SELECTION THROUGH COMPETITION*

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1. CO-OPERATION AND ANTAGONISM

IN considering the effects of selection on gene frequencies in populations it is customary to assume, implicitly if not explicitly, that the individuals and genotypes are not effective parts of one another's environments, or in other words that they have no differential effects on one another's fitness. This assumption is no doubt valid under certain circumstances and in certain connections—for example where the selective force lies in differing abilities of individuals to tolerate or withstand physical features of the environment. But if a species is successful the time must soon come when the availability of some necessary resource becomes a factor limiting further increase of the population and competition for this resource ensues between individuals. Genotypes differing in the competitive abilities with which they endow their possessors will then be favoured differentially by selection, and changes of gene frequency may ensue. It is indeed difficult to avoid the conclusion that competition in this broad sense must enter into most of the forms of selection that impinge on populations in the wild, and that it must therefore play its part in determining most genetical adjustments and equilibria.

Competition is of course not the only interaction between individuals, whether of the same or different species, that is of significance in relation to selection. In principle, these interactions may be of three kinds. First, individuals may co-operate to their mutual advantage. Co-operation may be between likes and the advantage so gained may give the genotype or genotypes favouring such co-operation an effectively increased fitness. Or co-operation may be between unlikes, in the sense that *A* and *B* together are on the average each fitter than *A* would be with a second *A* or *B* with a second *B*. Such a relation is symbiotic if between different species and selection must act both to adjust the partners towards increasingly effective complementarity in their relationship and to secure increasingly the maintenance of their association. Where the co-operation is between different types within a species a form of disruptive selection ensues leading to polymorphism, with its characteristic genetic architecture (Mather, 1955; Clarke and Sheppard, 1960; Thoday and Boam, 1959). Co-operative interaction of individuals is not, however, the subject of our present discussion.

Secondly, individuals may be antagonistic to one another and two classes of antagonism may be recognised in principle. It is to be expected that, by comparison with the situation where individuals are not interacting, a state of antagonism will not be to the advantage of either of two interacting individuals; but given that two individuals of different genotypes are in appropriate relationship, two classes of antagonism may in principle be recognised. In the one case, one of the two individuals may derive advantage while the other suffers disadvantage, in the sense that when *A* and *B* come

* Dedicated to Professor Th. Dobzhansky to mark his seventieth birthday.

together, A is on the average at an advantage by comparison with the situation where A has come together with a second A , and B is on the average at a disadvantage by comparison with the situation where B has come together with a second B . This is competition in the sense that the term is generally used.

The second kind of antagonism (that is, the the third type of interaction) is where both of the two unlike individuals suffer disadvantage in the sense that when A and B are together, A is on the average at a disadvantage by comparison with A when together with a second A , and B is on the average at a disadvantage by comparison with B when together with a second B . This relationship may be referred to as one of contest and it must tend towards the elimination from the population of one or other form depending on the relative frequencies of the two and the degree of disadvantage suffered. Where A and B are two true breeding genotypes and the disadvantage is equal for them, the less common of the two will tend to be eliminated, just as the less common of two alleles will tend to be eliminated where they produce a heterozygote which is at a disadvantage by comparison with the two homozygotes. But where the two true breeding forms A and B , while each having unit fitness when not in contest, have fitness reduced to $1 - k_A$ and $1 - k_B$ respectively when in contest, the population comprising a proportion u of A and $v = 1 - u$ of B , A will suffer a total disadvantage of vk_A , and B a total disadvantage of uk_B . Then A will tend to be eliminated when $vk_A > uk_B$ and B will tend to be eliminated when $vk_A < uk_B$. When $vk_A = uk_B$ the population is theoretically at equilibrium, but it is in unstable equilibrium and random variation in u and v will suffice to start the move towards elimination of one or other form.

Thus, contest must make for stability of populations by the elimination of new and rare variants, whether these depend on true breeding integrated genotypes or on individual alleles. The consequences of competition, to which we now turn, are however of more interest.

2. CONSEQUENCES OF COMPETITION

(a) *True breeding lines*

Consider a population comprising individuals of the two true breeding types A and B in the respective proportions u and $v = 1 - u$. Let the fitness of both A and B be unitary when they are not competing, but be $1 + k$ for A and $1 - k$ for B when an individual of A and one of B come into competitive relationship. Taking the simplest interaction of relationships in pairs, since u of the individuals are A and v are B , an individual of A will be paired with another A in u of cases and with B in v of cases. Then the average fitness of A individual will be $u.1 + v(1 + k) = 1 + vk$ and of B will be $v.1 + u(1 - k) = 1 - uk$. Since a proportion u of all individuals are initially A and a proportion v are initially B , the contribution of A to the next generation will be $u(1 + vk)$ and the contribution of B will be $v(1 - uk)$. The overall fitness of the population is thus $u(1 + vk) + v(1 - uk) = 1$ and the population will not be changing in numbers as a result of the competition. The proportion of A will, however, be changing since $u_1 = u_0(1 + v_0k)$ and $u_2 = u_1(1 + v_1k)$, etc., where u_0 refers to the initial population, u_1 to the first generation affected by the competitive and so on. Thus competition can and will change the constitution of the population without of itself affecting the overall number of

individuals the population comprises. The increment in A is $u_1 - u_0 = u_0(1 + v_0k - 1) = u_0v_0k$.

Competition is of course much more complex than a series of pairwise relations. A plant, for example, may feel the effects of competition from a number of other individuals growing at various distances from it and interacting with one another in their effects on it. We can, however, define a series of zones, which might be expected to approximate to concentric rings

FIG. 1.—The individual in the centre, P , is in competitive relationship with the four other individuals one in each of the zones, 1 to 4, centred on P and defined as each carrying, on the average, one individual. The competitive effect on P of the individual in zone 1 (and equally of P on this individual) depends on k_1 , that of the individual in zone 2 on k_2 and so on, the combined effect on P being the sum of the four individual effects. The individuals in zones 1 and 3 (bottom part of diagram) interact with one another in their competitive effects on P , but k_3 is defined to allow for their effect. The zones are shown, for convenience, as circular and of equal areas, but this need not of course always, or even commonly, be the case. (For further description see in the text.)

spaced round the primary individual, such that each zone on the average contains one individual whose competition with the primary individual results in an average advantage/disadvantage differential of k_1 for the first, or inner, zone, and k_2 , k_3 , etc., for the second, third, etc., zones numbering outwards from the centre. Then in general we expect $k_1 > k_2 > k_3$, etc., for the effects of competition will presumably be greater over shorter distances. How k_1 , etc., will be related to these distances is a matter of obvious practical as well as theoretical interest but one which we need not now pursue (fig. 1).

We assume that the individual in each zone has a chance u of being A and v of being B . Then when the primary individual is A its fitness will be raised by vk_1 as a result of its relation to the individual in the first zone, by vk_2 though its relation to the individual in the second zone, and so on. The fitness of a primary individual of type A will thus be $1 + vk_1 + vk_2 \dots = 1 + vS(k_1)$, which is of the form $1 + vk$ already derived for the pairwise relationship but now with $k = S(k_1)$. Similarly, if the primary individual is B its fitness will be $1 - uS(k_1) = 1 - uk$ where again $k = S(k_1)$. The results obtained from the simple pairwise comparison can thus be generalised to more complex situations. The assumption of additivity of k_1, k_2 , etc., clearly merits further consideration, but we may observe that k_2 as defined will necessarily reflect the effects of an individual in zone 1 in interposing itself between the primary individual and that in the second zone, where competitive consequences are measured by k_2 . Thus additivity of k_1 , etc., does not imply an absence of interaction involving three or more individuals, at least when these are in different zones. It should be observed, too, that the argument, though developed in terms of plants and the fixed distances between them, can be recast to apply to mobile species.

A further point may be made. If the density of the population changes, the zones can be redefined such that each still contains on the average a single individual. Increase in density will then make the zones and distances smaller and the value of k_1 , etc., greater, just as decrease in density will make the zones and distances greater and the values of k_1 , etc., less. An alternative approach might, however, be to maintain the same zones but introduce d_1, d_2 , etc., to represent the average frequency of an individual falling in zones 1, 2, etc. Then the advantage differentials would become $1 + vd_1k_1 + vd_2k_2$, etc., and $1 - ud_1k_1 - ud_2k_2$ which reduce to $1 + vS(d_1k_1)$ and $1 - uS(d_1k_1)$ which may in turn be written $1 + vS(k_1) = 1 + vdk$ and $1 - uS(k_1) = 1 - udk$ if $d = d_1 = d_2$, etc. Here d becomes a general measure of density whose effects are thereby taken into account; but only experiment could show whether this treatment is justifiable.

It is of interest to compare the effects on the frequencies of A and B of competition, as thus expressed, with those which arise when the difference in fitness is independent of the frequencies of the two types in the way commonly assumed in considering change under selection. Let the fitness of A be 1 and that of B be $1 - s$. (These could equally well be represented by the fitness of A being $1 + s$ and that of B being 1, if we wished to show the overall fitness of the population as increasing instead of decreasing: similar results would be obtained.) With u and v as the proportions of A and B in the initial population we find $\frac{u}{1 - vs}$ and $\frac{v(1 - s)}{1 - vs}$ as the proportions in the next generation.

The increment in A is thus $\frac{u}{1 - vs} - u = \frac{uvs}{1 - vs} = uvs(1 + vs + v^2s^2 \dots)$.

Returning to the change under competitive selection, the proportion of A in the population alters from u in the initial population to $u(1 + vk)$ in the next generation, giving an increment of uvk . Thus the graph of progress under competitive selection will be sigmoid and symmetrical round the mid-point, where $u = v = \frac{1}{2}$ and the rate of change is maximal. The graph of progress under independent selection will also be broadly sigmoid, because of the uv which appears in the expression of the increment, but the factor $(1 + vs + v^2s^2 \dots)$ ensures that it will not be symmetrical and the point of

maximal change will not be at the mid point. Furthermore, with equal values of uv , whereas change under competition is the same, no matter whether u or v is the larger, change under independent selection will be faster when v is the larger, that is when the advantageous type, A , is rarer than the disadvantageous type B . Or in other words, while under competitive selection the rate of establishment of a new, favourable variant and the

FIG. 2.—The increments (Δ) added by selection to the frequency u of an advantageous type, when selection depends on competition ($\Delta = uvk$), or is independent of relations between individuals ($\Delta = uv/1 - vs$). The curves relating Δ to u are shown for four values of s ($= 1, \frac{3}{4}, \frac{1}{2}, \frac{1}{4}$) and also for the value $k = 1$. For values of $k < 1$, the curve for $\Delta = uvk$ is correspondingly reduced in height.

rate of the final elimination of the type being replaced are equal, with independent selection a new, favourable variant is established more readily than its alternative is finally eliminated (fig. 2).

Finally, before leaving populations consisting of true-breeding lines we may note that the two-line case which we have been considering is easy to generalise to the multi-line case, as Mr Brian Bailey of this University has pointed out to me. Let u_i be the proportion of the i th line out of p lines and let the fitness differential of the i th and j th lines in competition be k_{ij} , with of course $k_{ij} = -k_{ji}$ for all i, j and $k_{ii} = 0$ for all i . Then the average fitness of individuals of the i th line is

$$\sum_{j=1}^p u_j (1 + k_{ij}) = 1 + \sum_{j=1}^p u_j k_{ij}$$

and the frequency of the i th line in the next generation will be

$$u_i \left[1 + \sum_{j=1}^p u_j k_{ij} \right].$$

The increment added by one generations selection is thus $u_i S u_j k_{ij}$.

(b) *Alleles*

With two alleles G and g there are prospectively three genotypes in the population and hence three competitive relations to be considered. When GG and Gg are in competition let their fitness be $1 + k_a$ and $1 - k_a$ respectively; with Gg and gg let them be $1 + k_b$ and $1 - k_b$; and finally with GG and gg let them be $1 + k_c$ and $1 - k_c$. Let the population mate at random and the frequencies in it of G and g be u and $v = 1 - u$ respectively. Then initially GG , Gg and gg will occur with frequencies u^2 , $2uv$ and v^2 . Then in any but a small population a GG individual will meet, in competitive relationship, GG in u^2 of cases, Gg in $2uv$ of cases and gg in v^2 of cases. The average fitness of GG will then be $u^2 \cdot 1 + 2uv(1 + k_a) + v^2(1 + k_c) = 1 + 2uvk_a + v^2k_c$ as in table 1.

TABLE 1

Genotypes	GG	Gg	gg
Frequency	u^2	$2uv$	v^2
Fitness	$1 + 2uvk_a + v^2k_c$	$1 - u^2k_a + v^2k_b$	$1 - 2uvk_b - u^2k_c$
Contribution	$u^2 + 2u^2vk_a + u^2v^2k_c$	$2uv - 2u^3vk_a + 2uv^3k_b$	$v^2 - 2uv^3k_b - u^2v^2k_c$
Gene frequencies	G	g	
Parents	u	v	
Offspring	$u(1 + u^2vk_a + v^3k_b + uv^2k_c)$	$v(1 - u^3k_a - uv^2k_b - u^2vk_c)$	

The average fitness of Gg and gg are also shown in the table. The contribution of GG to the gametes from whose random combination the next generation will be formed is given by the product of the genotypic frequency and fitness, as $u^2(1 + 2uvk_a + v^2k_c) = u^2 + 2u^3vk_a + u^2v^2k_c$, which together with the contributions made by Gg and gg are set out in the fourth line of table 1. It will be seen that the contributions of the three genotypes sum to unity, showing that there is no inbuilt tendency for the population either to increase or to decrease in size. The frequencies of G and g gametes are then easily found to be $u(1 + u^2vk_a + v^3k_b + uv^2k_c)$ and $v(1 - u^3k_a - uv^2k_b - u^2vk_c)$ and the increment by which the frequency of gene G has changed as a result of competition in this generation is then

$$u(u^2vk_a + v^3k_b + uv^2k_c) = uv(u^2k_a + v^2k_b + uvk_c).$$

The increment in frequency of g is, of course, the same but of opposite sign.

It is clear that if all the k 's are of the same sign the favoured gene will spread to fixation in the population even though the competition is not of itself tending to change the size of the population. If the k 's are not all of the same sign, one or other allele will tend towards fixation, though more slowly, unless values of u and v arise at which $uv(u^2k_a + v^2k_b + uvk_c) = 0$ when the population will come to equilibrium, either stable or unstable. Now, if the competitive differential between the two homozygotes is equal to the sum of those between the heterozygote and each of the homozygotes, *i.e.*,

$k_c = k_a + k_b$ (which might be expected to be the case at least when the k 's are reasonably small), the increment in the frequency of G becomes

$$uv[u^2k_a + v^2k_b + uv(k_a + k_b)] = uv(uk_a + vk_b).$$

Equilibrium is then reached when $uk_a = -vk_b$, that is when both homozygotes are at a competitive disadvantage (k_a being negative) or at a competitive advantage (k_a being positive) with respect to the heterozygote. The latter will be an unstable equilibrium but the former will be stable and represents the familiar case of a polymorphism maintained by heterozygote advantage. Indeed, the very relationship $uk_a = -vk_b$ is precisely the same as that which applies in the familiar case of selection independent of gene frequency.

Where one allele is fully recessive to the other, either k_a or k_b becomes 0, depending on whether G or g is the recessive, and the other becomes equal to k_c . Thus if g is recessive, $k_a = 0$ and $k_b = k_c$. The increment in a then becomes $uv.vk_b$. If the mutation rate from G to g is then μ , equilibrium will be reached when $uv^2k_b = \mu u$, which may be variously rewritten as $\mu = v^2k_b$ or $v^2 = \frac{\mu}{k_b}$. This again is the exact counterpart of the equilibrium attained by

the balance of mutation and selection in the familiar case where the selection is non-competitive. Equally if G is dominant and disadvantageous with mutation from g to G we find $uv = \frac{\mu}{k_a}$, which again repeats the relationship

familiar from consideration of non-competitive selection. Selection by competition thus repeats all the relationships familiar from earlier studies, where selective differences were assumed to be independent of interaction between individuals, and it does so even though the competition will not of itself tend to change the overall number of individuals in the population. In other words, populations intrinsically stable in size can show all the familiar phenomena of gene spread and equilibria, provided their density is sufficiently great to produce competitive interactions, which indeed must generally be the case.

Genotypes may of course differ in more complex ways in the effects they produce. Some of these effects may confer an unconditional advantage on one genotype over another, while other simultaneous advantages may be conditional on the occurrence of competitive relations. Where both the competitive and the independent advantages pull in the same direction the favoured allele will of course just proceed towards fixation with a speed dependent on the joint effect. Should, however, the competitive and the independent advantages be opposed it may be possible for an equilibrium to be struck. Equilibrium will in fact be attained when the opposing s and k just balance out. Now the independent or non-competitive selection may well be tending either to raise or to lower the size of the population. In the former case competitive selection will be tending to increase in strength, and in the latter case to decrease in strength. If, therefore, a gene favourable in respect of its non-competitive effects is tending to spread and to raise the size of the population, any disadvantageous competitive effects could be intensified and in due course a balance struck. In the same way a gene spreading because of advantageous competitive effects but tending to reduce population size because of disadvantageous non-competitive consequences,

could be brought to equilibrium as competition became less intense. It is of interest that, despite the dependence of the intensity of competitive selection on gene frequency, an equilibrium so struck may actually be independent of gene frequency. Consider the simple case of a recessive gene g with gene frequency v , reducing when homozygous non-competitive fitness by s but with an advantageous competitive differential of k , by comparison with GG and Gg . The fitness of gg will thus be $1 - s + (1 - v^2)k$ and that of GG and Gg jointly will be $1 - v^2k$. The contribution of gg to the next generation will be $v^2[1 - s + (1 - v^2)k]$ and that of the other two genotypes jointly will be $(1 - v^2)(1 - v^2k)$, the overall fitness of the population thus being $1 - v^2s$. Then equilibrium will be struck at $v^2 = v^2[1 - s + (1 - v^2)k]/(1 - v^2s)$ or $s(1 - v^2) = (1 - v^2)k$ which reduces to $s = k$ and is independent of v .

3. COMPETITIVE FITNESS AND GENETIC LOAD

As we have seen, competition can result in all the familiar equilibrium changes in gene frequency, for under the conditions of competitive selection individuals and alleles can display the necessary advantages over one another. Yet such changes and equilibria do not imply that the populations carry genetic loads in the commonly accepted sense of that term, for the fitness or lack of fitness of an individual is not a simple and unconditional property of its genotype. On the contrary, loss of fitness, and equally gain in fitness, will occur only when effective competition is taking place. If the genotype which is competitively above the population average in fitness were to dwindle the genotype which is below average would rise in fitness and might even become fully fit as its more effective competitors disappeared.

Under competition the fitness of an individual, and by derivation of its genotype, depends not just on itself but on the constitution of the population as a whole. When considering the multi-line case in Section 2(a) we saw that the competitive fitness of a line i could be expressed as $1 + \sum_{j=1}^p u_j k_{ij}$, where k_{ij} takes sign according to whether line j is competitively more or less efficient than i . This same expression can be used more widely for with any set of p genotypes, whether true-breeding or not, it will give the fitness of the i th genotype. Thus the fitness of any one genotype will be dependent on its interaction with the other genotypes, and on the frequencies with which these other genotypes occur. Any change in the constitution of the population will thus be reflected in the competitive fitness associated with any of the constituent genotypes.

Fitness will depend on the size, or more precisely on the density, of the population too. In general, competition will become more intense, and the resulting differences in fitness greater, as the density of the population rises. Any change, whether arising from an earlier action of selection or from the direct impact of external agencies, which increases the density of the population will increase the impact of competitive interaction and so sharpen the action of competitive selection. Equally a reduction in density will weaken the action of competitive selection and so reduce the fitness of the better than average genotypes and raise those of the less well-endowed types. This regulatory action may, of course, go further and tend towards a stabilisation of the density, though this would depend on effects not represented directly in the formulae we have used to express the consequences of competition.

In discussing the maintenance of a high level of polymorphic variation in populations without the entailment of an unsupportably high genetic load, Sved, Reed and Bodmer (1967) have recognised the importance of competitive selection and indeed point out that it would permit the maintenance of any given number of polymorphisms with a lower death rate than would independent individual selection. Their chief concern was, however, to argue for the concept of an upper limit to the fitness that an individual could display and they make the point that such an upper limit is not incompatible with competitive selection. While this is true, it is not obvious that the occurrence of an upper limit to fitness is of major significance to the understanding of the maintenance of variation where selection is by competition. Even if competitive advantage were cumulative over all the relevant loci that varied, very few individuals would have all or most of the advantageous alleles, and these few would hardly have any material effect on the competitive situation prevailing in the population which would consist very largely of types with intermediate competitive capacities. With competitive selection, low fitness depends necessarily on the occurrence at appropriately high frequencies of more competitive types.

It is further pointed out by Sved *et al.* that the results of studies of the effect of inbreeding in a variety of species (which presumably are normally outbreeders) do not give estimates of fitness as low as would be expected from the customary mathematical treatment. They go on to suggest that this may be due to the experiments having been carried out under non-competitive conditions. Where competition was introduced as by Latter and Robertson (1962), estimates of fitness were obtained more in keeping with theoretical expectation. This is of course no more than expected; for if inbreeding is imposed on a population, the more homozygous types might well in general be at a disadvantage in competition with the more heterozygous types which chiefly constitute the normal outbred population. They would thus appear less fit, yet they would be at no great disadvantage in competition with one another and so would show no special loss of fitness, at least in respect of competitive effects, under these circumstances. The moment, however, that the inbred group was again placed in competition with an outbred group, the loss of fitness would appear once more.

Fitness in a pure stand of inbreds will fall only in so far as it depends on non-competitive properties. These may of course be associated with competitive properties: a general loss of vigour consequent on inbreeding could result in a reduction of ability to stand up to hardships in general whether these arose from competition with better endowed individuals or from the vagaries of the physical environment. But in such a case the loss of fitness as measured by competition with an outbred population, or by competition with a vigorous marked type or with another related species, would necessarily overestimate the inescapable reduction which springs from effects independent of the interaction with other individuals, and would therefore result in underestimation of the inbred's capacity for survival *per se*.

Fitness in any population whose rate of reproduction is high enough for it potentially to more than hold its own is a relative thing. The higher fitnesses reveal themselves only by contrast with lower fitnesses and it would be hazardous and generally unwarrantable to assume that even the highest fitness observable in the wild or obtainable in experiment was incapable of

being exceeded. If fitness is relative, so must be any notion of genetic load and doubly so in so far as fitness itself turns on the interaction of individuals as in competition. Basically the same point would seem to underly Turner and Williamson's (1968) discussion of genetic load in relation to density dependent selection.

This relative quality of fitness is of significance in another connection too. The attempt has been made by Haldane (1957) to assess what he terms the cost of natural selection. He points out that breeders find it impossible to select heavily for a number of characters simultaneously in their plants and animals, because if they were to do so they would reduce the number of prospective parents below the limits at which their populations could be maintained. The same must apply in natural population where the toll of natural selection is high, as it is in Kettlewell's *Biston betularia* for example. Haldane then seeks to quantify these considerations by calculating the number of deaths that must occur over the generations as one allele replaces another at a single locus, and this he finds to be high, though of course varying with the situation. He takes 30 times the number of individuals in a generation as a reasonably general estimate of this cumulative total of deaths and goes on to point out that if the population is to survive with a number of loci undergoing substitution at the same time, the deaths must be spread over hundreds or even thousands of generations with the result that evolution in general is necessarily slow. While not seeking to dispute the conclusion that evolutionary change is generally slow, which is indeed establishable by observation, Haldane's calculations should not be accepted uncritically, as a theoretical basis for expecting this always to be the case. The sort of situation he discusses, which presupposes that a sharp change in the environment results in a gene, initially rare because it had been disadvantageous and held in the population by mutation, becoming advantageous and hence spreading at the expense of its common allele, cannot be accepted as the general or even the most common position in which a species finds itself. And selection experiments drawing on polygenic variation show that advance under selection can indeed be quite rapid without unduly hazarding the continuation of the population.

Furthermore, the high number of deaths at which Haldane arrives springs, even under the conditions he is discussing, from a combination of two assumptions that he makes, one explicitly but the other less obviously. These are respectively that the selective differential is relatively small and that the size of the population is constant. The outcome is the curious finding that the less the difference in fitness associated with two alleles, the more deaths must occur before the one replaces the other, or as he would say, the higher the cost of natural selection. This conclusion is an inevitable consequence of the assumptions, for if the disadvantages of a dominant allele were absolute, so that all its carriers died or failed to leave offspring, the number of "deaths" could not exceed the number of individuals carrying that allele in one generation and this itself obviously cannot be greater than the number in the population in that generation. This would be true no matter what change there ensued in the size of the population as a consequence. If, however, we consider genes which result in only a partial loss of fitness the situation is quite different. Given that the size of population falls as a result of the selection, the surviving disfavoured individuals leaving no more than a single offspring equivalent each, the number of "deaths" consequent on the

substitution of one allele for the other would again in the case of a dominant gene no more than equal the number of individuals carrying that gene in the initial population, though the "deaths" would now be spread over a number of generations. But if despite the selection the population is constant in size, as Haldane assumed it to be, each surviving individual carrying the disfavoured allele must leave more than one offspring equivalent in the next generation, even though the frequency of the allele in question is falling. A single survival in one generation will then mean more than a single death before the gene it was carrying is eliminated in later generations, and the cost of natural selection will be correspondingly increased. Furthermore, under these circumstances, the less the selective differential the greater will be the number of individuals leaving more than one offspring equivalent, the greater the ultimate number of deaths and the greater the apparent cost of natural selection. At the same time, if the size of the population is being maintained, it can hardly be held that its continued existence is being put into serious hazard, and we have the contradiction that as the cost of natural selection is going up the consequences to the population are less hazardous—a contradiction that arises from the assumptions.

Haldane's calculations have a certain pioneering interest, but they require re-examination in terms of a sounder theoretical structure and more realistic assumptions. The effect of fitness and selection on population size is a factor that must be taken into account. Competitive selection can hardly fail then to be taken into account for as we have seen it can result in all the familiar changes and equilibria without innately requiring a change in population size. It of course requires a potential reproductive rate greater than unity if it is to produce changes; but equally it has a self-regulatory quality in that any tendency for the density of the population to fall will be accompanied by a reduction in the rigour of competition and hence of selection so that the result would be to slow the change in gene frequency rather than to put the future of the population in hazard. The cost of natural selection would be much less than Haldane's calculation would suggest (even if we overlook the contradictions that we have seen to be intrinsic in his assumptions), and indeed the concept of the cost of natural selection, at any rate in Haldane's sense, becomes merely misleading.

4. SUMMARY

1. Individuals of different genotypes may interact in respect of their fitness. They may cooperate, so increasing one another's fitness, or they may be in contest and so reduce one another's fitness, or they may compete with one gaining and the other losing from the competition.

2. Such competition can result in all the familiar changes in the frequencies of true-breeding lines and of alleles in populations, and in the familiar equilibria of alleles also. At the same time these changes and equilibria can be achieved without any intrinsic tendency towards alteration in the overall fitness of the population.

3. Insofar as it depends on competitive ability, the fitness of an individual or of a genotype is only relative.

4. A genotype may give full fitness when in a population of one genetic constitution while in a population of different constitution, even in the same environment, its fitness may be reduced.

5. Where selection is by competition, therefore, the concepts of genetic load and cost of natural selection require re-examination.

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