposed to act, and then according to the 'unit of selection' – population, individual, or gene. Classifying according to time scale, we have at one extreme selective forces comparable to those which established industrial melanism or insecticide resistance in natural populations. Such a selective force could, at least in principle, increase the frequency of an allele for high recombination relative to its low-recombination allele in each generation. Such forces exist (Chapter 6), but it is far from clear that they are the only or the most important ones.

At the other extreme are forces which produce an increase in recombination only when their effects are summed over a period long enough for some populations to go extinct and for new ones to arise. I return to such forces in a moment. But there are, in addition, selective forces acting within a population, and over an intermediate time scale. There is no guarantee that the frequency of an allele for high recombination will increase in any one generation, but it will do so when summed over tens or hundreds of generations (Chapter 7). Such processes are hard to treat analytically, but they may well be the most important ones with which we are concerned.

A more usual way of classifying selective forces is in terms of 'group' and 'individual' selection. Thus, natural selection will operate on any set of entities with the properties of multiplication, heredity,

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and variation. If the entities in question are individuals, we can speak of 'individual selection'; it is this type of selection with which the vast majority of evolution theory is concerned. If the entities are groups – species, or reproductively isolated populations – we speak of 'group selection'. It was Fisher's view that sexual reproduction was the only adaptation which had evolved by group selection, but he at least hinted that recombination might be explained without group selection (see p. 119).

The reason why most evolutionists have been reluctant to accept group selection as a significant force in evolution is that the circumstances in which a trait advantageous to the group but deleterious to the individual could increase in frequency are restricted and unlikely to arise often in nature (Maynard Smith, 1976c; for a contrary view see Gilpin, 1975). My own active interest in the evolution of genetic systems dates from around 1964, when I recognised that it was illogical to reject Wynne-Edwards's (1962) views in ecology as group selectionist, if at the same time I accepted a group selectionist explanation of sex. G. C. Williams's interest in genetic systems seems to have been aroused in a similar way, although he appears to have been provoked more by Emerson (1960) than by Wynne-Edwards.

Is it then possible that sex is maintained by group selection? In its simplest form, the argument would go as follows. An individual female which abandons sexual reproduction obtains thereby an immediate short-term advantage. This advantage means that once a parthenogenetic strain is established, it will selectively displace the sexual species. In the long run, however, the new parthenogenetic species is doomed to extinction because of its inability to evolve. While parthenogenetic species go extinct, sexual ones speciate, thus maintaining the total number of species. The result of this process is that, at any one time, most species are sexual.

Thus, the group selection explanation of sexual reproduction accepts that there is a short-term advantage to parthenogenesis, and argues that this is more than balanced by the long-term advantages of sex. I now discuss these two points.

Consider first the twofold disadvantage of producing males. Although absurdly simple, the point is so fundamental and so often

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misunderstood that I repeat below the argument first presented by Maynard Smith (1971*b*). Suppose that, in a sexual species, with equal numbers of males and females, a mutation occurs causing females to produce only parthenogenetic females like themselves. The number of eggs laid by a female,  $k$ , will not normally depend on whether she is parthenogenetic or not, but only on how much food she can accumulate over and above that needed to maintain herself. Similarly, the probability,  $S$ , that an egg will survive to breed will not normally depend on whether it is parthenogenetic. With these assumptions the following changes occur in one generation:

	Adults		Eggs		Adults in next generation
Parthenogenetic	♀ ♀	$n$	→	$kn$	→ $Skn$
Sexual	{	$N$	→	$\frac{1}{2}kN$	→ $\frac{1}{2}SkN$
	{	$N$	↔	$\frac{1}{2}kN$	→ $\frac{1}{2}SkN$
	{	$N$	→	$\frac{1}{2}kN$	→ $\frac{1}{2}SkN$

Hence, in one generation, the proportion of parthenogenetic females increases from  $n/(2N+n)$  to  $n/(N+n)$ ; when  $n$  is small, this is a doubling in each generation.

Some of the confusion which has arisen over this came, I think, from the phrase 'the cost of meiosis'. In a species with isogametes there is no necessary twofold cost associated with meiosis, although the time taken to complete the meiotic divisions might constitute a cost. In a sense, a gene in a primary oocyte is running a 50% chance of being eliminated in a polar body, and could therefore gain a twofold selective advantage by suppressing meiosis. But I believe that the twofold advantage of parthenogenesis is best seen as the advantage of not producing males. Some situations in which the advantage does not accrue are discussed in Chapter 4, section A.

This short-term advantage of parthenogenesis has been noted in passing by a number of authors. However, it has not led them to reformulate the question of the evolution of parthenogenesis in what seems to me to be the natural way: i.e. why do parthenogenetic varieties not replace sexual ones? For most writers, the question seems to have remained, why are some parthenogenetic varieties able to establish themselves? There are, I think, two contributing reasons

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why the question is still put in this way. First, some authors have thought that a twofold increase in reproductive rate would be an advantage only when a population is increasing in numbers, a confusion which has not been helped by all the recent discussion of  $r$  and  $K$  selection and its relation to colonisation.

A second reason why the twofold advantage has, until recently, been either ignored or treated as unimportant is that most writers on the subject have felt that the short-term advantages of sexual reproduction in an unpredictable environment are so overwhelming that the problem is to explain why parthenogenesis exists, not to explain why it is not universal. I hope that Chapter 6 will dispel any idea that selection in an unpredictable environment necessarily or easily confers an advantage on sex. I am, however, very conscious of the fact that my method of formulating the problem of selection in an unpredictable environment may have caused me to draw misleading conclusions. But if anyone believes that, let him show how the problem should be expressed.

The second point to be established is that there are long-term selective advantages in sexual reproduction to the species. The nature and extent of these advantages are discussed in Chapters 2 and 3. The essential point, however, was made independently by Fisher (1930) and Muller (1932). In a population without genetic recombination, if two different favourable mutations,  $a \rightarrow A$  and  $b \rightarrow B$ , occur in separate individuals, there is no way in which they can be combined in a single descendant. An  $AB$  individual can arise only if a second  $A$  mutation occurs in a descendant of the original  $B$  mutant (or a second  $B$  in a descendant of the original  $A$ ). With recombination, an  $AB$  individual can arise by recombination in a common descendant of the two original mutants.

Thus the essential logical requirements for a group selection explanation exist, namely genetically isolated groups (sexual species, and parthenogenetic clones), and a trait which is individually disadvantageous but advantageous to the survival of the group. The only questions that remain are, first, is the group selection hypothesis plausible in numerical terms, and, secondly, is it supported by the comparative data?

The question of numerical plausibility is easily formulated. The

group selection hypothesis in its simple form requires that for every successfully established parthenogenetic clone, the sexual species which gave rise to it must go extinct. Since species extinction is not an everyday occurrence, this requires that parthenogenetic origins must likewise be rare. There is no easy way to estimate how frequently such origins occur. The problem is discussed in Chapter 4, sections B and C. The situation is somewhat different between plants and animals because most spontaneous parthenogenesis in animals is of a kind which gives rise to individuals of low viability because of genetic homozygosity. At least in animals it is not implausible to suppose that viable parthenogenetic varieties may arise sufficiently rarely.

Further support for the group selection argument comes from the arguments in Chapter 4, sections D and F. Section D is primarily concerned with showing that the taxonomic distribution of parthenogenetic varieties supports the hypothesis that such varieties are doomed to early extinction, presumably because of lack of evolutionary potential. Section F points out that the establishment of a successful parthenogenetic variety is often not followed by the extinction of the sexual species which gave rise to it, because the variety may be adapted to only a part of the ecological range of the parent species.

All this, however, merely points to the plausibility of the group selection hypothesis; it does not prove it to be true. To my mind, much the most powerful argument of an empirical kind against the hypothesis was proposed by Williams (1975); it is the 'balance' argument. Suppose that a population consists of facultatively parthenogenetic organisms which can reproduce either sexually or asexually. In such a case it is inconceivable that there should not be genetic variation capable of wholly suppressing sex if this were advantageous in the short run. Since sex continues, it must have some short-term advantages.

The argument is a powerful one. It is discussed in Chapter 4, section E. The difficulty is not with the argument but with the data. There are some unequivocal cases which support Williams's position. But, in assessing the data, it is essential to remember that a population consisting of a mixture of sexually reproducing individuals and obligate parthenogenetic ones does not support the balance

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argument because it is possible that the parthenogens contain a different (and narrower) range of genotypes. Further, a species in which individuals produce sexual and asexual eggs with different ecological roles (as, for example, the overwintering sexual and immediately hatching asexual eggs of rotifers, or the sexual dispersive seeds and asexual bulbils of onions) does not support the balance argument; it could be that sex is retained in these cases only because of its ecological correlates. If, in such cases, it can be shown that there are genetic variants capable of producing all ecological types asexually, this would support the balance argument.

I am sure that there must be much data on the balance argument with which I am unfamiliar, and still more which could easily be collected if its relevance were appreciated. In animals, in particular, there are many reports of populations in nature with a substantial excess of females. But are these facultative parthenogens, or are they mixtures of sexual individuals and obligate parthenogens?

I do not find it possible to give an unequivocal answer concerning the role of group selection in the maintenance of sexual reproduction. It has played some role, as evidenced by the taxonomic distribution of parthenogens; but it is not the only relevant force, as will be apparent from the review of the balance argument in Chapter 4, section E. But, whatever one may think of the role of group selection in the maintenance of sex, it cannot explain how it started, and it cannot explain the maintenance of high levels of genetic recombination within sexual populations.

So far I have discussed only the maintenance of sexual reproduction and genetic recombination. But what of their origins? Surely evolution theory should be concerned with the origins of adaptations, not merely with their maintenance once they have arisen. There is much in this objection; indeed one of the things I have learnt while writing this book is that my own insight into the field may have been obscured by an obsession, which I share with most population biologists, with equilibrium situations.

Yet there is really little alternative. Recombination probably originated some three thousand million years ago, and eukaryotic sex one thousand million years ago. Each origin may have been a unique series of events. We can speculate about such events, but cannot test

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our speculations. In contrast, selection must be acting today to maintain sex and recombination. We have to concentrate on maintenance rather than origins because only thus can we have any hope of testing our ideas. Nevertheless, something must be said about origins.

Although group extinction may eliminate the occasional 'loss' mutation, it cannot have brought together the series of adaptations concerned in the origin of sex. Long before the origin of eukaryotic sex, the prokaryotes had acquired the capacity for genetic recombination – that is, the pairing, breaking, and rejoining of homologous lengths of DNA. It seems clear that its original function was not the generation of evolutionary novelty, but the repair of damage. This point is discussed further on p. 36.

In the prokaryotes, the problem of bringing together in a single cell DNA from two different ancestors is solved in a variety of ways. But with the evolution of the eukaryotic cell, the problem had to be solved anew. The origin of meiosis, syngamy, and the haplo-diploid cell cycle remains one of the great puzzles of evolution theory. The best scheme I can offer is illustrated in Figure 1. Starting from a population of haploid single-celled organisms, one can at least guess at the main stages and the selective forces responsible. The first stage would be binary cell fusion to form a heterokaryotic cell, with two haploid nuclei of different ancestry. The selective advantage of such fusion would be analogous to the advantages of hybrid vigour, particularly the covering up, by complementation, of deleterious genes. As in the origin of recombination, the first step was to compensate for damage rather than to create novelty.

To evolve from a heterokaryon to a diploid it is required only that the two nuclei use a single spindle in mitosis rather than each its own spindle. This would have the obvious advantage of ensuring that one copy of each nuclear chromosome set was transmitted to each daughter cell, retaining the advantages of hybrid vigour; if two spindles were used, there would always be danger of two diploid 'homozygotes' resulting from mitosis.

So far, all is straightforward. But given the evolution of a successfully replicating diploid, why then evolve the pairing of homologous chromosomes, chiasma formation, reduction divisions

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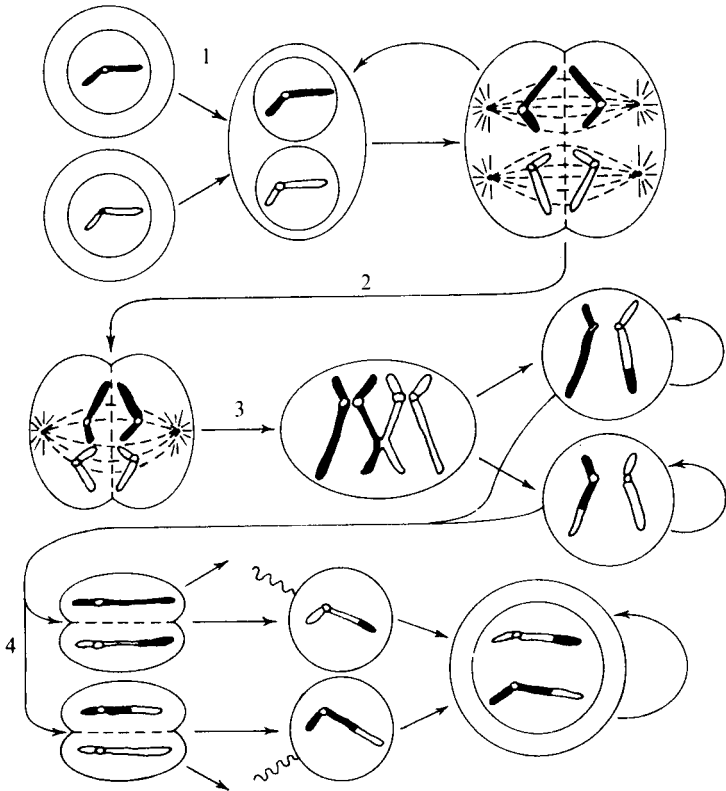


Figure 1. A scheme for the origin of sex. 1, Fusion of haploid cells to form heterokaryon. 2, Origin of diploid from heterokaryon. 3, Chiasma formation during mitosis ('somatic crossing over') generates variability within a clone. 4, Reduction and syngamy restores heterozygosity.

and subsequent syngamy? It is possible to suggest why it might be selectively advantageous to evolve homologous pairing at the 'four-strand' stage, accompanied by chiasma formation (the genes for which would already be present) between non-sister strands. The consequence of such 'somatic crossing over' is the generation of new genetic variants within a clone. But somatic crossing over generates new variation at the expense of genetic homozygosity. At this stage, a



final reduction division followed by syngamy would have been favoured because it again restored the lost genetic heterozygosity.

There remains the question of why, given a haplo-diploid cycle, both higher animals and plants spend the major part of the cycle as diploids. We do not know. Two possibilities are that diploidy protects against the deteriorative effects of somatic mutation, and that it in some way assists complex processes of cellular differentiation, but these are unsupported speculations.

I cannot pretend to much confidence in this explanation. Apart from other drawbacks, it argues in a circle: first, give up genetic variability for hybrid vigour; then abandon hybrid vigour for the benefits of variability; finally, regain hybrid vigour through reduction division and syngamy. It also runs contrary to the orthodox view that in primitive sexual eukaryotes the main part of the life cycle was spent as a haploid. But at least it may provoke better speculations.

More important than the failure of group selection to explain the origin of sex is its failure to explain the maintenance of genetic recombination. What evidence we have (reviewed in Chapter 5, section A) suggests that there is within-population genetic variation for the frequency and location of chiasmata. If it were advantageous in the short run to reduce or eliminate crossing over, it seems that the genetic variation exists upon which selection could act. True, there is no twofold disadvantage to recombination comparable to that associated with sex. Yet there almost certainly is a short-term disadvantage to recombination. In Chapter 5, the theoretical reasons for this are reviewed in section B, and some observational evidence supporting the theory in section C. In effect, what both theory and observation suggest is that it is selectively disadvantageous to break up the co-adapted genotypes of surviving adults by recombination.

Thus Williams's (1975) balance argument applies to the maintenance of recombination, whatever the difficulties may be of applying it to the maintenance of sex. Therefore, unless we are going to claim that chiasmata perform some essential mechanical role in meiosis – and it would be difficult to support such a claim in view of the absence of chiasmata in the meiosis of male Diptera – we are driven to seek some counterbalancing short-term advantage for recombination.

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The search for such short-term advantages is taken up in Chapters 6 and 7, which form the core of the book. There are basically three types of model: 'unpredictable environment'; 'sib competition'; and the 'Hill–Robertson effect' i.e. the effects of randomly generated linkage disequilibrium. Felsenstein & Yokoyama (1976) would reduce these to two by treating the 'sib-competition' model as a sub-class of the Hill–Robertson effect.

Each type of model has its difficulties. If my analysis is sound, an unpredictable environment will favour increased recombination only when the type of unpredictability is of a special and, in my view, implausible kind. Sib competition, which I believe to be the essential new feature of the models proposed by Williams (1975), can favour higher recombination in an environment which is unpredictable in a more plausible way. The major snag is that, as soon as one allows for the fact that alleles at a number of different loci may be concerned with adaptation to a single feature of the environment, the direction of selection is reversed and favours tighter linkage.

The Hill–Robertson effect is sufficiently universal to be the type of mechanism we are seeking. It arises in two forms:

- (i) the 'hitch-hiking' effect (Chapter 7), in which recombination favours the establishment of new favourable alleles; and
- (ii) 'Muller's ratchet' (Chapters 2 and 7, section C), in which recombination favours the elimination of deleterious alleles.

In both cases, it can be shown that there is short-term selection for higher recombination.

I fear that the reader may find these models insubstantial and unsatisfactory. But they are the best we have.

The topics discussed in Chapters 8–10 are in a sense subsidiary to the main theme. In these chapters, I take it for granted that sexual reproduction exists, and discuss the selective factors responsible for variations in the means of achieving it. Finally, in Chapter 11 I take up a new topic – the evolution of the mutation rate. The major part of the book is concerned with the evolution of those genes which determine the rate of recombination between other genes; Chapter 11 is concerned with the evolution of those genes which determine the accuracy of replication of other genes.