GENERAL

Population Size, Natural Selection and the Genetic Load

How great a genetic load can a population tolerate ? This subject has recently been discussed by several authors[1–5]. We believe that a most important point can be stated briefly: because most organisms produce far more offspring than are necessary to maintain a constant population density, and because population densities remain, very roughly, constant, many individuals die before they are mature; it does not matter whether they die of starvation, accidents or from genetic ailments; the population can still maintain itself. It is not so much that there is a genetic load which might threaten the species, but that there is an ecological load, resulting from density regulation, which because it must, as Darwin noted, produce natural selection, gives rise to the apparent genetic load. A population will be able to tolerate what seems to us a considerable genetic load, without being, on that account, in any danger of extinction. The genetic load is, for the most part, merely an expression of the fact that not all genotypes are equally viable when the population becomes crowded. Many individuals have to die in the process of density regulation, and if those which die differ genetically from those which survive, we will observe a "genetic load". This is the crux of the Malthus–Darwin concept of selection. This does not of course apply to genetic conditions which are markedly disabling at all population densities; there must be a decided limit (although a fairly high one, for the dead individuals simply leave more food or space for others, which otherwise would die) to the number of these which a population can contain. We suggest the term "loaded" for this last kind of selection.

It is quite possible that many of the biochemical polymorphisms of *Drosophila pseudoobscura*[1] are maintained by density-dependent natural selection—some genotypes, probably homozygotes, are less viable than others when the population is crowded, and it has been shown[2–4] that this could maintain polymorphisms at perhaps a thousand loci.

We must distinguish between density-dependent and frequency-dependent selection. If the organism contaminates its environment and if the contaminant is removed at a constant rate, then the denser the population the greater the amount of contaminant. If some genotypes suffer more than others from the contaminant, then this component of natural selection is purely density-dependent. If the fitness of a genotype depends entirely on its frequency relative to other genotypes, irrespective of population density (as with assortative mating or Batesian mimicry within moderate limits of population density), then selection is purely frequency-dependent. Both kinds of selection can maintain polymorphism[6,7].

It is probably commoner for selection to be both frequency and density-dependent. If the densities of different genotypes are limited by different factors in the environment, then the fitness (rate of survival and reproduction) of a genotype will depend on its density. The density of a genotype is a direct function of its frequency and of the density of the population, so that selection is dependent on both these two variables. Kojima and Yarbrough[8] misleadingly call this type of selection "frequency-dependent". Perhaps it should be called population-dependent.

Levene's[9] much quoted model of differential selection in different "niches" can be conceived as an elaborate kind of population-dependence. Models have been suggested in which a certain percentage of the population survives[2–4]; if the survivors tend to be heterozygous at more loci than are those which die, these loci remain polymorphic. Because the percentage surviving depends on population density, these models correspond with our density-dependent selection. In general the fitness of any genotype can be described by the equation

$$W = k_i + k_c + f_i(q) + f_c(q) + f_i(d) + f_c(d) + f_i(q,d)$$

where the ks are constants, $f(q)$ is a function of gene (or genotype) frequency, $f(d)$ is a function of population density, and i and c indicate, respectively, that the function is unique to that genotype or is shared by all genotypes in the population. (To make the equation completely general, we should include all the products of the $f(\ )$ terms.) Then k_i is the loaded selection, $f_i(q)$, $f_i(d)$ and $f_i(q,d)$ are the frequency-dependent, density-dependent and population-dependent parts of selection, $f_c(d)$ represents density-dependent population control and $f_c(q)$ represents gene-frequency-dependent population control (which will probably often be zero).

The great increase in variability observed in the butterfly *Melitaea aurinia* while the population was expanding[10] was probably, as Ford suggests, an indication that density or population-dependent selection had been much reduced, allowing survival of extreme phenotypes and an increase in numbers. The viability of *bar* genotypes in Drosophila varies with population density[11]. Harding *et al.*[12] have shown selection in *Phaseolus lunatus* which is certainly frequency-dependent and may also be density-dependent.

Selection which is frequency-dependent produces little or no genetic load at equilibrium, but does produce a genetic load at other gene frequencies. Density-dependent selection never produces a "genetic load" in the ecological sense (although it will do so in terms of human values), for selection acts only when the population has to be "thinned" in any case because it is becoming too dense. Although other forms of selection no doubt maintain some polymorphisms, we suggest that the majority must be maintained by population-dependent selection, with a small residue of "loaded" selection, and that this is how *Drosophila pseudoobscura*, and no doubt other species, can be polymorphic. The populations would be in no danger if they became inbred.

Loaded selection, frequency density and population-dependent selection do not, *ipso facto*, maintain polymorphism; but all of them can do so, in complete accord with the facts of genetics and ecology. In saying this, we are re-stating the arguments of Cain and Sheppard[13,14] and of Darwin. In view of the mounting importance of eugenic as well as demographic problems for man as a species, we think it of great importance to understand the relations between these various causes of death.

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