It is difficult to interpret this, because we have little idea of the value of $W_{max}/\bar{W}$. However, it is clear that sexual populations can, when selection is intense, evolve very much more rapidly than asexual ones. This difference will be greatest when a large number of different favourable genetic substitutions are possible, each by itself relatively unimportant.

D Genetic load, extinction and the Red Queen

In the last section, the basic assumptions were that if conditions change too rapidly the genetic load would increase, and that if the load became too great the population would go extinct. There are many population biologists who have come to regard the concept of a load as misleading, and who in consequence are likely to reject the whole approach. Are they correct?

In Chapter 4, section D, I argue that the taxonomic distribution of parthenogenetic forms leads inescapably to the conclusion that populations committed to parthenogenesis are more likely to go extinct; in saying this, I am only following the view propounded by Stebbins (1950), White (1973 and earlier), Darlington (1939), and others. If the conclusion is accepted, it follows that some populations go extinct because their genotype is not sufficiently well adapted to the contemporary environment, although, given a different genetic system, they could have adapted. It is surely reasonable to say that the difference between the actual genotype and the better-adapted genotype which could have evolved represents a genetic 'load'; it is this load which I have called the lag load. Why then has there been such hostility to the concept of a load?

The idea of a substitutional load traces back to Haldane's (1957) concept of a 'cost of selection'. Here the analogy is with animal breeding. A breeder who wishes to make his breed homogeneous for an initially rare mutant has no choice but to cull many individuals who do not carry the mutant. Haldane's contribution was to show that, if selection was not intense, the total number of individuals lacking a new mutant who must die, or fail to breed, before that mutant is fixed is in the region of ten to thirty times the population size. This, he suggested, placed a limit on the rate of evolutionary change; I have no doubt that he was correct.

As the theory of genetic loads was developed, particularly by Crow & Kimura (1970 and earlier), opposition arose for two reasons, one largely semantic and the other partly emotional. The semantic difficulty has already been touched on; it is surely ridiculous to say that a 'load' has increased when a favourable mutation occurs. I believe that this difficulty disappears if we use the lag load; the load was there already before the mutation occurred, and will be diminished when the mutation is fixed.

The real hostility to the concept arose when it was used to support the view that some allelic variation is selectively neutral. Lewontin & Hubby (1966) argued that the amount of enzyme polymorphism was too great to be maintained by selection favouring heterozygotes unless the selective advantages per locus are very small. Kimura (1968) argued that the apparent rate of molecular evolution was too great to be compatible with Haldane's cost, and hence that most substitutions must be neutral. Many geneticists seem to have felt that, if the load argument could be used to support the neutralist heresy, there must be something wrong with loads.

Two main criticisms of the load argument were made. The first concerns the way in which costs at different loci should be combined. Haldane (1957) had assumed that fitnesses are multiplicative; in this case loads can be added. It was pointed out by a number of people (King, 1967; Milkman, 1967; Sved, Reed & Bodmer, 1967, in relation to segregational loads; Sved, 1968 and Maynard Smith, 1968*b*, in relation to substitutional loads) that a given total intensity of selection (i.e. a given load) can maintain a much greater number of polymorphisms, or cause a greater number of gene substitutions, if the multiplicative assumption is dropped. In particular, this is so if one supposes that selection acts simultaneously on all loci, picking out those individuals who, summed over all loci, have the greatest fitness. This can be called 'threshold' selection, since all individuals with a genetic score above some threshold survive.

The second main criticism (Brues, 1964; Wallace, 1968) is concerned with the way in which selection acts, in particular with the distinction between 'hard' and 'soft' selection. If a particular genotype will always be killed by a particular selective agent (e.g. a particular temperature), this is hard selection. It is reasonable to

speak of a cost since the action of the agent reduces the population. In contrast, suppose that the environment is capable of supporting only some fixed number of individuals. Then supernumerary individuals will die in any case. If the ones which die are genetically different from the ones that survive, this constitutes selection, but it is soft selection. There is no cost, since the population is not decreased by the selection. In this latter case, fitnesses will be frequency-dependent; the argument is therefore related to the argument that frequency-dependent selection can maintain polymorphisms without a genetic load (e.g. Clarke, 1972).

The two criticisms are of course related. Victory in intraspecific competition is likely to be determined by the overall effect of genes at many loci (i.e. 'threshold' selection); in general, intraspecific competition leads to soft selection. Hence, 'soft' selection will tend to be 'threshold' selection and 'hard' selection to act independently on different loci, although these associations are by no means absolute. In most cases, selection will be somewhere between hard and soft and somewhere between multiplicative and threshold.

The interpretation of genetic loads is certainly modified by these criticisms, but I do not think that the whole concept is invalidated. Two things must be said about threshold selection. First, with threshold selection the cost required to bring about a number of gene substitutions is indeed reduced, but it does not vanish. Secondly, there is no evidence that in natural populations selection does in fact act in this way, although one can think of situations in which it may well do so. In particular, if one supposes that a species is simultaneously adapting to changes in its pathogens, its predators, its competitors and to climatic conditions, it is hard to see how selection could act in a threshold manner.

The distinction between hard and soft selection is highly relevant to the problem of extinction, and its relation to the genetic load. Thus, suppose that the main evolutionary changes occurring in a species affected characteristics (e.g. size, weapons, behavioural strategies) enabling individuals to occupy dominant positions in a hierarchy and so to increase their chances of survival and breeding. Since there would be genetic differences in fitness, there would be a lag load associated with the changes. Yet the changes which occurred would

not make the species less likely to go extinct; indeed precisely the opposite might be the case, because weapons evolved for intraspecific contests may be a handicap in other contexts (if they were not, hinds would have antlers). A parthenogenetic population which made these changes more slowly or not at all would be no more likely to go extinct.

Clearly, the whole concept of lag load and its relation to environmental change and extinction is irrelevant when the selective agent is intraspecific competition of this kind. But this does not invalidate the concept when applied to evolution in response to changes in the physical environment, or in diseases, predators, or competing species.

I cannot end a discussion of the relationship between evolutionary rate and extinction without considering the ideas of Van Valen (1973). He presents evidence for a 'law of constant extinction', according to which, for any group of related organisms with a common ecology (e.g. carnivorous mammals or bony fishes), there is a constant probability of extinction of any taxon (e.g. a genus or family; the fossil record is too incomplete to measure species extinction) per unit time. In an attempt to explain this empirical law, he appeals to the hypothesis of the 'Red Queen', according to which each evolutionary advance by any one species is experienced as a deterioration of the environment by one or more other species. Like the Red Queen, each species must therefore evolve as fast as it can merely to survive.

In general terms, the hypothesis is an attractive one. If it is correct, we would expect to find parthenogenetic and selfing species to be rarest in tropical environments and commonest in temperate and sub-arctic ones, since the challenge from other species is likely to be most intense in the former. Evidence that this is so is discussed in Chapter 6, section D.

There is, however, a difficulty in deriving a law of constant extinction from the Red Queen hypothesis. Van Valen suggests that the derivation depends on a 'zero sum' condition, according to which the increase in fitness of one species is exactly equal to the sum of decrements of fitness of all other species. In fact it can be shown (Maynard Smith, 1976*a*) that a law of constant extinction per unit