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Replicator Selection and the Extended Phenotype[1])

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Abstract

Adaptations are often spoken of as 'for the good of' some entity, but what is that entity? Groups and species are now rightly unfashionable, so what are we left with? The prevailing answer is DARWIN's: 'the individual'. Individuals clearly do not maximise their own survival so the concept of fitness had to be invented. If fitness is correctly defined in HAMILTON's way as 'inclusive fitness' it ceases to matter whether we speak of individuals maximising their inclusive fitness or of genes maximising their survival. The two formulations are mutually inter-translatable. Yet some serious mistranslations are quoted from the literature, which have led their authors into actual biological error. The present paper blames the prevailing concentration on the individual for these errors, and advocates a reversion to the *replicator* as the proper focus of evolutionary attention. A gene is an obvious replicator, but there are others, and the general properties of replicators are discussed. Defenders of the individual as the unit of selection often point to the unity and integration of the genome as expressed phenotypically. This paper ends by attacking even this assumption, not by a reductionist fragmentation of the phenotype, but, on the contrary, by extending it to include more than one individual. Replicators survive by virtue of their effects on the world, and these effects are not restricted to one individual body but constitute a wider 'extended phenotype'.

Introduction

Sociobiology is a name that has acquired irritating pretensions, but we shall probably have to learn to live with it. Whatever may have been E. O. WILSON's (1975) definition, the aspect of 'sociobiology' which has captured the imagination of biologists (other than the minority over-excited by political misunderstanding) is a particular neo-Darwinian view of social ethology. WILSON sums this up in his first chapter, 'The Morality of the Gene', where he identifies the central problem of sociobiology as the problem of altruism, and gives as the answer: 'kinship'. I would characterize the approach as the 'selfish gene' approach to ethology, and I unhesitatingly name as its founding genius W. D. HAMILTON. Not only did HAMILTON (1964, 1971, 1972, 1975) supply the theory of inclusive fitness, bulwark of WILSON's admirable new synthesis. JOHN MAYNARD SMITH has told us that HAMILTON (1967) was also

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an inspirer of the concept of the *evolutionarily stable strategy,* which has been developed by MAYNARD SMITH et al. (1973, 1974, 1976) into another central plank of modern sociobiological theory (DAWKINS 1976 a). With acknowledgment to Konrad LORENZ's well known tribute to Oskar HEINROTH, I would define sociobiology as the branch of ethology inspired by W. D. HAMILTON. But HAMILTON did not go far enough. Paradoxically, the logical conclusion to his ideas should be the eventual abandonment of his central concept of inclusive fitness. We should also move towards giving up the term 'kin selection' as well as group selection and individual selection. Instead of all of these we should substitute the single term 'replicator selection'.

Evolutionary models, whether they call themselves group-selectionist or individual-selectionist, are fundamentally gene-selectionist. They work within the population geneticist's assumption that natural selection acts by changing the relative frequencies of alleles in gene pools. The thing which changes in evolution is the gene pool, and the things between which nature fundamentally selects are alternative alleles. But genes don't literally float free in a pool, they go around in individual bodies and are selected by virtue of their effects on individual phenotypes. A biologist can count on a chorus of approving nods if he says that, in the last analysis, selection works on 'outcomes': it is the whole individual that has to survive, the whole individual who faces the cutting edge of natural selection. Superficially sensible as this sounds, it can be called in question. Selection means differential survival, and the units which survive in the long run are not individuals but *replicators* (genes or small fragments of genome). They survive by virtue of their phenotypic outcomes, to be sure, but these are best interpreted not exclusively at the individual level, but in terms of the doctrine of the *extended phenotype.* Replicator selection and the extended phenotype will be discussed in the last two sections of this paper, after some preliminary matters have been dealt with.

Individuals in their turn go around in larger units — groups and species. Some biologists have accordingly argued that inter-group selection is an important cause of adaptive evolution (WYNNE-EDWARDS 1962). WYNNE-EDWARDS (1977) has recently written: "The general consensus of theoretical biologists at present is that credible models cannot be devised, by which the slow march of group selection could overtake the much faster spread of selfish genes that bring gains in individual fitness. I therefore accept their opinion". But, whether or not we found models of group selection convincing, my point here is that in any case these models were always framed as special cases of *gene-selection* models. Inter-group selection and inter-individual selection, and all the other levels reviewed by LEWONTIN (1970) and WICKLER (1976), are different proximal processes whose claim to biological importance is judged on the extent to which they can be shown to correlate with what really matters — inter-allele selection. I believe it is often superfluous, and sometimes actually misleading, to discuss natural selection at these higher levels. It is usually better to go straight to the fundamental level of selection among *replicators* — single genes or fragments of genetic material which behave like long-lived units in the gene pool.

This amounts to a plea that the good example of population geneticists should be followed by those of us who want to discuss adaptation or function. We often wish to attribute 'benefit' to some entity. Thus an animal may be said to show parental care 'for the good of the species' or 'for the good of its own fitness'. The first of these is almost certainly wrong (WILLIAMS 1966), the second right if fitness is correctly defined in HAMILTON's way. But, in any

case, how much more compelling it is to say: 'Genes which make individuals more likely to perform parental care than their alleles work for the survival of copies of themselves in the bodies of the young cared for.' Or, more briefly and generally, genes work for their own benefit, using individual bodies as their agents. We substitute the easily understood notion of *survival* (gene survival) for the complex and difficult concept of *fitness* (individual fitness).

Fitness

In Herbert SPENCER's (1864) day the fittest survived, and the 'fittest' were understood in the everyday sense of the most muscular, fleetest of foot, brainiest. For SPENCER, fitness was passed on because the individuals best fitted to their way of life survived to reproduce. Fitness was the capacity to survive, and survival was a prerequisite for reproductive success. It was only later that fitness started to *mean* reproductive success, and the fitness of an individual could hence, without contradiction, be said to be increased by sexually attractive characters which detracted from individual survival.

Both in SPENCER's sense, and in the sense of reproductive success, fitness was attributed to *individuals*. But population geneticists developed an independent usage of the word, and they applied it not to individuals but to genotypes at a locus. This made sense, because you can count the number of occurrences of a particular genotype, say Aa, in a population, relative to its alternatives at the same locus. An equivalent count in the next generation, followed by a normalizing division sum, leads to a direct quantitative estimate of the fitness of Aa relative to, say, AA and aa. This is quite different from the idea of individual fitness. You can't count the number of times an individual occurs in a sexually reproducing population, for he only occurs once, ever. If you want to measure the 'fitness' of an individual you have to resort to something like counting the number of his fledged offspring. In the light of HAMILTON's inclusive fitness concept we can now see that this is a very crude approximation. Offspring turn out to be only a special case of close genetic relatives with a high probability of sharing one's own genes.

HAMILTON's rationale is best explained at the level of genes. Thus parental care and sibling care both evolved because genes for such caring behaviour tend to be present in the bodies of the individuals cared for. But HAMILTON expressed the idea at the level of the individual: the individual works so as to maximize his inclusive fitness. Inclusive fitness may be defined as that property of an individual organism which will appear to be maximized when what is really being maximized is gene survival. This is not his own definition, but Dr. HAMILTON allows me to say that it is the ideal inclusive fitness to which his actual concept was an approximation. Table 1 shows the two equivalent ways of expressing what happens in natural selection.

Table 1

'Unit of selection'	Quantity maximized
Individual	Inclusive fitness
Gene	Replication

For different purposes it is convenient to use sometimes the individual/inclusive-fitness formulation, sometimes the gene/replication formulation. We should become adept at translating rapidly between the two. Unfortunately,

some serious mistranslations have appeared in the literature. Since, for reasons which I have given at length (DAWKINS 1976a, following WILLIAMS 1966, pp. 22—25), I believe that the gene/replication formulation is to be preferred when there is any apparent conflict, I am prepared to say that these mistranslations have led their authors into actual biological error. Readers who do not accept this preference for replicator selection rather than 'individual selection' may at least agree that the following examples demonstrate confusion. As for outright error, the only disagreement should be over whether it is mine or the authors' whom I quote.

Confusion

It is an important part of my case that the concept of individual fitness has proved itself to be actively misleading. It is therefore necessary that I demonstrate from the literature that people have been misled. I do not intend this in a carping or ungracious spirit. My case is against a fashionable concept, and the more distinguished the authors who have been misled, the stronger the indictment against the concept.

The ordinary everyday usage of 'fitness' is so deeply ingrained that the special neo-Darwinian meaning is hard to get used to. Here is a distinguished American ecologist writing as recently as 1960. He first quoted WADDINGTON's (1957) definition of survival in the modern sense: "... survival does not, of course mean the bodily endurance of a single individual ... That individual 'survives' best which leaves most offspring." Then the eminent ecologist goes on: "Critical data on this contention are difficult to find, and it is likely that much new investigation is needed before the point is either verified or refuted." He apparently thought that WADDINGTON was making a statement of fact about survival, whereas WADDINGTON was really *defining* survival in the new sense of individual fitness. No wonder this poor ecologist had such trouble grappling with mammary glands: "It would be extremely difficult to explain the evolution of the uterus and mammary glands in mammals ... as the result of natural selection of the fittest individual." He goes on to recommend a group-selectionist interpretation. I think it would be discourteous to regard his confusion as anything but a black mark against the concept of individual fitness.

Here is another example of the trouble which can result from carelessly looking at adaptation in terms of individual benefit. It is often pointed out that some coefficients of relationship are exact while others are probabilistic. For instance the coefficient between brothers is $1/2$, but this "is an average figure: by the luck of the meiotic draw, it is possible for particular pairs of brothers to share more or fewer genes than this. The relatedness between parent and child is always exactly $1/2$" (DAWKINS 1976a, p. 98). GIBSON (1976) correctly stated this point, but then went on to draw an incorrect inference. She supposed that an adult might invest in a son rather than in a full sibling because nature might prefer "a sure thing (relatedness = 0.5 as in the case of the son) to gambling (average relatedness = 0.5 as in the case of the siblings)". But only an individual could see the son as a 'sure thing'. From the point of view of a single gene determining parental or brotherly behaviour, the son is no more a sure thing than the brother: both are gambles with 50 % odds (DAWKINS (1976b).

FAGEN (1976) made a similar mistake in the course of worrying about something called the 'doting grandparent problem'. The number of a grandparent's genes inherited by a given grandchild is $1/4$, but only *on average*.

Some grandchildren will inherit more than 1/4 of the grandparent's genome, others less. So, the author reasoned, "grandparents should tend to detect and favour those grandchildren having a disproportionate number of grandparental genes ... physical resemblance of grandchildren to grandparents should serve as an important releaser of doting (and is expected to lead to endless discussions of 'grandpa's chin' or 'grandma's eyes')." The fallacy is again easily seen. What matters is the replication of the gene or genes which make for doting. As PARTRIDGE and NUNNEY (1977) have pointed out, unless there is genetic linkage between genes for chins and genes for doting, grandpa should behave as if completely indifferent to whether any given grandchild has inherited his chin. In practice, linkage effects and uncertainty about whether an individual is a grandchild at all could lead to FAGEN's being right, but if so it would be for the wrong reason. FAGEN, like GIBSON, was misled by the following mathematical equivalence. The coefficient of relationship between two relatives is equivalent to two things. It is the average *proportion* of the genome of one which is shared by the other. It is also the *probability* that a given gene in one will be identical by descent with one in the relative. What matters is this probability. The proportion is merely incidentally equivalent, but all too often it is what people think in terms of.

I am grateful to L. PARTRIDGE for calling my attention to the last example, and to P. J. GREENE for showing me yet a third example of the same error in a paper devoted to 'exact versus probabilistic coefficients of relationship' (BARASH et al. 1978). In this paper, FAGEN's fallacy is repeated, but in a more stark and general form. More general because it makes the same point about relationships other than the grandparental one, and more stark because here it is not possible to save the argument by special pleading about linkage, pleiotropy, or detectability of relationship. Thus FAGEN could defend grandpa's chin by pointing out that it could help grandpa to decide whether a particular child was really his grandchild at all, or by suggesting that the genes controlling facial appearance might be linked to the genes for grandparental altruism. BARASH et al. have no such defence, since they were specifically concerned to emphasize the difference between exact and probabilistic coefficients of relationship, and the arguments about linkage etc. apply regardless of this distinction.

Now I want to mention a more subtle and important source of misunderstanding resulting from the individual fitness point of view. I refer to the so-called cost of meiosis. WILLIAMS (1971) has put it like this: "Suppose there were two kinds of females in a population; one produced monoploid, fertilizable eggs, and the other ... diploid eggs ... each with exactly the mother's genetic make-up. These parthenogenetic eggs would each contain twice as much of the mother's genotype as is present in a reduced and fertilized egg. Other things being equal, the parthenogenetic female would be twice as well represented in the next generation as the normal one. In a few generations, meiosis and sexual recombination should disappear ... Meiosis is therefore a way in which an individual actively reduces its genetic representation in its own offspring ... Sexual reproduction is analogous to a roulette game in which the player throws away half his chips at each spin."

It is with particular diffidence that I criticize a quotation from one of DARWIN's foremost heirs. I believe WILLIAMS's expression of a cost of meiosis is misleading because the important question is not what happens to the whole genome of a female, but what happens to the gene or genes determining sexuality versus asexuality (TREISMAN and DAWKINS 1976). By the way, to

avoid a mistake which has already appeared in the literature (BARASH 1976), I must hasten to agree with MAYNARD SMITH and WILLIAMS (1976) that this does not mean there is *no* cost of sex. WILLIAMS (1975) is right to stress that the existence of sexual reproduction really is a huge paradox, but it is not the same paradox as he originally said. A better expression of the true nature of the paradox is that of MAYNARD SMITH (1971), but Professor MAYNARD SMITH will agree with me that TRIVERS's (1976) way of explaining it is easier to understand. The true cost of sex is an economic cost resulting from the fact that fathers usually do not invest as much in their children as mothers do. Elsewhere I have gone into the nature of this cost, which I call the cost of paternal neglect (DAWKINS 1978), and which might more generally be called the cost of anisogamy. All that is relevant here is that it is different from WILLIAMS's cost of meiosis. We are misled into the formulation of a cost of meiosis because, once again, of the habit of thinking about individual fitness (genome survival) rather than gene survival.

Table 2: Probability that a gene on a particular type of chromosome (row titles) will be identical by descent to a gene in a relative (column titles). Male sex assumed heterogametic; if female heterogametic, reverse sex titles

	Sex	Chromosome	Brother	Sister	Father or son	Mother or daughter
Normal Diploid	♀	X	1/4	3/4	1/2	1/2
	♂	X	1/2	1/2	0	1
		Y	1	0	1	0
	Either	Autosome	1/2	1/2	1/2	1/2
Haplodiploid	♀	Any	1/4	3/4	1/2	1/2
	♂	Any	1/2	1/2	-	1

Here is an amusing little idea which would not occur to somebody who thought in terms of genome preservation rather than gene preservation. HAMILTON (1972) pointed out that, as far as a gene on an X-chromosome was concerned, its probability of being shared by two siblings of the homogametic sex was 3/4, not the usual 1/2 (see Table 2). For instance, in birds, a gene for brother to brother altruism, if it happened to be on an X-chromosome, should be favoured by the same strong selection pressure as would favour a gene for sister to sister altruism in a haplodiploid hymenopterous insect. This could favour the evolution of helping at the nest by elder brothers. HAMILTON modestly considered his idea too far-fetched to merit more than a paragraph, but it has recently been rediscovered and expounded at greater length (WHITNEY 1976) as has a Y-chromosome version of the same idea (WICKLER 1977). The 'green beard effect' (DAWKINS 1976a, p. 96) represents an extreme of this way of thinking. All these ideas, even if they appear far-fetched in practice, are perfectly respectable in theory, and you would never think of them if you based your ideas on individual fitness rather than on gene replication.

One of the most pernicious consequences of the 'individual selection' viewpoint is the notion that explanations in terms of 'kin selection' are somehow unparsimonious. ZAHAVI (1975) says of one of his own entertaining theories: "Such an interpretation may provide an alternative to other hypotheses which assumed complicated selective mechanisms, such as group selection or kin selection which do not act directly on the individual". When he says 'act directly on the individual' he must mean individual reproductive success, i.e., number of children and lineal descendants. He is implicitly using 'kin' to

refer to relatives *other* than offspring. WILSON (1975) incorporates this odd usage into an explicit definition, as I have criticized elsewhere (DAWKINS 1976a, p. 102). WOOLFENDEN (1975) similarly mars his discussion of Florida scrub jays helping at the nest by speaking of a 'controversy about group or kin selection versus individual selection'. The literature contains many efforts to explain facts in terms of 'individual selection' without having to 'resort' to kin selection. Of course 'resort' is an entirely inappropriate verb. 'Kin selection' is not a distinct kind of natural selection, to be invoked only when 'individual selection' cannot explain the facts. Both kin selection and individual selection are logical consequences of gene selection. If we accept neo-Darwinian gene-selectionism, kin selection necessarily follows. There is, indeed, no need for the term kin selection to exist, and I suggest that we stop using it.

We round off this section, as we began it, with mammary glands. "... mammary glands contribute to individual fitness, the individual in this case being the kinship group" (HULL 1976). WILSON (1975) goes so far as to define kin selection as a special case of group selection. But there is no 'kinship group' unless families happen to go around together — an incidental fact, not a necessary assumption. Individuals do not, in an all or none sense, either qualify or fail to qualify as kin. They have, quantitatively, a greater or less chance of containing a particular gene. If HULL *must* talk about individuals, the post-HAMILTON 'individual' in his sentence is certainly not a group. It is an animal plus $^1/_2$ of each of its children plus $^1/_2$ of each sibling plus $^1/_4$ of each niece and grandchild plus $^1/_8$ of each first cousin plus $^1/_{32}$ of each second cousin ... Far from being a tidy, discrete group, it is more like a sort of genetical octopus, a probabilistic amoeboid whose pseudopodia ramify and dissolve away into the common gene pool. We have reached the Darwinian equivalent of the Ptolemaic epicycles. It is time to go back to first principles. What really happens in natural selection?

Replicator Selection

We may define a *replicator* as any entity in the universe which interacts with its world, including other replicators, in such a way that copies of itself are made. A corollary of the definition is that at least some of these copies, in their turn, serve as replicators, so that a replicator is, at least potentially, an ancestor of an indefinitely long line of identical descendant replicators. In practice no replication process is infallible, and defects in a replicator will tend to be passed on to descendants. If a replicator exerts some power over the world, such that its nature influences the survival of itself and its copies, natural selection, and hence progressive evolution, may occur through differential replicator survival.

A DNA molecule is the obvious replicator. The mistakes which are made in its replication are the various kinds of gene mutation and also, since multicistron fragments of chromosome can qualify as replicators (see below), crossing over. The power which a gene exerts over its world is its influence on the synthesis of proteins which in turn influence the embryonic development of phenotypes. Since the gene rides inside the body whose development it influenced, its own long-term future is affected by its nature.

BATESON (1978) has criticised the view that an animal is the genes' way of making more genes by drawing an analogy which appears to reduce the

idea to an absurdity. Birds build nests, and nests protect new growing birds. So you might as well say that a bird is a nest's way of making new nests! But BATESON's amusing analogy is a false one. A nest is not a true replicator because a 'mutation' which occurs in the construction of a nest, for example the accidental incorporation of a pine needle instead of the usual grass, is not perpetuated in future 'generations of nests'. Similarly, protein molecules are not replicators, nor is messenger RNA.

A gene in the nucleus of a germ-line cell is a replicator, but a sexually reproducing individual organism is not. It does not make copies of itself. It propagates copies of its genes, but its genome is shredded to smithereens at meiosis. Because individual bodies are big things that we can watch moving about in apparently purposeful ways, we focus our attention on them. We forget the lesson of August WEISMANN: organisms are but the transient engines of long-term gene replication. The qualities of a good replicator may be summed up in a slogan reminiscent of the French Revolution: Longevity, Fecundity, Fidelity (DAWKINS 1976a, 1978). Genes are capable of prodigious feats of fecundity and fidelity. In the form of copies of itself, a single gene may persist for a hundred million individual lifetimes. Some genes survive better than their alleles, which is what natural selection is all about. But neither individual organisms, whose copying fidelity is destroyed by meiosis, nor groups of individuals for similar reasons, deserve to be called replicators at all.

Why 'replicator selection' rather than 'gene selection'? One reason for preferring replicator selection is that the phrase automatically pre-adapts our language to cope with non-DNA-based forms of evolution such as may be encountered on other planets, and perhaps also cultural analogues of evolution (DAWKINS 1976a, pp. 203—215). The term replicator should be understood to *include* genetic replicators, but not to exclude any entity in the universe which qualifies under the criteria listed above.

The other reason for avoiding 'gene selection' is that we must not be forced into the position of saying that the single gene, in the narrow molecular biologists' sense of cistron, is the unit of selection. The problem of what fragments of genome should be regarded as units of selection is discussed from time to time in the mathematical genetics literature. The details are complicated and yet to be finally resolved (LEWONTIN 1974), but whatever conclusions the geneticists come up with are of great importance to those of us who want to talk about adaptation. Here is one opinion:

"It is clear that when permanent linkage disequilibrium is maintained in a population, the higher order interactions are important and the chromosome tends to act as a unit. The degree to which this is true in any given system is a measure of whether the gene or the chromosome is the unit of selection, or, more accurately, what parts of the genome can be said to be acting in unison" (SLATKIN 1972).

Very well, if the geneticist says the chromosome functions as the unit of selection, so be it. The implication for whole-animal biology is that under these conditions adaptations might be interpreted as 'for the good of the chromosome'. This will not always be so, as SLATKIN indicates, and as TEMPLETON, SING and BROKAW (1976) put it:

"... the unit of selection is a function in part of the intensity of selection: the more intense the selection, the more the whole genome tends to hold together as a unit ... Thus selection under a broad range of conditions seems to preferentially operate upon linked blocks of genes."

So! Adaptation is sometimes for the good of the linked block of genes. By using the flexible word *replicator,* we can safely say that adaptation is for the good of the replicator, and leave it open exactly how large a chunk of genetic material we are talking about. One thing we can be sure of is that, except in special circumstances like asexual reproduction, the *individual organism* is not a replicator.

It is my contention that we should reserve the phrase 'unit of selection' for replicators, that is for entities which become either more or less numerous in the world as a result of selection. Replicators exert power over their world, and it so happens that, in the forms of life with which we are familiar, groups of replicators are to be found exerting this power via relatively discrete entities which we call individuals. Because these entities have a high degree of autonomy of behaviour and unity of structure, we are tempted to see them as the units of selection. But, for the reasons which we have seen, at least where reproduction is sexual, this is misleading. Individual bodies are units of replicator power. They are not replicators.

It is a remarkable fact that natural selection seems to have chosen those replicators that co-operate with each other, and go around in the large collective packages which we see as individual organisms. This is a fact that needs explaining in its own right, just as the existence of sexual reproduction needs explaining in its own right. Such extreme 'gregariousness' of replicators may not be true of life all over the universe, just as it probably was not true of the earliest forms of life on earth. I will not discuss it here, but I have the hunch that something like game theory may be the right way to think about interactions between replicators. MAYNARD SMITH (1974) has the right idea, but he should increase the time he spends on replicator games rather than individual games (DAWKINS 1976 a, pp. 91—93).

It has to be admitted that many biologists find attempts to dethrone the individual as the 'unit of selection' unsatisfactory. At one level this shows itself as a kind of gut reaction: "What you say is all very well in theory. But when I am out in the field what I actually see is individuals. I don't see a gene pool, I see animals. Each one has four legs, two eyes, and a skin round it. Each one has its own nervous system, and it behaves like a single coherent entity, as if it had a single goal, not like a sort of federal democracy of replicators." At a more profound level, no less biologists than Ernst MAYR (1963) and E. B. FORD (1975) have poured scorn on the idea of the gene, rather than the individual, as the unit of selection. Incidentally, MAYR's attack had the additional merit of provoking a splendidly spirited 'defence of beanbag genetics' from J. B. S. HALDANE (1964). I really do think the argument is based on a misunderstanding. I have no trouble at all in enthusiastically endorsing all MAYR's eloquently expressed views on the unity of the genome. Of course it is true that the phenotypic effect of a gene is a meaningless concept outside the context of many, or even all, of the other genes in the genome. Yet, however complex and intricate the organism may be, however much we may agree that the organism is a unit of *function,* I still think it is misleading to call it a unit of *selection.* Genes may interact, even 'blend', in their effects on embryonic development, as much as you please. But they do not blend when it comes to being passed on to future generations. I am not trying to belittle the importance of the individual phenotype in evolution. I am merely trying to sort out exactly what its role is. It is the all important instrument of replicator preservation: it is *not* that which is preserved.

Hesitantly, I will go further. It may be that, even in its role as the unit of gene action, the importance of the single individual phenotype has been exaggerated. If the word phenotype is defined physiologically, it is of course true that the phenotypic expression of a gene is confined to the one body in which it sits. But if we focus our interest on adaptation, and regard the 'phenotypic expression' of a gene as the power for its own preservation which it exerts over its surroundings, we are led to extend our view of what the word 'phenotype' should mean.

The Extended Phenotype

There is a hidden assumption running right through the whole idea of individual and inclusive fitness. This is that the individual, to the extent that it behaves in the best interests of *anybody's* genes, behaves in the best interests of its own (even if this means copies of its own genes in other individuals). There are rare cases of authors departing from this assumption. For instance ALEXANDER (1974), in his theory of parental manipulation, suggested that offspring should be expected to behave in the best interests of their parents' genes rather than their own. I have argued that ALEXANDER's main reason for expecting this is false (DAWKINS 1976a, pp. 145—149), and my verbal criticism has been confirmed in a mathematical model by PARKER and MACNAIR (1978). But although our refutation of ALEXANDER was justified within the framework of ordinary replicator selection theory, ALEXANDER's idea starts to look a lot more exciting within the framework of the extended phenotype, which I am now about to lay out. I begin with an example.

In the snail *Limnaea peregra*, the direction of coiling of the shell is controlled at a single locus. It is a classic case of simple Mendelian inheritance, right-handed coiling being straightforwardly dominant to left-handed coiling. Classic and simple except in one remarkable respect: control is exerted not by the individual's own genotype, but by its mother's. As FORD (1975) puts it: "We have here simple Mendelian inheritance the expression of which is constantly delayed one generation. It was long ago suggested that this phenomenon may be a widespread one controlling the early cleavage of the embryo until its own genes can take charge."

So, a gene can find phenotypic expression not in its own body but in a body of the next generation. This is a particular example of the concept of the extended phenotype. Here the route of the influence is presumably maternal cytoplasm, and other such 'maternal effects' are known. But I want to apply the idea not just to mother and child but to influences on other members of the species, members of other species, even inanimate objects. If we can do this convincingly we shall no longer be justified in regarding an individual as a machine programmed to preserve its *own* genes. It may be programmed to preserve somebody else's genes!

For didactic reasons I use examples which extend the idea of the phenotype gradually outwards in stages. Caddis larvae live in houses which they themselves build out of stones, twigs, or some other material. The form of the house is determined by the behaviour of the builder, and this in turn is presumably influenced by the builder's genes. The evolution of caddis houses came about through ordinary replicator selection — gene selection. There is nothing difficult about a genetics of caddis houses. All the ordinary genetic terms, dominance, epistasis, etc., would be perfectly applicable to traits such as stone colour or stick length. Each gene exerts its influence via building

behaviour, of course, and before that via control of protein synthesis. When I say the stones of houses are part of the phenotypic expression of genes, all I have done is to add one, rather minor, link to the end of an already long and complicated embryonic causal chain. Strictly speaking it is *differences* in houses that are controlled by differences in genes, but differences, in any case, are what geneticists study.

It is easy to see a caddis house as part of the phenotype of genes, because the genes ride inside that house. It is the outer fortification of the body which they helped to build for themselves. It just happens to be made of stone rather than skin. The fates of the genes that built it are bound up inside the house that they built, just as in an ordinary body made of cells rather than stone. It is also easy to imagine a genetic account of variation in bower-bird bowers. The genes for bower building do not ride inside their bower. Nevertheless, their chances of being passed on to the next generation may depend critically on the success of the bower in attracting females. The bower is part of the phenotypic expression of genes in the bird, and the success of the genes as replicators depends on their effects on the bower. So, we have seen that the phenotypic expression of a gene may extend to inanimate objects, and it may also extend outside the body in which it sits.

The genes of parasites do not 'build' the body of the host, but they can manipulate it. There is a large and interesting literature on parasites which influence the behaviour of the hosts in which they ride (HOLMES and BETHEL 1972). Sporocysts of flukes of the genus *Leucochloridium* invade the tentacles of snails where they can be seen conspicuously pulsating through the snail's skin. This tends to make birds, who are the next host in the life cycle of the fluke, bite off the tentacles, mistaking them, WICKLER (1968) suggests, for insects. What is interesting here is that the flukes seem to manipulate the behaviour of the snails. The normal negative phototaxis is replaced in infected snails by positive light-seeking. This probably carries them up to open sites where they are more likely to be eaten by birds, and this benefits the fluke.

I have so far used conventional 'individual level' language to describe this parasitic adaptation. The individual fluke is said to manipulate the behaviour of the individual snail for its own individual advantage. But now I want to rephrase it in replicator language, in this case gene language. A mutation in the fluke can be said to have phenotypic expression in the snail's body — it changes the snail's behaviour. The route of this phenotypic expression is tortuous and indirect, but not more so than the normal embryological details of phenotypic expression in a gene's 'own' body. We are quite accustomed to the idea that genes are selected for their distantly ramified phenotypic effects on their *own* body. I am saying that they may also be selected for their distantly ramified phenotypic effects on *other* bodies.

Now of course selection also acts on hosts to make them resist manipulation by parasites. We expect counteradaptations on the part of snails. Let us again move from the language of individuals to the language of replicators. Suppose a mutation arises in snails which restores negative phototaxis even in the presence of a manipulating fluke, counteracting the tendency of the fluke gene to produce positive phototaxis. Both genes are acting on the same phenotype — the snail phenotype. They are pushing it in opposite directions but, once again, this is nothing new. We are already familiar with the idea of conflict between genes *within* a single body. This is often discussed in terms of so-called 'modifier' genes. Any gene may modify the phenotypic expression of any other gene in the genome. A deleterious mutation is subject not

only to direct selection against itself. There may also be selection on other, modifier, genes, to reduce the phenotypic effects of the deleterious gene.

For instance, imagine a mutant gene on a mammalian Y-chromosome. The argument would work if it was an ordinary segregation distorter (HAMILTON 1967), but since it is a hypothetical gene we may dramatize its properties a little. Any individual possessing this hypothetical gene kills his own daughters and feeds them to his sons. The death of the daughters is of no consequence to the rogue Y mutant, since they never contain it. On the other hand the sons all contain it, so the rogue Y gene will tend to spread very rapidly, and it might incidentally lead to the extinction of the whole population. But suppose modifiers arise on other chromosomes. These tend to neutralise the phenotypic expression of the rogue Y gene. The modifiers are carried not only by males themselves, but also by half the females whose lives they save. Depending on circumstances, such modifiers might therefore spread through the gene pool. HAMILTON (1967) has suggested that something like this may be why so few genes on Y-chromosomes seem to have any detectable phenotypic expression. From our point of view the message is this: there can be conflicts of interest between the replicators in one individual's genome, and among the weapons at the disposal of a replicator is the modification of the phenotypic expression of another replicator. Now we can return to the fluke and the snail. The conflict between fluke genes and snail genes is no different from the conflict between genes within a single individual. In both cases the genes are struggling for *power over the phenotype*. In both cases they *modify* the phenotypic expression of other genes.

Fluke genes and snail genes ride inside the same body (though not inside the same cells). But, just as bower birds do not live inside their bowers, so parasites do not have to live inside their hosts. A cuckoo nestling manipulates the behaviour of its foster mother. Once again I now switch from individual language to replicator language. If a mutation arises in a cuckoo which brightens the colour of its gape so that it acts as a supernormal stimulus to a foster mother, the gene may be positively selected. The change in the behaviour of the foster mother is properly regarded as part of the phenotypic expression of the cuckoo gene. The parental behaviour of the foster mother is under the influence of many genes. Some of them are in her own body; some of them are in the cuckoo's body. They are struggling to push her behaviour in opposite directions. If a mutant arises in the host gene pool which causes individuals to stop treating bright gapes as supernormal, such a counteradapting mutation might be selected. I would call it a *modifier* of the cuckoo gene's phenotypic effects.

What we are talking about is *power*, replicator power. Those replicators survive which exert power over their world which leads to their own survival. Phenotypic expression is the name we are giving to the power of genes over their world and their future. The power of a gene within the body in which it sits is very considerable. Direct biochemical channels of power are available to it. No wonder we have got used to the idea that the phenotypic expression of a gene comes to an end at the wall of the individual body. Indeed, this makes very good sense if we are interested in physiological mechanisms in embryology. But if we are interested in adaptation, the logical conclusion to what I have been saying is that the whole world is potentially part of the phenotypic expression of a gene. It is only in practice that the power of a gene is limited to its immediate neighbourhood. Maybe we have underestimated the extended power of replicators.

The routes of power in the extended phenotype are less purely biochemical than the routes of power in the conventional local phenotype. In the extended phenotype we must look to *behaviour* rather than biochemistry. The study of animal communication turns out to be a branch of extended embryology. The same may be said of relationships between parasites and hosts, predators and prey, indeed it may be said of most of ecology. Bird song is the way it is because selection has acted on the distant phenotypic effects of genes in singing males: effects on the behaviour of rivals and females (DAWKINS and KREBS 1978). The peacock's tail is not the terminal phenotypic expression of the peacock's genes. It is only a way-station on the route to a more distant phenotypic expression in female behaviour. Genes in orchids express themselves phenotypically in the form of changes in bee behaviour, which result in the successful transference of pollen grains containing those same genes.

I end with a little flight of fancy through the ways of the extended phenotype. What is it about termites that led them to evolve eusociality? They are not haplodiploid, so that good old explanation won't do. HAMILTON's (1972) inbreeding theory seems plausible enough. Other theories have invoked the termites' need to congregate in order to infect themselves with symbiotic protozoa. But, for the sake of argument, let us use the protozoa in the service of a very different idea. The symbionts in a termite colony are usually an identical clone. They are very numerous, and may constitute up to a third of each individual termite's body weight (RIETSCHEL and ROHDE 1974). They would seem to be in an excellent position to manipulate their host's physiology. Who knows, perhaps it is the protozoan genes that are really running the termite nest, exerting phenotypic power over the behaviour of the termites, sterilizing the workers, making them behave eusocially.

To conclude: the replicator is the unit of selection. Adaptations are for the benefit of replicators. Individuals are manifestations of the power wielded by replicators over the world in which they live. The individual body is a convenient practical unit of combined replicator power. But we must not be misled by this parochial detail. In the light of the doctrine of the extended phenotype, the conceptual barrier of the individual body wall dissolves. We see the world as a melting pot of replicators, selected for their power to manipulate the world to their own long-term advantage. Individuals and societies are by-products.

Summary

'Sociobiology', in the sense in which the word has come to be used, may be defined as the branch of ethology inspired by W. D. HAMILTON. The time has come to carry his 'selfish gene' revolution to its conclusion, and give up the habit of speaking of adaptation at the individual level. Group selection, kin selection, individual selection, all may be swept away and replaced by *replicator selection*. Inclusive fitness is that property of an individual organism which will appear to be maximized when what is really being maximized is gene survival. The language of individual inclusive fitness is directly interchangeable with the language of gene replication, and it pays to learn to translate rapidly between the two languages. Examples are given of mistranslations in the literature. These have led to actual biological error, and the inherent confusingness of the concept of individual fitness is blamed. All remains clear if we stick to the language of replication. Genes are not the

only conceivable replicators, and some general properties of replicators are listed. Sexually reproducing individuals are definitely not replicators. Units of genetic material larger than cistrons may be. In general, adaptations should be thought of, not as for the good of the species, nor as for the good of the individual, but as for the good of the replicator. The last part of the paper develops the doctrine of the *extended phenotype*. Replicators such as genes manipulate their surroundings to their own advantage. Manifestations of such manipulation are called phenotypic. Conventionally, the phenotypic expression of a gene is considered to be limited to the individual body in whose cells it resides. If we are interested in physiological mechanisms this makes sense. But if we are interested in adaptation it pays to make an imaginative leap and see the phenotypic expression of a gene as extending outside the individual body wall. The study of animal communication, and most of ecology, turn out to be branches of extended embryology.

Zusammenfassung

Nach dem heutigen Sprachgebrauch kann man „Soziobiologie" als denjenigen Zweig der Ethologie definieren, der auf den Arbeiten von W. D. HAMILTON beruht. Es ist an der Zeit, die logische Konsequenz aus HAMILTONS Ideen zu ziehen: Wenn wir das Konzept vom „egoistischen Gen" akzeptieren, müssen wir uns abgewöhnen, den Begriff der Anpassung auf dem individuellen Niveau zu verwenden. Alle Vorstellungen von einer Selektion, die auf der Ebene der Gruppe, der Verwandtschaft („kin selection") oder des Individuums angreift, können über Bord geworfen werden; sie werden ersetzt durch die Vorstellung der Replikator-Selektion. Die Gesamt-Fitness („inclusive fitness") ist jene Eigenschaft eines einzelnen Organismus, die scheinbar maximiert wird, wenn es in Wirklichkeit auf das Überleben des Gens ankommt. Die Sprache, die im Zusammenhang mit der individuellen Gesamt-Fitness verwendet wird, ist unmittelbar gegen die Sprache der Genreplikation austauschbar; es lohnt sich, übersetzen zu lernen. Beispiele falscher Übersetzungen aus der Literatur werden aufgeführt. Solche Fehlübersetzungen haben zu biologischen Fehlern geführt, und Schuld daran ist die Tatsache, daß das Konzept der individuellen Fitness an sich verwirrend ist. Wenn wir konsequent die Sprache der Replikation beibehalten, ist alles klar. Gene sind nicht die einzigen Replikatoren, die man sich vorstellen kann; einige allgemeine Eigenschaften von Replikatoren werden aufgeführt. Individuen, die sich sexuell fortpflanzen, sind mit Sicherheit keine Replikatoren; Einheiten genetischen Materials, die mindestens ein Cistron umfassen, können es unter Umständen sein. Allgemein darf man den Vorteil einer Anpassung nicht in ihrer Wirkung auf die Art oder das Individuum suchen, sondern in ihrer Wirkung auf den Replikator.

Im letzten Abschnitt der Arbeit wird die These vom *erweiterten Phänotyp* vorgestellt. Replikatoren, z. B. Gene, manipulieren ihre Umwelt zu ihrem eigenen Vorteil. Der Effekt solcher Manipulation wird als phänotypische Ausprägung bezeichnet. Üblicherweise stellt man sich vor, daß sich ein Gen nur in demjenigen individuellen Körper phänotypisch ausprägt, in dessen Zellen es sitzt. Diese Betrachtungsweise ist dann sinnvoll, wenn wir an physiologischen Mechanismen interessiert sind. Interessieren wir uns jedoch für Anpassungen, so lohnt sich ein unkonventioneller Gedankengang: Man stelle sich vor, daß die phänotypische Ausprägung eines Gens über den Körper des Individuums

hinausreicht. Das Studium tierischer Kommunikation und der größte Teil der Ökologie erweisen sich dann als Teilgebiete einer erweiterten Embryologie.

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