as major transitions, in the sense of being changes in the way genetic information is stored and transmitted.

The fascinating thing about this story is the way in which many apparently unconnected changes, setting the scene for all subsequent evolution, were in a sense forced on the cell by the loss of the cell wall, an event that might have seemed at the time both trivial and regressive.

from Maynard Smith and Szathmáry

(1995)
The Origins of Life
Oxford University Press

CHAPTER 7
THE ORIGIN OF SEX

In animals and plants, and in eukaryotes generally, the essence of the sexual process is that a new individual starts from a single cell, the 'zygote', formed by the fusion of two sex cells, or 'gametes'. Typically, gametes have only one set of chromosomes—that is, they are 'haploid'—and consequently the zygote contains two sets of chromosomes—it is 'diploid'. Hence a new individual contains genetic information from two parents, and, in the longer term, each individual has received genes from many ancestors, and may contribute genes to many descendants. This has led to the concept of a 'gene pool', consisting of genes that may be in separate individuals today, but whose ancestors may have been together in the same individual in the past, and whose descendants may be together again in the future. The species, or set of potentially interbreeding individuals, thus constitutes an evolving unit, with a common gene pool.

The first point to make is that, although biologists often speak of 'sexual reproduction', the sexual process is in fact the precise opposite of reproduction. In reproduction, one cell divides into two: in sex, two cells fuse to form one. Sex is not even necessary for continued reproduction. Many single-celled organisms, and some animals and plants, reproduce indefinitely without sex. The production of eggs that develop without fertilization is called parthenogenesis, or virgin birth. Many insect species consist only of parthenogenetically reproducing females. Among reptiles, there are parthenogenetic species consisting entirely of females producing daughters genetically identical to themselves. One of the American whiptail lizards, *Cnemidophorus uniparens*, is such a species: it is thought to be of relatively recent origin, perhaps thousands rather than millions of years old, and to be descended from a female that was probably a hybrid between two sexual species. Parthenogenesis is even commoner in plants: for example, most dandelions, blackberries, and ladies' mantles reproduce without sex. It is curious that mammals never reproduce parthenogenetically, and that there are no parthenogenetic species of birds, although parthenogenesis is not unknown in domestic varieties. Thus, whatever may be the explanation of sex, it cannot be said that without it continued reproduction is impossible.
The problem

We are accustomed to associating sex with sexual differentiation—that is, with a difference between males and females. In animals and higher plants, this is justified. In animals, males produce small motile gametes (sperm), and females large non-motile ones (eggs). In hermaphroditic species—for example, many snails and flatworms—the same individual produces both eggs and sperm. But differentiation of the gametes, and hence of males and females, is not a universal feature of sexual reproduction. Most single-celled eukaryotes produce gametes of only one size—they are "isogamous," in contrast to the anisogamous animals and higher plants.

Our problem is to explain why sex arose, and why it is today so widespread. If it is not necessary, why do it? The problem is made harder by what has been called the "twofold cost of sex." To understand this cost, imagine a typical sexual species of lizard. A female can lay, perhaps, a hundred eggs during her lifetime, but on average, because the number of lizards remains roughly constant, only two of them will survive to breed, one a male and one a female. Thus, on average, each female will produce one daughter. Now imagine a mutant gene causing a female to be parthenogenetic, producing daughters genetically identical to herself. She, too, will, on average, lay a hundred eggs, of which two will survive. But both these will be parthenogenetic females. Initially, and barring accidents, the number of parthenogenetic females in the population will double in every generation. Rather quickly, parthenogens will replace sexuals. Thus, there is a twofold advantage associated with parthenogenesis, or, equivalently, a twofold cost of sex. Of course, in this argument we have ignored any countering advantages that may be conferred by sex. Part of the problem of the evolution of sex is to identify those countering advantages.

In the next section we will discuss what those advantages might be. But, first, there is an important point to make about the twofold cost. We assumed above that a parthenogenetic female can produce as many offspring as a sexual one. The assumption is reasonable for lizards, in which there is no parental care, but would be less plausible in passerine birds in which both parents care for the young. The point is that males contribute no nutrients to the fertilized egg, and so, in the absence of male parental care, they are expendable. The situation is very different in isogamous species, in which the two gametes contribute equally, in nutrients as well as genes, to the new individual. In an isogamous species, a parthenogen would have to contribute twice as much to each new egg, if that egg was to have as good a start in life as a sexually produced one. Thus in isogamous species there is no necessary twofold cost of sex.

Because the first sexual eukaryotes were certainly isogamous, it follows that the twofold cost is a problem only if we are concerned to explain the maintenance of sex in later, anisogamous organisms, but not when discussing the origin of sex. All the same, there must be some costs associated with sex, even in isogamous organisms. Apart from the necessity of a gamete finding a partner with which to fuse, growth and reproduction are interrupted by the complex process of meiosis whereby gametes with half the number of chromosomes are produced.

To ensure the proper distribution of chromosomes, the production of gametes is a complicated process, as anyone familiar with the accounts of meiosis in biology textbooks will be aware. Because of these complications, and the obvious disadvantages associated with them, it is not surprising that the origin and maintenance of sex continue to be a matter of controversy among biologists.

The advantages of sex

We now seek selective advantages of sex that might counterbalance the costs associated with the interruption of growth to produce gametes, the finding of a partner with which to fuse, and, in higher organisms, the twofold cost to females of producing males. Many suggestions have been made. These need not be mutually exclusive: after all, sex might confer more than one benefit. We will not list them all, but it would give a misleading picture of the present state of understanding if we were to concentrate on a single answer. If you find the story confusing, welcome to the club.

A major difficulty concerns the level at which selection is acting. Does selection act between individuals or between populations? We described this debate on pp. 19–21: the outcome was to persuade most biologists that, usually, between-population selection would be overwhelmed by between-individual selection, and that there was in any case little need to invoke it. But there remained the problem of sex, which had traditionally been explained by the advantages it confers on populations. More recently, there have been strenuous efforts to explain the evolution of sex without invoking group-level selection. In fact, things are more complicated than we had thought. Selection can act at any one of three levels:

1. Sex may benefit some populations at the expense of others, although it is of no advantage to a sexual individual relative to an asexual one within a population. For example, it may speed up evolution or reduce the load of deleterious mutations. Such advantages are long-term ones.

2. Sex may benefit individuals. For example, a sexual female produces offspring that are not all alike; if competition is intense, and only some genotypes have a chance of survival, this may pay. Such an advantage would be effective in the short term, in one or two generations.

3. Sex may benefit both individuals and populations. For example, a sexual population may be better able to cope with a changing environment, because it can produce more genetic diversity. This advantage would be effective in the long term, as it allows the population to adapt to new conditions.
3. Sex may benefit some genes at the expense of others in the same individual: we will explain this idea below.

Sex benefits populations

There are two ways in which this may be the case. The first is that a sexual population can evolve more rapidly to meet a changing environment. The reason is illustrated in Fig. 7.1. Suppose that two mutations, \( a \rightarrow A \) and \( b \rightarrow B \), are both selectively advantageous. Typically, the two mutations will occur in different individuals. Barring accidents, both will increase in frequency. In a sexual population, recombination can bring the two mutations together in a single individual: soon, the whole population will be \( AB \). In an asexual population, this cannot happen. An \( AB \) individual can arise only when a \( b \rightarrow B \) mutation occurs in an individual that is already \( A \), or vice versa. Calculations show that the effect on evolution rate can be substantial.

But this explanation requires that the environment should continuously be changing, forcing species to evolve to meet the challenge. Is it really true that the environment changes that rapidly? One idea is that the ‘environment’ of each species consists of other species: its competitors, predators, and parasites. When any one species changes, this is experienced by other species as a change in their environment, inducing them to change, and so on. The result is an arms race.

A second possible advantage is that sex may reduce the load of deleterious mutations in the population. Suppose that two individuals suffer from different harmful mutations, \( m_1 \) and \( m_2 \). If they mate, they can produce a normal offspring, with no mutations, by recombination: without sex, they can do so only in the unlikely event of a back mutation. We will call this the ‘engine-and-gearbox’ theory. You can buy two clapped-out motor cars, one with a broken gearbox and one with a broken engine, and make one functional car. The snag is that the child of a mating between individuals with different mutations might have both mutations, rather than neither. As Bernard Shaw said to the actress who suggested they have a child, which would have her beauty and his brains, ‘Yes, madam, but what if it had my beauty and your brains?’

Despite this snag, there are circumstances in which a sexual population will carry a smaller load of deleterious mutations than an asexual one. If you are happy to take this on trust, you should skip the rest of this paragraph, and the next. There are two situations in which the statement is true. First, consider a finite population, subject to continuous, slightly harmful mutations. Individuals can be classified as having 0, 1, 2, etc. harmful mutations. Those with no harmful mutations belong to the ‘optimal’ class. In a finite population, particularly if it is small, there is a chance, every generation, that no individuals belong to this optimal class. If there is no sex, the class cannot be reconstituted: a new optimal class, with one harmful mutation, now exists. It, too, may be lost by chance, and so on. The process is known as ‘Muller’s ratchet’, after Hermann J. Muller, the American geneticist who first suggested it. Sex, of course, can arrest the ratchet, and prevent continuing deterioration.

Sex may help even in an infinite population, but only if deleterious mutations act ‘synergistically’; that is, if it is OK to have \( m_1 \) or \( m_2 \) but not both (or, more generally, if mutations have worse effects in combination than would be expected from the sum of their effects on their own). It is difficult to see why this should be so without some mathematics. No one, however, doubts the conclusion: if deleterious mutations act synergistically, sex and recombination reduce the load of deleterious mutations. But do deleterious mutations act synergistically? There are reasons, both theoretical and empirical, to think that they do, but more evidence would be welcome.

There is no difficulty, then, in thinking of reasons why sex and recombination might benefit populations. The difficulty arises because the advantages would be long-term, and therefore cannot explain the origin of sex, even if they may help to explain its maintenance. If parthenogenetic females have a short-term advantage, they are likely to replace sexual ones, resulting in an asexual population, even if in the long-term this is harmful to the population. The problem is particularly severe in higher organisms, with a twofold cost of sex. This is the classic difficulty with ‘group selection’ explanations: short-term advantages to
The individual are likely to outweigh long-term advantages to the population. Nevertheless, there is a reason for thinking that group selection, favouring sexual populations, may have been important in the maintenance of sex. This comes from the taxonomic distribution of parthenogenetic. There are many parthenogenetic varieties and species, some parthenogenetic genera, but almost no higher taxonomic groups (families, orders) that are wholly parthenogenetic. This is exactly what we would expect if new parthenogenetic varieties arise from time to time, but are eliminated by between-group selection before they can produce a larger taxon.

It looks, therefore, as if group selection has been important in maintaining sex in higher organisms. But some reservations are needed. First, there are a few higher taxa that are wholly parthenogenetic. The most famous are the bdelloid rotifers, a whole order in which males have never been observed. It is important to find out what, if anything, is peculiar about these exceptional cases. Second, the group-selection explanation works only if the origin of a new parthenogenetic variety is a rare event. This is probably true. As we mentioned on p. 25, mammals are never parthenogenetic. The reason is essentially trivial. There is a curious phenomenon called 'gene imprinting'. In mammals, some genes 'remember' whether they were inherited from the father or mother. In some tissues only the father's gene is active, and in others only the mother's gene. Because the genes are essential, every child must have a father and a mother: this rules out parthenogenesis. The point of this example is that a secondary adaptation—imprinting of genes—has been built onto the sexual process, and makes reversal to parthenogenesis difficult or impossible. We could have given other examples of such 'sexual hang-ups'.

A third reservation is that in some species the same individual produces offspring both sexually and asexually. An example is the puzzle of Antennaria, of the family Compositae, in which the same flower head may produce asexual seeds as well as ovules that require fertilization by a pollen grain. In such cases there must be some short-term advantage in retaining sex: they are well worth further study.

A final reservation is crucial in a book concerned with origins. Group selection may be relevant to the maintenance of sex, once it has arisen, but cannot be relevant to its origin, which requires short-term advantages.

The benefit of sex to individuals
Why should an individual that reproduces sexually leave more surviving offspring? We have already mentioned one possible answer. Sexually produced offspring are all different, whereas parthenogenetically produced offspring are usually identical genetically. As the American George Williams pointed out, a parthenogenetic female is like a man who buys 100 tickets in a raffle, and finds that they all have the same number. It would be better, like a sexual female, to buy only 50 tickets, all with different numbers. Models of this process, which we will call the 'raffle-ticket' model, can be made to favour sex, but they do require that selection be very intense and very unpredictable. We think that an explanation along these lines will work only in rather exceptional circumstances.

Sex may be selected at an individual level for other reasons. In the last section, we suggested that sex could be advantageous to a population in two ways. A sexual population can evolve faster, and deleterious mutations may accumulate in an asexual population. Analogous advantages may accrue to individuals. In a rapidly changing environment, sexually produced offspring are more likely to have characteristics adapting them to the new circumstances: this is particularly likely if the challenge comes from rapidly evolving parasites. Sexually produced individuals may also have a lower load of deleterious mutations. There is still debate about whether such individual-level advantages are sufficient to maintain sex, particularly in face of the twofold cost of producing males.

There is another possible explanation of sex, of quite a different kind, that is popular with molecular biologists. Although we think it is based on a misunderstanding, it points to a process that may have been important in the origin of sex, so it is worth explaining. The idea is that sex exists to make possible DNA repair. First, we must distinguish between 'mutation' and 'damage' to DNA. A mutation is a change of one DNA molecule to another DNA molecule with a different base sequence: this is not the kind of change we are now considering. 'Damage' is a change of a DNA molecule into something that is not DNA at all. If it is not repaired, such damage may be fatal: the damaged molecule cannot be replicated so that all information is lost. The problem is how to repair it. Enzymes can recognize the damaged piece and excise it, but how can they replace it by new DNA carrying the original message—that is, with the original base sequence? If only one strand of the double helix is damaged, it can be replaced using the information on the undamaged strand. But what if both strands are damaged? Repair is then possible only by copying an undamaged DNA molecule with the same message. If the cell contains two copies of its DNA, then double-strand damage can be repaired by copying the undamaged strand.

Such double-strand repair does occur. It is found not only in eukaryotes but also in bacteria. How can this be, if bacteria have only a single chromosome? The answer is that, when we say that bacteria have only one chromosome, we mean that they have only one kind of chromosome, with only one genetic message. But, most of the time, a bacterial cell contains two identical chromosomes: if one is damaged, it can be repaired using information from the other. The point is that double-strand repair requires diploidy—two copies of the genetic message—but not sex. Double-strand repair is ancient and important. We shall
invoke it in our scenario for the origin of sex in the next section. But we see it as an explanation for diploidy, not sex.

Sex and selfish genes

Most genes replicate only when the cell replicates. There are, however, transposable elements, or ‘transposons’, which jump, or transpose, to a new site in the genome, while leaving the original copy behind: after transposition, there are two copies, where there was only one before. Consider such a transposon in an asexual organism. By transposing, it can increase the number of copies of itself in the organism but not the number of organisms in which it is present. Its long-term survival depends entirely on whether its host organism leaves descendants. But suppose such a transposon could cause its host cell to fuse with another cell. It could then transpose to the chromosomes derived from the other cell. If the cell then divided, the transposon would be present in both daughter cells, although it entered from only one parent. In other words, by causing cell fusion, the transposon would give itself an additional means of increase. Such an element could spread through a population, even if, as would be expected, it reduced somewhat the fitness of its host. What has all this to do with sex? Donal Hickey and Michael Rose suggested in 1988 that sexual fusion originated, not because of any benefit to the cells that fused, but because it benefited a transposon that was present in one of the two cells that fused, but which was transmitted to all their joint descendants.

The idea is attractive for two reasons. It provides an immediate selective advantage for sexual fusion: we need not appeal to the long-term advantage to the population. Further, there is a precedent for such genetic elements in bacteria. Plasmids are accessory genetic elements, usually carrying only a few genes, present in most bacteria. They cannot cause their host cells to fuse, fusion being difficult for a cell with a rigid cell wall, but some plasmids do cause the cell they are in to attach to another bacterium. When this happens, a copy of the plasmid DNA is passed to the other cell. Occasionally, DNA of the bacterial chromosome can also be passed to the recipient cell. So the scenario proposed by Hickey and Rose is not too far-fetched. If they are right, it leads to a somewhat paradoxical conclusion. Sexual fusion originated because it benefited a transposon, at the expense of the host cell. In the long-term, sex survives because it benefits the population, although, again, the short-term interests of the individual would be better served by parthenogenesis.

In the next section, we suggest a way in which sexual fusion could have originated. Our proposal does not depend on selfish transposable elements, but, as we explain, such elements could have played a part.

The various advantages for sex that we have proposed in this section are summarized in Table 7.1.

<table>
<thead>
<tr>
<th>Table 7.1 Theories of the evolution of sex</th>
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<tr>
<td>1. Selection favours sexual populations</td>
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<tr>
<td>(a) Sexual populations can evolve more rapidly</td>
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<tr>
<td>(b) Asexual populations accumulate deleterious mutations</td>
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<tr>
<td>2. Selection favours individuals that reproduce sexually</td>
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<tr>
<td>(a) It pays an individual to produce a variable progeny (the lottery model)</td>
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<tr>
<td>(b) Sex makes repair of damaged DNA easier</td>
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<tr>
<td>(c) Even within a population, selection may favour sexual individuals for the reasons under (1) above; in a changing environment, their offspring may be better adapted to the new circumstances, or, in an unchanging environment, their offspring may have fewer harmful mutations</td>
</tr>
<tr>
<td>3. Selection favours genes that cause individuals to undergo sexual fusion (or to produce gametes that fuse) because then a gene present in one of the fusing cells can transfer to the other.</td>
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A theory for the origin of sex

Figure 7.2 shows a possible scenario for the origin of sex. The first stage shows a single-celled organism, with a haploid—diploid life cycle. For part of the time the cells grow as haploids, with a single set of chromosomes, and at other times as diploids. Thus the population cycles between haploid and diploid states. In this respect it resembles some modern sexual organisms—for example, some seaweeds—but the means of changing from haploid to diploid, and from diploid to haploid, are different. The haploid becomes a diploid not by fusion with another haploid but simply by replicating each chromosome without dividing into two cells. This simple process, known as ‘endomitosis’, occurs in some cells today.

The diploid is converted into a haploid by a simplified version of meiosis. Because it is the origin of meiosis, which, it seems to us, is the hard thing to explain, we digress for a moment to discuss the problem. Before meiosis, each cell contains two copies of each kind of chromosome—two As, two Bs, and so on: after meiosis, each cell contains one of each kind. How is this to be achieved? To understand the answer, imagine that you are a schoolteacher with a class of children, of whom two are called Anne, two Bill, two Charles, and so on through the alphabet. To organize a game, you wish to divide the class into two teams, each containing one Anne, one Bill, one Charles, and so on. You could append to the wall a list of the two teams, with surnames, and tell the children to read the list, and assemble accordingly. But there is a much easier way. Tell each child to find the partner with the same name, and then to separate, one member of each pair going to one assembly point, and one member to the other. This is exactly what happens in meiosis. Each chromosome pair with its similar partner, or ‘homologue’, and the members of the pair are then pulled...
occurring today in some single-celled protists. We do not know whether crossing over occurs in the meiosis of these protists; genetic studies are needed to find out.

The haploid–diploid cycle that we propose as an ancestral state is therefore quite plausible. Both endomitosis and a one-step meiosis are found in existing organisms. But what selective advantages could such a cycle confer? Presumably, it was an adaptation to a changing environment, perhaps to the annual cycle. There are good reasons why, in some environments, it would pay to be a diploid. As we explained earlier, damaged DNA can be repaired only if there is an undamaged molecule to copy; that is, it requires diploidy. DNA damage is more frequent if the oxygen concentration is high, so diploidy could be explained as an adaptation to periods of high oxygen. But what were the corresponding advantages of haploidy? Haploid cells are smaller and, therefore, have a higher ratio of surface area to volume. For this reason, haploid yeast cells grow faster than diploids at low nutrient concentrations. But there is a snag to this explanation. Yeast cells feed by the diffusion of molecules through their surface. But we argued in the last chapter that the first eukaryotes fed by engulfing particles. So we are faced with a problem. Perhaps the best hope lies in a study of present-day protists with a haploid–diploid cycle.

In a sense, the first stage that we hypothesize has nothing to do with sex, which requires that DNA from different ancestors be brought together in a single descendant. The virtue of suggesting such a stage is that it helps to explain the origin of a simple form of meiosis.

In the second stage in Fig. 7.2, endomitosis has been replaced by cell fusion as a means of restoring diploidy. This is a genuine sexual cycle. We can suggest two reasons for the change. The obvious one is that fusion would cover up the effects of recessive deleterious mutations. Two different haploid cells would carry different mutations: if the mutations were recessive, a diploid formed by the fusion of two such cells would not suffer the ill effects of either mutation. This is the mechanism that underlies the phenomenon of 'hybrid vigour', whereby hybrids between inbred lines are more vigorous than their parents. The effect would have been less striking than it is when crossing diploid inbred lines today, because seriously deleterious mutations would be eliminated by selection from haploid cell lines. But the advantages of fusion over endomitosis could still be substantial. A second possible reason for cell fusion was mentioned on p. 86: a selfish transposon could have caused cell fusion as a means of ensuring its own propagation.

Once diploidy is restored by cell fusion rather than endomitosis, the two homologous chromosomes, although similar, will be slightly different, so that crossing over will produce new genotypes. In the third stage in Fig. 7.2, the simple one-step meiosis without crossing over has been replaced by a two-step
meiosis with recombination. This raises two questions. What are the advantages of recombination? Why, in modern meiosis, is there an initial doubling of every chromosome, requiring two cell divisions to restore haploidy?

The advantages of recombination are now rather well understood. On pp. 81–85, we explained that a sexual population has two potential advantages over an asexual one. It can evolve faster, and may have a lower load of deleterious mutations. Suppose now that we have a sexual population, with cell fusion and meiosis, and suppose also that there are genes that alter the rate of recombination. The latter assumption is plausible: every sexual population that has been carefully studied has been found to have genetic variability in the rate of recombination. It turns out that the same circumstances that favour sexual populations over asexual ones will favour genes increasing the rate of recombination within a sexual population. If the environment is changing rapidly, or if deleterious mutations act synergistically, selection will favour increased rates of recombination.

The second question is harder to answer. Why a two-step meiosis? Every elementary textbook describes the curious process whereby, first, every chromosome replicates, so that there are four copies of each kind of chromosome; then crossing over occurs between homologous strands, so that genes inherited from different parents can be transmitted to the same offspring; and then two cell divisions produce haploid gametes. But the same texts rarely comment on how odd this process actually is. The primary function of meiosis is to halve the chromosome number; why then start by doubling it? It is not obvious that recombination requires a four-strand stage. As mentioned above, we do not know whether crossing over occurs in the one-step meiosis of archaeozoa, but it is hard to see why it should not. We know of two suggested explanations of the two-step nature of meiosis. They are too complex to explain here, but are described briefly in Chapter 9 of our book _The major transitions in evolution._

One final point about meiosis is important. The process of recombination is a complex one, requiring that two strands of DNA be lined up precisely, cut at precisely the same point, and rejoined with a change of partners. The process requires enzymes. How could such a complex system evolve, because it would be worse than useless until complete? As usual, the answer is that the components of the system evolved earlier, to perform a different function. The enzymes used in recombination are the same enzymes that repair damaged DNA, a process that also requires the precise cutting and splicing of DNA molecules. Meiosis is peculiar to eukaryotes, but most of the enzymes involved were present in prokaryotes, performing a different function.

We think that the theory summarized in Fig. 7.2 is plausible. There are unanswered questions, but the answers may come from a study of existing organisms. In particular, we need to know more about the ecology of organisms with alternating haploid and diploid phases, and about the genetics of the Archaea.

**Mating types and sexual differentiation**

Almost all sexual protists have two ‘mating types’, + and -. Gametes of the + type will fuse only with -, and vice versa. This has the effect that a gamete will not fuse with another too similar to itself. This is easy to understand. If, as we suggested, the function of gamete fusion is to confer hybrid vigour, fusion of identical gametes, and in particular of gametes that have just arisen by the meiotic division of a single cell, should be avoided. But it is at first sight puzzling that there are just two mating types. Why not many types, any one of which could fuse with any other? This would increase the proportion of gametes with which any particular gamete could fuse. To digest for a moment: something like this happens in flowering plants. Most are hermaphrodite, producing both seeds and pollen, but are self-incompatible: that is, they cannot pollinate themselves. Usually there are many self-incompatible types, and pollen from any one of them can pollinate any of the others. This is just what one would expect. So why only two mating types?

A possible answer is that it is easier to devise a system with two mating types than with many, but evolution is not usually so un inventive. Whatever may have been the case in the first sexual organisms—and two mating types may well be primitive—today the explanation seems to be that mitochondria, and chloroplasts if they are present, are inherited from one parent only. For example, in the well-studied green alga, _Chlamydomonas_, mitochondria are inherited only from the + mating type and chloroplasts only from the - type. This rule of uniparental inheritance of intracellular organelles is almost universal. In animals, mitochondria are maternally inherited. In plants, chloroplasts are usually transmitted in the ovule, not the pollen, but there are exceptions: in conifers, chloroplasts are transmitted in the pollen. It is therefore not surprising that, unlike flowering plants but like mammals, conifers are never parthenogenetic —another example of the difficulty of reverting from sex to parthenogenesis.

Why should uniparental inheritance of organelles be so nearly universal? The likely answer is that, if mitochondria, say, were inherited from both parents, the stage would be set for the evolution of ‘selfish’ organelles. At cell division, mitochondria are randomly passed to the two daughter cells; there is no device ensuring that one copy of each mitochondrion passes to each daughter cell, as is the case for chromosomes. Therefore a mitochondrion that multiplied more rapidly within the cell, but which was less effective in making ATP, would spread through the population. Such mitochondrial mutants do occur, but
because of uniparental inheritance they cannot spread, although they may kill
the cell they are in.

Given the need for uniparental inheritance of organelles, it is hard to evolve
more than two mating types, one that provides the organelle and the other that
does not. In 1992, Laurence Hurst and William Hamilton published a remark-
able set of observations showing that this is indeed the right explanation. In
ciliated protozoa (for example, the slipper animalcule, *Paramecium*), there is
usually no gamete fusion. Instead, two cells lie side by side, or ‘conjugate’, and
each passes a haploid nucleus to the other, without any cytoplasmic mixing.
The conjugating cells then separate. Each is a diploid, with a chromosome set
from both parents, but each with its own mitochondria. In these ciliates, there
are multiple incompatibility types, as expected because there is no risk of the
spread of selfish organelles. The proof goes further. In one group of ciliates, the
hypotrichs, there are two alternative sexual processes, conjugation and gamete
fusion. There are multiple mating types for conjugation, but only two deter-
mining gamete fusion. It is pleasing when peculiar and otherwise baffling facts
such as these make sense in terms of a theory that was developed in ignorance
of them.

In animals and plants, there is a division of labour between a motile gamete
and a gamete carrying the food stores needed for the development of a large
adult. There are mathematical models showing the circumstances in which such
a division of labour would pay: they confirm that adult size is a crucial variable
favoured by anisogamy. There is again some support for the theory by com-
paring related isogamous and anisogamous species. *Volvox* is a genus of green algae,
related to *Chlamydomonas*, but forming multicellular colonies—hollow spheres
of green ciliated cells. In species with small colonies, the gametes are all motile
and all the same size. In colonies of intermediate size, the gametes are again
motile but of varying size. In species with the largest colonies, there are large
non-motile gametes and small motile ones. In *Volvox*, we can see on a small
scale the evolutionary path once travelled by the ancestors of the animals and
plants. It is interesting that the Bible gets it right. Males were the first sex:
females were secondary.

Given anisogamy, secondary sexual characters may evolve. The central logic
is that eggs are costly to produce and sperm cheap. Males, therefore, may have
more to gain than females by mating repeatedly. This asymmetry often leads to
differences in size, weapons, and ornaments between the sexes. This is a grossly
oversimplified summary of a complex subject. For example, there are ways of
investing in offspring other than putting nutrients into the gametes. If, as in sea
horses, males incubate the eggs, females may compete for males rather than the
other way round.

To summarize, we can detect three stages in the history of sexual differen-
tiation. First, the evolution of two mating types, + and −, driven by the need for uni-
parental inheritance of intracellular organelles. Second, the evolution of males
and females, producing motile and food-storing gametes, respectively. Third,
the evolution in some lineages of secondary differences between the sexes, driven
in part by competition for mates, and in part by a division of labour in raising
the young.