

12. Evolution of sex

Suppose that in a sexual population a mutant female arises that reproduces asexually, e.g. through parthenogenesis. If she produces the same number of offspring, she will have twice as many daughters as the sexual females. Since these daughters inherit the mutation, they will also reproduce asexually, and the mutation will rapidly spread in the population (Figure 12.2). This obvious disadvantage of sexual reproduction has been denoted the ‘two-fold cost of sex’. Yet most species reproduce sexually. Why? Even today, this paradox of sex is still one of the greatest mysteries in biology. It has also found considerable interest in the public (Figure 12.1). The largest part of this lecture will be devoted to the question ‘Why sex?’.



Figure 12.1 Title pages of the German weekly magazine *Der Spiegel* (38/2003, 41/2005) address the important question: Why sex?

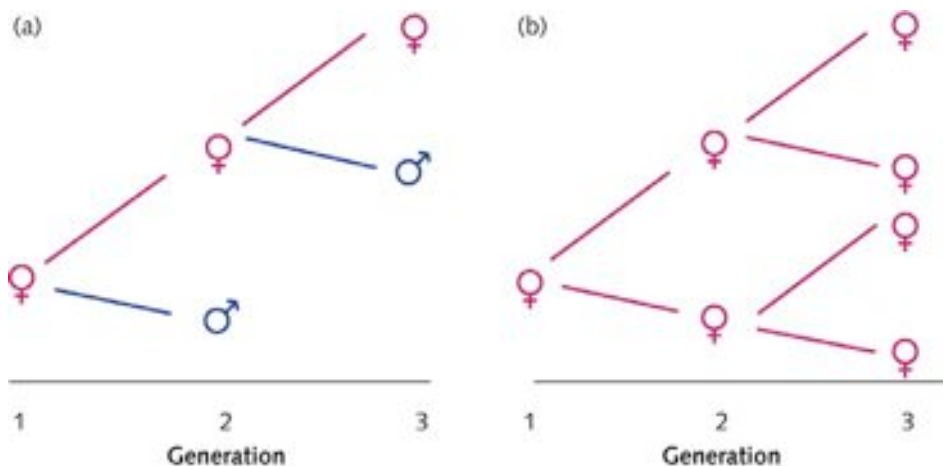


Figure 12.2 The two-fold cost of sex.

12.1. Why sex?

The 'two-fold cost of sex'

Let us start with a closer look at the 'two-fold cost of sex'. Is this really true? There are several simplifications and assumptions implicit to this concept. First of all, we should more correctly speak of a 'two-fold cost of meiosis' rather than a 'two-fold cost of sex'. For example, simple forms of sex that do not involve meiosis, such as the exchange of genetic material between two individuals of *Paramecium* come without a two-fold cost. More generally, only when there are males and females, which produce small, 'cheap' sperm and large, 'expensive' eggs, respectively, a two-fold cost arises. This situation is denoted as *anisogamy* (i.e. having gametes of different sizes). By contrast, a two-fold cost does not arise in isogamous organisms, so it is rather a cost of males than a cost of sex. Most likely, the first sexual organisms were isogamous. This implies that the two-fold cost of sex is irrelevant for the *origin* of sex (which occurred before anisogamy).

Moreover, the model makes two important assumptions: Firstly, a female's reproductive mode should not affect the *number of offspring* she can raise. For example, there should be no help received from fathers when raising the offspring (i.e. no paternal care), which would likely increase this number. Throughout the animal kingdom, paternal care is rather an exception than the rule, so this assumption may not be too critical. Secondly, the female's reproductive mode should not affect *offspring fitness*. This assumption is a particularly critical one, as we will see in the following. Offspring fitness could have long-term effects, if grand-offspring inherit a potential fitness benefit, etc. It is thus important to note that the cost of sex is short term (affecting the next generation), whereas potential evolutionary benefits may 'accumulate' over generations!

A long-term benefit of adaptation?

The following experiment addresses the second assumption. Is there a long-term benefit of sex that might compensate for its short-term cost? Flour beetles *Tribolium castaneum* were used in this experiment.³² They were exposed to a new environment; flour containing the insecticide malathion. Since this species does not reproduce asexually, an 'asexual' strain was simulated by replacing the beetles with individuals from a separate reservoir population, thus that every generation was genetically identical to the generation before (Figure 12.3). This resembles the absence of sexual reproduction. To introduce a cost of 'sex', any one beetle in the 'asexual' strain was replaced with three beetles (which is an even three-fold cost, just to be sure). Beetles were directly competing and distinguishable by their colour (red or black, which was alternated between the treatments in replicate experimental rounds).

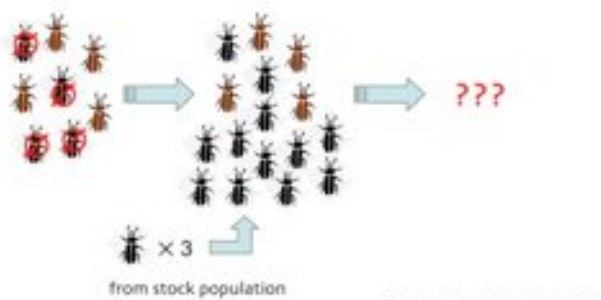


Figure 12.3 Design of an experimental test of the hypothesis that adaptation to a new environment has long-term fitness benefits. See text for details.

³² Dunbrack, R.L., *et al.* (1995) The Cost of Males and the Paradox of Sex - Experimental Investigation of the Short-Term Competitive Advantages of Evolution in Sexual Populations. *Proceedings of the Royal Society of London Series B-Biological Sciences* 262, 45-49

The outcome of this experiment was pretty clear (Figure 12.4). The ‘sexual’ strains had an initial disadvantage, but on the long term, they always won! The speed with which they outcompeted the ‘asexuals’ increased with the concentration of insecticide. In control treatments where both colours were replaced, but only one had a three-fold advantage, this colour always won.

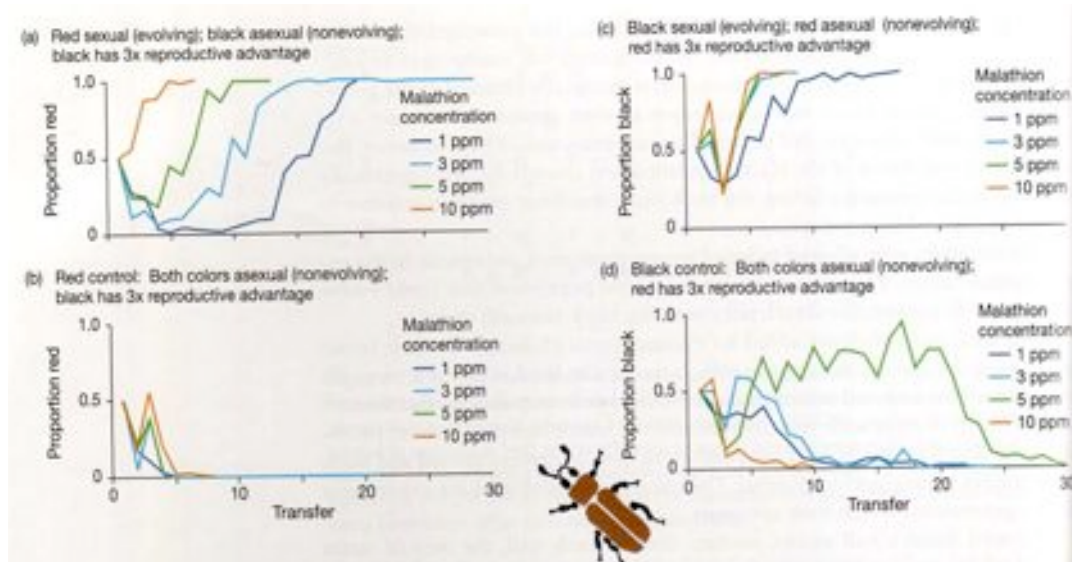


Figure 12.4 Results of an experimental test of the hypothesis that adaptation to a new environment has long-term fitness benefits. See text for details.

Sex means recombination

The above experiment shows that the strain that could adapt to a new environment had an advantage on the long run. This could imply that sexual reproduction is beneficial when adaptation to a changing environment is necessary. However, the experiment did not directly address potential benefits of sex. Before focussing more closely on this question, we need to clarify what sex actually is. There is a rather simple answer that is not so far away from reality: Sex means recombination!

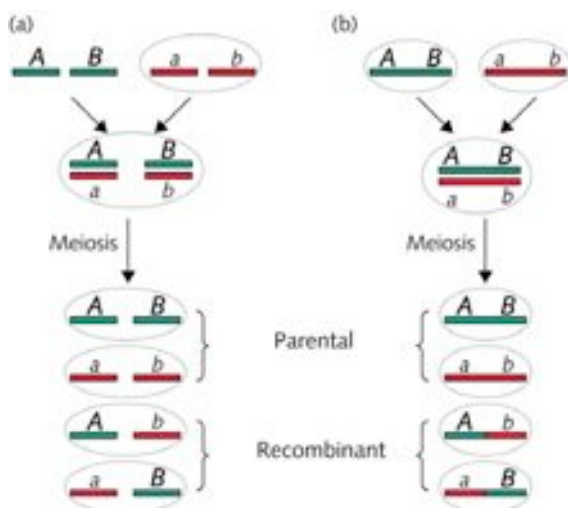


Figure 12.5 Recombination between two loci on different chromosomes may result from Mendelian independent assortment (a) and recombination between loci on the same chromosome from crossing-over (b).

In most organisms recombination takes place during meiosis. Two important processes lead to recombination during meiosis I, the first round of division (Figure 12.5): *Segregation* ensures that every gamete contains a complete haploid set of chromosomes and that the paternally and maternally derived chromosomes are equally represented among the gametes. Importantly, paternally and maternally derived chromosomes are independently assorted, giving rise to gametes with new combinations of chromosomes. While segregation mixes whole chromosomes, *crossing-over* is the exchange of chromosome parts. This process thus leads to new combinations of alleles on the same chromosome. When the chromosomes are pairing up together for a short time before being separated during synapsis, chromosomal crossing-over occurs. During this time, non-sister chromatids of homologous chromosomes may exchange segments at random locations called chiasmata (Figure 12.6).

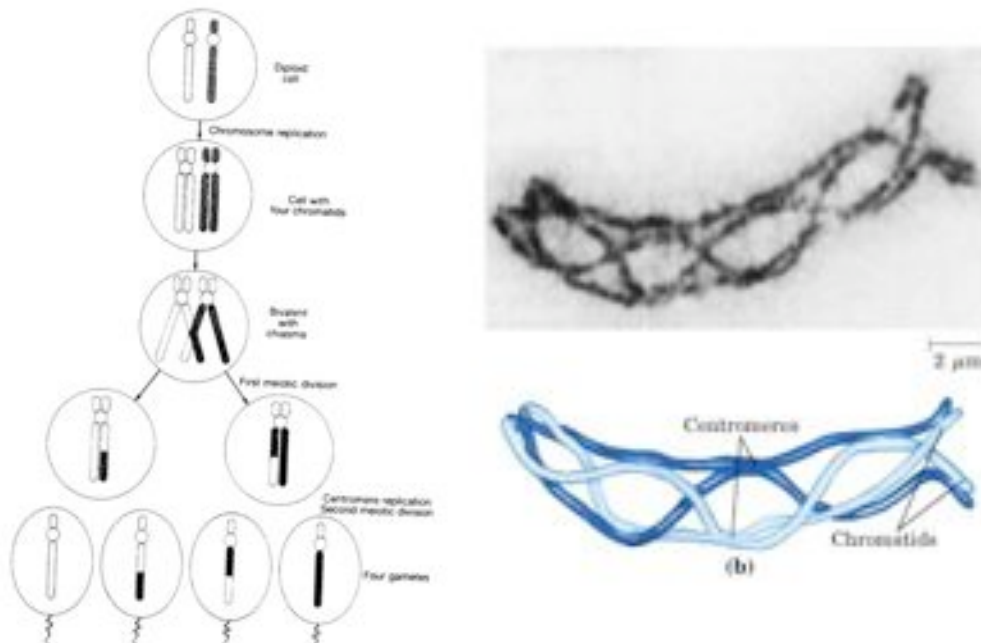


Figure 12.6 Recombination between loci on the same chromosome occurs through the process of crossing-over (right), which is observable as chiasmata formed between bivalents during meiosis (left).

Recombination eliminates linkage disequilibrium.

More important for us than the details of the genetic processes are the population genetic effects of recombination. What effect does recombination have? The answer is straightforward: Recombination eliminates linkage disequilibrium. Per definition, *two loci are in linkage disequilibrium (D) when there is a non-random association between the alleles at these loci*, i.e. when we know the genotype at one locus, it provides a clue about the genotype at the other. For example, when two rare mutations occur together more often than expected by chance, these are in linkage disequilibrium. Note this is a population genetic definition about allele frequencies in a population, not a genetic statement about the ‘physical’ linkage of two loci.

Assume there are two loci with two alleles each (A,a and B,b respectively). *D* is then defined as a deviation from the random expectation for the frequency of the genotypes:

$$D = g_{AB} g_{ab} - g_{Ab} g_{aB}$$

$$D = ps qt - pt qs$$

with allele frequencies: $A: p$
 $a: q = 1-p$
 $B: s$
 $b: t = 1-s$

with: $-0.25 < D < 0.25$

If D is negative, the extreme genotypes AB and ab are under-represented. If D is positive, they are over-represented.

What is the effect of recombination on D ? We need to calculate the change in gene frequencies from one generation to the next through recombination. To begin with, let us first consider only the genotype AB , i.e. we get from g_{AB} to g'_{AB} in the next generation (based on two-locus Hardy-Weinberg equilibrium). All possible genotypes are listed in Table 12.1.

Table 12.1 All possible genotypes for two loci, shown as all possible combinations of gametes. Red = genotypes that can give rise to AB gametes.

	AB	Ab	aB	ab
AB	AB/AB	AB/Ab	AB/aB	AB/ab
Ab	Ab/AB	Ab/Ab	Ab/aB	Ab/ab
aB	aB/AB	aB/Ab	aB/aB	aB/ab
ab	ab/AB	ab/Ab	ab/aB	ab/ab

Table 12.2 Calculation of the frequencies with which the genotypes make AB gametes

	AB	Ab	aB	ab
AB	AB/AB	AB/Ab	AB/aB	AB/ab
Ab	Ab/AB	Ab/Ab	Ab/aB	Ab/ab
aB	aB/AB	aB/Ab	aB/aB	aB/ab
ab	ab/AB	ab/Ab	ab/aB	ab/ab

$g_{AB} g_{AB}$ (points to AB/AB)
 $1/2 (2 g_{AB} g_{aB})$ (points to AB/Ab and Ab/AB)
 $1/2 (2 g_{AB} g_{aB})$ (points to aB/AB and Ab/aB)
 $r 1/2 (2 g_{Ab} g_{aB})$ i.e. when they recombine at a rate r (points to AB/aB and Ab/aB)
 $(1-r) 1/2 (2 g_{AB} g_{ab})$ i.e. only those that did not recombine make AB (points to AB/ab and ab/AB)

We are interested in the frequency of AB in the next generation, but only part of these genotypes can produce AB gametes. These are the circled ones in Table 12.2, which give rise to AB at the indicated frequencies. g'_{AB} is the sum of all these frequencies, which can be simplified to: $g'_{AB} = g_{AB} - r D$.

With similar calculations, we get:

$$g'_{ab} = g_{ab} - r D; \quad g'_{Ab} = g_{Ab} + r D; \quad g'_{aB} = g_{aB} + r D$$

We can now calculate the new D' in the next generation as:

$$D' = g'_{AB} g'_{ab} - g'_{Ab} g'_{aB}$$

$$D' = (g_{AB} - r D) (g_{ab} - r D) - (g_{Ab} + r D) (g_{aB} + r D)$$

$$D' = (g_{AB} g_{ab} - g_{Ab} g_{aB}) - r D (g_{AB} + g_{ab} + g_{Ab} + g_{aB})$$

$$D' = D - r D = D (1 - r)$$

This means that recombination eliminates D (i.e. brings D closer to 0) in proportion to the recombination rate r . An empirical demonstration in *Drosophila* shows the elimination of D in a sexual population (Figure 12.7).

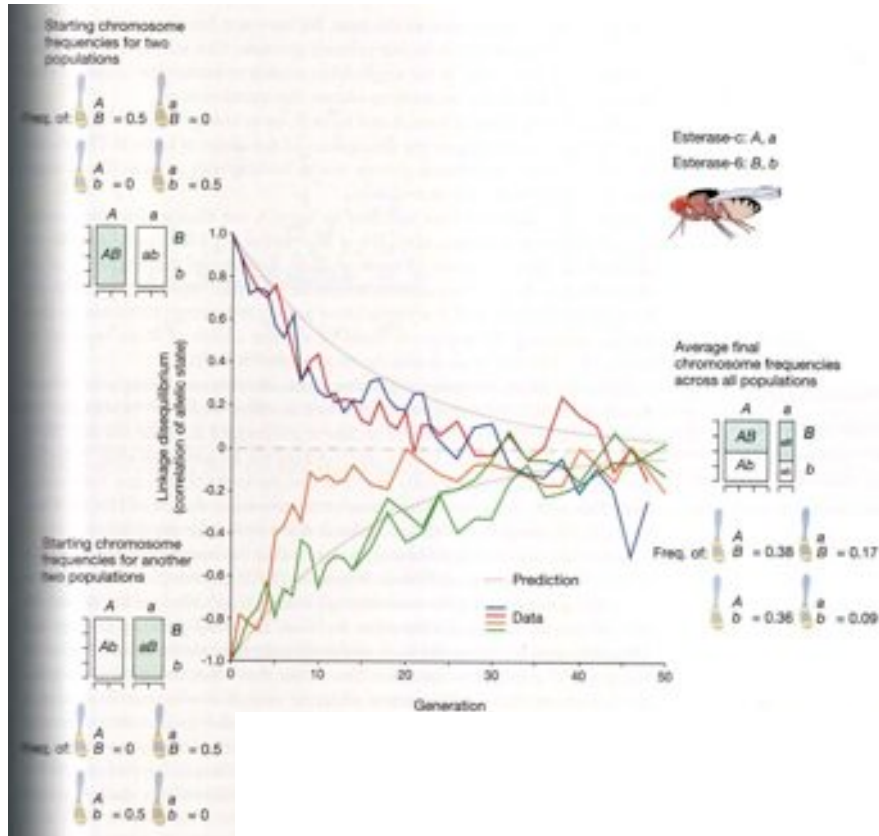


Figure 12.7 An empirical demonstration that sexual reproduction eliminates linkage disequilibrium. Each of several populations of fruit flies began in complete linkage disequilibrium. Over 50 generations, all populations approached linkage equilibrium. Note that linkage disequilibrium is here given as the correlation of allelic state, ranging from - 1.0 to 1.0 rather than from - 0.25 to 0.25, as would be the case for the standard notation (see text).

The higher r , the faster will D be eliminated. We should thus also expect that r is higher when elimination of D is most needed. If sex (i.e. recombination) is particularly beneficial for adaptation to changing environments (as indicated by the experiment on *Tribolium*; Figure 12.4), then we might expect that in artificial selection experiments (which represent in fact adaptation to a new environment), r might increase, as a correlated response. This seems indeed to be the case (Figure 12.8).

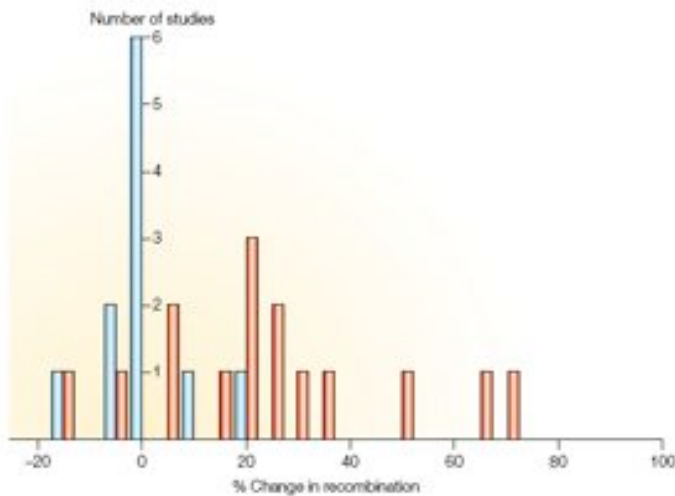


Figure 12.8 Artificial selection favours increased genetic recombination. This graph summarizes the results of experiments in which researchers artificially selected traits unrelated to sex and recombination. Among the experiments in which selection produced a significant change in recombination rate (red bars), the majority of the changes were increases.

Benefits of sex: mutation-based approaches

A ‘classical’ explanation for the benefits of sex is based on the *Fisher-Muller theory*: Sexual populations evolve faster, because independently arising *beneficial mutations* combine more quickly in the same genotype (Figure 12.9). However, most mutations are deleterious, while beneficial mutations are rare. Therefore, an asexual lineage will likely out-compete sexuals before a potential long-term benefit from the combination of such rare beneficial mutations becomes important.

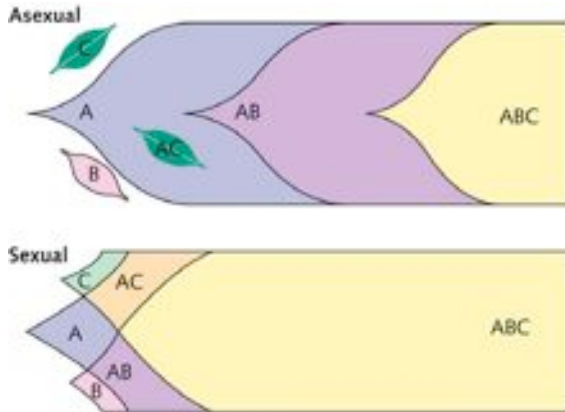
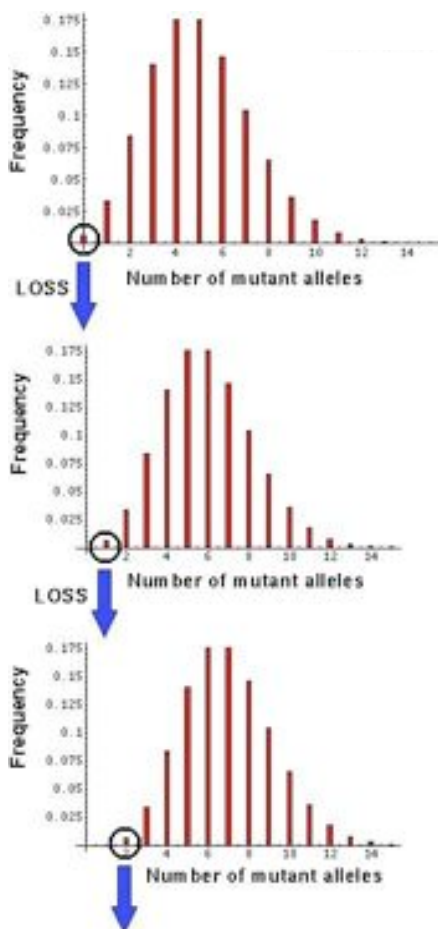


Figure 12.9 Sex can combine beneficial mutations that arise independently in different individuals; with asexual reproduction the mutations must be accumulated sequentially in the descendants of a single mutant individual.



‘*Muller’s Ratchet*’ is another classical explanation for sex. It is based on *deleterious mutations* and the effect of *genetic drift* (the random loss of genotypes in finite populations). It denotes a mechanism operating in finite asexual populations whose effect is that the number of deleterious mutations can only increase over time (Figure 12.10). Recombination is beneficial, because it can re-create the group with the lowest mutational load. There is some empirical evidence supporting Muller’s Ratchet. Bacterial cultures have been shown to accumulate deleterious mutations (Figure 12.10). In one of such experiments, 444 cultures of asexually growing bacteria (*Salmonella typhimurium*) were subjected to periodic bottlenecks that exposed the cultures to genetic drift. After ca. 1700 generations, 1 % of the cultures had seriously reduced fitness (longer generation time). Although this is not a particularly strong effect, it does in principle confirm Muller’s Ratchet. However, the process is slow and mutation rates are probably too low in natural populations.

Figure 12.10 Muller’s Ratchet: the random loss of the class of individuals with the lowest number of deleterious mutations shifts the population towards higher mutational load.

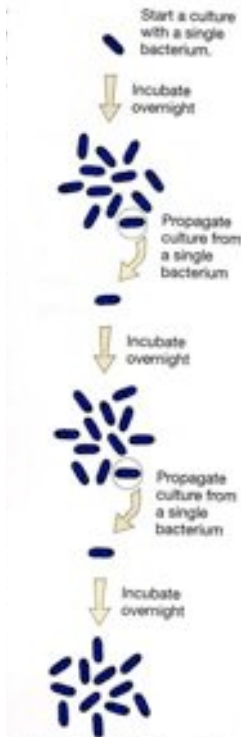


Figure 12.11 Experimental evidence for Muller's Ratchet. A bacterial population subjected to periodic bottlenecks (i.e. a single bacterium) provides an opportunity for genetic drift to operate.

Benefits of sex: selection-based approaches

The knowledge we have obtained in the previous paragraph about the influence of recombination on *linkage disequilibrium* D will help us to get deeper insight into modern hypotheses for the benefits of sex. We have seen that recombination eliminates D , i.e. brings it closer to 0. Thus, there can only be a benefit of sex when $D \neq 0$. To find out under what circumstances sex is beneficial, we thus have to ask: What creates D ? The following processes are most important:

1. Drift (mutation and selection in finite populations)
2. Selection (on multi-locus genotypes)
3. Gene flow (population admixture)

Current hypotheses for the benefits of sex focus on selection as the most important cause. These models are based on the notion that the change in D is the main benefit of sex, but this depends on the value of D and another important value, epistasis, which we will introduce below. First, with regard to D alone, it has often been argued that sex would be beneficial because it "*increases genetic variance*". However, this is not always true. More precisely, the effect of sex on genetic variance depends on D :

If $D = 0$:

Genes in the population are already well mixed, thus sex has *no effect* on genetic variation.

If $D < 0$:

Extreme genotypes AB and ab are under-represented; sex produces them and thus increases variance.

If $D > 0$:

Extreme genotypes AB and ab are over-represented; sex reduces them and thus decreases variance.

Thus, only if $D < 0$, sex will increase genetic variance. There is a *long-term advantage to sex and recombination whenever D is negative*, because it improves the response to selection and increases mean fitness on the long run. However, for a more detailed approach we will now also include potential short-term effects on fitness.

Benefits of sex depend on linkage disequilibrium and epistasis

For the discussion of potential benefits of sex, we need to deal with selection on multi-locus genotypes, i.e. selection that affects more than one locus simultaneously. The relevance of selection on multi-locus genotypes is obvious, since we have seen that recombination eliminates linkage disequilibrium, i.e. associations between loci. To understand the effect of *selection on multiple loci*, we need a term that describes fitness interactions between alleles at these different loci: epistasis.

Epistasis ϵ is a measure of fitness interactions between alleles at different loci.

$$\epsilon = W_{AB} W_{ab} - W_{Ab} W_{aB}$$

Epistasis describes a situation where some *combinations* of genes are more ‘fit’ than others. For example, if *A* and *B* are the wild-type genotypes at the two loci, then the deleterious mutations *a* and *b*, respectively, might be more harmful when occurring together (*ab*), compared with the expectation from each of their effects when alone (we then speak of negative epistasis). By contrast, if the double mutant *ab* has higher fitness, we speak of positive epistasis (Figure 12.12).

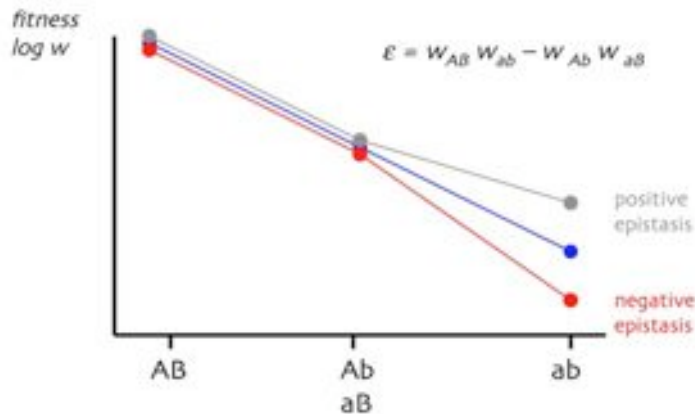


Figure 12.12 A graphical representation of epistasis

Note that epistasis ϵ is a measure of the *fitness* of gene combinations, whereas linkage disequilibrium D is about allele frequencies in a population. When selection has occurred, epistasis will normally create linkage disequilibrium. For example, in the case of positive epistasis, individuals having both mutations have higher fitness, and will therefore be over-represented in the next generation. We should thus expect a positive correlation between epistasis and linkage disequilibrium ($D \sim \epsilon$), at least in a large population that is subject to constant and weak directional selection.

To resolve the question of benefits of sex, we need to find out the conditions of epistasis and linkage disequilibrium that favour recombination³³. This question has been addressed with *modifier models*. These models determine the fate of a modifier M that alters the recombination rate r between two selected loci (A and B) by an amount δr . Alleles a and b , when present alone, change the survival probability of an individual by an amount of s_a and s_b , respectively.

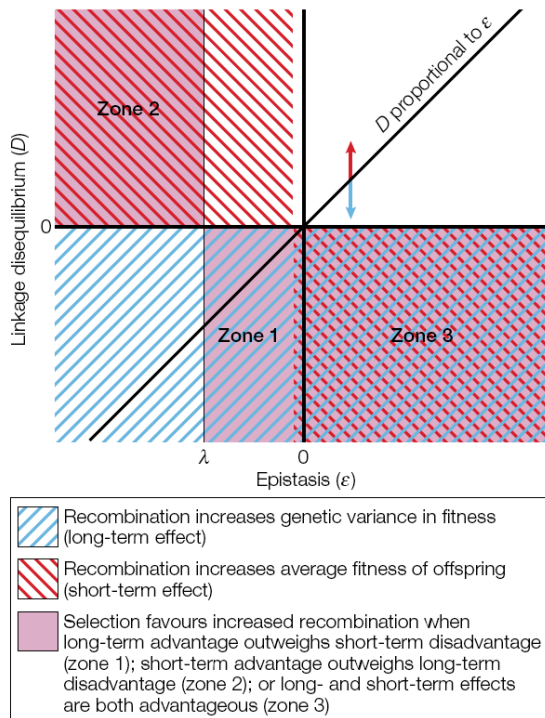
In the simplest haploid model, selection on the modifier is given by: $(\delta r/r_{MAB}) D (\lambda - \epsilon)$

with: r_{MAB} = rate of recombination between alleles at M , A and B

$$\lambda = -s_a s_b (1/r_{MA} + 1/r_{MB} - 1)$$

This equation determines the conditions under which selection favours an increase in the rate of recombination. It shows that the long-term effect is very sensitive to the rate of recombination between the modifier and the selected loci, because this determines whether the modifier remains associated with the allelic combinations that it creates long enough. A very useful graphical representation is given in Figure 12.13. The shaded zones 1, 2 and 3 are combinations of ϵ and D where sex (i.e. recombination) is favoured.

³³ Otto, S.P., and Lenormand, T. (2002) Resolving the paradox of sex and recombination. *Nature Reviews Genetics* 3, 252-261



- Zone 1: As said above, most populations will converge along the line where $D \sim \epsilon$. In this case, only zone 1 is relevant, where ϵ is weak and negative. The long-term benefit of sex (eliminating neg. D) outweighs its short-term disadvantage (creating extreme genotypes such as ab that have lower fitness, since ϵ is neg.).

- Zone 2: Extreme genotypes are over-represented (pos. D), but have lower fitness (neg. ϵ). Sex removes them and thus has a short-term advantage that outweighs the long-term disadvantage (elimination of pos. D).

- Zone 3: Extreme genotypes are rare (neg. D) and have higher fitness (pos. ϵ). Sex creates them and thus has a short-term benefit. Moreover, it here also has a long-term advantage (elimination of neg. D increases variance).

Figure 12.13 A graphical scheme addressing the question: When does selection favour sex and recombination in a single large population? See text for details.

Empirical studies have failed to show that weak and negative epistasis (criteria for zone 1) is common. It follows that we need to find out in what situations populations might be in zones 2 or 3. In these zones, D and ϵ have opposite sign. As said, this condition should be rare in most large populations that are subject to constant and weak directional selection, which leads to a positive correlation between epistasis and linkage disequilibrium ($D \sim \epsilon$, as indicated by the diagonal line in Figure 12.13). However, selection that varies over time or space can generate situations where D and ϵ have opposite sign. Host-parasite co-evolution is a particularly good candidate.

The Red Queen

Situations that fulfil the above criteria may arise when epistasis fluctuates rapidly, which is particularly likely in situations of *Red Queen* dynamics, e.g. when parasites and hosts co-evolve (Figure 11.13). Since co-adaptational cycles of parasite and host allele frequencies are based on *frequency-dependent selection* allele combinations that had higher fitness, due to parasite resistance, in the previous generation will be more common in the current generation. If parasites have adapted, these more common genotypes will have lower fitness in the current generation. However, although intuitively true, evidence that host-parasites co-evolution can create such rapid changes in epistasis is still scarce. Models indicate that fluctuations in epistasis should be fast (over a few generations) and parasites should be highly virulent for this process to work. Some correlative evidence at least supports the general idea that sexual reproduction is more common in populations that suffer more strongly from parasites (Figure 12.14).³⁴

³⁴ Lively, C.M. (1992) Parthenogenesis in a freshwater snail - reproductive assurance versus parasitic release. *Evolution* 46, 907-913

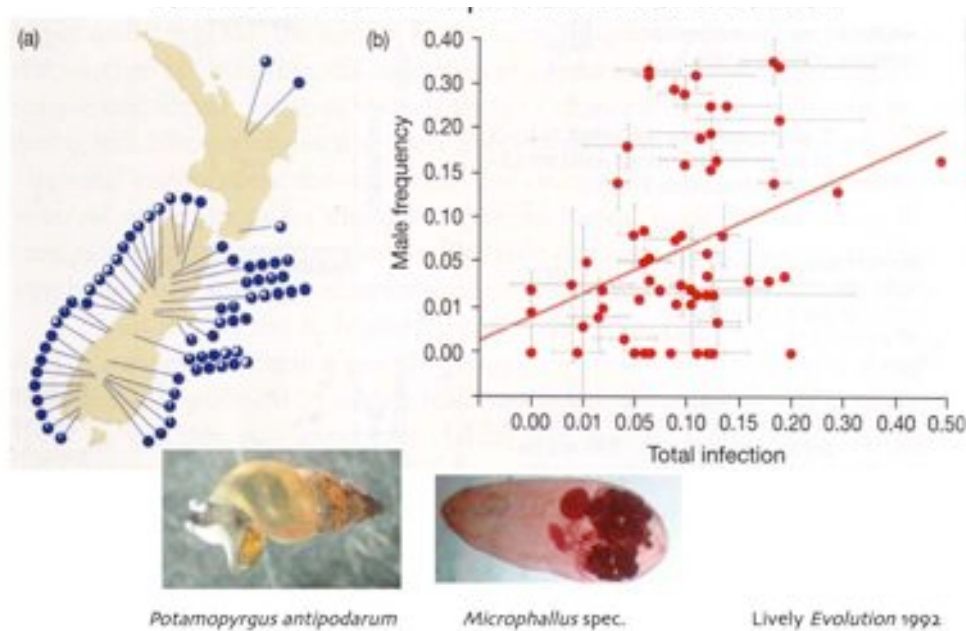


Figure 12.14 The frequency of sexual individuals in populations of a host snail is positively correlated with the frequency of its trematode parasites. (a) Locations of the 66 lakes sampled; in each population's pie diagram, the white slice represents the frequency of males. Since populations consist of both parthenogenetic and sexual females, the frequency of males is associated closely with the frequency of sexual reproduction. (b) Frequency of males in each population versus the proportion of snails infected with the trematode.

Next to host-parasite co-evolution, *spatially heterogeneous selection* in combination with migration could also generate a mismatch between the genetic associations (D) that are present in a patch and the form of local selection (ϵ).

Further ecological hypotheses for the benefit of sex

Ecological hypotheses for the benefit of sex generally focus on either temporal or spatial variation in the physical or biotic environment (Figure 12.15). The argument of the *lottery model* is that asexual reproduction is like buying a number of tickets with the same number, while sexual reproduction is a diversified approach. The mean "payoff" to each strategy may be similar, but the variance for asexual reproduction should be greater. In biological terms this would translate into a higher rate of lineage extinctions for asexual species. The problem with lottery models is that the conditions under which sexual reproduction is advantageous appear to be fairly restrictive.

Graham Bell's '*Tangled Bank*' concept derives its name from the closing passage of Charles Darwin's *Origin of Species* (1872):

*"It is interesting to contemplate a **tangled bank**, clothed with many plants of many kinds, with birds singing on the bushes, with various insects flitting about, and with worms crawling through the damp earth, and to reflect that these elaborately constructed forms, so different from each other, and dependent upon each other in so complex a manner, have all been produced by laws acting around us."*

In our context, '*Tangled Bank*' concept proposes an advantage for sexual reproduction in saturated environments (where soft selection predominates). Biological diversity provides ecological opportunity and successful individuals are those who can produce diverse offspring, thereby reducing sibling competition.

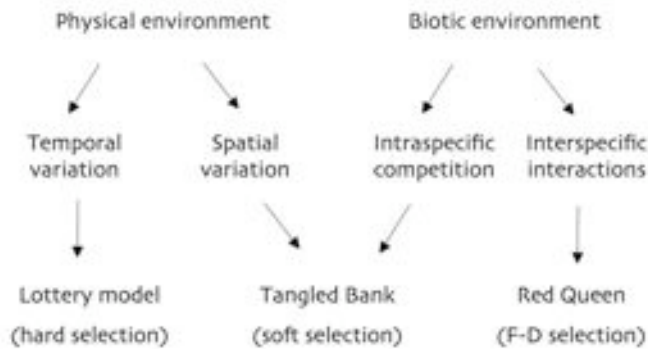


Figure 12.15 An overview of environmental hypotheses for the benefits of sex.

Red Queen vs. Tangled Bank

Under the Red Queen scenario, we might expect that (parasitic) environments are more contrary for species with longer generation times. This might lead to a positive correlation between generation time and recombination. By contrast, the Tangled Bank might predict that the larger the number of offspring, the higher the level of competition among offspring, which should create a positive correlation between litter size and recombination. Burt & Bell attempted to test this prediction, using a comparison between mammalian species (Figure 12.16).³⁵

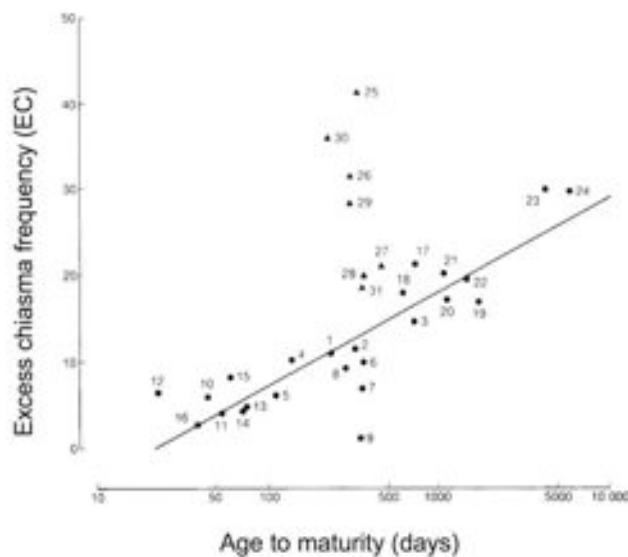


Figure 12.16 Semilog plot of mammalian excess chiasmata frequencies (as a measure for the rate of recombination) as a function of age to maturity. Only male mammals were considered. Circles, nondomesticated species; solid line, least square regression; triangles, domesticated species, not included in the regression. (numbers next to data point denote species names but are not relevant here).

They found no relationship between offspring number and recombination, but a positive correlation between generation time and recombination, which supports the Red Queen hypothesis. However, there were several objections against their interpretation:

- Species are no independent data points, phylogenetic contrasts should be used (Burt & Bell did so in a later analysis, and the general pattern remains).

³⁵ Burt, A., and Bell, G. (1987) Mammalian chiasma frequencies as a test of 2 theories of recombination. *Nature* 326, 803-805

- Alternative theories cannot be excluded, e.g. Muller's ratchet also predicts the positive correlation observed here, because species with long generation time tend to have smaller effective population size.
- Relative generation time of hosts and parasites is relevant, not absolute generation time of hosts. Long-lived hosts have long-lived parasites!

These objections show that it is in general difficult to draw definitive conclusions from correlative studies. What we need are more experimental studies on this subject. A recent experimental study in *Tribolium* flour beetles supports the expectation that recombination rate should increase in hosts that co-evolve with parasites (Figure 12.17).

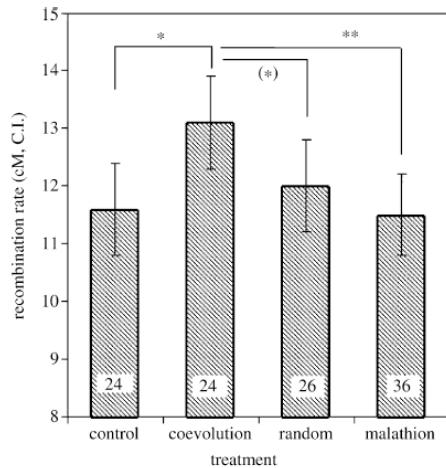


Figure 12.17 Recombination (in cM) varied with treatment (internal numbers are sample sizes, i.e. beetle lines). The treatments consisted of control lines, lines that coevolved with the parasitic microsporidian *Nosema whitei*, lines that were exposed to a random mixture of non-coevolving parasites, and lines subjected to directional selection on the insecticide malathion. Coevolution differed from control, but not from random while random was marginally different from malathion.

12.2. Forms of sexual and asexual reproduction

Sexual reproduction is by far the most widespread mode of reproduction. During evolution, sexuality seems to have evolved several times from asexual ancestors. Only very few taxa seem to reproduce completely asexual (Figure 12.18) and there is considerable debate whether or not these might have as yet unobserved, un-frequent sex. By contrast, there are quite a lot of species that occasionally reproduce asexually, thereby potentially combining the benefits of both modes of reproduction.



Figure 12.18 Certain species of Bdelloid rotifers (left) and ostracods (middle) are supposed to be ancient asexuals. Aphids (right) reproduce asexually through parthenogenesis most of the time, but shift to sexual reproduction under certain environmental conditions.

Haplontic and diplontic sexual life cycles

In most taxa, sexual reproduction involves meiosis. It depends on the position in the life cycle where meiosis occurs whether we are dealing with sexual haplontic or diplontic organisms (Figure 12.19)

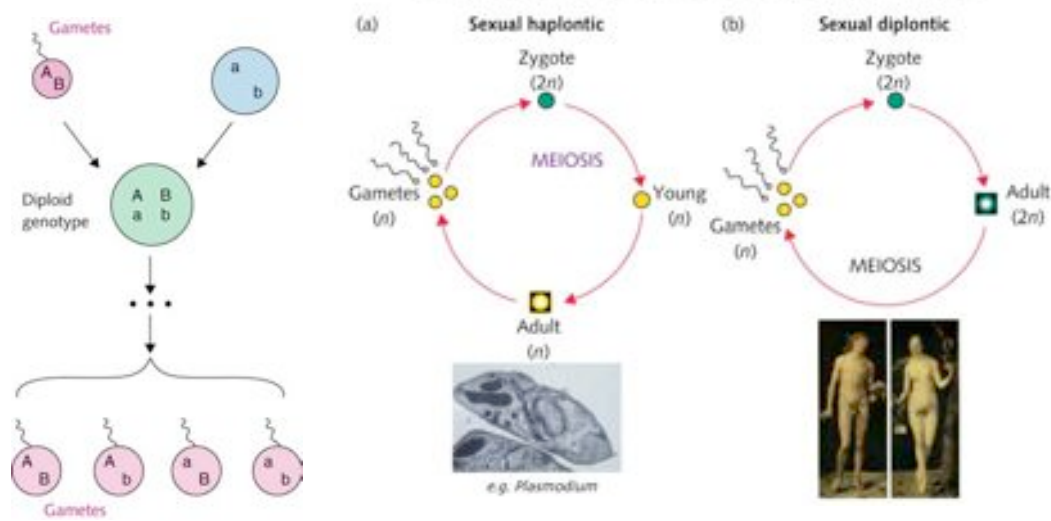





Figure 12.19 Left: Sexual reproduction normally involves that two haploid gametes fuse to form a diploid zygote. Right: The essential difference between haplontic and diplontic sexual life cycles is the position in the cycle where meiosis occurs. (a) If zygotes directly undergo meiosis, the resulting cells function as haploid offspring that produce gametes by mitosis. This is the case in some protozoans. (b) If zygotes do not undergo meiosis, they develop into diploid offspring that produce gametes by meiosis. This is the case in metazoans such as humans.

Reproductive systems

Meiosis is absent in asexual, apomictic species. Examples include Dandelions, (*Taraxacum officinale*) and the snail *Potamopyrgus*. Both these species reproduce sexually as diploids but have parthenogenetic triploids. Meiosis followed by syngamy between gametes produced by different individuals is the 'normal' mode of sexual reproduction (amphimixis). However, syngamy may also occur between meiotically produced nuclei of the same individual (automixis), which limits the effect of recombination. An example is the cape honeybee (*Apis mellifera capensis*): while workers of almost all subspecies of honeybee are able to lay only haploid male eggs, workers of this species are able to produce diploid female eggs by thelytokous parthenogenesis. Another example is the antler smut fungus *Microbotryum violaceae*. Figure 12.20 lists the consequences of these diverse modes of reproduction.

Mode of reproduction	Progeny genomes		
	Diversity	Heterozygosity	Costliness
 Apomixis ¹	Minimal	Maximal	Minimal
 Automixis ²	Intermediate	Low	Low
 Amphimixis ³	Maximal	High	Maximal

¹ Apomixis: the absence of both meiosis and syngamy (fertilization)
=> asexual, no recombination, parthenogenetic

² Automixis: syngamy between meiotically reduced nuclei descending immediately from the same zygote => limited recombination, parthenogenetic

³ Amphimixis: syngamy between gametes produced by different individuals
=> 'true sexual reproduction'

Figure 12.20 Modes of reproduction and its effects on diversity and heterozygosity of offspring.

12.3. Sex allocation

Sex allocation theory predicts the allocation of lifetime reproductive effort among male and female offspring or between male and female function. In gonochorists³⁶, it addresses the questions: What is the optimal *sex ratio*? In hermaphrodites³⁷, it asks: How much should an individual invest to male and female function, and when should a sequential hermaphrodite change sex?

Sex ratios

The evolutionary stable sex ratio is 50:50 under random mating, external fertilization and no parental care.³⁸ When a sex ratio is not 50:50, then enough fitness must be gained through the rarer sex to balance the fitness gained through the commoner sex (Shaw-Mohler Theorem, 1953). Adaptive sex ratio manipulation by mothers has been reported for several species. For example, high-ranking red deer produce more sons. The reason is that sons (but not daughters) of high-ranking female red deer have higher reproductive success. It thus pays for high-ranking females to produce more sons (Figure 12.21).³⁹

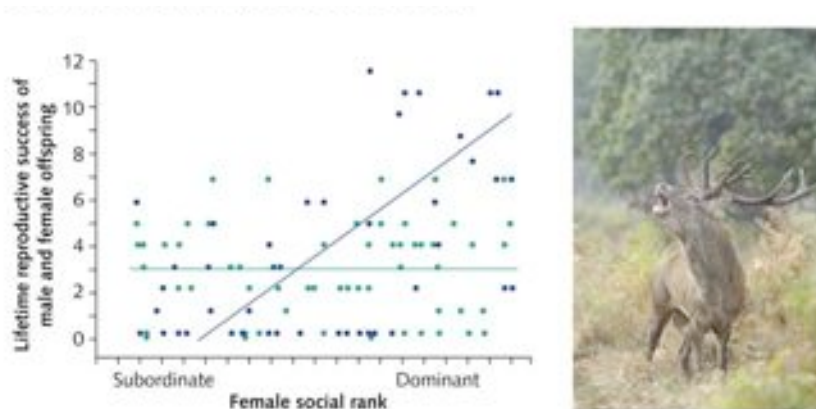
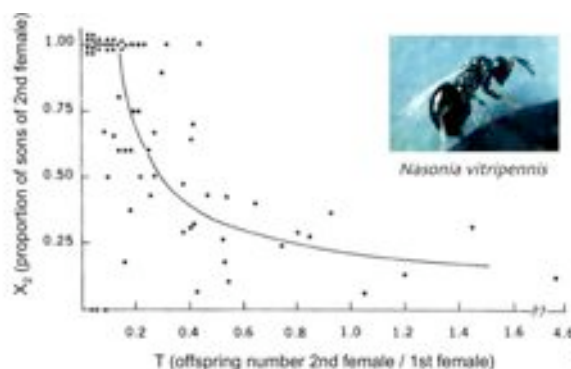


Figure 12.21 Offspring lifetime reproductive success, daughters (green) versus sons (blue), for female red deer of different social ranks.



Local mate competition occurs when the offspring of one parent compete with each other for mates. In an extreme case, one son can inseminate all daughters, thus a second son would be wasted. The optimal sex ratio is then one son and as many daughters as possible. Werren⁴⁰ showed that a hymenopteran parasitoid (*Nasonia vitripennis*) adjusts the sex ratio to local mate competition (Figure 12.22).

Figure 12.22 Sex ratio dynamics of the parasitic wasp *Nasonia vitripennis* in response to varying level of local mate competition.

³⁶ *Gonochorism / dioecy*: Individuals are either males or females. This might be advantageous because it is less costly to specialize on one sexual function.

³⁷ *Hermaphroditism / monoecy*: Individuals are both male and female, either simultaneously or sequentially. This is advantageous e.g. when mates are hard to find.

³⁸ Fisher, R.A. (1930) *The genetical theory of natural selection*. Clarendon Press

³⁹ Cluttonbrock, T.H., and Iason, G.R. (1986) Sex-Ratio Variation in Mammals. *Quarterly Review of Biology* 61, 339-374

⁴⁰ Werren, J.H. (1980) Sex-Ratio Adaptations to Local Mate Competition in a Parasitic Wasp. *Science* 208, 1157-1159

Sex change in sequential hermaphrodites

Sequential hermaphroditism may evolve where the advantage of being a particular sex changes with age or with size (Figure 12.23).

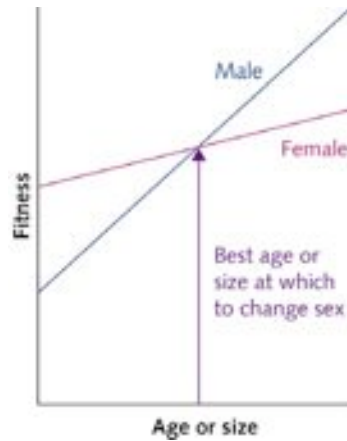


Figure 12.23 The size-advantage model for sequential hermaphroditism: it is best to be born as the sex that loses less by being young and small, and to change to the sex that gains more by being old and large.

The gastropod *Crepidula* (Figure 12.24), found in rocky inter-tidal habitats, is a sequential hermaphrodite that is first male, then female (*protandry*). Females gain more by being large. In the picture, a female is at the bottom, at the top is a male, and intermediates in the middle change from male to female. By contrast, the Blueheaded wrasse (*Thalassoma bifasciatum*) is first female, then male (*protogyny*), because it pays to be large as a male to be able to defend a large harem. The picture shows a large harem-holding male and smaller females. When the male is removed, the largest female attains male function within weeks.



Figure 12.24 Left: Protandry. *Crepidula* females gain more by being large. A female is at the bottom, at the top is a male, intermediates in the middle change from male to female. Right: Protogyny. A large harem-holding male and smaller females of *Thalassoma bifasciatum* (Blueheaded wrasse). When the male is removed, the largest female attains male function within weeks.

12.4. Take home messages

- *There is a “two-fold cost of sex”.*
- *Sex means recombination.*
- *Recombination eliminates linkage disequilibrium.*
- *Most hypotheses for the benefit of sex are based on mutations (Fisher-Muller, Muller’s Ratchet) or selection (Red Queen, Tangled Bank).*
- *Models show that sex is advantageous if epistasis is either weakly negative or if epistasis and linkage disequilibrium have opposite sign.*
- *Ancient asexuals are rare, but quite a few species occasionally reproduce asexually.*
- *Under simple assumptions, the sex ratio is 50:50. Sex ratio manipulation may be advantageous e.g. when there is local mate competition.*
- *Sex allocation theory explains why sequential hermaphrodites are either protandrous or gynandrous, depending on how the advantage of being a particular sex changes with age or with size.*

12.5. Literature

Recommended textbooks for this chapter:

Freeman, S. & Herron J.C. (2004) *Evolutionary Analysis*. 3rd ed. Pearson, Upper Saddle River, NJ.

Stearns, S.C. & Hoekstra, R. F. (2005) *Evolution: an introduction*. 2nd ed. Oxford University Press, Oxford.

Further reading:

Maynard Smith, J. (2002) *Evolutionary Genetics*. 2nd ed. Oxford University Press, Oxford / New York.

Otto, S.P., and Lenormand, T. (2002) Resolving the paradox of sex and recombination. *Nature Reviews Genetics* 3, 252-261