Group Selection, Altruism, and the Levels of Organization of Life

Richard D. Alexander, Gerald Borgia

Annual Review of Ecology and Systematics, Volume 9 (1978), 449-474.

Stable URL:
http://links.jstor.org/sici?sici=0066-4162%281978%299%3C449%3AGSAATL%3E2.0.CO%3B2-B

Ann. Rev. Ecol. Syst. 1978. 9:449–74

❖4149

GROUP SELECTION, ALTRUISM, AND THE LEVELS OF ORGANIZATION OF LIFE

Richard D. Alexander and Gerald Borgia
Division of Biological Sciences and Museum of Zoology, University of Michigan, Ann Arbor, Michigan 48109

INTRODUCTION

As the early papers of Hamilton (35) attracted increasing attention, a trend began in identification of the unit of natural selection that paralleled the "atomistic" approach to population genetics [Mayr (57), p. 377]. The gene began to be treated as the sole unit of selection, with arguments at genotypic or higher levels seen as imperfect, holistic, or unnecessarily complicated. On the other hand, unsupportable holism also persisted. Thus, in a book concerned with sex and evolution, Ghiselin (30) scarcely mentioned subgenotypic units of any kind.

Some recent discussions on these issues have focused on the "levels" at which selection is most effective. Generally, the question has been asked only in relation to populations and individuals, often in efforts to understand cooperative or altruistic social behavior. Thus, Lack (45) and Williams (77) argued that selection at the individual level is sufficient to explain evolutionary adaptation. Although biologists have generally tended to agree (e.g. 51), evolution by differential extinction of groups has recently been modelled or discussed anew by several authors (11, 18, 21, 28, 30, 32, 39, 48, 49, 51, 53, 55, 79–82, 84, 85). E. O. Wilson (86), for example, has argued that "In the past several years a real theory of interpopulation selection has begun to be forged, with both enriched premises and rigorous model building. . . . Insofar as the new theory considers the results of counteraction between group and individual selection, it will produce complex, nonobvious results that constitute testable alternatives to the hypothesis of individual selection. My own intuitive feeling is that interpopulation selection is important in special cases. . . ."

In contrast to evolutionary biologists concerned primarily with social behavior, population geneticists have attempted to identify the selective "unit" (51). While it is commonly asserted that selection operates chiefly at the individual level, it is also obvious that the genetic composition of individuals disintegrates during sexual reproduction and that the identifiable heritable units that reconstitute in descen-

0066-4162/78/1120-0449$01.00

dants represent only fragments of the original genotype. Franklin & Lewontin (27) emphasized what Ford (25), Sheppard (61), Mayr (56, 57), and other ecological geneticists had long argued—that units larger than genes are regularly transferred from one generation to the next and that therefore heritability is high for such linkage groups or "genetic chunks" (52; see also 63). Mayr (56) and Williams (77) followed Fisher (24) and Haldane (33) in emphasizing the importance of the genetic environment in the effects of selection on gene frequencies.

The discrepancy between the widely accepted unit of selection (the individual) and the obvious units of inheritance (chromosomes and parts of chromosomes), while certainly not new to geneticists, has nevertheless been ignored in most recent evolutionary genetic models, which have assumed the gene to be both the unit of evolution and the unit of selection, and tacitly the "level" at which selection is most effective [e.g. (19), but see (47, 52)]. Sometimes there may be no significant loss of predictive ability in treating genes as if selection acted directly upon them. In other cases the resulting predictions may be inadequate, as when the interests of the individual gene conflict with those of the rest of the genome in which it resides. The imperfect state of our analyses of these points is revealed by the absence, to now, of any discussion of the sense in which frequencies of conflicts of interest between units at different levels of organization (such as gene and genome) may themselves be products of selection.

THE CONCEPT OF GROUP SELECTION

To the extent that group selection means simply the occurrence of differential extinction (or reproduction) of groups of individuals, no biologist is likely to deny its existence. As Williams (77, 78) notes, however, *differential extinction of groups can account for the direction of evolutionary change in a trait only when groups differ in the trait and when this difference accounts for the difference in extinction rate.* We regard this as the criterion for group selection. Moreover, as Williams and others imply, differential extinction of groups has seemed most interesting when the direction of change it causes runs counter to that effected by selection at individual or genic levels, in such fashions as to cause phenomena giving the appearance of altruism. (Part of the reason for attention to such cases may also be that when group and individual selection operate in opposite directions their relative potencies are likely to be easier to measure). A simple but unavoidable paradox arises in modeling the kind of group selection that meets these requirements: Neighboring groups must simultaneously be independent enough to develop differences that will account for differential extinction, yet near enough to one another that (*a*) extinguished groups can be replaced by emigrants from surviving groups or (*b*) new patches of habitat that would have been populated by emigrants from extinguished groups can be populated by emigrants from extant groups. To the extent that conspecific emigrants can recolonize neighboring areas, however, they can also dilute differences between neighboring groups, thus simultaneously reducing the significance of selective (as opposed to accidental and therefore nondirectional) intergroup extinction, and enhancing selection at individual and genic levels.

The critical question about group selection of individuals remains essentially unanswered: How important is it in determining the traits of organisms, and are there some situations, some traits, or some kinds of organisms for which it is predictably more important? Approaches to these questions have most often taken the form of modelling the population structure seemingly most conducive to group selection, then seeking that structure in nature. Additionally, most authors writing on this topic have restricted themselves largely to groups distinct from one another only because of habitat patchiness, as opposed to groups resulting from other circumstances that favor group living (2). Lewontin (51) in a somewhat different approach sought reasons for different intensities of selection in the properties of units at different levels in the hierarchy of organization of life, and noted that rapidity of response to selection depends upon heritability, variation, and the rates of reproduction and mortality per unit time. He concluded that "the primary focus of evolution by natural selection is the individual." The change in Lewontin's thinking about selection at individual and group levels between 1965 and 1970, paralleling that of most evolutionary biologists, can be illustrated by this quotation from his 1965 paper [(50), p. 309]:

> By all odds the most important cases of interdeme selection are those in which the character that increases the probability of survival of the deme as a unit is itself being selected against within the population. . . . A striking one is selection of feeding pressure in an herbivore. Clearly, within a population the animal that gets the most food with the least effort is selected for, yet the end result of such selection pressure would be a population of super herbivores who would rapidly deplete the vegetation and cause extinction of the population as a whole. Only interdeme selection, it would seem, could hold down the rate of food consumption in the species.

In 1970 he analyzed precisely the same circumstance in quite different terms [(51), p. 13], concluding that "self-regulation of numbers, on which most of the argument for group selection is built, simply does not occur."

ANALYSES OF GROUP SELECTION OF INDIVIDUALS

Recent analyses of group selection have taken three forms. First are attempts to explain apparent cases in nature, of which two remain problematical (51). Second are efforts to model population structure conducive to group selection. Third are experiments demonstrating conditions under which group selection is effective (71).

Two Supposed Cases

Lewontin [(51), pp. 14–15] notes that, "although . . . we do not need population selection to explain evolution [and] . . . in most instances it would be an inefficient process, there is still the possibility . . . [and] in fact [there are] two well-documented cases of interdeme selection . . . [these being] evolution of the "t" gene in *Mus musculus* . . . [and] the evolution of the rabbit *Oryctolagus cuniculus* and a disease virus, myxoma, in Australia. . . ." These two cases have been cited by recent authors, and for many biologists they represent the only apparent demonstrations that group

selection of individuals occurs naturally. Some of the attributes of these cases, however, and some unanswered questions, cast doubt on such an interpretation.

In the case of the mouse *t* alleles there are two problems. First, little is known of reproductive effects of *t* alleles other than their tendency to be present in 85% rather than the expected 50% of the sperm produced by males heterozygous for *t*. Since males homozygous for *t* are sterile, Lewontin concludes that "powerful individual selection" reduces the frequency of *t* alleles while "powerful organelle selection" increases it. But we are given no information on the vigor, mating success, or other reproductive attributes of either males or females heterozygous for *t* alleles. Other phenotypic effects are known, such as tail-shortening or absence of tails (20). Without information on relative reproductive success, which cannot easily be gained in the laboratory, we are not compelled to accept Lewontin's suggested explanation of the intermediate frequencies that would be produced in wild populations from the combined effects of gametic and individual selection. An apparent case of this kind of balance was, however, discovered by Heribert-Nilsson (41) in the primrose (*Oenothera*). One allele produces a faster-growing pollen which outcompetes that produced by the other allele in reaching the ovum and fertilizing it; but it also produces a plant that is less winter hardy than that resulting from the less successful pollen. Haldane (33) presumed that the selective conflict, expressed at the different levels of gamete and zygote (individual), results in both alleles remaining in the population.

Second, Lewontin argues that selection at the deme level is involved in the observed frequencies of *t*-alleles in wild populations by noting that "because of the very small size of mouse populations and the domination of a territory by one or two males, it often happens that a small deme will have only *t/t,* sterile males. That deme will then become extinct so that *t* genes are removed from the species by population selection."

It is significant that Lewontin speaks of "one or two" males per "deme." The question arises as to how frequently a female mouse's litter derives from matings with two males (i.e. littermates have different fathers), or how frequently a female who has mated with one sterile male either has a second male available for mating or actually mates with him. If Lewontin's "demes" are actually individual males with their harems, and two-male "demes" are simply cases of hanger-on, essentially nonbreeding males or of misinterpretation by the observer (i.e. the so-called second male is not actually a part of the breeding group), then the selection he is describing is not population or group selection at all, but selection on individual males and females. Rather than "demes" becoming extinct as a result of their males all becoming sterile, a sterile male fails to produce offspring, and one or more females, unable to recognize and respond to their mate's sterility, also fail to produce offspring.

For a different reason the myxoma virus case may also be inappropriately termed interdeme selection. Lewontin states that reduction of "virulence" in the virus "cannot be explained by individual selection." First, virulence must be inseparable from rate of multiplication for Lewontin's argument to make sense. Ability or tendency by the virus to kill its hosts, as such, is not in the interest of the virus, since

"the virus cannot survive in a dead rabbit" and is not transmitted when the host dies because "mosquitoes do not bite dead rabbits."

Even if virulence is equated with rate of multiplication, interdemic selection in the usual sense is not indicated. Lewontin refers to each rabbit as "a deme from the standpoint of the virus." But the virus reproduces without sexuality, so that a rabbit infected by a single virus particle, or a few identical particles, will contain a clone of genetically identical viruses except for mutational changes. Lewontin's group selection model actually requires that, as a rule, less- and more-virulent viruses be mixed in the same rabbits and that those with increased proportions of less-virulent viruses acquire such virus populations largely as a result of rates of infection rather than as a result of the reproduction of the viruses once they enter the rabbit. Because rates of multiplication would favor the more-virulent virus, the changes in rates of infection would have to favor the less-virulent virus as a result of differential longevities of rabbits containing fewer or more of the two viruses; thus the rate of differential extinction of Lewontin's "demes" would have to be rapid enough to overcome the more rapid rates of multiplication of more-virulent viruses in mixed populations within rabbits.

If the population of rabbits is composed largely of individuals infected with pure more-virulent and pure less-virulent strains (i.e. clones), the relevant selection on the virus might be more appropriately described as occurring at the individual level. Thus, if this virus has evolved for a long time in pure clones, a clone of identical viruses would be no more appropriately regarded as a group or deme than would be the cells of a metazoan organism, since each member of a clone should evolve to sacrifice as much for a clone member as for itself. Because vertebrates and other familiar organisms are almost devoid of clones of individuals, it is difficult for us even to think in terms of the extremes of altruism that are predicted by consistent cloning.

Recent Models

Several recent authors have attempted to model the appearance of altruistic traits as a result of group selection (11, 32, 49, 53, 79, 81). Characteristically these models assume the most favorable conditions for group selection. Thus, the rates of reproduction and mortality of populations are reduced to near those of individuals—e.g. by assuming that habitats last only one or a few generations. The likelihood of relevant heritable differences between groups is sometimes maximized by assuming replacement of extinct groups, or filling of empty habitat, randomly across the population rather than by neighboring populations (e.g. 79). If populations do not break up and reform frequently, then individual and genic selection within groups will tend to eliminate altruism among founding individuals; if founding individuals tend to locate near their natal populations, then neighboring groups will be similar and less likely to be differentially extinguished owing to their trait differences. Unless differential extinction of groups (or differential production of migrants by them) correlates with trait differences, group selection cannot occur, although individually deleterious traits could be fixed by drift (87).

In many cases these recent models also assume very small subpopulations or groups, which reduces the proportion of the total genetic variance contained in each group, thus also enhancing the likelihood of heritable differences between groups (e.g. 53). In some cases they assume a high degree of population viscosity, with or without actual formation of subpopulations (e.g. 39). In some cases small subpopulations are assumed to form as a result of a few females' depositing all of their young in an empty habitat (79). These kinds of assumptions are forced on group-selection theorists: In order to get models that work they must have situations in which supposedly altruistic traits benefit other individuals also carrying the alleles that lead to altruism. In other words, if social interactants are sufficiently likely to be genetic kin, then genes leading their bearers to altruism will spread by kin selection and the inclusive fitness of the bearers will be raised. Accordingly, the relevant selection is returned to individual and gene levels, and models with this feature are not models of group selection. Consistent with these arguments, in some models individuals are also postulated to "assort positively" with their "own type in settling from the migrant cloud" [(39), p. 139]. Hamilton (39) thus considers an inclusive-fitness model "more general than the 'group selection,' 'kin selection,' or 'reciprocal altruism' approaches" because it "can deal with an ungrouped viscous population where, owing to restricted migration, an individual's normal neighbors and interactants tend to be his genetical kindred." One of the reasons Hamilton believes his model is able to deal with this situation is that "it obviously makes no difference if altruists settle with altruists because they are related (perhaps never having parted from them) or because they recognize fellow altruists as such, or settle together because of some pleiotropic effect of the gene on habitat preference." However, genes for the same habitat preference as altruists, but no altruism, would replace those leading to both altruism and habitat preference. For this and other reasons, detailed later, we are skeptical of some of the proximate mechanisms for altruism among kin suggested in this model. As we will also argue later, an inclusive-fitness model depending upon nondiscriminative altruism among individuals who are more or less symmetrical in their ability to provide social benefits may not be very general. [For a postulated example of this application, however, see Fisher (24) on distastefulness in caterpillar sibling groups.]

That groups are often composed of kin does not mean that kin selection and group selection are in any sense synonymous (e.g. 74, 77, 79, 85). As West-Eberhard (73) points out, "In the same trivial sense that kin selection is group selection, all of natural selection is group selection, since even 'individual' selection really concerns the summed genetic contribution of a group—the individual's offspring." Moreover, although kin selection can occur in continuously distributed populations, group selection cannot. For reasons elaborated later, we agree with Maynard Smith (54) that it is more appropriate to distinguish kin selection and group selection than to blur their differences by considering them together.

In some recent models for the evolution of altruism, traits that involve social acts benefiting others are regarded as altruistic even when their net effect is beneficial to their possessors (53, 82), meaning that they raise their possessors' inclusive fitness in the population at large. Such traits are not altruistic in the usual biological sense, and models proposed for their spread need not involve group selection (16, 67).

EXPANDING THE CONCEPT OF GROUP SELECTION

Group selection is commonly regarded as a problem involving the relative potencies of selection at the two levels of the individual and the social group or population. Individuals cooperate in social, economic, political, and kin groups, and these various groups may form coalitions at still higher levels. Life, however, is organized not in a two-ranked hierarchy but in a many-ranked one. Thus, genes function in groups called chromosomes, and chromosomes in groups called genomes, and these merge into hierarchies of individuals, demes, populations, species, communities, and ecosystems. Interactions among subunits within a genome may parallel the interactions of individuals or coalitions within social groups. By comparing the operation of selection at different levels in the hierarchy, we may better understand how differential reproduction has produced the current structures and organization of living systems. Traditional arguments concerned with group selection have focused on interactions among individuals to produce group effects. Under what we call an expanded view of group selection, interactions among entities at all levels in the hierarchy of living systems may be considered in terms of their contribution to patterns of reproduction. Long-continued potent selection at any level in the hierarchy of life is likely to enhance the integrity of entities at that level and reduce the likelihood of conflicts of interest with units at lower levels. This effect of selection may be responsible for many of the commonplace concepts of biology: gene, chromosome, individual, and family; and in some sense it lies behind the identification of cohesive functional elements such as tissues and organs, various appendages, reflexes, and reflex groups, and of individuality in behavioral acts. The complexities involved in extending the concept of group selection to include the interactions of subgenomic genetic units, and the value of this procedure, are illustrated by Lewontin's 1974 conclusion [(52), p. 307], in a chapter entitled "The genome as unit of selection," that "relative selective values of substitution at a locus cannot be judged from the frequencies of the alleles in nature because *the selection of the chromosome as a whole is the overriding determinant of allelic frequencies.*" (Italics in original.)

Selection Among Species

Some constituents in the hierarchy of organization of life evidently owe their identity not so much to their status as selective units as to externally imposed selective forces such as habitat patchiness and the vagaries of climate and weather. Species, for example, almost certainly form as a result of genic and individual selection. Nevertheless, as Hamilton (37) noted, group (interpopulational or interdemic) selection within species may be a more precarious concept than group selection between species; the reason is that reproductive isolation between species negates the requirement of an unlikely intraspecific population structure for intraspecific group selection. Thus, species can co-exist, "poised" to replace one another, without losing their differences through amalgamation. Indeed, they can continue to diverge, and they can even cause one another's extinction. One implication is that while ecological communities may often be significantly affected by differential extinction of species, species are not necessarily likely to have been greatly influenced by differential extinction of populations or demes (see 81). Another is that conspecific populations

that behave toward one another as if they were different species—maintaining separate territories, discouraging movement of individuals between groups, displaying intergroup aggression, and especially showing efforts to extinguish and replace one another—are thereby enhancing the likelihood of significant group selection, or indicating its importance in their past.

Units or groups such as species, then, may be established through individual or genic selection, yet persist or fail as a result of competition with other species—hence, through a kind of group selection. Dawkins (19) has denied that differential species extinction can properly be termed group selection because, as he puts it, species are not "replicators." But of course they are: Species give rise to species; species multiply.

From this perspective we may reconsider Fisher's (24) view of the selective background of sexuality. Recent investigators seem to agree that Fisher's arguments about the evolution of sexuality are unsupportable because of the way in which they invoke selection at the population or species level (e.g. 54, 78). But when a sexual population produces an asexual one—when a sexual individual asexually produces an offspring capable of continuing to reproduce asexually—unless the asexual descendants can revert more or less easily to sexuality, the asexual population is exactly like a second species, and the competition between sexuality and asexuality (that is, the question whether or not sexuality is maintained) is in some sense between the asexual genotype (throughout the asexual population except for mutations) and the whole collection of recombining sexual genotypes. This is as clearly group selection as is competition between two distinct species. Williams [(78), p. 110] invokes this kind of selection as a partial explanation for the maintenance of sexuality in low-fecundity populations, as Fisher had invoked it long before by comparing the relative rates at which beneficial mutations can spread through competing sexual and asexual populations.

Part of the reason for considering the whole collection of genotypes in the sexual population in the above argument is that, in comparing sexual and asexual genotypes as competitors, one cannot measure simply the collective value of a brood in the sexual line to determine the fitness of the parent. Also involved is how different is the average fitness of the brood members from the rest of the population—or, more specifically, from that of potential mates. A set of offspring enormously superior to the asexual genotype(s) might, on this account, be limited to producing a set of grandchildren inferior to the asexual genotype(s). This is a hazard of specialized sex (meaning loss of the ability to become quickly asexual when a superior genotype is produced, or when fitness is high and the environment is stable), and to some extent it binds together the members of a sexual population and gives special importance to the average fitness of sexual genotypes. The average fitness of genotypes in sexual populations, despite its wide use in population genetics, actually has little significance, except in interspecific competition that is definitely leading to unilateral extinction, or when there are asexual genotypes with which to compete. If there are no asexual genotypes, then, unless fitness differences involve some kind of "absolute" values (i.e., unless they refer to differential survival rather than differential reproduction), the variance in (heritable) fitness will determine the rate of

evolution (24). Situations involving the above "absolute" and "relative" fitness values correspond roughly to Wallace's (72) "hard" and "soft" selection, which he used to explain why genetic load is a misleading concept when it involves arithmetic which implies that the fitness of any polymorphic population is lowered because of the "genetic deaths" that result from heterozygote advantage (segregational load) or that are necessary to remove the deleterious allele by selection (substitutional load) (see also 12, 13). If there are competing asexual genotypes, however, the fittest sexual genotypes will be hurt in their competition with the asexual genotypes by having only inferior genotypes with which to recombine. One way out of the dilemma is selective mating. If phenotypes produced by superior genotypes are able to restrict their matings to other superior genotypes they need not be dragged down all the way by the average fitness of the entire sexual population. Selective mating, however, increases the cost of sexuality.

Thus, Fisher's argument would have been appropriate had he applied it to the maintenance of sexuality rather than to its origin—in other words, if he had assumed that sexuality was the primitive condition and had referred only to forms in which asexual lines are generated from sexual lines, without the ability to change with facility between sexuality and asexuality.

Selection of Asexual Genomes

In the expanded view of "group" selection suggested here, the extreme example below the species level, as evidenced by integrity of groups, may be the populations of genes that make up secondarily asexual genotypes. Although they may not represent the easiest cases to review in causal historical terms, we can use them to consider conditions optimal to group selection, as well as to exemplify an extreme outcome of these conditions.

First, it may be useful to justify considering genes in genotypes as groups in discussing "group" selection. We tend to view only alternative alleles as competitors. Even genes at different loci, however, are potentially reproductively competing entities, at least historically, and in this sense genes in genomes may be viewed as parallels to individuals in populations. At infrequent intervals, the genes in sexual genomes disperse and recombine, forming new genomes in a fashion roughly parallel to the dispersal and recombination of individuals in species living in temporary habitats. The regularity and organization of the periodic recombinations of genes in genomes, and the complexity of genic cooperation between recombinations that produces the emergent populational effect that we call the phenotype, may at first cause this comparison with individuals to seem bizarre. Unlike individuals in species, genes in genomes (usually) cooperate without differential reproduction and it is this cooperation that contributes to our view of an individual as a well-defined unit in the reproductive hierarchy. At any time, however, sexual genomes are susceptible to the appearance of genes or other units (here termed "outlaw" genes or units) that, because of recombination, may reproduce at some expense to the rest of the genome. There are obvious limits to the damage such elements can inflict while still successfully spreading, but they can have important effects on individual reproduction. Reproductive units capable of suppressing other units that generate

effects deleterious to the rest of the genome will be favored for that suppression; in a later section we describe systems that seem to produce this type of control.

Unlike the populations of genes that make up sexual genotypes, those in completely asexual (apomictic) genotypes never recombine; hence, the genome is certainly the unit of selection. Changing any gene creates a new genotype, and the fate of every gene is inextricably linked to the fates of all other genes in its genotype. The interests of the genes within an asexual genotype are evidently synonymous, meaning that they scarcely (if at all) compete with other genes in their own genotypes; they compete only with those in other genotypes. (In this sense the apomictic genotype is functionally a gene.) That asexuality appears usually to be secondary increases the evident appropriateness of viewing asexual genotypes as products of a history of group selection.

In some sense, then, it can be seen that all units of higher levels in the hierarchy of organization of life are essentially aspects of the environments in which units at lower levels are selected. [Williams (77) made this point for the gene.]

SEXUAL GENOTYPES AND THE OUTLAW CONCEPT

The consequence of reproductive conflicts at genic and genomic levels, with selection remaining potent at genomic levels, is that genes that favor themselves at the expense of all other alleles in the genome (outlaw genes) are likely to have their effects nullified, at the very least to the extent that they are outnumbered by the other genes in the genome. An allele mutating to neutralize partly or completely the effects of a gene acting contrary to the interests of all other genes in the genome (including itself) would be favorably selected for that effect; the chances of such neutralizing mutations' occurring, and of synergistic effects or combining of partial effects from different neutralizing mutations, would be much larger than the chances of counteraction by a single outlaw gene. This "power of the collective" [Leigh (46, 47) called it a "parliament of the genes"] exists in any situation in which a large number of units, at any level in the hierarchy of life, share a similar interest contrary to that of a single unit or smaller group. For example, even though parents are generally more able to manipulate their offspring in their own interests than vice versa [(2), (68), p. 256, (69), p. 255, (70), footnote 21], large broods of adult offspring might be able to force a parent to behave according to interests contrary to the parent's own if the collective interests of the brood members are the same. Trivers & Hare (69) argue that such offspring domination occurs in the social Hymenoptera in regard to the colony's investment in reproductives of the two sexes (also 7, 22, 59). Offspring domination is probably most likely in cases in which the conflict involves adult offspring, no longer in any sense dependent on parental care; in such cases, typical of at least some hymenopteran interactions, the conflict may be more appropriately viewed simply in terms of the relatedness of the interactants rather than as parent-offspring conflict.

Some alleles that cause an offspring to gain at its parent's expense may be able to spread (9). Such an allele will be an outlaw not in its own genome but within its

brood and in terms of the parental genome. The allele, therefore, must not only overcome any suppressing effects by the parent, which may be quite powerful (2), but must also compete with its alternatives both in lines not containing it and in mixed broods.

Support for the existence of selection against "outlaws" is provided by reports that, in some dipteran females, aberrant chromosomes are preferentially shunted into polar bodies during meiosis (15, 65). In humans, only one of the X chromosomes is active in somatic cells; if one X chromosome is damaged it generally becomes the deactivated member of the pair (26, 31, 34).

Genes on the Y chromosome are particularly likely to be able to operate to their own advantage, contrary to the interests of other genes in their own genome (37). Because this chromosome occurs only in the heterogametic sex, and alone is passed intact through generations of descendants of that sex, the possibility exists for genes on it to mutate and cause their bearers to favor descendants carrying the mutation (40). Any effects of genes on any chromosome that increase the phenotypic distinctiveness of the sexes would incidentally increase the potential for a Y mutant to produce this effect. Such a gene might cause its bearer to give more care to descendants of the same sex, or to give care only to descendants of that sex; or it might cause the bearer to produce only offspring of the same sex. Any such gene would be approximately three times as effective as similar genes on the other (X) sex chromosome, because each X chromosome spends approximately one third of its time in the heterogametic sex (37), and in humans even more so because of the recognizability of the Y chromosome, given established cultural inheritance patterns [e.g. males may pass inheritance directly to grandsons or great-grandsons through all-male lines of descendants (40)].

If the above arguments about outlawry are correct, effects of genes on the Y chromosome might be expected to lead to a general suppression of their activity by modifiers throughout the genome, thus contributing to the observed general inertness of the Y chromosome. Hamilton (37) attributed this general inertness to the genomic response to Y mutants when they cause either meiotic drive or differential success of Y-bearing sperm. The effect postulated here refers to favoring of Y-bearing zygotes; it is likely more restricted than that postulated by Hamilton because it would only be effective when descendants beyond first-generation offspring can be assisted (since first-generation offspring carrying the male's X chromosome can also be positively identified).

To illustrate further the behavior of what we are calling "outlaw" genes and systems within the genome, we may consider the meiotic drive of sex chromosomes in *Drosophila* (29, 66), which causes biases in the representation of chromosomes among the gametes of an individual male. In *Drosophila* the male genotype is XY, that of the female XX. Nevertheless, in some *Drosophila* populations X chromosomes have been found in nearly all mature male gametes, and such sperm produce almost all females. These X chromosomes realize at least a short-term advantage. The rest of the genome, however, suffers, since the potential for producing broods with optimal sex ratios is eliminated. In other *Drosophila* populations, certain X chromosomes induce disintegration of the Y chromosomes, leading to death of the

Y-bearing spermatocyte or sperm. The effect is a loss of fitness to the genotype, owing to (*a*) sex ratio distortion, (*b*) energetic loss, and (*c*) a probable reduced success in sperm competition with other males (because half of the sperm of a male with this X chromosome are inviable). White [(75), p. 476] has referred to these processes as "murder of one chromosome by another" (see also 47).

Control mechanisms for dealing with driving sex chromosomes have been found in *D. paramelanica* (64). Driving X chromosomes in populations of this species differentiate into two types, northern and southern, based on their response to control by Y chromosomes. Northern X loses its capability to drive when paired with southern Y; when an X is paired with a Y from its own locality there is no inhibition of drive. Northern Y does not suppress drive in either X. Additionally, an autosomal trait suppresses drive in both types of X. Alleles involved in suppressing chromosomal drive apparently also exist in some populations of the mosquito, *Aedes aegypti* (42), and Hamilton (37) discusses suppression of driving chromosomes by other elements in the genome.

Selection against differential success of Y-bearing sperm or zygotes could feasibly have contributed to the disappearance of the Y chromosome in many insects. Thus, the general evolutionary sequence leading to the haplodiploid insects might have been the following: 1. establishment of separate sex chromosomes, with the male XY; 2. modification of the Y chromosome to a generally inert condition; 3. loss of the Y chromosome, leaving females with all of the genetic materials necessary to make sons; 4. production of haploid sons parthenogenetically [or, as in some aphids, diploid sons by a meiotic parthenogenetic process in which only an X chromosome is eliminated via the polar body, creating the XO male genotype (58)]. Loss of the Y chromosome in the ancestor of haplodiploid insects, with its peculiar short-term advantages, thus apparently led to the males of haplodiploid species now being unable to produce sons at all, having lost this particular battle of the sexes because in their ancestors part of the male genome behaved contrarily to the interests of the more powerful remainder.

Driving effects most certainly occur in the homogametic sex and in autosomes, but they should be less apparent there, largely because they would not lead to sex ratio distortions. A driving autosome would either replace its homolog or be suppressed by modifiers, most likely on the homolog. Considerations of these sorts, however, lead to the suggestion that whatever symmetry exists in the events of mitosis and meiosis may be largely the result of stalemates between competitive elements in the genome.

PARALLELS IN SOCIAL-GROUP AND GENOME ORGANIZATION

As with generalized responses to outlawry by individuals living in social groups (e.g. legislation against predictable classes of disruptive behavior such as murder or usury), generalized responses to deleterious mutants (whether or not the mutant contributes to its own reproduction at the expense of the genotype as a whole) would have the additional advantage to the group as a whole (here, the group of genes) of providing a means of avoiding disasters, or of capitalizing on opportunities that

are individually unique but fall within the class protected by the "law" or the "repair" (or maintenance) mechanism. The response would evolve as a result of recurring past deviations (e.g. murders or mutations) and would be maintained because of its value in dealing with continuing deviations; thus, it could prevent an "outlaw" gene from spreading at all.

Thus, we may raise the question whether the evolution of dominance and recessiveness of allelic effects by selection of modifiers at other loci (24) could evolve to be expressed as a generalized resistance to dominance in mutants, or whether it is strictly a genic effect, with specific modifiers affecting each allele. The possibility of generalized response counters the criticisms of Fisher's theory of dominance modification that second- or third-order modification would be weak and genetically expensive, and would only be favored during brief transitory periods of heterozygosity (e.g. 17). Such a possibility was raised by Sheppard [(61), p. 122]: ". . . dominance may sometimes be evolved as the result of selection of a gene-complex facilitating normal development under extreme environmental fluctuations because such a gene-complex also tends to maintain normal development when the agent disturbing development is not the external environment but a rare mutant."

Other likely candidates as generalized mechanisms that have evolved to work against outlaw effects include: canalizing influences such as DNA repair mechanisms that evidently consistently return uncomplemented mutant molecules to the original condition in somatic cells of multicellular organisms, several possible anticancer mechanisms (14), DNA repair mechanisms that may occur in germ-line cells (23), and intracellular effects involving selective removal of DNA from both somatic and germ-line cells (60). Unless they have been retained because of specific effects on particular molecules, such mechanisms are likely to be polygenic and they may often be the result of selection maintaining the genetic status quo at the level of the genome or very large subunits within it. Similarly, immune responses that are so complex or so generalized that they are commonly described as involving the recognition of "foreignness" may effectively bar outlawry as a result of either growth of mutant tissues or viral invasion (10).

SOCIAL ALTRUISM

Probable Origins

Demonstrations that undirected altruistic tendencies would spread in viscous populations if individuals tended to interact with close relatives have led to implications that (*a*) sociality commonly grows from acts of undirected altruism that are faintly beneficial to the inclusive fitness of the actor because of slight incidental numerical favoring of individuals with the same trait, and (*b*) that altruism directed equally to any interactant is a simpler, more likely primitive condition, or a more parsimonious assumption, than is altruism directed only to certain closely related interactants. Thus, Hamilton (39) regards the basic problem of achieving a suitable frequency of altruism in a viscous population as similar to that of spreading a powerfully kin-selected mutant. This might be true if there were no preexisting situations involving powerful kin selection, or if alleles leading to discriminative nepotism could be kept out of the population.

Powerful kin selection is universal among sexual organisms in the form of maternal investment in eggs and offspring. A mutant causing its bearer to assist only its own juvenile offspring has a 50% likelihood of helping itself, which is an enormous advantage at the outset; and it is most likely to represent aid with potent returns because (*a*) juveniles are so easily assisted by parents, (*b*) parents (in sexual species) usually have several or many offspring they might assist, and (*c*) few parents are precisely adapted to predictable environments; most have some opportunity to contribute to offspring survival with remainders of reproductive effort that would otherwise be lost.

In higher animals, complex sociality is concentrated in highly parental organisms and tightly knit sibling groups; it may be restricted to them. Such circumstances place together the most closely related individuals of a species in terms of genes identical by descent. In the parent-offspring interaction, moreover, the two kinds of individuals are maximally asymmetrical in age, stage, vulnerability to mortality from predators and climate, and overlap of resource use. Accordingly, parents (adults) are in a maximally favorable position to dispense inexpensive aid to offspring (eggs) that maximally resemble the parents genetically. The ubiquity of varying extents of parental investment across the animal and plant kingdoms supports the argument that the parent-offspring interaction is the major source of social donorism or phenotypic altruism. Except for sibling groups [e.g. (24), p. 177–78] we know of no cases in which altruism seems likely to have originated between individuals of like age and stage, which therefore lack the asymmetry of competitiveness and inexpensive aid that characterizes the parent-offspring interaction. Sibling interactions, moreover, may not be entirely divorced from parental influence.

Temporariness of habitat and patchiness in distribution together tend to create conditions conducive to group selection [generation time near that of habitat duration and likelihood of heritable differences between groups (see 51)]. However, these conditions also tend to break up families. As a consequence they interfere with the ability of individuals to direct altruism toward relatives. Parental care and other forms of nepotism seem more likely to lead to altruism in populations that are stable and viscous. Population stability and viscosity, however, are least conducive to the evolution of altruism as envisioned in the model of D. S. Wilson (79), and a precursor of parent-offspring sociality is contrary to the model of undirected nepotism discussed by Hamilton (39) for these kinds of populations. The kinds of altruism discussed by Hamilton and D. S. Wilson seem most likely in populations living in discontinuous habitats; and altruism in higher animals, whether directed or undirected, seems more likely to involve stable interactions of family members.

We are also unaware of any clear evidence, in animal groups that do not reproduce by fission or budding, of the kind of undirected altruism specified in the models of D. S. Wilson and Hamilton, and also implied in the arguments of authors who tend to merge kin selection and group selection (e.g. 84), except as a likely outgrowth of within-family or directed nepotism.

Contrasting directed and undirected altruism suggests either (*a*) that the two kinds of altruism have mutually exclusive origins or (*b*) that the weak forces of undirected altruism postulated by D. S. Wilson (79, 80, 81) and Hamilton (39) are

more likely to be derivatives than forerunners of powerful kin selection in contexts like parental assistance to offspring or cooperation between siblings. As Hamilton's [(35), p. 24] second generalization suggests, undirected altruism would tend to be replaced by directed nepotism because the latter, as soon as it appears, renders the former an inferior alternative. Once altruism had evolved, however, from, say, parent-offspring or sibling interactions, situations would arise in which ability to practice nepotism discriminately had not been evolved, but in which altruism was elicited in a nondiscriminative fashion.

A complicating factor, for understanding sociality in groups of individuals of only slightly closer than average relatedness, is that even the most strictly "selfish" herd (38) may, erroneously as well as accurately, give the appearance of involving mild or diffuse undirected altruism. Thus, if one individual tolerates another's presence, it may be difficult for an observer to know whether the first or the second individual is receiving the benefits (e.g. see 16).

Probable Mechanisms

Hamilton's (35) model of inclusive fitness does not require that nepotists recognize genetic relationships of potential beneficiaries, despite his argument that "the social behaviour of a species evolves in such a way that in each distinct behaviour-evoking situation the individual will seem to value his neighbor's fitness against his own according to the coefficients of relationship peculiar to that situation." The solution to this seeming paradox can be understood from the details of human sociality. Mothers who observe the birth of their own children and immediately develop an unmistakable means of recognition directly establish the identity of this particular relative; but all other assumptions of relationship in human societies depend upon circumstantial evidence. We are told who our various relatives are, or we decide on the basis of various kinds of responses to them by others. Thus, one's putative siblings may be identified as those individuals who are cared for by the same adult female, and a man's putative offspring are those individuals accepted by the woman with whom he has been living. Even so, humans could scarcely have been aware, before the advent of modern genetics, of the precise or probable fractional relationships of supposed genetic relatives. To the extent that they have behaved appropriately to the various relationships, it is because stable social relationships have paralleled genetic relationships, allowing the former to predict the latter (4, 5). We may assume that similar circumstances account for the realization of Hamilton's model of inclusive fitness wherever it is appropriate for nonhuman species.

There are other possible ways in which kin selection might work. Hamilton (35) discussed the possibility of genes' producing phenotypes able to recognize and tending to help bearers of the same genes that produce them. For two reasons such "genetic recognition systems" are unlikely. First is the complexity of the effects required (35), and second is the fact that any such genes (or genetic units) would cause effects contrary to the interests of all others in the genome (4). Thus, such a genetic unit must: (*a*) influence some perceptible feature of the phenotype; (*b*) cause the perception of the feature; (*c*) bring about the appropriate social response.

Although Hamilton described these requirements, he was equivocal about the possibility of establishment of such units. Thus, he suggested that "exactly the same a priori objections might be made to the evolution of assortative mating. . . ." It seems to us, however, that this parallel is not justified. First, there is yet no proof that any case of assortative mating depends on genes somehow causing all of the above effects without some kind of learning. Second, in mate choice there is presumably no conflict of interests within the genome. Third, with assortative mating a frequency-dependent advantage is usually associated with each mating type; otherwise the different types would not be maintained. In genetic-recognition systems, the appearance of alleles that are interpreted as identical by donors but do not themselves donate would cause genetic-recognition mechanisms of benefit distribution to disappear. The gain for such "cheating" alleles is highest when they are rare and the donors are abundant. However, cheaters may remain abundant when donors become rare, so that donors rarely obtain benefits from other donors. Conceivably, alleles that cause their bearers to donate may produce complex signal mechanisms that segregate true donors from cheaters. Such systems would be vulnerable because of the ease of producing phenotypes that lose the tendency to donate while retaining the signal.

Genetic recognition systems are also unlikely because of problems in integrating individual behavior in the distribution of benefits. If more than one allele influences flows of benefits to other individuals, conflicts are certain to arise because potential recipients will not always be genetically identical at all loci involved in the control of benefit distribution. Although an allele at one locus might control the distribution of benefits in a manner that enhances only its own reproduction, other alleles in its genome would gain from neutralizing this control, and mere numerical superiority should be sufficient to tip the balance in their favor.

Patterns of benefit distribution in populations do not correspond to those expected if genetic-recognition systems were common. Studies on human populations (4), ground squirrels (62), prairie dogs (43), and wasps (59) indicate that closer relatives are treated preferentially. By contrast, fixation for "genetic recognition" alleles would lead to rather uniform distribution of benefits to all interactants, and relatives would be preferred only while such traits were on their way to fixation. Such systems could account for variations in nepotistic behavior associated with social structure only if there is (*a*) rapid recurrence of mutations leading to genetic recognition or (*b*) common association with disadvantageous characters through either pleiotropy or linkage. The first condition is unlikely because of the necessity of complex recognition mechanisms, and there is no reason to expect the second.

Dependence of the classical inclusive fitness model (35) on social behavior appropriate to genetic relationships explains the success of transfers of young of different broods into the broods of adults unrelated to them, and the appearance of parental ability to discriminate among offspring at the time when offspring commonly become mixed in nature (44).

In the classical model of inclusive fitness there is agreement within the genome in the pattern and degree of aid given to possible recipients. If donorism depends solely on average probability of identity at each locus, then all intragenomic units share the same interests. Fixation of traits coding for control of such kin-selection

mechanisms would yield the common patterns of preferential treatment of close relatives and apparent inability to distinguish among relatives of the same class, even though individuals of that class differ in actual proportions of alleles identical to those of the donor.

If assessment of relatedness is circumstantial rather than direct, then cheating is less likely because (*a*) behavior that brings an individual benefits from nonrelatives may reduce the likelihood of recognition, thus benefits, from true relatives, and (*b*) selection will improve the ability of donors to identify appropriate recipients. Thus, parents of potentially cheating offspring will be excluded from sites where mixups may occur, and social relations will be established at times when certainty of genetic relationship is greatest.

It has been suggested that selection should work to enhance effectiveness of donors in determining relatedness of potential recipients (35, 39). Even so, kin-selection models generally appear to work through the distribution of benefits based on average relatedness of, for example, siblings, rather than preferential treatment of individual siblings that happen to have greater relatedness than that of the average sibling. Given apparent limits on the ability of nepotists to detect variance within classes of related individuals we conclude that donors' discriminative abilities commonly fall below the actual variance among relatives within discriminative classes, in terms of genes identical by descent.

Phenotypic cues may sometimes be used to determine relatedness, as with paternity in humans. This is an extreme case, since a man shares either half or no genes identical by descent with his spouse's offspring; and because of the long human gestation period men share with other male mammals a certain reduced confidence of paternity. We may note, incidentally, the concern, sometimes expressed jokingly, over whether or not a human baby, especially a boy, resembles its mother's spouse as compared to whether or not it resembles its mother.

If phenotypic recognition occurs we predict that genes responsible probably do not give their bearers the ability to recognize their own particular effects, but a generalized ability to locate phenotypic similarities reflecting genetic overlap. So long as this kind of recognition of relatives, which evidently would have to be learned, only tests for whether or not to accept particular relationships (such as the father-son relationship), it will tend to benefit all genes in the genome equally. Mutants that diminish this effect would not be favorably selected.

Suppose that certain alleles give to their bearers tendencies or abilities to use phenotypic cues to assess the relatedness of potential recipients of nepotism. If the assessment involves distinguishing different classes of relatives such as full- or half-siblings, then all genes in the genome will, on average, be equally benefited. If, however, such genes operate to assess the proportional genetic overlap of relatives within a single class—for example, different full siblings— then only the genes responsible for phenotypic similarity will be benefited. Even the genes yielding the tendencies to assess the similarity, unless they are responsible for it, will be disfavored for their behavioral effect, as will all other genes in the genome (5).

If nepotism depends on circumstantial evidence of relatedness, as we have argued here, then inclusive fitness (as opposed to fitness through direct descendants alone) is meaningful only to the extent that social interactions permit accurate assessments

of genetic relationships. The interesting consequence is that the essential ontogenetic basis of appropriate patterns of nepotism in any species need be no more complex or deterministic than learning through ordinary positive and negative reinforcement schedules (4).

Inclusive-Fitness-Maximizing and the Capacity for Culture

It is a widespread assumption that humans everywhere possess approximately the same "capacity for culture." This assumption is based on the evidence that individual humans can be moved among the societies on earth, and reared in a system other than the one in which their immediate ancestors evolved; if they are not identified and discriminated against by the other members of that system, they are likely to function perfectly well.

It is also assumed that if the capacity for culture is approximately the same in all humans, and if this is what has chiefly evolved in humans, then evolution and natural selection must have ceased long ago to have any important influence on human behavior.

Should these things be true we are immediately puzzled by the degree to which cultural patterns, such as those involving variations in the symmetry of treatment of cross- and parallel-cousins, marriage rules, inheritance patterns, infanticide, and other aspects of culture (4) seem to correspond so closely to predictions from selection theory.

Consider the genetic changes that might occur in connection with inclusive-fitness-maximizing through altruism to genetic relatives. Ideally, one should weigh relatedness against ability to translate benefits into genetic reproduction and compare the result among relatives available or likely to become available for assistance. If the relevant facts about relatives are learned in various ways, then one expects an accumulation of genes leading to aptness in acquiring and and understanding all of the right kinds of information about relatives. These genes might be expected to become tightly linked into a supergene and the same genes—or the same supergene —would be equally valuable to everyone if the useful kinds of information and the learning situations were similar for all individuals. Genes leading toward actual inclusive-fitness-maximizing by assisting relatives should, in fact, become fixed in the population, even if their action were extremely indirect through a wide variety of learning processes. In a theoretical extreme one can imagine the fixation in all humans of a single supergene that gives to every individual the ability and tendency, in normal environments, to discriminate relatedness and needs of relatives optimally for his own genetic interests. Even if this is an unlikely possibility because of cultural variations among societies and temporal changes within societies, it is a useful postulate for considering the effects of selection on inclusive-fitness-maximizing behavior.

The paradox in the above scheme is that it leads to synonymy in precisely the genes responsible for abilities and tendencies to discriminate among relatives, and for producing competition among individuals. One supposes that once the above supergene had been fixed, tendencies would spread to ignore genetic differences and

treat everyone equally because all would share the genes actually responsible for discrimination. No mutant leading to such behavior, however, could invade the system just described. Each new mutant on a supergene for nepotism would reintroduce a significance for discriminatory behavior on the basis of genes identical by immediate descent among interacting relatives. It would do this because at the start its own distribution would be based on immediate descent. This means, paradoxically, that mutants causing any departure whatsoever from the kind of inclusive-fitness-maximizing behavior just described would, by this particular effect on their phenotypes, render themselves inferior alternatives to their previously existing alleles. The only way in which a supergene for nepotism of the sort described could be altered or replaced would be if changes in the cultural environment diminished the tendency of the old supergene to accomplish inclusive-fitness-maximizing, so that a mutant could improve it.

These arguments show why Hamilton (35) was correct to focus his analysis upon relatedness in genes identical by immediate descent, and they point the way to determining how to deal with the questions of genetic overlap from inbreeding and convergence owing to parallel or convergent selection. One needs only to consider the fates of mutants affecting nepotistic behavior. Such mutants represent the means by which the altruism of nepotism generates, increases, and becomes directed with precision. The successive waves of such mutants will always maximize their own spread by treating relatives as if their own likelihood of occurring in the relative depends upon the proportion of genes identical by immediate descent. This is because each new mutant will indeed tend to be present in just those proportions: No better odds will occur.

GROUP SELECTION AND EMERGENT PROPERTIES

The above comparisons suggest a classification of instances of group selection that may lend direction to the kind of theorizing mentioned by E. O. Wilson (86) (see above). The difference between the sexual or asexual genotype as a group and the usual deme of organisms in nature is that the genome is itself an evolved phenomonon while the deme [e.g. the "trait group" of D. S. Wilson (79, 80)] is an effect imposed upon the population of which it is a part by extrinsic ecological contingencies. In other words, the organization and integrity of the genotype appear to be products of selection reinforcing the gene-group as the actual unit of selection and reducing conflicts between the genomic and the subgenomic level. "Trait group" is also a biased term, implying that the demes under consideration differ in traits leading to differential reproduction of the demes, when this may not be a general or maintained condition.

Separation of evolved and incidental groupings may be facilitated by considering, for example, whether particular organisms (*a*) are actively forming social groups or (*b*) are thrust together passively by the vagaries of climate, weather, and habitat distribution; whether individuals (*a*) gain by the presence of neighbors or (*b*) are simply forced to tolerate their proximity; and whether gain from active grouping

(*a*) results chiefly from predators or other extraspecific forces or (*b*) is a matter of group-against-group competition within the species (2, 3). Group unity may be promoted, or evolve, because of group selection, or group selection may be promoted because of (gain by individual units in promoting) group unity. The difficulty of decisions in particular cases does not detract from the fact that these distinctions represent important biological foci.

Evolved units may take on emergent properties. Perhaps it is significant that biological senescence has so far been clearly identified only at the individual (genotypic) level (36, 76). It is probably a prerequisite of senescence that selection has occurred consistently at the level of the unit undergoing it, and this may mean, in turn, that heritable attributes paralleling the phenotype of the individual organism should be identifiable as products of the action of social groups. Such attributes as colony "life" cycles and nest architecture in the social insects, and some features of culture (e.g. laws, symbols, and other formalized and codified conventions) in human societies are suggestive. The effect, however, may also be entirely illusory. In the case of social insects we are drawn to the unsettled question of whether single maternal genotypes or the collection of parental and offspring genotypes determine colony attributes (2, 7, 59, 69). In the former case senescence of the colony may correlate with senescence of the maternal phenotype. In the case of human culture, evidence of senescence in the sense of Williams (76) seems doubtful.

The distinction between groups of evolutionary units (whether groups of genes or groups of individuals) that are incidental effects and those that are evolved products of selection is an important one. For example, the latter category may include some social groups of humans. Group living in humans (at least today) seems both inevitable and enormously specialized. It is difficult to avoid the speculation that human individuals, as with genes, long ago began to gain in the reproductive race by cooperating to compete. That genes have been at it much longer and have become much more specialized is indicated by the integrity of the individual organism, so overwhelming that philosophers have regarded the individual as the most distinctive entity in the universe. (Chauvinism is probably also involved, since only individuals have evolved the ability to philosophize.) We take completely for granted the evident standoff competition among genes, which usually results in either an even distribution of representatives of the different loci to the genome population during meiosis and mitosis, or else the automatic death or deformity of the resulting individual. In contrast, despite the complexity of organization and patriotic fervor that characterize some human political states and family clans, differential reproduction by the individuals comprising them, and competitive striving evidently related to it, remain prominent. Genes in genomes also operate so much more simply, as, perhaps, to make the comparison strained: Genes appear that enhance or thwart effects of other individual genes or groups of genes, and spread if the effect helps themselves even if it does so only by helping the genotypes in which the new genes occur. But individual humans reflect on the past, predict the future, compare and discuss their deliberations, and make conscious decisions about cooperation, nepotism, ostracism, cheating, and outlawry—all, of course, through the actions of their genotypes in their particular developmental environments.

COMPETITION OF LIKE GROUPS

Commonality of interests leads to group unity and enhances the likelihood or potency of group selection. Reflecting upon genotypes and human social groups leads one to the speculation that commonality of interests may reach a maximum when the principal "hostile forces" (in Darwin's sense) result from competition by parallel or similar groups, as with other social groups within the same species or other groups of genes in a population of genes. This might be true because (*a*) groups of individuals represent an easy way of multiplying appropriate competitive forces against other individuals and groups; (*b*) reproductive competition and overlap of resource utilization cannot diminish among conspecific individuals as they do between species as a result of evolutionary divergence; (*c*) within groups of like individuals, multiplication of interests prevalent throughout the group against the interests of selfish individuals is more possible [see below; (3, 39)]; or (*d*) groups within species can be formed of closely related individuals competing against more distant relatives.

Actions of individuals in obligately group-living species necessarily evolve as compromises exemplified by two hypothetical questions: (*a*) How can I help myself *directly by helping my own phenotype* and those of genetic relatives, and (*b*) how can I help myself *indirectly by contributing to the unity and perpetuation of my social group?* In human social groups, at least, the picture is further complicated by a third question: (*c*) How can I help myself and my relatives by furthering novel or temporary within-group coalitions that tip the balances of power toward our interests?

Whenever the interests of the individual gene (or linkage group) coincide with those of the rest of the genome it is useful, and probably sufficient, to derive selective hypotheses from the simple question: Would a gene causing a certain tendency spread (e.g. one leading to better escape from predators, one maintaining sexuality, or one enabling an offspring to cheat its parent)? It is not easy, however, to determine when such coincidences of interest are the case. Thus, commonality of interests may at first seem likely for all aspects of genomic cooperation in producing the phenotype—that is, for all somatic effort. On the other hand, such commonality is definitely not the case in nepotism, since some genes will be represented in partial relatives and others will not. Because, in sexual organisms, reproductive effort is usually a matter of nepotism to partial relatives, and somatic effort has presumably evolved solely as a contribution to reproductive effort, differences of interest among genes can be prevalent even in the guiding of ontogenies. For genes as well as for individuals in social groups, therefore, the kind of question posed above will lead to accurate predictions only when the investigator considers fully the abilities of other units (genes, chromosomes, individuals, groups of individuals) to counteract the effect whose success he wishes to predict, should it be contrary to their interests. To the extent that natural selection has produced coalitions of genetic units or individuals, whose numbers and cooperative interactions toward common interests enable them to deny success to subunits with conflicting interests or abilities to reproduce differentially, the coalitions themselves may be properly described as the

units of selection. Evidently this is commonly the case for genomes, and in this sense the individual rather than the gene or any other subunit is properly termed the unit of selection. The essential question is the extent to which commonality of interest is produced and maintained by selection. In Lewontin's terms (51), individuals in sexual species (*a*) retain the *rapid cycle time* of subgenomic units, (*b*) possess *high heritability* most of the time (i.e. except during gamete formation), and (*c*) enormously increase their *variability* by recombination.

GROUP SELECTION AND CULTURE

Human social groups represent an almost ideal model for potent selection at the group level (1, 2, 3, 84). First, the human species is composed of competing and essentially hostile groups that have not only behaved toward one another in the manner of different species but have been able quickly to develop enormous differences in reproductive and competitive ability because of cultural innovation and its cumulative effects. Second, human groups are uniquely able to plan and act as units, to look ahead, and to carry out purposely actions designed to sustain the group and improve its competitive position, whether through restricting disruptive behavior from within the group or through direct collective action against competing groups. Human groups, in other words, have the organization and foresight to accomplish the kinds of behavior tacitly, and evidently erroneously, attributed to organisms in general by Wynne-Edwards (88); rather than being the only organism lacking intrinsic ability to regulate their populations, as Wynne-Edwards (89) suggests, humans may actually be the only species possessing such ability. Of course, majorities, or power groups, prevail in human societies by rendering disadvantageous certain individual behavior that would otherwise be reproductively advantageous; an example is the imposition of socially controlled monogamy, apparently unique to the human species (8).

Recent legislation in India requiring sterilization of individuals once they have produced a specified number of offspring suggests the counter example: In any case in which the background and success of such a law depend on a conscious desire of a majority of group members to prevent expansion of the population (which was certainly not the case in India), the law would properly be labelled evidence of self-regulation of numbers by the group itself. Human populations obviously possess this capability [see also (2), p. 376]. Such self-regulation operates through the power of the collective to punish selfishness and reward altruism—hence, to reinforce the interests of a majority of the group or of a power clique. The extent to which such activities represent or promote group selection depends partly on the fates of the members of unsuccessful or threatened groups. If, in one extreme, they disappear without issue, as in the cases of the Tasmanians and Patagonians, then the concept of group selection is appropriate; if they are consistently able to join successful groups without severe detriments to their reproduction, that is, if coalitions are temporary and shifting, then group selection is not indicated.

Learning and culture are commonly viewed merely as systems of evolved plasticity. The unique adaptive significance of culture, however, may be that in sexually

recombining organisms it can be simultaneously more heritable than genes and more abruptly changeable. Thus, wealth and power may be acquired suddenly, but they may also be more easily passed to the next generation than the phenotypes necessary to acquire them. To the extent it derives from traditionally transmitted learning, culture can also change cumulatively in the absence of genetic change. Moreover, whether culture is conservative or revolutionary can be determined by immediate contingencies; culture, to a greater degree than learning in general, incorporates the feedback between need and novelty that the genes seem never to have evolved in regard to rates and directions of mutational changes. All aspects of phenotypes illustrate this feedback to a lesser degree than does culture. The ultimate adaptability in this direction seems to be the potential to make conscious and deliberate decisions on the basis of predictions extended into the distant future.

Cultural invention is thus a source of power imbalances that may appear abruptly and continually rebuild the differences between neighboring human populations (or cultural trait groups) to a greater than random level. Culture is the great unbalancer that may have reinforced tendencies of humans to live and compete in groups, to make their systems of learning into group phenomena, and to engage in an unusual and unusually ferocious group-against-group competition throughout human history. Murder and war are likely to keep recurring only when their perpetrators are likely to gain, or to believe they will gain. These behaviors may be essentially human phenomena because culture alone leads frequently to imbalances that make such all-out aggression apparently profitable.

This view also emphasizes the significance of plagiarism in the spread of cultural change. Unlike phenotypes acquired in other fashions, those that can be acquired by certain kinds of learning can also be copied by others. The evolved human capacity for culture includes abilities not only to invent and to learn from parents and other active and passive teachers, but also constantly to observe and interpret success and failure and to profit from using the ideas, inventions, and successful practices of others. Such behavior, while representing intragroup competition, also tends to standardize behavior within groups, and thus to increase both the significance of between-group differences and, perhaps, the likelihood of significant selection at the group level (6).

CONCLUDING REMARKS

Humanity is unlikely to understand itself adequately except through knowing exactly what its genes have evolved to accomplish in particular environments, especially in particular social environments. As a result there may be few problems in biology more basic or vital than understanding the background and the potency of selection at different levels in the hierarchies of organization of living matter. The approaches currently being used by evolutionary ecologists and behaviorists in assessing the likelihood of effective selection at the level of groups or populations of individuals may also be used to advantage by those concerned with function at intragenomic levels. The kind of selectionist techniques used recently to analyze the behavior of nonhuman organisms may in the near future be widely applied toward

understanding not only human social phenomena, but a variety of phenomena of classical biology such as mitosis, meiosis, sex determination, segregation distortion, linkage, cancer, immune reactions, and essentially all problems in gene function and in ontogeny.

"The fitness at a single locus ripped from its interactive context is about as relevant to real problems of evolutionary genetics as the study of the psychology of individuals isolated from their social context is to an understanding of man's socio-political evolution" [(52, p. 318].

ACKNOWLEDGMENTS

For suggestions concerning the manuscript we thank Diane DeSteven, John L. Hoogland, Richard D. Howard, Cynthia Kagarise, Bobbi S. Low, Katharine M. Noonan, Daniel Otte, Paul W. Sherman, Donald W. Tinkle, Charles D. Michener, and, especially, James Blick, D. Caldwell Hahn, William D. Hamilton, Egbert Leigh, and Mary Jane West-Eberhard.

Literature Cited

1. Alexander, R. D. 1971. The search for an evolutionary philosophy of man. *Proc. R. Soc. Victoria* 84:99–120
2. Alexander, R. D. 1974. The evolution of social behavior. *Ann. Rev. Ecol. Syst.* 5:325–83
3. Alexander, R. D. 1975. The search for a general theory of behavior. *Behav. Sci.* 20:7–100
4. Alexander, R. D. 1977. Natural selection and the analysis of human sociality. In *Spec. Publ. 12, The changing scenes in the natural sciences, 1776–1976,* ed. C. E. Goulden, pp. 283–337. Philadelphia: Acad. Nat. Sci.
5. Alexander, R. D. 1977. Evolution, human behavior, and determinism. *Proc. Bienn. Meet. Philos. Sci. Assoc.* (1976), 2:3–21
6. Alexander, R. D. 1978. Evolution and culture. In: *Evolutionary Biology and Human Social Behavior,* ed. N. A. Chagnon, W. G. Irons. North Scituate, Mass: Duxbury Press.
7. Alexander, R. D., Sherman, P. 1977. Local mate competition and parental investment patterns in the social insects. *Science* 196:494–500
8. Alexander, R. D., Hoogland, J. L., Howard, R. D., Noonan, K. L., Sherman, P. W. 1978. Sexual dimorphisms and breeding systems in pinnipeds, ungulates, primates, and humans. See Ref. 6
9. Blick, J. 1977. Selection for traits which lower individual reproduction. *J. Theor. Biol.* 67:597–601
10. Bodmer, W. F. 1972. Evolutionary significance of the HL-A system. *Nature* 237:139–44, 183
11. Boorman, S. A., Levitt, P. R. 1973. Group selection on the boundary of a stable population. *Theor. Pop. Biol.* 4: 85–128
12. Brues, A. 1964. The cost of evolving vs. the cost of not evolving. *Evolution* 18:379–83
13. Brues, A. 1969. Genetic load and its varieties. *Science* 164:1130–36
14. Cairns, J. 1975. Mutation selection and the natural history of cancer. *Nature* 255:197–200
15. Carson, H. L. 1946. The selective elimination of inversion-dicentric chromatids during meiosis in the eggs of *Sciara impatiens. Genetics* 31:95–113
16. Charnoff, E. L., Krebs, J. R. 1975. The evolution of alarm calls: altruism or manipulation? *Am. Nat.* 109:107–12
17. Crosby, J. L. 1963. The evolution and nature of dominance. *J. Theor. Biol.* 5:35–51
18. Darlington, P. J. 1975. Group selection, altruism, reinforcement, and throwing in human evolution. *Proc. Natl. Acad. Sci. USA* 72:3648–52
19. Dawkins, R. 1976. *The Selfish Gene.* New York: Oxford Univ. Press. 224 pp.
20. Dunn, L. C. 1957. Studies on the genetic variability in populations of wild house mice. II. Analysis of eight additional alleles at locus *t. Genetics* 42:299–311

21. Eshel, I. 1972. On the neighbor effect and the evolution of altruistic traits. *Theor. Pop. Biol.* 3:258–77
22. Evans, H. E. 1977. Extrinsic versus intrinsic factors in the evolution of insect sociality. *Bioscience* 27:613–17
23. Evans, H. J. 1975. Symposium No. 15. Genetics. Introduction by the chairman. *Genetics* 79:171–78
24. Fisher, R. A. 1930. *The Genetical Theory of Natural Selection.* New York: Dover. 291 pp. 2nd ed.
25. Ford, E. G. 1971. *Ecological Genetics.* London: Chapman and Hall 3rd ed. 410 pp.
26. Fraccaro, M., Lindsten, J. 1964. The nature, origins and genetic implications of structural abnormalities of sex chromosomes in man. In *Cytogenetics of Cells in Culture,* ed. R. J. C. Harris, pp. 97–110. New York: Academic.
27. Franklin, I., Lewontin, R. C. 1970. Is the gene the unit of selection? *Genetics* 65:707–34
28. Gadgil, M. 1975. Evolution of social behavior through interpopulational selection. *Proc. Natl. Acad. Sci. USA* 72:1199–201
29 Gershensen, S. 1928. A new sex ratio abnormality in *Drosophila obscura. Genetics* 13:488–507
30. Ghiselin, M. T. 1974. *The Economy of Nature and the Evolution of Sex.* Berkeley: Univ. Calif. Press. 346 pp.
31. Gianelli, F. 1963. The pattern of X-chromosome deoxyribonucleic acid synthesis in two women with abnormal sex chromosome complements. *Lancet* 1:863–65
32. Gilpin, M. E. 1975. *Group Selection in Predator-Prey Communities.* New York: Princeton Univ. Press. 110 pp.
33. Haldane, J. B. S. 1932. *The Causes of Evolution.* London: Longmans, Green and Co. 235 pp.
34. Hamerton, J. L. 1968. Significance of sex chromosome–derived heterochromatin in mammals. *Nature* 219:225–28
35. Hamilton, W. D. 1964. The genetical evolution of social behaviour. I, II. *J. Theor. Biol.* 7:1–52
36. Hamilton, W. D. 1966. The moulding of senescence by natural selection. *J. Theor. Biol.* 12:12–45
37. Hamilton, W. D. 1967. Extraordinary sex ratios. *Science* 155:477–88
38. Hamilton, W. D. 1971. Geometry for the selfish herd. *J. Theor. Biol.* 31:295–311
39. Hamilton, W. D. 1975. Innate social aptitudes of man: An approach from evolutionary genetics. In *Biosocial Anthropology,* ed. R. Fox, pp. 133–55. New York: Wiley and Sons.
40. Hartung, J. 1976. On natural selection and the inheritance of wealth (and Commentary). *Curr. Anthropol.* 17: 607–22
41. Heribert-Nilsson, N. 1923. Zertatronsversuche mit Durchtrennung des Griffels bei *Oenothera lamarckiana. Hereditas* 4:177–89
42. Hickey, W. A., Craig, G. B. 1966. Distortion of sex ratio populations of *Aedes aegypti. Can. J. Genet. Cytol.* 8:260
43. Hoogland, J. L. 1977. *The evolution of coloniality in white-tailed and black-tailed prairie dogs* (*Scuiridae:* Cynomys leucurus and C. ludovicianus). Ph.D. thesis, Univ. Michigan, Ann Arbor, Michigan. 292 pp.
44. Hoogland, J. L., Sherman, P. W. 1976. Advantages and disadvantages of bank swallow (*Riparia riparia*) coloniality. *Ecol. Monogr.* 46:33–58
45. Lack, D. 1954. *The Natural Regulation of Animal Numbers.* New York: Oxford Univ. Press. 343 pp.
46. Leigh, E. 1971. *Adaptation and Diversity.* San Francisco: Freeman, Cooper, and Co. 288 pp.
47. Leigh, E. 1977. How does selection reconcile individual advantage with the good of the group? *Proc. Natl. Acad. Sci. USA* 74:4542–46
48. Levin, B. R., Kilmer, W. L. 1974. Interdemic selection and the evolution of altruism: a computer simulation study. *Evolution* 28:527–45
49. Levins, R. 1970. Extinction. In *Some Mathematical Questions in Biology. Lectures on Mathematics in the Life Sciences,* ed. M. Gerstenhabre, 2:77–107. Providence, RI: Amer. Math. Soc.
50. Lewontin, R. C. 1965. Selection in and of populations. In *Ideas in Modern Biology,* ed. J. A. Moore, pp. 299–311. New York: Nat. Hist. Press
51. Lewontin, R. C. 1970. The units of selection. *Ann. Rev. Ecol. Syst.* 1:1–18
52. Lewontin, R. C. 1974. The genetic basis of evolutionary change. New York/ London: Columbia Univ. Press. 346 pp.
53. Matessi, C., Jayakar, S. D. 1976. Conditions for the evolution of altruism under Darwinian selection. *Theor. Pop. Biol.* 9:360–87
54. Maynard Smith, J. 1971. The origin and maintenance of sex. In *Group Selection,* ed. G. C. Williams, pp. 163–75. Chicago: Aldine-Atherton

55. Maynard Smith, J. 1976. Group selection. *Q. Rev. Biol.* 31:277–83
56. Mayr, E. 1954. Change of genetic environment and evolution. In *Evolution as a Process,* ed. J. Huxley, A. C. Hardy, E. B. Ford, pp. 157–180. London: Allen and Unwin
57. Mayr, E. 1975. The unity of the genotype. *Biol. Zentralbl.* 94:377–88
58. Morgan, T. H. 1912. The elimination of sex chromosomes from male-producing eggs of phylloxerans. *J. Exp. Zool.* 7:239–352
59. Noonan, K. M. 1978. The sex ratio of parental investment in colonies of the social wasp, *Polistes fuscatus. Science* 199:1354–56
60. Sager, R., Kitchen, T. 1975. Selective silencing of eukaryotic DNA. *Science* 189:426–33
61. Sheppard, P. M. 1969. *Natural Selection and Heredity.* London: Hutchinson. 192 pp. 3rd ed.
62. Sherman, P. W. 1977. Nepotism and the evolution of alarm calls. *Science* 197:1246–53
63. Singh, C. F., Templeton, A. R. 1976. A search for the genetic unit of selection. In *Isozymes,* ed. C. L. Markert, 4:115–29. New York: Academic
64. Stalker, H. D. 1961. The genetic system and modifying meiotic drive in *Drosophila paramelanica. Genetics* 46:177–202
65. Sturtevant, A. H., Beadle, G. W. 1936. The relation of inversions in the X-chromosome of *Drosophila melanogaster* to crossing-over and disjunction. *Genetics.* 21:554–604
66. Sturtevant, A. H., Dobzhansky, T. 1936. Geographical distribution and cytology of sex ratio in *D. pseudoobscura* and related species. *Genetics* 21:473–90
67. Trivers, R. L. 1971. The evolution of reciprocal altruism. *Q. Rev. Biol.* 46:34–57
68. Trivers, R. L. 1974. Parent-offspring conflict. *Am. Zool.* 14:249–64
69. Trivers, R. L., Hare, H. 1976. Haplodiploidy and the evolution of the social insects. *Science* 191:249–63
70. Trivers, R. L., Willard, D. E. 1973. Natural selection of parental ability to vary the sex ratio of offspring. *Science* 179:90–92
71. Wade, M. S. 1976. Group selection among laboratory populations of *Tribolium. Proc. Natl. Acad. Sci. USA* 73:4604–7
72. Wallace, B. 1968. *Topics in Population Genetics.* New York: Norton. 481 pp.
73. West-Eberhard, M. J. 1976. Born: Sociobiology. *Q. Rev. Biol.* 51:89–92
74. West-Eberhard, M. J. 1975. The evolution of social behavior by kin selection. *Q. Rev. Biol.* 50:1–34
75. White, M. J. D. 1973. Animal cytology and evolution. Cambridge, Mass.: Harvard Univ. Press. 961 pp.
76. Williams, G. C. 1957. Pleiotropy, natural selection, and the evolution of senescence. *Evolution* 11:398–411
77. Williams, G. C. 1966. *Adaptation and Natural Selection.* Princeton, N.J.: Princeton Univ. Press. 307 pp.
78. Williams, G. C. 1975. *Sex and Evolution.* Princeton, NJ: Princeton Univ. Press. 200 pp.
79. Wilson, D. S. 1975. A theory of group selection. *Proc. Natl. Acad. Sci. USA* 72:143–46
80. Wilson, D. S. 1975. New model for group selection. *Science* 189:8701
81. Wilson, D. S. 1976. Evolution on the level of communities. *Science* 192: 1358–60
82. Wilson, D. S. 1976. Structured demes and the evolution of group-advantageous traits. *Am. Nat.* 111:157–85
83. Wilson, E. O. 1973. The queerness of social evolution. *Bull. Entomol. Soc. Am.* 19:20–22
84. Wilson, E. O. 1973. Group selection and its significance for ecology. *Bioscience* 23:631–38
85. Wilson, E. O. 1975. *Sociobiology: The New Synthesis.* Cambridge, Mass.: The Belknap Press of Harvard Univ. Press. 697 pp.
86. Wilson, E. O. 1975. The origin of sex. *Science* 188:139–40
87. Wright, S. 1945. Tempo and mode in evolution: A critical review. *Ecology* 26:415–19
88. Wynne-Edwards, V. C. 1962. Animal dispersion in relation to social behaviour. Edinburgh: Oliver and Boyd. 653 pp.
89. Wynne-Edwards, V. C. 1965. Self-regulating systems in populations of animals. *Science* 147:1543–48