3

Replicators and vehicles

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The theory of natural selection provides a mechanistic, causal account of how living things came to look as if they had been designed for a purpose. So overwhelming is the appearance of purposeful design that, even in this Darwinian era when we know 'better', we still find it difficult, indeed boringly pedantic, to refrain from teleological language when discussing adaptation. Birds' wings are obviously 'for' flying, spider webs are for catching insects, chlorophyll molecules are for photosynthesis, DNA molecules are for ... What *are* DNA molecules for? The question takes us aback. In my case it touches off an almost audible alarm siren in the mind. If we accept the view of life that I wish to espouse, it is the forbidden question. DNA is not 'for' anything. If we wish to speak teleologically, all adaptations are for the preservation of DNA; DNA itself just *is*. Following Williams (1966), I have advocated this view at length (Dawkins 1976, 1978), and I do not want to repeat myself here. Instead I shall try to clear up an important misunderstanding of the view, a misunderstanding which has constituted an unnecessary barrier to its acceptance.

The identity of the 'unit' of selection' has been controversial in the literature both of biology (Williams, 1966; Lewontin, 1970; Leigh, 1977; Dawkins, 1978; Alexander & Borgia, 1978; Alexander, 1980) and philosophy (Hull, 1981). In this paper I shall show that only part of the controversy is real. Part is due to semantic confusion. If we overlook the semantic element we arrive at a simplistic hierarchical account of the views that have been expressed in the literature. Living matter is nested in a hierarchy of levels, from ecosystem through species, deme, family, individual, cell, gene, and even nucleotide base pair. According to this analysis each one of the protagonists in the debate on units of selection is perched on a higher or a lower rung of a ladder, sniping at those above and below him. Thus Gould (1977) remarks that in the last fifteen years

> 'challenges to Darwin's focus on individuals have sparked some lively debates among evolutionists. These challenges have come from above and from below. From above, Scottish biologist V.C. Wynne-Edwards raised orthodox hackles fifteen years ago by arguing that groups, not individuals, are units of selection, at least for the evolution of social behavior. From below, English biologist Richard Dawkins has recently raised my hackles with his claim that genes themselves are units of selection, and individuals merely their temporary receptacles.'

At first blush, Gould's hierarchical analysis has a neatly symmetrical plausibility. Much as my sense of mischief is tickled by the idea of being allied with Professor Wynne-Edwards in a pincer-attack on Darwin's individual, however, I reluctantly have to point out that the dispute between individual and group is different in kind from the dispute between individual and gene. Wynne-Edwards's attack from above is best seen as a factual dispute about the level at which selection is most effective in nature. My attack from below is a dispute about what we ought to *mean* when we talk about a unit of selection. Much the same point has been realised by Hull (1981), but I prefer to persist in expressing it in my way rather than to adopt his terminology of 'interactors' and 'evolvors'.

To make my point I shall develop a distinction between *replicator survival* and *vehicle selection*. Anticipating the conclusion, there are two ways in which we can characterise natural selection. Both are correct; they simply focus on different aspects of the same process. Evolution results from the differential survival of *replicators*. Genes are replicators; organisms and groups of organisms are not replicators, they are vehicles in which replicators travel about. Vehicle selection is the process by which some vehicles are more successful than other vehicles in ensuring the survival of their replicators. The controversy about group selection versus individual selection is a controversy about the rival claims of two suggested kinds of vehicle. The controversy about gene selection versus individual (or group) selection has been a controversy about whether, when we talk about a unit of selection, we ought to mean a vehicle *at all*, or a replicator. In any case, as I shall later argue, there may be little usefulness in talking about discrete vehicles at all.

Replicators

A replicator may be defined as any entity in the universe of which copies are made. Replicators may be subclassified in two overlapping ways (Dawkins, 1982, chapter 5). A germ-line replicator, as distinct from a dead-end replicator, is the potential ancestor of an indefinitely long line of descendant replicators. Thus DNA in a zygote is a germ-line replicator, while DNA in a liver cell is a dead-end replicator. Cutting across this

classification, an active, as distinct from a passive, replicator is a replicator that has some causal influence on its own probability of being propagated. Thus a gene that has phenotypic expression is an active replicator. A length of DNA that is never transcribed and has no phenotypic expression whatever is a passive replicator. 'Selfish DNA' (Dawkins, 1976, p. 47; Doolittle & Sapienza, 1980; Orgel & Crick, 1980), even if it is not transcribed, should be considered passive only if its nature has absolutely no influence on its probability of being replicated. It might be quite hard to find a genuine example of a passive replicator. Special interest attaches to *active germ-line replicators*, since adaptations 'for' their preservation are expected to fill the world and to characterise living organisms. Automatically, those active germ-line replicators whose phenotypic effects happen to enhance their own survival and propagation will be the ones that survive. Their phenotypic consequences will be the attributes of living things that we see, and seek to explain.

Active, germ-line replicators, then, are units of selection in the following sense. When we say that an adaptation is 'for the good of' something, what is that something? Is it the species, the group, the individual, or what? I am suggesting that the appropriate 'something', the 'unit of selection' in that sense, is the active germ-line replicator. The active germ-line replicator might, therefore, be called the 'optimon', by extension of Benzer's (1957) classification of genetic units (recon, muton and cistron).

This does not mean, of course, that genes or other replicators literally face the cutting edge of natural selection. It is their phenotypic effects that are the proximal subjects of selection. I have been sorry to learn that the phrase 'replicator selection' can be misunderstood along those lines. One could, perhaps, avoid this confusion by referring to replicator survival rather than replicator selection. (In passing I cannot help being reminded of Wallace's (1866) passionate plea to Darwin to abandon 'natural selection' in favour of 'survival of the fittest', on the grounds that many people thought 'natural selection' implied a conscious selecting 'agent' (see also Young, 1971). My own prejudice is that anybody who misunderstands 'replicator selection' is likely to have even more trouble with 'individual selection').

Natural selection does not inevitably follow whenever there exist active germ-line replicators. Certain additional assumptions are necessary, but these, in turn, are almost inevitable consequences of the basic definition. Firstly, no copying process is perfect, and we can expect that replicators will sometimes make inexact copies of themselves, the mistakes or mutations being preserved in future descendants. Natural selection, of course,

depends on the variation so created. Secondly, the resources needed to make copies, and to make vehicles for propagating copies, may be presumed to be in limited supply, and replicators may therefore be regarded as in competition with other replicators. In the complicatedly organized environments of eukaryotic cells, each replicator is a competitor specifically of its alleles at its own locus on the chromosomes of the population.

There is a problem over how large or how small a fragment of genome we choose to regard as a replicator. Is it one cistron (recon, muton), one chromosome, one genome, or some intermediate? The answer I have given before, and still stick by, is that we do not need to give a straight answer to the question. Nobody is going to be hanged as a result of our decision. Williams (1966) recognised this when he defined a gene as 'that which segregates and recombines with appreciable frequency' (p. 24), and as 'any hereditary information for which there is a favorable or unfavorable selection bias equal to several or many times its rate of endogenous change' (p. 25). It is clear that we are never going to sell this kind of definition to a generation brought up on the 'one gene one protein' doctrine, which is one reason why I (Dawkins, 1978) have advocated using the word replicator itself, instead of gene in the sense of the Williams definition. Another reason is that 'replicator' is general enough to accommodate the theoretical possibility, which one day may become observed reality, of non-genetic natural selection. For example, it is at least worth discussing the possibility of evolution by differential survival of cultural replicators or 'memes' (Dawkins, 1976; Bonner, 1980), brain structures whose 'phenotypic' manifestation as behaviour or artefact is the basis of their selection.

I have lavished much rhetoric, or irresponsibly purple prose if you prefer, on expounding the view that 'the unit of selection' (I meant it in the sense of replicator, not vehicle) must be a unit that is potentially immortal (Dawkins, 1976, chapter 3), a point which I learned from Williams (1966). Briefly, the rationale is that an entity must have a low rate of spontaneous, endogenous change, if the selective advantage of its phenotypic effects over those of rival ('allelic') entities is to have any significant evolutionary effect. For a replicator such as a small length of chromosome, mutation and crossing over within itself are hazards to its continued replication, in exactly the same sense as are predators and reluctant females. Any arbitrary length of DNA has an expected half-life measured in generations. The world tends to become full of replicators with a long half-life, and therefore full of their phenotypic products. These products are the characteristics of the animals and plants which we see around us. It is these that we

wish to explain. Of those phenotypic products, the ones that we, as whole animal biologists, are particularly interested in are those that we see at the whole animal level, adaptations to avoid predators, to attract females, to secure food economically, and so on. Replicators that tend to make the successive bodies they inhabit good at avoiding predators, attracting females, etc., tend to have long half-lives as a consequence. But if such a replicator has a high probability of internal self-destruction, through mutation in its broad sense, all its virtues at the level of whole animal phenotypes come to naught.

It follows that although any arbitrary length of chromosome can in theory be regarded as a replicator, too long a piece of chromosome will quantitatively disqualify itself as a potential unit of selection, since it will run too high a risk of being split by crossing over in any generation. A replicator worthy of the name, then, is not necessarily as small as one recon, one muton, or one cistron. It is not a discrete, all or none, unit at all, but a segment of chromosome whose length is determined by the strength of the 'whole animal level' selection pressure of interest. As Francis Crick (1979) has written, 'The theory of the "selfish gene" will have to be extended to any stretch of DNA'.

It further follows that critics of the view advocated here cannot score debating points by drawing attention to the existence of within-gene (cistron) crossing over. I am grateful to Mark Ridley for reminding me that most within-gene crossovers are, in any case, indistinguishable in their effects from between-gene crossovers. Obviously, if the gene concerned happens to be homozygous, paired at meiosis with an identical allele, all the material exchanged will be identical, and the effect will be indistinguishable from a crossover at either end of the gene. If the gene is heterozygous, but differs from its allele by only one nucleotide, a within-gene crossover will be indistinguishable in effect from a crossover at one of the two ends of the gene. Only on the rare occasions when the gene differs from its allele in two places, and the crossover occurs between the two places, will a within-gene crossover be distinguishable from a between-gene crossover. The general point is that it does not particularly matter where crossovers occur in relation to cistron boundaries. What matters is where crossovers occur in relation to heterozygous nucleotides. If, for instance, a sequence of six adjacent cistrons happens to be homozygous throughout an entire species, a crossover anywhere within the six will be exactly equivalent to a crossover at either end of the six.

The possibility of widespread linkage disequilibrium, too, does not weaken the case (Clegg, 1978). It simply increases the length of chromo-

somal segment that we expect to behave as a replicator. If, which seems doubtful, linkage disequilibrium is so strong that populations contain 'only a few gametic types' (Lewontin, 1974, p. 312), the effective replicator will be a very large chunk of DNA. When what Lewontin calls l_c, the 'characteristic length' (the distance over which coupling is effective), is only 'a fraction of the chromosome length, each gene is out of linkage equilibrium only with its neighbours but is assorted essentially independently of other genes farther away. The characteristic length is, in some sense, the unit of evolution since genes within it are highly correlated. The concept is a subtle one, however. It does not mean that the genome is broken up into discrete adjacent chunks of length l_c. Every locus is the center of such a correlated segment and evolves in linkage with the genes near it' (Lewontin, 1974, p. 312). In the same spirit, I played with the idea of entitling an earlier work 'The slightly selfish big bit of chromosome and the even more selfish little bit of chromosome' (Dawkins, 1976, p. 35).

I used to think that, in species with asexual reproduction, the whole organism could be thought of as a replicator, but further reflection shows this to be equivalent to the Lamarckian heresy. The asexual organism's *genome* may be considered a replicator, since any alteration in it tends to be preserved. But an alteration in the organism itself is quite likely to have been imprinted from the environment and will not be preserved. It is not replicated. Asexual organisms do not make copies of themselves, they work to make copies of their genomes.

An adaptation is a tool by which the genes that made it have levered themselves through the past, into the present where it demands our explanation. But the tools and levers do not rattle around loose in the world, but come neatly packaged in tool-kits: individual organisms or other vehicles. It is to vehicles that we now turn.

Vehicles

Replicators are not naked genes, though they may have been when life began. Nowadays, most of them are strung along chromosomes, chromosomes are wrapped up in nuclear membrances, and nuclei are enveloped in cytoplasm and enclosed in cell membranes. Cells, in turn, are cloned to form huge assemblages which we know as organisms. Organisms are vehicles for replicators, survival machines as I have called them. But just as we had a nested hierarchy of would-be replicators – small and large fragments of genome – so there is a hierarchy of nested vehicles. Chromosomes and cells are gene vehicles within organisms. In many species, organisms are not dispersed randomly but go around in groups. Multi-species

groups form communities or ecosystems. At any of these levels the concept of vehicle is potentially applicable. Vehicle selection is the differential success of vehicles in propagating the replicators that ride inside them. In theory selection may occur at any level of the hierarchy.

One of the clearest discussions of the levels of selection is that of Lewontin (1970), although his paper, like my own first discussion of the matter (Dawkins, 1976), suffers from its failure to make a clear distinction between replicators and vehicles. Lewontin does not mention the gene as one of the levels in his hierarchy, presumably because he rightly regards it as obvious that it is changes in gene frequency that ultimately matter, whatever level selection may proximally act on. Thus it is easy, and probably largely correct, to interpret his paper as being about levels of *vehicle*. On the other hand, at one point he says the following:

> 'The rate of evolution is limited by the variation in fitness of the units being selected. This has two consequences from the point of view of comparison between levels of selection. First, the rapidity of response to selection depends upon the heritability of differences in fitness between units. The heritability is highest in units where no internal adjustment or reassortment is possible since such units will pass on to their descendent units an unchanged set of information. Thus, cell organelles, haploid organisms, and gametes are levels of selection with a higher heritability than diploid sexual genotypes, since the latter do not perfectly reproduce themselves, but undergo segregation and recombination in the course of their reproduction. In the same way, individuals have a greater heritability than populations and assemblages of species' (Lewontin, 1970, p. 8).

This point makes sense only if the units being referred to are would-be replicators; indeed it is the same point as I made a few pages back. This suggests that Lewontin was not entirely clear over whether he was talking about units of selection in the sense of replicators (entities that become more or less numerous as a consequence of selection) or vehicles (units of phenotypic power of replicators). The same is suggested by the fact that he cites M.B. Williams's (1970) axiomatization of Darwin's theory as indicating that 'the principles can be applied equally to genes, organisms, populations, species, and at opposite ends of the scale, prebiotic molecules and ecosystems'. I would maintain that genes and prebiotic molecules do not belong in the hierarchical list. They are replicators; the rest are vehicles.

An organism is not a replicator, not even a very inefficient replicator with a high probability of endogenous change. An organism's *genome* can be regarded as a replicator (a very poor one if reproduction is sexual), but to treat an organism as a replicator in the same sense as a gene is tantamount to Lamarckism. If you change a replicator, the change will be passed on to its descendants. This is clearly true of genes and genomes. It is not true of organisms, since acquired characteristics are not inherited.

The reason we like to think in terms of vehicle selection is that replicators are not directly visible to natural selection. Gould (1977) put it well, albeit he mistakenly thought he was scoring a point against the whole replicator concept: '... I find a fatal flaw in Dawkins's attack from below. No matter how much power Dawkins wishes to assign to genes, there is one thing he cannot give them – direct visibility to natural selection. Selection simply cannot see genes and pick among them directly. It must use bodies as an intermediary.' The valid point being made is that replicators do not expose themselves naked to the world; they work via their phenotypic effects, and it is often convenient to see those phenotypic effects as bundled together in vehicles such as bodies.

It is another matter whether the individual body is the only level of vehicle worth considering. That is what the whole group selection versus individual selection debate is about. Gould comes down heavily in favour of the individual organism, and this is the main one of the would-be units that I shall consider.

Of all the levels in the hierarchy of vehicles, the biologist's eye is drawn most strongly to the individual organism. Unlike the cell and the population, the organism is often of a convenient size for the naked eye to see. It is usually a discrete machine with an internally coherent organization, displaying to a high degree the quality which Huxley (1912) labelled 'individuality' (literally indivisibility – being sufficiently heterogeneous in form to be rendered non-functional if cut in half). Genetically speaking, too, the individual organism is usually a clearly definable unit, whose cells have the same genes as each other but different genes from the cells of other individuals. To an immunologist, the organism has a special kind of 'uniqueness' (Medawar, 1957), in that it will easily accept grafts from other parts of its own body but not from other bodies. To the ethologist – and this is really an aspect of Huxley's 'individuality' – the organism is a unit of behavioural action in a much stronger sense than, say, half an organism, or two organisms. The organism has one central nervous system. It takes 'decisions' (Dawkins & Dawkins, 1973) as a unit. All its limbs conspire harmoniously together to achieve one end at a time. On those occasions when two or more organisms try to coordinate their efforts, say when a pride of lions cooperatively stalks prey, the feats of coordination among individuals are feeble compared with the intricate orchestration, with high spatial and temporal precision, of the hundreds of muscles within each individual. Even a starfish, whose tube-feet each enjoy a measure of autonomy and may tear the animal in two if the circum-oral nerve ring has been surgically cut, looks like a single entity, and in nature behaves as if it had a single purpose.

For these and other reasons we automatically prefer to ask functional questions at the level of the individual organism rather than at any other level. We ask, 'Of what use is that behaviour pattern to the animal?' We do not ask, 'Of what use is the behaviour of the animal's left hind leg to the left hind leg?' Nor yet do we usually ask 'Of what use is the behaviour of that pair of animals to the pair?' We see the single organism as a suitable unit about which to speak of adaptation. No doubt this is why Hamilton (1964*a*, *b*), in his epoch-making demonstration that individual altruism was best explained as the result of gene selfishness, sugared the pill of his scientific revolution by inventing 'inclusive fitness' as a sop to the individual organism. Inclusive fitness, in effect, amount to 'that property of an individual organism which will appear to be maximized when what is really being maximized is gene survival' (Dawkins, 1978). Every consequence that Hamilton deduced from his theory could, I suggest, be derived by posing the question: 'What would a selfish gene do to maximize its survival?' In effect, Hamilton was accepting the logic of gene (replicator) selection while affirming his faith in the individual organism as the most salient gene vehicle.

Presumably it would, in principle, be possible to imagine a group-level equivalent of individual inclusive fitness: that property of a group of organisms which will appear to be maximized when what is really being maximized is the survival of the genes controlling the phenotypic characters of the group. The difficulty with this is that, while we can conceive of ways in which genes can exert phenotypic power over the limbs and nervous systems of the bodies in which they sit, it is rather harder to conceive of their exerting phenotypic power over the 'limbs' and 'nervous systems' of whole groups of organisms. The group of organisms is too diffuse, not coherent enough to be seen as a unit of phenotypic power.

And yet to some extent the individual organism, too, may be not quite such a coherent unit of phenotypic power as we have grown to think. It is certainly much less obviously so to a botanist than to a zoologist:

> 'The individual fruit fly, flour beetle, rabbit, flatworm or elephant is a population at the cellular but not at any higher level. Starvation does not change the number of legs, hearts or livers of an animal but the effect of stress on a plant is to alter both the rate of formation of new leaves and the rate of death of old ones: a plant may react to stress by varying the number of its parts' (Harper, 1977).
>
> Harper feels obliged to coin two new terms for different kinds of 'individual'. 'The "ramet" is the unit of clonal growth, the module that may often follow an independent existence if severed from the parent plant' (Harper, 1977, p. 24).
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> The 'genet', on the other hand, is the unit which springs from one single-celled zygote, the 'individual' in the normal zoologists' sense. Janzen (1977) faces up to something like the same difficulty, suggesting that a clone of dandelions should be

regarded as equivalent to a single tree, although spread out along the ground rather than raised in the air, and divided up into separate physical 'plants' (Harper's ramets). Janzen sees a clone of aphids in the same way, although Harper presumably would not: each aphid in a clone develops from a single cell, albeit the cell is produced asexually. Harper would therefore say that a new aphid is produced by an act of *reproduction*, whereas Janzen would regard it as having *grown* like a new limb of its parent.

It might seem that we are now playing with words, but I think Harper's (1977, p. 27) distinction between reproduction by means of a single-celled (asexual or sexual) propagule, and growth by means of a multicellular propagule or runner, is an important one. What is more, it can be made the basis of a sensible criterion for defining a single vehicle. Each new vehicle comes into being through an act of reproduction. New parts of vehicles come into being through growth. The distinction has nothing to do with that between sexual and asexual reproduction, nor with that between ramet and genet.

One act of reproduction, one vehicle

I do not know whether Harper had the same thing in mind, but for me the evolutionary significance of his distinction between growth and reproduction is best seen as arising out of a view of development which I learned from the works of J.T. Bonner (e.g., 1974). In order to make complex adaptations at the level of multicellular organs – eyes, ears, hearts, etc., – a complex developmental process is necessary. An amoeba may give rise to two daughters by splitting down the middle, but an eye, or a heart, cannot give rise to two daughter eyes, or two daughter hearts, by binary fission. Eyes and hearts are so complex that they have to be developed from small beginnings, built by orderly cell division and differentiation. This is why insects whose life cycle takes them through two radically different bodies, like caterpillar and butterfly, do not attempt to transform larval organs into corresponding adult organs. Instead development restarts from undifferentiated imaginal discs, the larval tissues being broken down and used as the equivalent of food. Complexity can develop from simplicity, but not from a wholly different kind of complexity. The evolution of one complex organ into another can take place only because in each generation the development of individuals restarts at a simple, single-celled beginning (Dawkins, 1982, chapter 14).

Complex organisms all have a life cycle which begins with a single cell, passes through a phase of mitotic cell division in which great complexity of structure may be built up, and culminates in reproduction of new single-celled propagules of the next generation. Evolutionary change consists in genetic changes which alter the developmental process at some crucial stage

in the life cycle, in such a way that the complex structure of the organism of the next generation is different. If organisms simply grew indefinitely, without returning cyclically to a single-celled zygote in a sequence of generations, the evolution of complexity at the multicellular organ level would be impossible. For lineages to evolve, individuals must develop from small beginnings in each generation. They cannot just grow from the multicellular bodies of the previous generation.

We must beware of falling into the trap of 'biotic adaptationism' here (Williams, 1966). We cannot argue that a tendency to reproduce rather than grow will evolve in order to allow evolution to happen! Rather, when we look at complex living things we are looking at the end products of an evolutionary process which could only occur because the lineages concerned showed repeated reproduction rather than just growth. A related point is that repeated cycles of reproduction are only possible if there is also death of individual vehicles, but this is not, in itself, a reason that explains why death occurs. We cannot say that the biological function of death is 'to' allow repeated reproduction and hence evolution (Medawar, 1957). But given that death and reproduction do occur in a lineage, evolution in that lineage becomes possible (Maynard Smith, 1969).

Is the distinction between growth and reproduction a rigid one? As so far defined it seems so. A life cycle which restarts with a single cell represents a new reproductive unit, a new discrete vehicle. All other apparent reproduction should be called growth. But couldn't there be a new life cycle that was initiated, not by a single-celled propagule but by a small multicellular propagule? When a new plant grows from a runner sent out by an old plant, is this reproduction or growth? If Harper's definition is rigidly applied, everything depends on an embryological detail. Are all the cells of the new 'plant' the clonal descendants of one cell at the growing tip of the runner? In this case we are dealing with reproduction. Or is the runner a broad-fronted meristem, so that some cells in the new plant are descended from one cell in the old plant, while other cells in the new plant are descended from another cell in the old plant? In this case the Harper definition forces us to classify the phenomenon as growth, not reproduction. It is, in principle, not different from the growth of a new leaf on a tree.

That is what follows from the Harper definition, but is it a sensible definition? I can think of one good reason for saying yes. It makes sense if we are regarding reproduction as the process by which a new vehicle comes into existence, and growth as the process by which an existing vehicle develops. Imagine a plant that sends out vegetative suckers which are broad-fronted meristems, and suppose that this species never reproduces sexually. How might evolutionary change occur? By mutation and selec-

tion in the usual way, but *not* by selection among multicellular organisms. A mutation would affect the cell in which it occurred, and all clonal descendants of that cell. But because the runner is broad-fronted, new 'plants' (ramets) would be heterogeneous mosaics with respect to the mutation. Some of the cells of a new plant would be mutant, others would not. As the vegetation creeps over the land, mutant cells are peppered in haphazard bunches around the 'individual' plants. The apparent individual plants, in fact, are not genetic individuals at all. Since they are genetic mosaics, the largest gene vehicle that can be discerned as having a regular life cycle is the cell. Population genetics would have to be done at the cellular level, not at the 'individual' level. And vehicle selection could give rise to adaptive modification at the cellular level, but not at the level of the whole 'plant'. The whole 'plant' would not function as a vehicle propagating the genes inside it, because different cells inside it would contain different genes. Cells would function as vehicles, and adaptations would not be for the good of the whole plant but for the good of smaller units within the plant. To qualify as a 'vehicle', an entity must come into being by reproduction, not by growth.

That is my justification for the importance of the Harper definition. But now suppose that the runner is a narrow bottleneck of mitotic cell descent, so that the life cycle consists of an alternation between a growth phase and a small, if multicellular, restarting phase. 'Individual plants' would now be statistically unlikely to be genetic mosaics. In this case vehicle selection at the level of whole plants could go on, in a statistical sense, since most, though not all, plants would be genetically uniform. Genetic variation within the cells of individual plants would be less than that between cells of different plants. A kind of 'group selection' (J. Hartung, personal communication) at the cellular level could therefore go on, leading to adaptation at the level of the multicellular vehicle, the level of the 'individual plant'. We might, therefore, tolerate a slight relaxation of Harper's criterion, using 'reproduction' whenever a life cycle is constricted into a narrow bottleneck of cells, even if that bottleneck is not always quite as narrow as a single cell.

We are now, incidentally, in a position to see a reason, additional to those normally given, why the individual organism is so much more persuasive a unit of natural selection (vehicle) than the group of organisms. Groups do not go through a regular cycle of growth (development), alternating with 'reproduction' (sending off a small 'propagule' which eventually grows into a new group). Groups grow in a vague and diffuse manner, occasionally fragmenting like pack-ice. It is significant that

models of group selection which come closest to succeeding tend to incorporate some reproduction-like process. Thus Levins, and Boorman & Levitt (reviewed by Wilson, 1973) postulate a metapopulation of groups, in which populations 'reproduce' by sending out 'propagules' consisting of migrant individuals or small bands of individuals. Moreover, 'group selection' in the sense of D.S. Wilson (1980) can only work if there is some mechanism by which genetic variation between groups is kept higher than genetic variation within groups (Maynard Smith, 1976; Grafen, 1980, and in preparation). This point is analogous to the one I made in my discussion of 'cellular selection' in plants with narrow runners. In practice the most likely way for intergroup variation to be higher than intragroup variation is through genetic relatives tending to associate together. In this case we are dealing with what has been called kin-group selection. Is 'kin selection', then, an authentic case where we have a vehicle larger than the individual body, in the same way as group selection would be if it existed?

Kin selection and kin group selection

There are those who see kin selection as a special case of group selection (E.O. Wilson, 1973; D.S. Wilson, 1980; Wade, 1978). Maynard Smith (1976) disagrees, and emphatically so do I (Dawkins, 1976, 1978, 1979). Maynard Smith is too polite in suggesting that the disagreement is merely one between lumpers and splitters. Hamilton (1975) at first reading might be thought to be endorsing the lumping of kin and group selection. To avoid confusion I quote him in full

> 'If we insist that group selection is different from kin selection the term should be restricted to situations of assortation definitely not involving kin. But it seems on the whole preferable to retain a more flexible use of terms; to use group selection where groups are clearly in evidence and to qualify with mention of "kin" (as in the "kin-group" selection referred to by Brown, 1974)' (Hamilton, 1975, p. 141, citation of Brown corrected).

Hamilton is here making the distinction between kin selection and kin-group selection. Kin-group selection is the special case of group selection in which individuals tend to be closely related to other members of their own group. It is also the special case of kin selection in which the related individuals happen to go about in discrete family groups. The important point is that the theory of kin selection does not *need* to assume discrete family groups. All that is needed is that close relatives encounter one another with higher than random frequency, or have some method of recognizing each other (Maynard Smith, this volume). As Hamilton says, the term kin selection (rather than kin-group selection) 'appeals most where pedigrees are unbounded and interwoven.'

I have previously quoted Hull (1976) on mammary glands: 'mammary glands contribute to individual fitness, the individual in this case being the kinship group'. Hull is here using 'individual' in a special, philosopher's sense, as 'any spatio-temporally localized, cohesive and continuous entity'. In this sense 'organism' is not synonymous with 'individual' but is only one of the class of things that can be called individuals. Thus Ghiselin (1974) has argued that species are 'individuals'. The point I wish to make here is that the 'kinship group' is an 'individual' only if families go about in tightly concentrated bands, rigidly discriminating family members from non-members, with no half measures. There is no particular reason for expecting this kind of rigid family structure in nature, and certainly Hamilton's theory of kin selection does not demand it. As I suggested when I originally quoted Hull (Dawkins, 1978), we are not dealing with a discrete family group but with

> '... an animal plus $\frac{1}{2}$ of each of its children plus $\frac{1}{2}$ of each sibling plus $\frac{1}{4}$ of each niece and grandchild plus $\frac{1}{8}$ of each first cousin plus $\frac{1}{32}$ 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.

Where they exist, tightly knit family bands, or 'kin groups', may be regarded as vehicles. But the general theory of kin selection does not depend on the existence of discrete family groups. No vehicle above the organism level need be postulated.

Doing without discrete vehicles

It will have been noted that my 'vehicles' are 'individuals' in the sense of Ghiselin and Hull. They are spatiotemporally localized, cohesive and continuous entities. Much of my section on organisms was devoted to illustrating the sense in which bodies, unlike groups of bodies, are 'individuals'. My sections on vegetatively propagating plants and on kin-groups suggested that while they sometimes *may* be discrete and cohesive entities there is no reason, either in fact or in theory, for expecting that they usually will be so. Kin selection, as a logical deduction from fundamental replicator theory, still leads to interesting and intelligible adaptation, even if there are not discrete kin-group vehicles.

I now want to generalise this lesson: although selection sometimes chooses replicators by virtue of their effects on discrete vehicles, it does not have to. Let me repeat part of my quotation from Gould (1977) 'Selection simply cannot see genes and pick among them directly. It must use bodies as an intermediary.' Well, it must use *phenotypic effects* as intermediaries, but do these have to be bodies? Do they have to be discrete vehicles at all? I have suggested (Dawkins, 1982) that we should no longer think of the phenotypic expression of a gene as being limited to the particular body in which the gene sits. We are already accustomed to the idea of a snail shell as phenotypic expression of genes, even though the shell does not consist of living cells. The form and colour of the shell vary under genetic control. In principle the same is true of a caddis larva's house, though in this case building behaviour intervenes in the causal chain from genes to house. There is no reason why we should not perform a genetic study of caddis houses, and a question such as 'are round stones dominant to angular stones?' could be a perfectly sensible research question. A bird's nest and a beaver dam are also extended phenotypes. We could do a genetic study of bower bird bowers in exactly the same sense as we could do a genetic study of bird of paradise tails. I continue this conceptual progression further in the book referred to, concluding that genes in one body may have phenotypic expression in another body. For instance, I argue that genes in cuckoos have phenotypic expression in host behaviour. When we look at an animal behaving, we may have to learn to say, not 'How is it benefiting its inclusive fitness?', but rather 'Whose inclusive fitness is it benefiting?'.

Gould is right that genes are not naked to the world. They are chosen by virtue of their phenotypic consequences. But these phenotypic consequences should not be regarded as limited to the particular individual body in which the gene sits, any more than traditionally they have been seen as limited to the particular cell in which the gene sits (red blood corpuscles and sperm cells develop under the influence of genes that are not inside them). Not only is it unnecessary for us to regard the phenotypic expression of a gene as limited to the body in which it sits. It does not have to be limited to any of the discrete vehicles which it can be described as inhabiting – cell, organism, group, community, etc. The concept of the discrete vehicle may turn out to be superfluous. In this respect, if I understand him aright, I am very encouraged by Bateson (this volume, page 136) when he says 'Insistence on character selection and nothing else does not commit anyone to considering just the attributes of individual organisms. The characters could be properties of symbionts such as competing lichens or mutualistic groups such as competing bands of wolves'. However, I think Bateson could have gone further. The use of the word 'competing' in the last sentence quoted suggests that he remains somewhat wedded to the idea of discrete vehicles. An entity such as a band of wolves must be a relatively discrete vehicle if it is to be said to compete with other bands of wolves.

I see the world as populated by competing replicators in germ lines. Each replicator, when compared with its alleles, can be thought of as being

attached to a suite of characters, outward and visible tokens of itself. These tokens are its phenotypic consequences, in comparison with its alleles, upon the world. They determine its success or failure in continuing to exist. To a large extent the part of the world which a gene can influence may happen to be limited to a local area which is sufficiently clearly bounded to be called a body, or some other discrete vehicle – perhaps a wolf pack. But this is not necessarily so. Some of the phenotypic consequences of a replicator, when compared with its alleles, may reach across vehicle boundaries. We may have to face the complexity of regarding the biosphere as an intricate network of overlapping fields of phenotypic power. Any particular phenotypic characteristic will have to be seen as the joint product of replicators whose influence converges from many different sources, many different bodies belonging to different species, phyla and kingdoms. This is the doctrine of the 'extended phenotype'.

Conclusion

In the present paper I have mainly tried to clear up a misunderstanding. I have tried to show that the theory of replicators, which I have previously advocated, is not incompatible with orthodox 'individual selectionism'. The confusion over 'units of selection' has arisen because we have failed to distinguish between two distinct meanings of the phrase. In one sense of the term unit, the unit that actually survives or fails to survive, nobody could seriously claim that either an individual organism or a group of organisms was a unit of selection; in this sense, the unit has to be a replicator, which will normally be a small fragment of genome. In the other sense of unit, the 'vehicle', either an individual organism or a group could be a serious contender for the title 'unit of selection'. There are reasons for coming down on the side of the individual organism rather than larger units, but it has not been a main purpose of this paper to advocate this view. My main concern has been to emphasise that, whatever the outcome of the debate about organism versus group as *vehicle*, neither the organism nor the group is a *replicator*. Controversy may exist about rival candidates for replicators and about rival candidates for vehicles, but there should be no controversy over replicators *versus* vehicles. Replicator survival and vehicle selection are two aspects of the same process. The first essential is to distinguish clearly between them. Having done so we may argue the merits of the rival candidates for each, and we may go on to ask, as I briefly did at the end, whether we really need the concept of discrete vehicles at all. If the answer to this turns out to be no, the phrase 'individual selection' may be judged to be misleading. Whatever the upshot of the latter debate about the extended phenotype, I hope here to have removed an unnecessary source of semantic confusion by exposing the difference between replicators and vehicles.

Summary

1. The question of 'units of selection' is not trivial. If we are to talk about adaptations, we need to know which entity in the hierarchy of life they are 'good' for. Adaptations for the good of the group would look quite different from adaptations for the good of the individual or the good of the gene.
2. At first sight, it appears that 'the individual' is intermediate in some nested hierarchy between the group and the gene. This paper shows, however, that the argument over 'group selection' versus 'individual selection' is a different kind of argument from that between 'individual selection' and 'gene selection'. The latter is really an argument about what we ought to *mean* by a unit of selection, a 'replicator' or a 'vehicle'.
3. A Replicator is defined as any entity in the universe of which copies are made. A DNA molecule is a good example. Replicators are classified into Active (having some 'phenotypic' effect on the world which influences the replicator's chance of being copied) and Passive. Cutting across this they are classified into Germ-line (potential ancestor of an indefinitely long line of descendant replicators) and Dead-end (e.g., a gene in a liver cell).
4. Active, germ-line replicators are important. Wherever they arise in the universe, we may expect some form of natural selection and hence evolution to follow.
5. The title of replicator should not be limited to any particular chunk of DNA such as a cistron. Any length of DNA can be treated as a replicator, but with quantitative reservations depending on its length, on recombination rates, linkage disequilibrium, selection pressures etc.
6. An individual organism is not a replicator, because alterations in it are not passed on to subsequent generations. Where reproduction is asexual, it is possible to regard an individual's whole genome as a replicator, but not the individual itself.
7. Genetic replicators are selected not directly but by proxy, via their phenotypic effects. In practice, most of these phenotypic effects are

bundled together with those of other genes in discrete 'Vehicles' – individual bodies. An individual body is not a replicator; it is a vehicle containing replicators, and it tends to work for the replicators inside it.

8. Because of its discreteness and unitariness of structure and function, we commonly phrase our discussions of adaptation at the level of the individual vehicle. We treat adaptations as though they were 'for the good of' the individual, rather than for the good of some smaller unit like a single limb, or some more inclusive vehicle such as a group or species.
9. But even the individual organism may be less unitary and discrete than is sometimes supposed. This is especially true of plants, where it seems to be necessary to define two different kinds of 'individuals', 'ramets' and 'genets'.
10. An individual may be defined as a unit of *reproduction*, as distinct from *growth*. The distinction between reproduction and growth is not an easy one, and it should not be confused with the distinction between sexual and asexual reproduction. Reproduction involves starting anew from a single-celled propagule, while growth (including vegetative 'reproduction') involves 'broad-fronted' multicellular propagation.
11. Kin selection is quite different from group selection, since it does not need to assume the existence of kin-groups as discrete vehicles. More generally, we can question the usefulness of talking about discrete vehicles at all. In some ways a more powerful way of thinking is in terms of replicators with *Extended Phenotypes* in the outside world, effects which may be confined within the borders of discrete vehicles but do not have to be.
12. The concept of the discrete vehicle is useful, however, in clarifying past discussions. The debate between 'individual selection' and 'group selection' is a debate over rival vehicles. There really should be no debate over 'gene selection' versus 'individual (or group) selection', since in the one case we are talking about replicators, in the other about vehicles. Replicator survival and vehicle selection are two views of the same process. They are not rival theories.

I have benefited from discussion with Mark Ridley, Alan Grafen, Marian Dawkins, and Pat Bateson and other members of the conference at King's College. Some of the arguments given here are incorporated, in expanded form, in chapters 5, 6 and 14 of *The Extended Phenotype* (Dawkins, 1982).

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