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Models of Evolution [and Discussion]

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Models of evolution

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Different ways in which evolution can be modelled will be reviewed. Two asexual models – 'Muller's ratchet' and a model due to Eigen and Schuster – both lead to the conclusion that the accuracy of replication must reach a limiting value, but the details are different. In classic population genetics models, difficulties arise if fitnesses depend on interactions with others. Two approaches – 'trait group' methods, and game theory – are discussed. If the interacting individuals are relatives, there is again a choice between the exact 'neighbour-modulated fitness' approach and the more intuitive 'inclusive fitness' method. A more drastic change in the nature of the model arises if the units of the evolving system are not individual organisms, but either genes or species. There are conceptual difficulties which must be clarified before species selection can be analysed mathematically.

This paper is a plea for pluralism in the ways in which we model evolution. By considering some of the main issues in contemporary evolutionary biology, I attempt to show two things. First, a given problem can often be approached in different ways, and a comparison of these may reveal more about the problem than would either method by itself. Second, any attempt to formalize an evolutionary problem mathematically obliges one to make, explicitly or implicitly, assumptions about what are the 'units of selection', and about how the total population is regulated; these assumptions have a crucial effect on the conclusions drawn.

The topics in evolutionary biology which in recent years have excited most interest and most research are, I think, as follows: the causes of molecular variability, the evolution of sex and breeding systems, the evolution of the genome, and the relation between macro- and micro-evolution. It is striking that, for each topic except the first, a major part of the difficulty lies in deciding how the process should be modelled, and in particular in deciding what are the appropriate 'units of selection' (Lewontin 1970). In this paper, I discuss some of the ways in which we can model evolution.

1. The asexual model

Darwin's theory of evolution by natural selection postulates the existence of a population of entities with the properties of multiplication (one entity can give rise to two), heredity (entities of kind A usually give rise to A, of kind B to B, and so on), and variation (heredity is not exact). Provided that the different kinds, A, B, etc., have different probabilities of surviving and reproducing, changes will occur in the kinds of entities present, which we call evolution. If heredity is exact,

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these evolutionary changes will ultimately cease, or perhaps tend to a repeated cycle of frequencies. If heredity is not exact, so that multiplication gives rise to ever new kinds of entity, evolutionary change may continue indefinitely.

This model omits much that we know to be important; in particular, it omits sex and genetic recombination, and also any reference to the genotype–phenotype distinction. Nevertheless, analysis of the model has led to some interesting results, particularly in making more precise the word 'usually' in the definition of heredity given above. How accurate must heredity be to permit evolution by natural selection?

The most thorough analysis of this model is by Eigen & Schuster (1979), who are interested in the origin of life from populations of replicating molecules. It is reasonable for them to ignore the genotype–phenotype distinction, because, if the relevant entities are replicating polynucleotides, genotype and phenotype are aspects of the same object, that is, its nucleotide sequence, and its folded structure and chemical properties, respectively. They conclude that, provided that the accuracy of replication is above a critical value, the population will come to consist of a unique sequence of maximum fitness, together with a cloud of similar sequences derivable from it by mutation; such an aggregate of sequences they call a 'quasi-species'.

Rather than give their full mathematical treatment, I here treat a highly simplified case, but one which leads to the same general conclusions. Suppose that some unique sequence, S, produces copies at a rate A, and that all other sequences produce copies at a lower rate, a. A sequence produces an exact copy of itself with probability Q. If x_0 and x_1 are the numbers of copies of S and non-S respectively, then, ignoring deaths, and the small probability that a non-S sequence will give rise to an S by mutation,

and
$$\left.\begin{aligned}\dot{x}_0 &= AQx_0,\\ \dot{x}_1 &= ax_1+A(1-Q)x_0.\end{aligned}\right\} \tag{1}$$

We now introduce the fact that the total number of entities cannot increase without limit, because of resource limitation. The simplest way to introduce such limitation is to assume that deaths exactly balance births in the population as a whole; then the total death rate must be Ax_0+ax_1. If death rates are the same for all types, so that selection acts only on births,

$$\left.\begin{aligned}\dot{x}_0 &= AQx_0-\{x_0/(x_0+x_1)\}(Ax_0+ax_1),\\ \dot{x}_1 &= ax_1+A(1-Q)x_0-\{x_1/(x_0+x_1)\}(Ax_0+ax_1).\end{aligned}\right\} \tag{2}$$

Hence, at equilibrium

$$A(1-Q)x_0 = (AQ-a)x_1.$$

It follows that, if an equilibrium is to exist with the optimal type present,

$$Q > a/A. \tag{3}$$

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Eigen & Schuster (1979) give a more general treatment, allowing for sequences with varying birth and death rates, but reach a result essentially similar to (3), with a replaced by a weighted average of the fitnesses of the mutant forms. This gives the critical accuracy of the hereditary process needed if evolution is to proceed.

Note that in this model, the proportion of the population with the optimal sequence is

$$x_0/(x_0+x_1) = (AQ-a)/(A-a). \tag{4}$$

If the accuracy Q is only just above the critical value, this fraction will be small.

It follows from this analysis that, for a given error rate per base replication, there is an upper limit of genome size. In the absence of enzymes, this limit may be quite low: perhaps less than 100 bases. Hence the 'Catch-22' of the origin of life: no large genome without enzymes, and no enzymes without a large genome. The possibility that mutation may set a limit on genome size has emerged from another direction, through consideration of 'Muller's ratchet' (Felsenstein 1974). Consider a population of N asexual organisms, of which x_0 have the optimal, fittest genotype, x_1 differ from the optimal type by one mutation, and x_i by i mutations. Then

$$N = \Sigma x_i.$$

Muller argued that, if x_0 is small, there is in every generation a chance that all x_0 individuals die without leaving progeny. If so, then in the absence of genetic recombination, and ignoring back mutation, there is no way in which the optimal type can be reconstituted. Thus an asexual population will gradually degenerate. The analogy of a ratchet is appropriate, because of the irreversible nature of the steps.

Suppose (Maynard Smith 1978) that the fitness of an individual with i mutations is $(1-s)^i$, and that the per genome mutation rate is U, then it can be shown that, at equilibrium between selection and mutation in an infinite population, the frequency of individuals with 0, 1, 2... mutations is distributed as a Poisson with mean U/s. Hence, in a finite population, the expected number of individuals in the optimal class is

$$E(x_0) = N\,\mathrm{e}^{-U/s}, \tag{5}$$

and the mean fitness of the population (relative to a fitness of 1 for the optimal genotype) is

$$\overline{W} = \mathrm{e}^{-U}. \tag{6}$$

Equation (5) does imply a limit on U, and hence on genome size, in a finite population. If x_0 is small (< 100, approximately) then the ratchet will operate.

It is interesting to compare the ratchet model with that of Eigen & Schuster. In an infinite population, the only limit on U in the ratchet model is set by (6); this is a less severe limitation than (3). Thus $a/A = 1-s$, and $Q = \mathrm{e}^{-U}$, so that (3) becomes $\mathrm{e}^{-U} > 1-s$. Suppose, for example, that $s = 0.1$, then a value of $Q = 0.8$ would by fully compatible with the survival of an infinite population on the ratchet model, but would not satisfy (3), and so would not permit the maintenance of the optimal type in the Eigen–Schuster model. The reason for the difference is that

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in the ratchet model fitness falls of continuously with number of mutations, whereas in my simplified version of the Eigen–Schuster model all mutants are equally fit, and so offer serious competition to the optimal genotype.

If s is small, it is quite possible that the ratchet will operate in a finite population, and yet $\overline{W}$ will not be unreasonably small. The relevance of this is as follows. In a sexual population the optimal class can be reconstituted by genetic recombination, and so the ratchet will not operate. However, the expected genotypic distribution, and the value of $\overline{W}$, are unaltered. Hence the value of U may be compatible with the survival of a sexual population, but not an asexual one. This raises several questions:

(*a*) what effect would molecular recombination have on the origin of life?

(*b*) If recombination has permitted the evolution of larger genomes, are secondarily derived parthenogens doomed to long-term extinction by the ratchet? Charlesworth (1978) has pointed out that sections of the genome (such as the Y chromosome) prevented from recombination are also susceptible to the ratchet.

(*c*) How much recombination is needed to avert the ratchet? It can be shown that (5) applies to a population of selfing diploids as it does to parthenogens (Heller & Maynard Smith 1979). However, most natural selfers show a low frequency of recombination. Is this sufficient to avert the ratchet?

2. Sexual populations

Classical population genetics adds to the model of §1 sexual reproduction and genetic recombination, usually in a diploid population. Such models are familiar. In such models, 'fitness' is a property of a genotype, and not of a gene or a population. It is specific to an environment, or set of environments, and is defined over a single generation: usually from zygote to zygote.

I want now to discuss some cases that call for an extension or modification of the classical approach, either because of the difficulty of ascribing fitnesses to individuals, or because the entities being considered are not individual organisms.

(*a*) *Fitnesses determined through interactions with others*

In general, the fitness of an individual will depend on interactions with others. It is convenient to start an analysis of such cases with a model (table 1) proposed by D. S. Wilson (1975). Zygotes are produced by random mating, but subsequently come together in 'trait groups', upon which selection acts. In Wilson's original model, two types were considered, say A and B, such that each A individual affected its own fitness by d, and that of all other members of the trait group by r, whereas B individuals had no such effects. Thus in a group containing np A individuals and $n(1-p)$ Bs, the fitness of an A individual is $C+d+(np-1)r$, and of a B individual is $C+npr$.

Wilson showed that, if individuals assort into groups randomly and if the population is infinite, then A will replace B selectively if d is positive. The value of r is irrelevant. This conclusion is readily seen by using Hamilton's way (Hamilton 1964) of looking at such problems: a gene causing an individual to increase by r the fitness of a *random* sample of the population will not thereby increase or decrease its own frequency in future generations.

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I have been critical of Wilson's paper (Maynard Smith 1976), mainly for semantic reasons. It seems inappropriate to describe the process as 'group selection' since I would prefer to confine that term to cases in which the entities with the properties of multiplication, heredity, and variation, whose evolution is being described, are in fact the groups, and not individuals.

TABLE 1. ADDITIVE AND SYNERGISTIC INTERACTIONS IN TRAIT GROUPS OF TWO INDIVIDUALS

trait group	*A*	*A*	*A*	*B*	*B*	*B*
fitnesses (additive)	$(c-d+r)$	$(c-d+r)$	$(c-d)$	$(c+r)$	c	c
fitnesses (synergistic)	$(c-d+r+b)$	$(c-d+r+b)$	$(c-d)$	$(c+r)$	c	c

In other words, if the model of figure 1 is to be treated as a case of group selection, one would analyse it in terms of the variables $x_0, x_1, x_2 \ldots x_n$, giving the frequencies of the different kinds of group (i.e. those containing $0, 1, 2 \ldots n$ *A* individuals), and derive equations for the values $x'_0, x'_1 \ldots x'_n$ in the next generation. The generation interval would then be period T_1 in figure 1. In practice, it is much easier to use the frequencies of genes or genotypes as the basic variables, and period T_2 as the generation interval; this, of course, is what Wilson does.

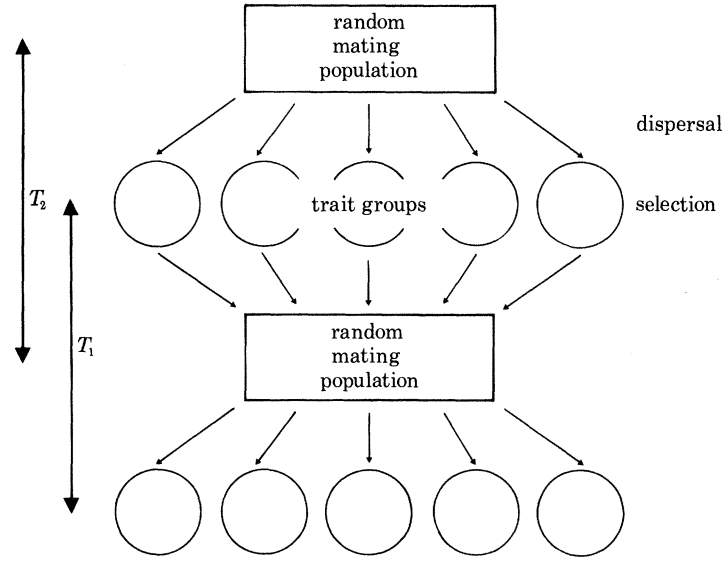

FIGURE 1. Wilson's trait-group model (Wilson 1975).

Leaving aside this purely semantic issue, however, Wilson's model has had some fruitful consequences. First, as a kind of 'null model', it brings out particularly clearly the situations in which the interaction term *r is* relevant to gene frequency change. There are two such situations.

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(i) interactions not additive. For example, table 1 illustrates this for trait groups of two individuals. The fitness of a pair of *A*s is greater than would be predicted from the interaction between an *A* and a *B*. Such synergistic interactions can lead to the evolution of cooperation even when interactions are between non-relatives.

(ii) Group assortment may not be random. Such cases, and in particular cases of 'kin selection', are discussed in the next section.

TABLE 2. THE SEX RATIO GAME, ASSUMING FOUR OFFSPRING PER FEMALE

type of female	*H*	*H*	*H*	*F*	*F*	*F*
number of offspring	3♀, 1♂	3♀, 1♂	3♀, 1♂	2♀, 2♂	2♀, 2♂	2♀, 2♂
grandchildren per child	4, 12	4, 12	4, $\frac{20}{3}$	4, $\frac{20}{3}$	4, 4	4, 4
total grandchildren	12+12	12+12	12+$\frac{20}{3}$	8+$\frac{40}{3}$	8+8	8+8
	24	24	18.7	21.3	16	16

pay-off matrix		*H*	*F*
	H	24	18.7
	F	21.3	16

A second development of Wilson's trait group model has been the analysis of such cases in terms of within- and between-group variance of fitness (Wade 1980). Curiously, this approach has been seen as in conflict with the game-theoretic approach to similar problems (see, for example, Colwell 1981). As I see it, the two methods are alternative, and equally correct. Consider, for example, the case of the sex ratio with local competition for mates, originally analysed by Hamilton (1967) using a game-theoretic approach, and re-analysed by Colwell (1981) as an example of 'group selection'. Table 2 assumes that the offspring of two unrelated females mate randomly *inter se* before dispersal (groups of two are treated for simplicity; either method can readily be extended to groups of n females). Two kinds of female are considered: 'Fisher' females, *F*, producing a 1 : 1 sex ratio, and 'Hamilton' females, *H*, producing the game-theoretic unbeatable ratio of 3♀♀ to 1♂.

Both the group selection and the game theoretic approach to this problem start by defining the 'fitness' of a female, not as the number of children she has (which is the same for *H* and *F* females), but as the number of genomes transmitted to grandchildren. The group-selection approach to the problem is to say that, in mixed groups, *F* does better than *H*, but all-*H* groups do better than all-*F* groups. The between-group effect outweighs the within-group effect, and it is for this reason that *H* replaces *F*.

In the game-theoretic approach, one draws up the payoff matrix as shown, and it is at once apparent that *H* is an evolutionarily stable strategy. More generally, to find the stable sex ratio for groups of n females, one writes down an expression for W_S, the fitness of a female producing a sex ratio of S in a group whose other members produce a ratio S^*. One then seeks a ratio S^* such that W_S is a maximum when $S = S^*$; S^* is then the stable ratio.

These methods are not correct and incorrect, respectively, but alternative correct methods. I suspect that the game theoretic method will prove more intuitive and more powerful, but that remains to be seen.

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(b) Interactions with relatives: inclusive or neighbour-modulated fitness?

Additional difficulties arise if interacting individuals are relatives. Hamilton (1964) proposed that, to analyse such cases, we should replace classical fitness by 'inclusive' fitness, defined as the expected number of offspring produced by an individual, discounting any effects (positive or negative) of social interactions with relatives, and adding any additional offspring produced by relatives by virtue of the activities of the individual, each weighted by the appropriate coefficient of relationship. In a wide range of cases, this method correctly predicts the direction of change, and approximately estimates its rate.

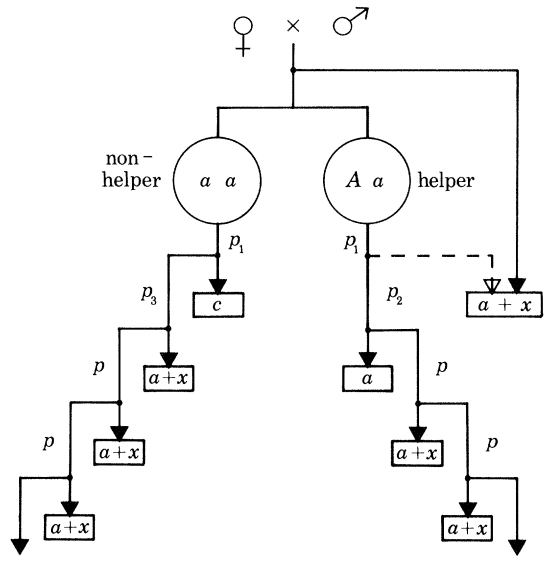

FIGURE 2. A model of helping at the nest. Genotype *aa* breeds in its first year; genotype *Aa* helps its parents to raise their young in its first year, and breeds in subsequent years. For simplicity, it is assumed that parents form new pairs each year: an *Aa* genotype helps its mother; if its mother is dead, it breeds in its first year. Probabilities of survival: p_1, first year; p_2, p_3, second year, for helpers and non-helpers respectively; p, all subsequent years. Number of offspring raised: c, first-year breeder; $a+x$, subsequent years, where x is the number of additional young raised because of helpers. Note that x is not the same for all families, since it depends on the number of helpers.

The above definition of inclusive fitness is a loose one, but still seems unduly cumbersome. It has led to a copious crop of errors (Dawkins 1979; Grafen 1982); it gives only approximate results; it is applicable only when the effects of different interactions are additive (Cavalli-Sforza & Feldman 1978). This has led several authors; (Cavalli-Sforza & Feldman 1978; O'Donald 1982) to propose that it be abandoned in favour of a return to classical fitness, that is, an estimate of the actual number of direct offspring produced by each genotype. After all, this is unambiguous, and must give the correct answer. There is only one snag. Suppose, for example, there is a pair of alleles, *A* and *a*, such that *A* causes its carrier to

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help neighbours. Then, in calculating the fitness of a genotype, say AA, one must allow for the fact that neighbours of AA are more likely to carry the gene A, and hence more likely to help.

Hamilton (1964) used the term 'neighbour-modulated fitness' for this classical fitness, modified to allow for help from neighbours. For practical reasons, he preferred to use inclusive fitness, because, although harder to define, it is easier to calculate and to think about. There are two reasons for this. One, illustrated in Maynard Smith (1982), is that to find the neighbour-modulated fitness one must calculate the probabilities that neighbours have particular genotypes, and not merely the appropriate coefficients of relationship.

Even when this difficulty is removed, it may still be hard to calculate neighbour-modulated fitness. This will be illustrated by an example, in which, by considering only the spread of a rare dominant gene, the former difficulty is removed. The case (figure 2) is the evolution of 'helping at the nest'. The assumptions in the figure have deliberately been made as simple as possible. To decide whether a rare gene A for helping will spread, we ask whether the substitution of an allele a by A will increase or decrease inclusive fitness. Writing W_{aa} and W_{Aa} for the inclusive fitnesses,

$$W_{Aa} = p_1 p_2(a + pa + p^2a + \ldots) + kp_1 y = p_1 p_2 a/(1-p) + kp_1 y,$$

and

$$W_{aa} = p_1[c + p_3(a + pa + p^2a + \ldots)] = p_1 p_3 a/(1-p) + p_1 c,$$

where y is the number of additional offspring raised by parents if helped by one helper, and k is the appropriate coefficient of relationship. If pairs are re-formed every year, $k = \frac{1}{2}$. Note that, in calculating the inclusive fitnesses of an Aa individual, we do *not* include the additional x offspring arising because help may be received from that individual's other children, but only the additional offspring the individual helps its parents to have. Note also that we do not have the calculate the probability that an individual will have a live parent to help, because if no parent exists, the gene A is not expressed, and so does not alter fitness.

Allele A will spread if W_{Aa} is greater than W_{aa}, or

$$c < \tfrac{1}{2}y + a(p_2 - p_3)/(1-p). \tag{7}$$

It is possible to get the same result using neighbour-modulated fitnesses, but it is difficult, and I doubt whether I should have found it if I had not already known the answer. The calculation is too long to give here, but requires, among other things, that one adjusts p_1 to ensure that the population size is stationary, and calculates the probability that an Aa individual of one year old has a half-sib to help. The full calculation is left as an exercise for the reader. The point being made is that it is a great deal easier to find the solution using inclusive fitness.

We thus find ourselves in a somewhat ironic position. On the one hand, neighbour-modulated fitness is easier to define with precision, it gives exact solutions, and does not require additive fitness effects. Yet when one attempts to model a specific case, it is usually much easier to use inclusive fitness. Perhaps the main service that population geneticists can perform is to specify the circumstances in which inclusive fitness methods can safely be applied.

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3. Levels of selection

I now turn to cases in which the entities that possess heredity, multiplication and variation, and that are therefore treated as the units of which the evolving population is composed, are no longer individual organisms. There are two main cases: the entities may be replicating nucleic acid molecules, or they may be species or other reproductively isolated populations.

The need to consider the former case arises because it is now apparent that there are pieces of DNA that can replicate out of phase with the cells in which they find themselves. If Mendel's laws are obeyed, it is largely a matter of convenience whether we set up our models in terms of the frequencies of genes or the genotypes: usually the former method saves complications if mating is random, but not otherwise. But if Mendel's laws break down, this is no longer so. I have no space to discuss such cases here: one particular problem is analysed by Charlesworth at this symposium.

Interest in inter-species selection has been re-awakened by the suggestion (Stanley 1979; Gould & Eldredge 1977) that it is responsible for major trends in evolution. The proposal is that (*a*) most morphological changes occur at the time of speciation (i.e. lineage splitting), (*b*) the direction of these changes are random relative to long-term 'macro-evolutionary' trends ('Wright's rule'), and (*c*) major trends are caused by the fact that species with certain characteristics are either less likely to go extinct, or more likely to speciate, than others. Thus there seems, at first sight, a perfect analogy between species selection and the evolution of a population of asexual individuals, with speciation replacing reproduction, extinction replacing death, and the random origin of new traits at speciation replacing mutation.

In view of the comparative rarity of speciation and extinction, compared with birth and death, I doubt whether such a selective process could have any very substantial effect in producing the complex adaptive syndromes characteristic of major groups. However, apart from this purely quantitative difficulty, there are more fundamental difficulties with the model.

(*a*) If species evolve particularly rapidly at their time of origin, this is probably because they are moving into a new ecological niche and are exposed to new selective forces. If so, the changes could hardly be random. However, let us accept for the sake of argument that the changes are randon, at least relative to the direction of major trends. To fix ideas, consider the extension of the secondary bony palate in the evolution from mammal-like reptiles to mammals. 'Randomness' implies that new species with less-developed palates would arise as often as ones with better-developed palates, which is itself strange. But the real difficulty is that, if speciation changes are random, most new species would differ from their ancestors in other ways, unrelated to any macroevolutionary trend, and not at all in the bony palate. That is, unless the palate was changing because of within-species selection, there is no reason why, in most speciation events, it should change at all. To model species selection, as has usually been done, by assuming that a specified trait changes at every speciation event is analogous to modelling the evolution of a population of cells on the assumption that some trait changes by mutation at every cell division.

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(*b*) If the evolution of the secondary palate is to be explained by species selection, then it must either decrease the likelihood of extinction, or increase that of speciation, of species possessing it. The secondary palate is an adaptation enabling an animal to breathe and chew at the same time. Now species do not chew or breathe: individual animals do these things. Thus even if we were to accept that changes in the palate arose randomly at the time of speciation, it would still be true that the survival of species with better palates would be caused by selection favouring individuals. There are traits (for example, sexual reproduction) that do affect species as entities, and not just individuals, but most major evolutionary trends do not concern them.

(*c*) It is not obvious that an increase in the number of species in one taxon relative to another implies that selection has favoured the former. This is best explained by an example. Vrba (1983) discusses the evolution of two groups of antelopes. One group of food specialists, the Alcephalini, has speciated repeatedly. Today there are seven species. The other group, the impala, consists of a single generalist species, which has never split. Yet there are probably more individual impala than of all other species combined. Thus, when considering the evolution of an asexual population, it is usual to assume a fixed total population size, regardless of the types present. If one type increases in numbers relative to another, we regard this as an indication that it will replace its competitor. In the case of the antelopes, it is less likely that the total number of species in Africa is regulated than that the number of individuals is regulated (although this is not certain). If so, it is not clear that the increase in the number of species of Alcelaphini is an indication that they will replace the impala.

If the relevance of species selection to evolution is to be evaluated, we need mathematical models of the process. Yet there are queries that have to be answered before mathematical modelling can usefully be undertaken. A mathematical model is only as good as its assumptions.

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Discussion

J. F. Y. Brookfield (*Department of Genetics, University of Leicester, Leicester LW1 7RH, U.K.*). In the models where the size of the genome is limited by mutation to genotypes of lower fitness, the size of s, the selection co-efficient against these new genotypes, is independent of genome size. What is the effect on the predictions of these models of allowing s to vary systematically with genome size?

J. Maynard Smith. In the Muller's ratchet model, the permissible mutation rate for a given genome size is lower if the selective disadvantage per mutant, s, is small. Therefore, if we assume that the average value of s falls as the genome size increases, the effect will be to place a sharper limitation on genome size for a given error rate.

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