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GROUP SELECTION IN STRUCTURED POPULATIONS

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Many species of animals have properties which enable their populations to persist for long periods of time. For example, territoriality may space out the members of a breeding population so that they are less likely to exhaust their food supply. One school of thought interprets these properties as “biotic” adaptations which have arisen because they tend to perpetuate the group and whose evolution is controlled by group selection (see, e.g., Dunbar 1960; Wynne-Edwards 1962, 1963; Nikolskii 1969). Another group of theorists maintains that these population phenomena arise as a consequence of individual selection and represent the summation of “organic” adaptations (see Cole 1954; Maynard Smith 1964; Williams 1966; Ghiselin 1974). Differing views are presented in Williams’s collection of papers (Williams 1968). It will be admitted that the issue is an important one and that the division between the protagonists is deep and serious.

The difficulties encountered by the theory of group selection are of two sorts. First, there are general criticisms which center on the facts that groups usually have much longer “generation times” than individuals; that there are by definition fewer groups than individuals, allowing greater play to stochastic forces; and that the variance of characters within groups is usually much greater than the variance of their mean values between groups. These criticisms are summarized by Crow and Kimura (1970) and by Emlen (1973). The second difficulty is that no very plausible mechanism for effective group selection has been proposed, at least until recently.

Group selection may be defined as the differential genetic survival of groups of unrelated individuals. Just as individual selection is the inevitable consequence of inherited differences in fitness between individuals, so group selection follows necessarily from the existence of heritable differences in fitness between groups. Any model of group selection, therefore, will be expected to require at least two necessary conditions. First, there must be more than one group within some larger entity, or there would be no basis for selection. Second, there must be genetic variance in fitness between groups; if the character in question were determined by alternative alleles at a single locus, this is equivalent to requiring that the alleles should not have the same frequency in all groups. Whenever these conditions are met, group selection of some sort will follow. However, I shall at first follow the usual convention and

restrict the usage of the term to situations in which an opposed individual selection pressure is ineffective, if only on the rather unsatisfactory grounds that adaptation should be interpreted at the lowest level of biological organization compatible with the evidence (see Williams 1966, chap. 4). A group-related adaptation will be recognized only if, as well as functioning to promote the fitness of the group, it is actually antagonistic to individual fitness. What is required from models of group selection is that a character should evolve because it increases the fitness of the group, despite decreasing the fitness of any individual who bears it. A more stringent condition might be that such a character should become fixed in the population as a whole, having arisen once by mutation in a single group.

THE TRAIT-GROUP MODEL

The classical model of group selection is due to Sewall Wright (1945). He imagined a set of populations, each so small that individually disadvantageous alleles were occasionally substituted by random drift. These populations exchange a few migrants in every generation, so that an allele fixed by chance in one population is constantly reintroduced into other populations where it may also, in time, drift to fixation. If the allele in question is "altruistic," that is, confers some benefit on the group, those populations which maintain it at high frequency may succeed in competition with others, and the whole set of populations may eventually come to consist entirely of altruistic individuals. The model does not allow a gene to pass deterministically from mutation frequency to fixation and seems to rely on a rather improbable combination of population parameters. Essentially similar models, in which the probability of extinction of semi-isolated populations is related to the frequency of altruists, have been discussed by Levins (1970), Eshel (1972), Boorman and Levitt (1973), Levin and Kilmer (1975), and Gadgil (1975). The consensus view seems to be that the requirements for group selection are so stringent that it is unlikely to be a very common or widespread phenomenon. A minority report has been entered by Gilpin (1975), who suggests that more sophisticated models of population dynamics in two-species systems lead quite readily to the evolution of "prudent" predators which do not overexploit their prey.

More recently, it has been suggested by Wilson (1975) that group selection may act effectively through the differential reproductive contribution of groups to a common pool, rather than through extinction. This idea has been criticized on logical grounds by Maynard Smith (1976, p. 279) who argues that, in a finite universe, "... in the long run, evolution by group selection requires group extinction, just as evolution by individual selection requires individual death." In fact, it is only the very reasonable assumption of a finite universe which requires individual death; individual selection can operate very efficiently on inherited variation in fecundity, although in an infinite universe this will not lead to the fixation of genes but only to an indefinitely close approximation to fixation. Granted that individual death must occur, evolutionary change (via selection on fecundity) may happen even if the deaths are wholly nonselective.

But if it is granted that individual death (selective or not) occurs, then the assumption of a finite universe can hold whether or not there is group extinction; all groups could maintain their integrity indefinitely in the absence of stochastic forces, as long as none of them is able to increase in numbers without limit. Even if it is granted that selective group extinction, when it occurs, is a powerful determinant of group selection, it is not a logical prerequisite.

Wilson (1975, p. 144) claims that, given a quite realistic population model, an altruistic gene may pass deterministically from mutation frequency to fixation despite an adverse effect on individual fitness. His population model relies on the concept of "trait-groups," which he describes as follows:

... individuals are spatially restricted during most of their life-cycle, with the exception of their dispersal phase, when what was previously a boundary is easily transcended. As an example, a caterpillar is restricted to one or a few plants, but as a butterfly it spans whole fields.... most ecological interactions, in terms of competition, mating, feeding and predation are carried out during the nondispersal stages in the smaller subdivisions, which I term "trait-groups." In some cases the trait-groups are distinct and easily recognised, such as for vessel-inhabiting mosquitos and dung insects. In other cases they are continuous ...

Few will doubt that this model describes the population structure of a wide variety of species. Among fish, obvious examples are provided by the anadromous salmons, and perhaps by the lampreys and sturgeons, and by the catadromous eels; but most fish seem to form larger and smaller aggregations during different stages of the life history (see, e.g., Gunderson 1972).

Wilson's treatment is appropriate to the case in which immature stages are restricted to the trait-groups, the adults dispersing to form a panmictic breeding unit (deme); I shall also consider the converse situation, in which the immature stages disperse while the adults breed within trait groups. These will be called the "eel" and "salmon" models, respectively, from their rudimentary similarity to the population structure of these two forms. Wilson proceeds to derive a condition that the weighted mean fitness of altruists (taken over all trait groups) exceeds that of nonaltruistic individuals in the deme as a whole. He finds that, given random placement of the two types into trait-groups, this condition is satisfied if the altruistic gene confers higher absolute fitness, whatever its effect on relative fitness; if the variance in gene frequency between groups is greater than random (binomial), altruism may evolve even if it lowers absolute fitness. I believe Wilson's treatment to lack rigor, although his conclusions are broadly correct. Selection occurs within each group, and the gene frequency in the population as a whole after selection is the weighted average of the gene frequencies in each group after selection. The weighted mean fitness of a gene taken over all groups has no obvious relationship to the change in gene frequency within any given group. The correct procedure is to calculate the expected change in gene frequency within each group and then to obtain the frequency in the deme as a whole as a weighted average.

A similar model is proposed by Charnov and Krebs (1975). However, they seem to make the implausible assumption that the total benefit received by an altruist in a group containing n other altruists is the same as that received by a

nonaltruistic individual in the same group, which will be contributed by $(n + 1)$ altruists. Both models are related to the "hay-rick" model of Maynard Smith (1964) (see also Wynne-Edwards 1964).

RESULTS FROM THE "EEL" AND "SALMON" TRAIT-GROUP MODELS

For simplicity, consider a haploid organism with discrete generations; extension to diploidy is straightforward. The totality of organisms in a particular place constitutes the population (= deme), which is divided into a number of groups (= trait-groups); groups are completely isolated during one stage of the life history and intermingle freely during the other stage. At a particular locus, one of two genes may be present: the A gene determines altruistic behavior and the B gene selfish behavior. We consider first the eel model, in which animals live and reach maturity within each group but form a common pool of mature adults within which mating takes place at random; subsequently zygotes resettle into discrete patches in the environment, reconstituting the groups. This is the case analyzed by Wilson (1975). He supposes that the effect of the A gene on its bearer is an increment in fitness f_a , while each A individual contributes an increment in fitness f_r to each other individual in the group. Since this is primarily a discussion of altruistic traits, it will be assumed that f_r is positive, although this in no way affects the algebra. In the i th group, there are N_i individuals among whom the frequency of the A gene is a_i . Given a baseline fitness, λ , this leads to definitions of the fitnesses of A and B individuals, respectively, in the i th group:

$$w_{A,i} = \lambda + f_a + N_i(a_i - 1/N_i)f_r,$$

$$w_{B,i} = \lambda + N_i a_i f_r.$$

Because reproduction occurs outside the groups, and there are assumed to be no differences in fecundity associated with the locus under selection, the fitnesses are intended as rates of survival. In the i th group the change in gene frequency between settling in the group and leaving it to reproduce is given by the usual equation: $\Delta a_i = a_i(w_{A,i} - \bar{w}_i)/\bar{w}_i$, where \bar{w}_i is the weighted mean fitness of individuals in the i th group. Since the total number of individuals remaining in the i th group after selection is $N_i^* = \bar{w}_i N_i$, by definition, the total number of A individuals in the group after selection is

$$N_i^* a_i^* = N_i^* (a_i + \Delta a_i),$$

$$= N_i a_i w_{A,i}$$

as expected, since fitnesses are defined to be rates of survival. If the A gene is to increase in frequency in the population as a whole, it must satisfy the condition

$$\sum_i N_i^* a_i^* / \sum_i N_i^* > \sum_i N_i a_i / \sum_i N_i.$$

Substituting in this inequality, and rearranging, we obtain a sufficient condition for group selection:

$$(f_d - f_r)/f_r > \sum_i \sum_j N_i N_j^2 a_j (a_i - a_j) / \sum_i \sum_j N_i N_j a_j (1 - a_i).$$

To investigate the meaning of this condition, suppose that the population is divided equally between the groups, so that $N_i = N_j = N/n$, where N is the total population size and n is the number of groups. The condition then becomes

$$(f_d - f_r)/f_r > N \sum_i \sum_j a_j (a_i - a_j) / n \sum_i \sum_j a_j (1 - a_i).$$

Now suppose that the A gene is associated with a decrement in fitness, relative to non- A genotypes, such that $f_d < f_r$. A necessary (but not sufficient) condition that the A gene should be favorably selected is then

$$N \sum_i \sum_j a_j (a_i - a_j) / n \sum_i \sum_j a_j (1 - a_i) < 0.$$

The fate of the A gene will depend on the numerical values of f_d and f_r . The inequality above is a sufficient condition that there should exist values of f_d and f_r such that an "altruistic" gene will tend to increase in frequency; for any given situation in which the values of f_d and f_r are specified only by the constraint $f_d < f_r$, it is a necessary, but not a sufficient, condition that group selection will be effective. It is equivalent to the condition

$$n \sum_i a_i^2 > \left(\sum_i a_i \right)^2.$$

This is satisfied by any finite variance in gene frequency between groups. A character which lowers individual fitness, relative to that of other individuals in the same group, may be favorably selected. But if the character in question causes a reduction in absolute fitness ($f_d < 0$), then a condition (with the same meaning as that given above) that it should increase in frequency is

$$N \sum_i a_i^2 - n \sum_i a_i > (N/n - 1) \left(\sum_i a_i \right)^2.$$

This is satisfied if the variance in gene frequency between groups is greater than random (binomial).

The two conditions given above are identical with those obtained by Wilson, although it should be emphasized that in any given situation they are necessary but not sufficient. Now suppose that the A gene is present only in the k th group, so that $a_i = a_j = 0$; $i, j \neq k$. Then a sufficient condition for its increase in frequency in the population as a whole is

$$f_d > f_r \left[1 - \frac{N(n-1)a_k}{n(n-a_k)} \right].$$

This is rather easily satisfied if a_k is large, since this would imply that gene frequencies are underdispersed. But if a_k represents the frequency of a unique mutation, its value will be $1/(N/n) = n/N$. Substituting this in the above inequality, we obtain

$$f_d > f_r \left[1 - \frac{N(n-1)}{n(N-1)} \right],$$

for which $f_d > 0$ is necessary but not sufficient.

It should also be pointed out that the fitness model suggested by Wilson, whether or not it leads to an increase in the frequency of the *A* gene, may not lead to its fixation. The expression for the fitness of the *B* gene is a sum of two components: the "baseline fitness," λ , which lies in the interval $0 \leq \lambda \leq 1$; and the product, $N_i a_i f_r$, which may be any finite positive number. But since fitness, $w_{B,i}$, is defined as a rate of survival, it must itself lie in the interval $0 \leq w_{B,i} \leq 1$. This implies that once the *A* gene has reached a frequency $(1 - \lambda)/N_i f_r$, any further increase in its frequency will not increase the mean fitness of the group. Its effect on group fitness beyond this critical value is undefined by Wilson's model, but it might easily be negative. This would lead to the maintenance of a stable polymorphism within the population as a whole, but this is the outcome of a flaw in the model rather than a biologically realistic prediction.

To summarize, the conditions under which a gene for altruism will pass deterministically from mutation frequency to fixation, given the population structure suggested by Wilson, are set out above. These are conditions that must be satisfied by the population at the beginning of each generation. Initially, a rare gene must cause an increase in absolute fitness, although it is not necessary that it should increase relative fitness. At moderate frequencies this condition is relaxed, and even a lowered absolute fitness may be favorably selected if gene frequencies are underdispersed.

We now consider the salmon model. Adults reproduce within the groups; the zygotes disperse and mingle freely before resettling within groups. This has the technical advantage that it is now convenient to use rate of increase as the criterion of population fitness. The within-group fitness of bearers of the *A* gene is s , defined relative to that of bearers of the *B* gene; the finite rate of increase of the i th group, in which the *A* gene is present at frequency a_i , is $\lambda(1 + ha_i)$, where h is a constant. Using the same nomenclature as before, we can define

$$a_i^* = a_i s / [1 - a_i(1 - s)],$$

$$N_i^* = \lambda N_i (1 + ha_i).$$

We require a condition such that

$$\sum_i N_i^* a_i^* / \sum_i N_i^* > \sum_i N_i a_i / \sum_i N_i.$$

Substituting in this expression, and performing the necessary algebra, we find a sufficient condition to be

$$\sum_i \sum_j N_i N_j a_j (a_j^* / a_j - 1) / \sum_i \sum_j N_i N_j a_j (a_i - a_j) > h.$$

If all groups are initially equal in size, this becomes

$$\sum_i (a_i^* - a_i) / \sum_i \sum_j a_j (a_i - a_j^*) > h;$$

if $s < 1$ and $h > 0$, then a necessary (but not sufficient) condition is

$$n \sum_i a_i a_i^* > (\sum a_i)^2,$$

which approaches the corresponding condition for the previous model as s approaches unity and leads to the same conclusion: a necessary (but not sufficient) condition for the increase in frequency of the A gene is that it should increase absolute fitness. For $s < 1$ the condition is more stringent than before (since $a_i^* < a_i$), and successful group selection requires a correspondingly greater variance in gene frequency. If the A gene is present only in the k th group, where it has a frequency a_k , then the above inequality becomes $(a_k^* - a_k) > h a_k (a_k - n a_k^*)$. Further, if a_k is a mutation frequency u , then the frequency of A in the k th group after selection is exactly $su / (1 + su - u)$, which approaches su as u approaches zero. Substituting su for a_k^* yields $s > (1 + hu) / (1 + hnu)$, and we note that if the mutation is unique, $u = n/N$, the condition becomes $s > (N + hn) / (N + hn^2)$. Since this does not require that $s > 1$, the initial condition for the spread of the A gene can be satisfied if the gene determines a decrease in relative fitness, provided that it has a positive effect on absolute fitness ($h > 0$), by raising the rate of increase of the group as a whole.

DISCUSSION

Using the trait-group model, Wilson is able to describe natural selection as a continuum which is bounded at one extreme by pure individual selection and at the other by pure group selection. There is no question but that individual selection is usually the more effective force and that group selection becomes more plausible the more closely the groups approximate individuals, that is, the more closely the trait-group model approaches the model of a panmictic population whose members intermingle freely at all stages in their life history. In terms of the argument presented above, the reader may satisfy himself that the conditions under which group selection will occur are progressively relaxed as n tends to its limit N and as the variance in gene frequency between groups tends to its limit $\bar{a}_i(1 - \bar{a}_i)$, when all groups are exclusively A or B .

Thus, for a given total population size, group selection becomes more plausible as the number of individuals per group diminishes. As this number becomes very small, sampling effects will increase in importance, and drift may be sufficiently powerful to create groups in which the frequency of the A gene exceeds the range of low frequencies over which group selection in the strict sense is unlikely in the eel model.

Maynard Smith (1976) has recently questioned the usefulness of Wilson's model on terminological grounds. He argues that to regard natural selection as a continuum between pure individual selection and pure group selection

confounds two essentially different processes; and that the underdispersion of gene frequencies required by the model implies kin selection rather than true group selection. As to the first criticism, the difference between these two authors seems to be that they view evolutionary change at different levels: Maynard Smith examines the selective process and concludes that it can be separated, at least conceptually, into two distinct components; Wilson inspects the result of the selective process, the process of change in gene frequency, and concludes that it is a perfect continuum, insofar as its two components may be "mixed" in any proportions. The one is an analytic, the other a synthetic, view of the same phenomenon, and there seems no reason that the two should not coexist.

But one may take issue with Maynard Smith's definitions of group and kin selection, which rely not on any essential taxonomic difference between the two processes but on what are alleged to be the different population structures they require in order to operate. Thus, group selection is defined as a process which may occur in a deme comprising many small, partially isolated groups, while kin selection may operate if relatives live close to one another. Now this is like defining an explosion in terms of a gas leak; it draws attention to an interesting concomitant of the process (assuming that our theory of explosions is true) at the expense of failing to describe the process itself. I take it that group selection refers to the differential genetic survival of groups of unrelated individuals, while kin selection operates between groups of relatives. Of course, "unrelated" is not intended as an absolute term, because the average relatedness of individuals will always exceed zero in a finite population. Rather, in group selection *sensu stricto* the average relatedness of individuals within a given group should not exceed the average relatedness of individuals within the deme as a whole. This requirement will be upset by any stochasticity in the placement of individuals into groups and by any degree of reproductive isolation between the groups, but this consideration applies to all models of group selection and not only to the trait-group model. It will apply with particular force to a model of small, almost completely isolated groups without a dispersal stage, within which inbreeding and stochastic effects will combine to increase average relatedness far above the mean value for the deme as a whole.

Wilson's model requires that genes should be underdispersed, which will follow if the members of a given group are on average related to one another more closely than they are related to individuals in other groups. Indeed, Maynard Smith (1976) refers the model in its entirety to kin selection, pointing out that the counterexample given by Wilson (1975) is unconvincing. However, there are other ways of generating a greater-than-binomial variance in gene frequency between groups. For example, partial isolation between groups will ensure that only a fraction of the individuals in a given group is derived from the gene pool of the deme as a whole; but in this case the trait-group model will tend to resemble Sewall Wright's model of partially isolated populations, in which the conditions for group selection are known to be stringent. If the gene in question also causes any systematic change in a character which affects spatial segregation, then this pleiotropic effect will inflate the variance of gene

frequency between groups; Wilson (1977) discusses a number of possibilities. The same result would follow if the *AB* locus were linked to a locus determining the type of environmental patch within which an individual tends to settle or if for any other reason these two loci were in linkage disequilibrium before selection. Finally, on the appearance of a unique mutation kin selection cannot be invoked, since there are by definition no other individuals identical by descent at this locus; nevertheless, conditions are shown above that an altruistic gene may initially increase from mutation frequency. One way of viewing the trait-group model is as a generalization of the kin-selection argument, referring to groups of individuals which are on average similar in state at a given locus, whether or not they are similar by descent.

A further objection made by Maynard Smith (personal communication) to the trait-group model is that it postulates that each individual altruist in a trait-group can contribute an amount, f_r , to each other member of the group, regardless of how many there are, at a constant cost to itself. This is a powerful criticism. Suppose that each altruist has a certain fixed amount, f_r , of benefit to contribute and that this is shared equally among all other members of the group. In terms of the eel model, this is equivalent to dividing the term furthest to the right in the expressions for $W_{A,i}$ and $W_{B,i}$ by a quantity $(N_i - 1)$. If we adopt the assumption that all trait groups are of equal size, then $f_r^* = f_r / (N/n - 1)$ is a legitimate substitution for f_r , and a sufficient condition for group selection is

$$(f_d - f_r^*)/f_r^* > N \sum_i \sum_j a_j(a_i - a_j)/n \sum_i \sum_j a_j(1 - a_i).$$

Suppose that $f_d < 0$. Then this condition is more stringent than it formerly appeared (since $f_r^* < f_r$ and $d[(f_d - f_r^*)/f_r^*]/df_r^* > 0$) and becomes more stringent as group size increases (since $d[(f_d - f_r^*)/f_r^*]/d(N/n) < 0$). Thus, a more realistic appraisal of the effect of altruism on group fitness in the eel model reduces the likelihood of group selection and further emphasizes the importance of small groups. This objection does not apply to results from the salmon model.

Group selection has always been of interest to biologists because it seems to explain how certain traits which happen to be of interest to human beings might have evolved. Its true significance lies deeper. If we consider the dynamics of gene number rather than of gene frequency (see Williamson 1972), then by the rules of differentiation

$$d \sum_i N_i a_i / dt = \sum_i [N_i (da_i / dt) + a_i (dN_i / dt)].$$

The first term on the right-hand side represents individual selection and the second term group selection. Group selection will rarely determine the direction of change in gene number, because a_i is small relative to N_i , but it will always influence the rate of change in gene number, and if the population is extensively subdivided its effect may not be negligible. As with gene number, so with gene frequency. The conditions under which group selection determines the direction of evolutionary change may be rather restricted, although they

appear to be less restricted than is usually supposed, but group selection may exert a significant influence on the dynamics of any given evolutionary change in a much wider set of circumstances. I would like to put this point of view more strongly. Wilson's trait-group model makes formal the concept of selection as a continuum which embraces individual selection on the one hand and group selection on the other. The distinction between these two modes of selection in practice is therefore a matter of degree, which is to say, arbitrary. In populations which can be described by the trait-group model, evolution will be determined by forces which can be represented by a point on this continuum. The real question, then, is not whether individual or group selection is the more important, but, how does population structure influence evolution?

SUMMARY

A population model proposed by Wilson (1975) is reanalyzed. The model describes a population which is divided into completely isolated groups during one phase of the life history but whose members intermingle freely during another phase. Sufficient conditions are demonstrated that an altruistic genotype should be favorably selected, including its increase from mutation frequency. Neither the model nor the conditions for group selection appear to involve any biological absurdities, although these conditions appear to be more stringent than was originally suggested by Wilson. This treatment also suggests that a rigid distinction between individual and group selection would be artificial.

LITERATURE CITED

- Boorman, S. A., and P. R. Levitt. 1973. Group selection on the boundary of a stable population. *Theoret. Pop. Biol.* 4:85-128.
- Charnov, E. L., and J. R. Krebs. 1975. The evolution of alarm calls: altruism or manipulation? *Amer. Natur.* 109:107-112.
- Cole, L. C. 1954. The population consequences of life history phenomena. *Quart. Rev. Biol.* 29:103-137.
- Crow, J. F., and M. Kimura. 1970. *An introduction to population genetics theory*. Harper & Row, New York.
- Dunbar, M. J. 1960. The evolution of stability in marine environments: natural selection at the level of the ecosystem. *Amer. Natur.* 94:129-136.
- Emlen, J. M. 1973. *Ecology: an evolutionary approach*. Addison-Wesley, Reading, Mass.
- Eshel, I. 1972. On the neighbour effect and the evolution of altruistic traits. *Theoret. Pop. Biol.* 3:258-277.
- Gadgil, M. 1975. Evolution of social behavior through interpopulation selection. *Proc. Nat. Acad. Sci.* 72:1199-1201.
- Ghiselin, M. T. 1974. *The economy of nature and the evolution of sex*. University of California Press, Berkeley.
- Gilpin, M. E. 1975. *Group selection in predator-prey communities*. Princeton University Press, Princeton, N.J.
- Gunderson, D. R. 1972. Evidence that Pacific Ocean perch (*Sebastes alutus*) in Queen Charlotte Sound form aggregations that have different biological characteristics. *J. Fisheries Res. Board Can.* 29:1061-1070.
- Levin, B. R., and W. L. Kilmer. 1975. Interdemic selection and the evolution of altruism: a computer simulation study. *Evolution* 28:527-545.

- Levins, R. 1970. Extinction. Pages 77-107 in M. Gerstenhaber, ed. Some mathematical problems in biology. American Mathematical Society, Providence, R.I.
- Maynard Smith, J. 1964. Group selection and kin selection. *Nature* 201:1145-1147.
- . 1976. Group selection. *Quart. Rev. Biol.* 51:277-283.
- Nikolskii, G. V. 1969. Theory of fish population dynamics as the biological background for the rational exploitation and management of fishery resources. Oliver & Boyd, Edinburgh.
- Williams, G. C. 1966. *Adaptation and natural selection*. Princeton University Press, Princeton, N.J.
- . 1968. *Group selection*. Aldine, New York.
- Williamson, M. H. 1972. *The analysis of biological populations*. Arnold, London.
- Wilson, D. S. 1975. A theory of group selection. *Proc. Nat. Acad. Sci.* 72:143-146.
- . 1977. Structured demes and the evolution of group-advantageous traits. *Amer. Natur.* 111:157-185.
- Wright, S. 1945. Tempo and mode in evolution: a critical review. *Ecology* 26:415-419.
- Wynne-Edwards, V. C. 1962. *Animal dispersion in relation to social behavior*. Oliver & Boyd, Edinburgh.
- . 1963. Intergroup selection in the evolution of social systems. *Nature* 200:623-626.
- . 1964. Reply to J. Maynard Smith. *Nature* 201:1147.