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Essays on Darwinism
3: Genic and Organismic Selection

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Abstract

The notion that *genes* play a uniquely distinguished rôle in biological evolution has been championed by Richard Dawkins (1976; 1989b). Furthermore, Dawkins has argued that this idea can be generalised in a way which makes it applicable to any properly Darwinian evolutionary process, at least if that process gives rise to a growth in adaptive complexity (Dawkins 1983). It is evident, therefore, that if Dawkins' analysis is correct, it has profound implications for any attempt to realise a growth of adaptive complexity in *artificial systems* by Darwinian means.

This essay is concerned with a detailed evaluation and critique of Dawkins' claims, and, to a lesser extent, of the related analysis carried out by David L. Hull (1980; 1981). It provides a reformulation which, it is claimed, captures the core of valid insights which these workers have achieved while, at the same time, avoiding certain confusions and misconceptions which might otherwise be read into their views.

The essay draws on concepts introduced in two previous essays (McMullin 1992a; 1992b); the three essays are therefore best read in conjunction with each other.

Contents

1 Introduction

In this essay I review the debate between so-called "genic" and "organismic" selection. The *problem* being addressed here is what I have previously (McMullin 1992b) called *Darwin's Problem* (or P_d): it is the problem of the phylogenetic growth of adaptive complexity (in at least some organismic lineages). Further, the *solution* presented in that previous essay is still accepted here in its essential respects—i.e. that this growth arose primarily, if not exclusively, by a process of unjustified variation and natural selection. The *only* point at issue here is which biological entities can or should be considered as the Darwinian *actors* (D-actors—see McMullin 1992a; this terminology is briefly reviewed in the next section) in this process. In the analysis of (McMullin 1992b) the D-actors were taken to be organisms (hence *organismic* Darwinism). In this essay I want to consider whether there are any "rival" candidates for this rôle, and, if so, whether we can establish any preferences among them.

I will be concentrating almost exclusively on the contributions of David L. Hull and Richard Dawkins to the analysis of this issue. I should therefore make absolutely clear, at the outset, my admiration and respect for these two men and their work. It is only because of this respect that, even as a relative outsider to the fields in question, I venture to offer criticism of aspects of their views, and some limited suggestions for improvement and clarification. But certainly, if I have succeeded in making any worthwhile contribution at all, it is only because I have been able to build on the insights which Hull and Dawkins had already carved out.

2 Terminology

This section provides a short summary of terminology introduced in (McMullin 1992a); please consult that essay for more detailed discussions.

Very briefly, *actors* are individuals which reproduce, with some degree of heritability. A Similarity-lineage or *S-lineage* is a lineage of actors which includes, at each generation, *only* those offspring which are "similar" to their parent(s) in some specified way. Distinct, heritable, "similarities" (similarity-classes or *S-classes*) thus distinguish distinct S-lineages. In the general case, any given actor may be a member of many distinct S-lineages. In certain circumstances an S-lineage may grow consistently until limited by resource availability; and, in so doing, may exclude or eliminate one or more other S-lineages. This is S-lineage *selection*. *S-value* is a parameter of an S-lineage such that differences in S-value are predictive of the rate and ultimate outcome of selection.

The birth of an actor with some heritable characteristic not possessed by any of its parents is called *S-creation*. S-creation initiates new S-lineages. If S-creation is unjustified (not informed by anticipatory models of S-value) the actors are called Darwinian- or *D-actors*. A lineage of D-actors, incorporating multiple distinct S-lineages, whose evolution can be usefully described in terms of selection events between those S-lineages, is called a *D-lineage*. A system of D-actors, forming D-lineage(s), is called a *D-system*.

3 Replicating Confusion

There has been considerable ambiguity, if not confusion, in the literature of evolutionary biology regarding specific technical usage of the terms *replication* and *replicator*. I hope to clear up some of this ambiguity in the course of my detailed discussion of genic and organismic selection; but before proceeding with that, I should like to illustrate these problems of interpretation in a general way.

As far as I am aware, the abstract, technical, idea of a *replicator* was first introduced by Dawkins (1976; 1978a). Hull subsequently elaborated the idea (Hull 1980; 1981), and Dawkins has since extended his own analysis somewhat further (Dawkins 1982a). I shall generally be relying on these sources in the discussion which follows.

I shall argue that "replicator" has been used to refer to at least two distinct kinds of entity, corresponding roughly to my (D-)actors and (S-)lineages; and that the distinction between these two kinds of entities (which I consider crucial) has not been satisfactorily recognised or respected.

Where necessary in the following, I shall explicitly distinguish the two possible meanings of "replicator" as *A-replicator,* for actor-replicator, and *L-replicator,* for lineage-replicator.

The ambiguity of usage can be conveniently illustrated by considering the concept of replicator *longevity*, introduced by Dawkins. In *The Selfish Gene*, Dawkins first defines longevity as relating to the lifetime of an individual replicator, i.e. an A-replicator:

> Certain molecules [supposed primordial replicators], once formed, would be less likely than others to break up again. These types would become relatively numerous in the soup, not only as a direct logical con-

> sequence of their 'longevity', but also because they would have a long time available for making copies of themselves. Replicators of high longevity would therefore tend to become more numerous and, other things being equal, there would have been an 'evolutionary trend' towards greater longevity in the population of molecules.
>
> Dawkins (1976, p. 18)

He goes on to argue that there would be an overall trend toward the evolution of "varieties" (classes or lineages?) of replicator with high "longevity/fecundity/copying-fidelity" (Dawkins 1976, p. 19).[1]

Henceforth, I shall call this first form of longevity *A-longevity,* for actor-longevity.

Somewhat later in the same source, Dawkins specifies that "Copying fidelity is another way of saying longevity-in-the-form-of-copies *and I shall abbreviate this simply to longevity*" (Dawkins 1976, p. 30, emphasis added). Now *this* version of longevity evidently refers to a replicator viewed as a lineage ("in-the-form-of-copies"), or L-replicator. Henceforth, I shall call this *L-longevity,* for lineage-longevity.

So far, any confusion is latent: as long as we remember that Dawkins is using "longevity" in two quite different ways, and judge his meaning from the context, it should not cause too much trouble. In particular, we might reasonably suppose that the slogan "longevity/fecundity/fidelity" will always refer to A-replicators, not to L-replicators—i.e. the "longevity" in question will be A-longevity rather than L-longevity. I say this for two distinct reasons. Firstly, A-longevity is the sense of longevity with which Dawkins first introduced the slogan. But secondly, and more significantly, Dawkins claims that L-longevity is effectively synonymous with copying *fidelity* (a dubious equation in any case, but let it stand). It follows that, if the longevity in the slogan were interpreted as L-longevity, the slogan would become synonymous with "fidelity/fecundity/fidelity"—which is at least redundant and confusing, if not actually incoherent.

Unfortunately, however, Dawkins did indeed subsequently use the slogan in precisely this confusing way:

> The qualities of a good replicator may be summed up in a slogan reminiscent of the French Revolution: Longevity, Fecundity, Fidelity [Dawkins 1976; 1978b]. 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.*
>
> Dawkins
> (1978a, p. 68, emphasis added)

So we have the slogan, which I have just argued *must* imply A-replicator, followed immediately by an elaboration that obviously implies L-replicator. Given Dawkins' own confusion here, it is hardly surprising that Hull then compounded the error further:

> According to Dawkins [Dawkins 1978a, p. 68], the qualities of a good replicator may be summed up in a slogan reminiscent of the French Revolution: Longevity, Fecundity, Fidelity. As striking as this slogan is, it can easily be misunderstood. The fidelity which Dawkins is talking about is copying-fidelity, and the relevant longevity is longevity-in-the-form-of-copies [Dawkins 1976, p. 19, p. 30].
>
> Hull (1981, p. 31)

Hull's last citation here refers to the two locations in *The Selfish Gene* (1976 edition), which I have already identified above, where "longevity" was defined—but he omits to mention that these are two different, *and incompatible,* definitions!

Hull is surely correct that Dawkins' slogan may be easily "misunderstood"—for, on my view, both he (and Dawkins himself) have suffered from just such misunderstanding. The interpretation Hull gives here is not the "correct" one—i.e. that which accompanied the original formulation of the slogan in (Dawkins 1976, p. 19), and which referred to A-replicators—but the confusing and redundant one which refers to L-replicators (Dawkins 1976, p. 30; 1978a, p. 68).

Hull also uses Dawkins' slogan "longevity, fecundity and fidelity" in another paper Hull (1980, p. 317), but, on this occasion, citing only (Dawkins 1978a) as the source. Again, Hull goes on to specify that the "relevant longevity concerns the retention of structure through descent" (i.e. L-longevity). Again, he does not comment on the fact that (according to Dawkins) this version of longevity is synonymous with (Dawkins' version of) fidelity, and is

[1] Note carefully that Dawkins' "fecundity" here refers to A-replicators; it should not be confused with my *S-fecundity* which properly refers only to S-lineages, or L-replicators.

therefore redundant and confusing as a separate criterion for judging replicators, of *either* kind.

The problem is further compounded by Dawkins (1982a, p. 84) where he quotes, at length, from (Hull 1980), and specifically endorses Hull's interpretation of longevity in this context—thus reinforcing the confusion he himself originated in (Dawkins 1978a).

I should point out that, although Hull may have been led astray by Dawkins on the use of the term *longevity*, as such, he also quite independently originated a related confusion himself. Recall that the underlying problem is not really concerned with longevity as such, but with the distinction between A-replicator and L-replicator. So consider this passage:

> Certain entities (replicators) pass on their structure largely intact from generation to generation. These entities either interact with their environments in such a way as to bias *their* distribution in later generations or else produce more inclusive entities that do.
>
> Hull
> (1980, p. 315,emphasis added)

Clearly, Hull means A-replicator here. Indeed, Hull emphasises this interpretation—that a replicator is an individual, which may form a component of a lineage, but is not itself a lineage in general—in several distinct passages. But in the passage quoted above he refers to these entities biasing "their" distribution in later distributions. Now an L-replicator might properly be said to have a "distribution" (i.e. a size); but an A-replicator, as such cannot have a "distribution" in any coherent sense—most especially not in a "later generation".

As it happens, Dawkins typically (but by no means exclusively) adopts the opposite usage—replicator in the sense of L-replicator rather than A-replicator. But, on the other hand, Dawkins also alternates between the two usages with bewildering speed. Thus, we have the following two comments (quoted from consecutive paragraphs):

> A *germ line replicator* (which may be active or passive) is a replicator that is potentially the ancestor of an indefinitely long line of descendant replicators...
> [*Evidently this refers to A-replicators.*]
>
> ... But whether it succeeds in practice or not, any germ line replicator is *potentially immortal.* It 'aspires' to immortality but in practice is in danger of failing.
> [*Yet now we must be talking about L-replicators.*]
>
> Dawkins (1982a, p. 83)

This confusion between A-replicator and L-replicator is counterpointed (presumably with unconscious irony) by Dawkins' approving remark, that *Hull* (1980; 1981) is "particularly clear about the logical status of the lineage, and about its distinction from the replicator and the interactor" (Dawkins 1982a, p. 100).

In conclusion: the objective of this section has been simply to establish that, though Dawkins and Hull make considerable use of the term *replicator,* their usage is quite generally ambiguous as between A-replicator and L-replicator, and calls for very careful interpretation. The recognition of this fact is an essential prerequisite for the analysis of their theories and arguments in following sections.

4 Two Questions

I suggest that the following two substantive questions in evolutionary biology have been wrongly confounded, and that the terminological confusion discussed in the previous section has contributed significantly to this. The first question may be put as follows:

1: *Is the unit of selection an actor or an (S-)lineage?*

This is an issue, specifically raised by Dawkins, which I believe to be of central importance to any abstract analysis or understanding of Darwinism. I have already considered this question at some length (McMullin 1992a, Section 11). I accepted Dawkins' particular formulation of this question ("The central theoretical problem of teleonomy will be that of the nature of the entity for whose benefit adaptations may be said to exist"—Dawkins 1982a, p. 81). I will not repeat that discussion, but simply note my conclusion (which I take to be identical with Dawkins' conclusion, though expressed in different terminology): that *only* S-lineages can be said to be units of selection in this substantive sense.

I consider this to be a very important insight into the Darwinian process. It is a substantial clarification compared to earlier views of Darwinian theory which presumed that actors were the units of selection. Indeed, a primary objective in building up the

abstract formalism of the D-system was precisely to allow this insight of Dawkins' to be expressed in the clearest possible terms. While Dawkins himself credits Fisher, Hamilton, G.C. Williams and others, with the original inspiration (Dawkins 1989b, p. ix), I may say that my own debt here is entirely to Dawkins.

To precisely the extent that we interpret the term "gene" as capturing, in the biological world, the abstract notion of *continuity through generations* represented by an S-lineage, then it is true to say that "genes" are the units of selection in the biological world. I claim that this was the interpretation Dawkins properly had in mind when he formulated the doctrine of the *The Selfish Gene*; that, indeed, it is identical with my doctrine of the "selfish S-lineage" (McMullin 1992a, Section 11).

The second question at issue is quite different:

2: *Is there a uniquely distinguished "level" of organisation (biological or otherwise) which characterises entities that can qualify as D-actors?*

I suggest that this is the question which Hull set out to answer. As we shall see, Hull's answer seems a little ambivalent; it might be paraphrased as "*probably* not"; I shall attempt to clarify this answer somewhat, but will essentially agree with it.

It is not clear whether Dawkins, on the other hand, ever consciously recognised this second question—not, at least, as a *separate* question. However he does *seem* to suggest an answer (implicitly or otherwise)—namely that *genes* are uniquely distinguished as the only biological entities which should properly be said to be D-actors. I shall argue that such a claim is quite mistaken (but also that it is questionable whether Dawkins really intended to make it).

Specifically, to the extent that we interpret "gene" as a material part of an organism (a fragment of DNA, say), then I shall accept that such genes *might* be usefully considered as D-actors (in "suitable" circumstances); but I reject absolutely the idea that they are *uniquely* qualified for this rôle. I suggest that there are always alternative candidate D-actors (particularly, but not exclusively, organisms); alternatives which will be *equivalent* in the precise sense of yielding a selection dynamics which is either identical, or differs only by a bijective transformation of the state variables. Preferences among these different candidates therefore arise *only* from pragmatic considerations relating to the particular circumstances in which the theory of (natural) selection is being applied.

It is, of course, no accident that the term "gene" turns up in two quite different senses in these two questions. For Dawkins, genes are the prototypical examples of replicators—and his conflation of both (D-)actor and S-lineage into the single term "replicator" is more or less mirrored in his usage of the term "gene" (though with some extra complications as will be discussed later). Again, we should try to distinguish between (at least) actor-genes or A-genes, and lineage-genes or L-genes. The former may (or may not) play the rôle of D-actors, and, correspondingly, the latter may (or may not) play the rôle of S-lineages. Dawkins first argues, correctly in my view, that only S-lineages can qualify as units of selection. He then equates this with a claim that only "genes" (or "replicators") can qualify as units of selection; I can accept this also, if it is read in the sense of "only L-genes *(as opposed to A-genes)*" or "only L-replicators *(as opposed to A-replicators)*". But Dawkins then seems to go on to parlay this into a claim that only "genes"—now specifically meaning A-genes—can qualify as *(D-)actors*. I believe this to be definitely in error. The confusion is deep seated and subtle; but identifying and resolving it is the key objective of this essay.

To repeat: my intention here is to consider (and ultimately rebut) the idea that a certain level of biological organisation—the "genes" or the "DNA" or the "genetic material"—uniquely fills the rôle of D-actor (in the solution of Darwin's Problem specifically). I refer to this flawed idea as *genic* selectionism—as opposed to the *organismic* selectionism discussed in (McMullin 1992b). My position will be the pluralist one that genic and organismic selection, properly viewed, are not competitors or rivals, but merely alternative, formally interchangeable, descriptions of the same underlying biological reality; that, indeed, there may exist an indefinite number of other, similarly equivalent, descriptions; but that, *whichever* of these viewpoints may be adopted, there will be a crucial distinction between D-actors and S-lineages, with only the latter being properly regarded as units of selection, or entities for whose benefit (Darwinian) adaptations may be said to exist. This latter question, of D-actor versus S-lineage as the unit of selection, thus cuts at right angles to the question of gene versus organism as D-actors; confounding these two questions leads only to confusion and error.

5 Candidate D-actors

5.1 Formal Equivalence

I should now like to clarify my claim that there exists a variety of biological entities which are more or less equivalent candidates to play the rôle of D-actor in the Darwinian solution to P_d—including, amongst others, genes and organisms.

Let me consider the question abstractly at first. Suppose that we have identified a D-system—i.e. we have identified a class of entities which qualify as D-actors, exhibiting a selective dynamics. Let us further suppose that these D-actors are such that they are in one-to-one correspondence with some other class of entities, and that it is possible to identify the S-class(es) of the D-actors (which is to say, ultimately, identify the S-values of the corresponding S-lineages) by inspection of these other entities. If this is the case then, clearly, we can, if we wish, regard the latter entities as the D-actors (defining S-procreation for these entities in the obvious manner), and our dynamic model of the system (and its predictions, particularly in terms of selective displacements) will remain completely unchanged—the same number of S-lineages will be identified, with the same S-sizes, undergoing the same selection dynamics.

We can also envisage more general cases. The relationship between the different entities need not be a simple one to one mapping. In principle, it can be arbitrarily complicated, just as long as it is bijective—which is to say that, given a description of the population structure in terms of one kind of entity, the structure in terms of the other is uniquely determined—for in this case the two descriptions are formally interchangeable. Of course, with more complex transformations, the dynamics in one state space or the other may no longer be recognisably "selective"; this suggests one minimal criterion for possibly preferring one representation over another—namely that we would prefer a representation in which the operation of selection is most clearly visible.[2]

Let us consider the specific case of organisms and genes (and/or genomes). I accept here, as my starting point, the arguments of a previous essay (McMullin 1992b) for believing that organisms can and do form S-lineages which can selectively displace each other in suitable circumstances—i.e. that organisms, *at least,* can be validly regarded as D-actors in the solution of P_d.

I shall initially restrict attention to cases where the organism S-lineages are disjoint (either because reproduction is asexual, or reproduction is sexual but the S-lineages under consideration are reproductively isolated—i.e. belong to different species). Consider the genomes of the organisms. To keep matters simple, I suppose that we are dealing only with unicellular organisms, so that, by "genome" I mean a definite material component of the organism (being essentially some collection of DNA and/or RNA molecules). Clearly, there is now a one to one correspondence between organisms and genomes.

Let us accept, furthermore, that the organism characteristics which distinguish S-class are related to the genomes such that the S-class of an organism can be determined (in principle at least) by examination of its genome. This assumption is made plausible by the modern theory of inheritance which stipulates, in effect, that only those organism characteristics which are *correlated* with some genome characteristics can be heritable (and, of course, only characteristics which are at least potentially heritable, can subserve S-classification).

Note carefully that I do not comment here (one way or the other) on whether there is any unique or universal mapping from genomes to organisms; merely that such a mapping may be established "locally" (in space and time)—i.e. for specified organism lineages over some specified period of time (which is sufficiently long for certain selection events of interest to work through). I do not even claim that some such local mapping is always possible—merely that it may be sometimes possible (and that these are, ultimately, the relevant cases to the solution of P_d).

If all this is granted then it follows that, in such cases, we can regard the genomes as D-actors, and the resulting S-lineages will have precisely the same S-sizes, and precisely the same selection dynamics, as the original organism S-lineages. There is no sense in which either kind of entity may be said to be inherently or formally preferable for the rôle of D-actor—though there may well be *pragmatic* reasons for focusing on one rather than the other in any particular case.

It may be remarked that, although this argument has been phrased in terms of whole genomes, it may well be that, in particular cases, the relevant S-classifications can be achieved just by consideration of characteristics of one or more *fragments* (contiguous or otherwise) of the genome. For example, the

[2] This is closely analogous to the search for a "normal" form of a state description, in which the state variables are maximally decoupled. See, for example, the so-called *quasi-species* of Eigen & Schuster (1979) for a particular illustrative example of this process actually applied to a D-system; but compare also my comments in (McMullin 1992a, Section 6.5).

significant difference between two genomes might well consist in a single nucleotide substitution, at a particular, identifiable, locus along a particular chromosome. We could then perfectly sensibly regard the nucleotides occupying this particular locus in each genome as D-actors, forming competing S-lineages etc.

And, of course, it need not stop there: the relevant difference between two nucleotides can, in general, be established just by looking at the the attached base (i.e. whether Cytosine, Adenine, Guanine or, in the DNA case, Thymine). Or, there again, Cytosine and Thymine (say) could, in theory, be resolved by the presence of an NH_2 group in place of an O atom respectively: we could then regard these, suitably located, O atoms and NH_2 groups as perfectly good D-actors.

This kind of argument would not be significantly altered if a number of distinct loci in the genome had to be taken into account in order to achieve S-classification. The *sets* of "occupants" of these specified loci in each genome (whether polynucleotides, single nucleotides, bases, etc.) could then be treated as D-actors, provided they are sufficient, in the particular case of interest, to distinguish (S-classify) the selectively significant S-lineages.

Consider next the case of multicellular organisms, but still with disjoint S-lineages (i.e. where the organism S-lineages of interest are reproductively isolated from each other). This permutation may be of limited biological significance, but it is a convenient conceptual stepping stone.

Matters are now slightly more complicated in that there is no single "genome" (in the sense of an identifiable material part of the organism). Typically, there are DNA molecules in each cell, and new cells may be manufactured on an on-going basis throughout the lifetime of the organism. Let us call the DNA molecules in a single cell a *c-genome*. Now, let us call the set of *all* c-genomes "belonging" to a single organism (over its complete life-time) an *o-genome*.[3] By this definitional contrivance, we can again assert that there is a one-to-one relationship between organisms and their (o-)genomes.

It then follows, just as in the unicellular case, that we can regard the (o-)genomes as D-actors, and the resulting o-genome S-lineages will have precisely the same S-sizes, and precisely the same selection dynamics, as the original (organism) S-lineages. Again, there is no sense in which either kind of entity may be said to be inherently preferable for the rôle of D-actor.

It may even be possible to treat the c-genomes as the D-actors in this case. Granted, the various c-genomes of a single organism may differ in various respects (this underlies the process of cellular differentiation); but they also exhibit various strong similarities—most notably, a very high similarity, if not complete identity, of base sequence. Let us suppose, for the sake of the argument, that the S-class of an organism can be reliably inferred (in principle at least) by examination of *any* of its c-genomes (i.e. the relevant organism characteristics are correlated with c-genome characteristics which are identical for all cells of the given organisms). Suppose further that the number of c-genomes per organism (of given S-classification) is approximately constant; then counting c-genomes (instead of o-genomes) would give a reasonably accurate, scaled, S-size of the organism S-lineage. Such scaling of the state variables by constant multiples is, of course, a bijective transformation, so regarding the c-genomes as the D-actors would indeed yield yet another formally equivalent, interchangeable, dynamic description of the selection process. I mention this possibility for completeness, but it is very difficult to envisage a situation in which it would have any particular merit—that is, a case in which there would be any practical advantage in attempting to count c-genomes rather than o-genomes (or organisms).[4]

It should be clear that, although this argument has been made in terms of DNA molecules, it can apply, *mutatis mutandis*, to many other kinds of entity, provided the following two general conditions are satisfied:

- The entities must support S-classification(s) equivalent to the original organism S-classification(s); in effect, they must exhibit characteristics which allow the S-class(es) of the corresponding organism D-actor to be reliably inferred.
- The entities must bear a fixed numerical relationship to the corresponding organisms; that is, the S-sizes of entity S-lineages must be related by constant scale factors (possibly depending on S-class) to the S-sizes of the original organism S-lineages.

[3] An o-genome can be formally thought of as a lineage of sorts, with c-genomes as its members—but *not* an S-lineage, as such.

[4] Regarding c-genomes as D-actors in this sense (formally equivalent to organism D-actors) should not be confused with selective theories of embryological development, in which the c-genomes of a *single* organism are S-classified in a way reflecting cellular differentiation. This may well be an important embryological mechanism, but is not relevant to the present discussion.

Thus, anything from biochemical reaction paths, through gross morphological features, through extra-somatic artefacts (shells, webs, etc.), through behavioural patterns, through social structures, *might* conceivably be regarded as valid D-actors in suitable cases.

Significant further complications arise when we consider the possibility of *sexual* reproduction—in the sense of meiosis or recombination of the chromosomes.

Again, for conceptual simplicity, I shall first consider a somewhat contrived case, of relatively little biological importance—namely, organisms (multicellular or otherwise) with a strictly *haploid* genotype (each c-genome consists of a set of unique chromosomes, rather than of homologous chromosome *pairs*, or even higher multiples). This qualification of the structure of the genome was not relevant in the previous cases, but becomes important when we consider sexual reproduction.

I assume that recombination occurs between homologous parental chromosomes to yield modified chromosomes which are then passed to offspring. I further assume that this can be satisfactorily modelled by reproduction and (Mendelian) segregation of distinct particulate "genes" (with varying degrees of linkage between them, depending on chromosomal organisation). A "gene" here should be thought of as an A-gene in the sense introduced previously; it physically corresponds to some characteristic (usually base sequence) of an identifiable, "short", fragment of some chromosome in the genome. In the case of multicellular organisms, we may technically distinguish c-genes from o-genes (and, indeed, c-chromosomes from o-chromosomes) in precisely the same way as for c-genomes and o-genomes.

The difference which arises in this case, at the organism level, is that selectively significant characteristics may be transmitted to offspring more or less independently of each other. This means that coherent, organism, S-lineages may now *intersect* to a greater or lesser extent—a single organism may be a member of many selectively distinguished S-lineages. We must now enquire as to the dynamic relationships between these S-lineages, and, in particular, whether any will compete in such a way as to result in selection. That is, we consider whether selection can or will occur within a single interbreeding population (which I call *phyletic* selection, following Eldredge & Gould 1972).

The general answer is that these dynamic relationships may be very complicated, and will *not* generally realise any simple form of selection. However, we can distinguish some special cases where phyletic selection, as such, may arise.

We focus initially on organism characteristics which are correlated solely with alternative genes at a single Mendelian "locus"—i.e. such that any single organism will display exactly one of these characteristics.[5]

By definition, organism S-lineages anchored on such characteristics *will* still be disjoint (with a haploid chromosome any single organism must have one gene or the other at the relevant locus, and must therefore be uniquely a member of one S-lineage or the other). If the characteristics are selectively distinct (i.e. the corresponding S-lineages have significantly different S-value) then selection will occur.

By exactly the same argument as was applied in the previous cases we can now conclude that such a selection dynamics, if it arise at all, can be equally well described by regarding the (allelomorphic) (o-)genes as D-actors, instead of the whole organisms. In fact, so far, this case is essentially identical to the earlier case which allowed that, even with reproductively isolated organism lineages, more or less short genetic fragments (or, generally, sets of such fragments) could serve as D-actors in suitable circumstances (namely if they sufficed to make the relevant S-classifications). The only difference under sexual reproduction is that, due to the possibility of recombination, only characteristics (S-lineages) correlated uniquely with a single locus can generally display this clear selection dynamics. But the point remains that, if such characteristics exist, precisely the same dynamic description will result whether the D-actors are considered to be the genes, the genomes, the organisms, or any of the other possibilities previously identified—for these distinct entities still exhibit one-to-one correspondences, and the relevant S-classifications can still (by hypothesis) by carried out by examining any of them.

This situation does not change at all if we suppose that there exist other selectively significant organism characteristics, which are correlated with other independently segregating genetic loci (i.e. there is no *linkage* between the loci), *and* if we can assume that the contributions of the characteristics correlated with these different loci to overall S-value of the corresponding S-lineages, are *additive* (i.e. there are no so-called *epistatic* interactions). If I understand it correctly, this is another way of formally stating that, in such circumstances, the dynamics of the S-lineages relating to different loci interact *lin-*

[5] The interpretation of what qualifies as a single, Mendelian, "locus" is somewhat obscure; I shall simply stipulate that genetic fragments which are "short" enough to have a "small" probability of being disturbed by recombination are at least one plausible case.

early (if at all) and therefore can be *decoupled* from each other. Should this happen to be the case, we can think of the system as consisting of a set of effectively independent D-systems, with the S-lineage dynamics in any one having no effects on any other. This is (arguably) made most clear by thinking of each genetic locus as defining an independent D-system in itself; that is, if we regard the genes as D-actors (instead of genomes or organisms), then we do not have to deal with the "complicated" idea of intersecting S-lineages.

This is (finally) a positive argument in favour of regarding genes as "uniquely" qualified candidate D-actors. However, I consider it to be rather weak. In the first place, even in this case, it is still perfectly possible to regard genomes or organisms as S-lineages—the only disadvantage is that one must more or less consciously recognise that, in that case, each D-actor may be simultaneously a member of an indefinitely large number of different, intersecting, S-lineages. But, in any case, the scenario is extremely contrived, and its biological significance is very doubtful. It "works" only on an assumption of linear interactions between loci in the S-lineage dynamics, and this, in turn, seems to rely on the absence of both linkage and epistasis—whereas, in practice, linkage and/or epistasis seem to be almost invariably present.

This is not to say that phyletic evolution cannot occur in the face of linkage and/or epistasis, but merely that regarding genes as D-actors need not offer any particular simplification in this case. If there are non-linear interactions of this sort then the "complication" of intersections between different S-lineages defined at the genome or organism levels must be mirrored by formally equivalent couplings between the dynamics of (admittedly disjoint) S-lineages defined at the gene level.

So, on this view, genes have no decisive merits as D-actors; but, equally, neither have they any special disadvantages. However, even this is not the case for the *shifting balance* model of (phyletic) evolution, due to Sewall Wright. This model was originally formulated in the 1920's (Provine 1986), but Wright has continued to develop and elaborate the theory since—see (Wright 1982) for a recent review. In this model,[6] significant events of phyletic selection can relate to S-classes which are distinguished at more than a single (Mendelian) genetic locus. To whatever extent this process does occur in practice, single (Mendelian) genes would not even be *candidate* D-actors—for they would not allow the necessary S-classification to be carried out. Genomes and organisms, on the other hand, may continue as more or less satisfactory candidate D-actors.

Let me now turn to the final case, perhaps the case of most practical interest—phyletic selection in a sexual species with a *diploid* (or, more generally, polyploid) genome.

At this point we lose the simple numerical relationship between the S-sizes of organism (or genome) S-lineages, and S-sizes of putative gene lineages—for each organism carries two (or more) genes, which are possibly selectively distinct, at each locus. Now, not even organism S-lineages grounded on a single genetic locus can be disjoint—a heterozygote harbours both heritable characteristics (though they may not both be fully "expressed") and must therefore be included in both S-lineages. The conditions for selection occurring even relative to a single locus become quite complex to establish—though it *can* be done, at least in some special cases, as analysed in detail in (McMullin 1992b). This revolved around establishing that competition could occur—in the sense that changes in S-size of the two S-lineages were constrained to be *opposite* (though generally unequal).

If we now consider the alternative of treating single genes as the D-actors we discover quite a different situation. Any single gene will, of course, be strictly in one S-lineage or the other, and not both—whether it occurs in a homozygote or otherwise. Thus, the *gene* S-lineages, established by (allelomorphic) genes, will (still) be disjoint. In the case of a fixed population size, we have the original simple competitive situation that any change in the size of one S-lineage must be matched by an equal and opposite change in the size of the other. We regain (more or less) a simple model of selective dynamics between disjoint S-lineages.

So: in the case of phyletic selection in a *diploid* species, the choice of genes as D-actors potentially has a definite advantage, relative to a choice of genomes or whole organisms. Such a choice involves a substantive *transformation* of state variables—a transformation which yields a state description in which the operation of selection may be significantly easier to recognise. A transformation of state variables arises precisely because, for the first time, our alternative candidate D-actors are no longer in a simple numerical relationship with the original D-actors (organisms). So, this is a case where genes do seem to have a definite claim to be preferred as D-actors.

[6] I introduce the shifting balance model here because this is the point in the argument where it first becomes logically applicable and relevant; however, the model is, of course, normally formulated in the more general case of *diploid* organisms.

I must repeat: although genes may be "preferred", it is still perfectly *possible* to treat the organisms or genomes etc. as D-actors. The latter would still yield a state description which can be transformed into a description in terms of genes, and *vice versa*. I assume here that the mating pattern is known (for example, randomised—yielding a Hardy-Weinberg relationship between heterozygote and homozygote distributions), for otherwise the transformation between a gene D-actor model and a genome (or organism) D-actor model would be indeterminate. This is not, however, an additional or *ad hoc* assumption; the S-values of the gene S-lineages obviously depend on how the genes are shuffled into organisms—the relative proportions of hetero- and homozygotes etc.—so the mating pattern is implicated in the formulation of the dynamical equations anyway, regardless of which entities are to be notionally described as the D-actors.

In any case, at best we have now identified a somewhat stronger reason for regarding genes as D-actors—that it yields a simpler, more clearly selective, state description in certain circumstances. But although this is a stronger reason than could be proposed in the case of a haploid genome, I still do not regard it as being at all compelling. The state descriptions in terms of the other candidate D-actors are still formally equivalent (merely slightly more opaque). Furthermore, the *defects* of the gene level view, already identified for the haploid case, still apply with (at least) equal force: that is, given non-linear interactions between the dynamics relative to distinct loci, the supposed simplification of the gene level description will be greatly compromised; and if Wright's shifting balance process is significant then a gene level description will positively obscure the operation of selection relative to a description in terms of genomes or organisms.

I have now fairly exhaustively reviewed the significance of the choice of D-actor (primarily as between organism, genome, and gene) and concluded that, in all cases which I have considered, there is no crucial *formal* effect on the resulting system description or model. In fact, the only case where there is a substantive formal distinction at all is for phyletic selection among diploid (or polyploid) organisms; and that can be considered as a more or less simple, bijective, transformation of state variables which may (or may not) simplify the form of the dynamic equations somewhat. In conclusion: none of this provides any grounds for claiming that any of the candidate D-actors which have been considered have any special or unique claim on that rôle.

5.2 A Pragmatic Distinction

I should now like to consider an essentially *pragmatic* or methodological argument for preferring one candidate D-actor over another. This has already been implicitly referred to above, in the context of the effect of breeding pattern on the relationship between gene and genome (or organism) descriptions for diploid organisms: it is the apparent conflict between S-classification on the one hand, and measuring or estimating S-value on the other.

S-classification relies on identifying *heritable* distinctions between D-actors. It is generally difficult to establish whether, or to what extent, gross characteristics of whole organisms are heritable. Similarly, an organism may have significant heritable characteristics which are virtually undetectable at the organism level. The latter is especially the case with dominance in diploid (or polyploid) organisms; if dominance is complete, the heterozygotic organism *appears* indistinguishable from the dominant homozygote, at least in terms of gross morphology and behaviour etc. In general, if we are restricted to examining gross characteristics of an organism, then controlled breeding experiments will be necessary to reliably achieve S-classification.

By contrast, heritable characteristics can be identified with great confidence (if not great ease) by examining genes and/or genomes. Thus, if our greatest concern or difficulty in any particular application of selection theory is with actually carrying out S-classification, we may be inclined to consistently regard genes and/or genomes etc. as the D-actors.

Conversely, measuring or estimating S-value for an S-lineage depends on an understanding of the relationship between the particular heritable characteristic(s) (which distinguish the S-lineage) and S-fecundity and/or S-mortality. In general this is very difficult if not impossible to recognise from consideration only of the genes and/or genomes. In fact, it is typically only at the level of the whole organism (or, in the case of a parasite or symbiote, perhaps at the level of some *other* organism, or for a social organism, at the level of a colony) that it becomes possible to make reliable assessments of relative S-value. Thus, if our greatest concern is with measuring or comparing S-value then we may well be inclined to consistently regard whole organisms (or perhaps organism artefacts etc.) as the D-actors of interest.

Of course, in practice, *both* aspects mentioned here must always be considered, so the supposed conflict is somewhat illusory. A class of entities is a class of D-actors only if the entities satisfy *both* the heritability requirement (so that coherent S-lineages

are established at all) *and* the requirement for significant differences in S-value (so that, under competitive conditions, a quasi-deterministic, selective, dynamics will result). Which is to say that, while there may be pragmatic reasons for emphasising either genes or organisms (or various other entities) as D-actors, there is no genuine conflict, no possibility of considering either kind of entity to the exclusion of the other. This is the real force of the formal equivalence which has been established in the previous section.

5.3 On Genetic Absolutism

> We have often heard it said that genes contain the "information" that specifies a living being. This is wrong for two basic reasons. First, because it confuses the phenomenon of heredity with the mechanism of replication of certain cell components (DNA), whose structure has great transgenerational stability. And second, because when we say that DNA contains what is necessary to specify a living being, we divest these components (part of the autopoietic network) of their interrelation with the rest of the network. It is the network of interactions in its entirety that constitutes and specifies the characteristics of a particular cell, and not one of its components. That modifications in those components called genes dramatically affect the structure is very certain. The error lies in confusing essential participation with unique responsibility.
>
> Maturana & Varela (1987, p. 69)

I will present a somewhat different kind of argument for genic selectionism in this section. It is related to the issues already considered, but might best be called aesthetic, or perhaps metaphysical. In any case, I shall claim that it is fatally flawed. I introduce it, not because of any merit it has in its own right, but because it provides a necessary contrast for a final, and much more subtle, argument, which is to be presented in the following section.

We ask, roughly, whether any of the rival entities considered here are *minimal, sufficient* candidates. By "sufficient" I mean that these entities are sufficient in themselves (given extra-organismic "environmental" constraints) to identify the selective dynamics of the system. Sufficiency demands both that S-classification can be carried out (including prediction of S-values), and that the S-sizes of the resulting S-lineages can be established (which is to say that the D-actors in each S-lineage can be unambiguously counted), purely by reference to the designated D-actors. By "minimal" I mean something like smallest physical size and/or organisation—"simplest".

So far, in discussing the relationship between organisms and their parts (particularly genes and genomes) I have been concerned solely with *correlation*. I have stipulated that all heritable organism characteristics are correlated with some genetic characteristics—which is to say that, at least "locally" (relative to specified organism lineages, localised in space and time), there exists a mapping between genomes and organisms, and thus between genomes and S-value. This was, of course, a necessary assumption to allow genomes as even *candidate* D-actors.

Now consider the possibility of a much stronger stipulation. Correlation implies some underlying *causal* structure. Let us suppose that this structure is entirely *from* genomes *to* organisms—i.e. that organism characteristics (or, at least, all heritable "regularities" thereof) are caused exclusively by (regularities of) genome characteristics.

Causation is, of course, a notoriously problematic concept, and I do not wish to lay much weight on it. For my purposes, since I am ultimately going to reject the argument anyway, I will rely on a completely informal notion of causation. Specifically, I stipulate that, if the "causal" relationship outlined above holds, a minimal implication is that there *is* a unique and universal mapping from genomes to organisms (and thus to S-value). Genomes must have intrinsic or *absolute* effects in the terrestrial world (including, *inter alia*, "causing" whole organisms to come about). This is what I call *genetic absolutism* (for terminology, compare Bateson's "absolute genetic unit"—see Bateson 1986, p. 82).

Genetic absolutism is closely related to, but should be very clearly distinguished from, genetic *determinism* (see, for example, Dawkins 1982a, Chapter 2). Genetic absolutism does not require that the effects of genomes (much less individual genes) be deterministic, only that they exhibit "regularities"; these regularities can be purely statistical. That is, genetic absolutism is perfectly consistent with a stochastic or probabilistic *in*-determinism. Furthermore, genetic absolutism in no way implies that the effects of genomes are independent of the *general* (extra-organismic) environmental context; indeed, it is obvious that genomes could not have interesting effects except in a more or less amenable environment. So, again, genetic ab-

solutism is compatible with an arbitrary degree of environmental plasticity or contingency. The *only* significant claim of genetic absolutism is that the *organisms* (associated with the genomes under discussion) can be completely factored out (they are *not* part of the "environment"). Which is to reiterate that S-value can be reliably inferred purely from characteristics of the genome in association with the (extra-organismic) environment. Genetic determinism would imply genetic absolutism, but not *vice versa*.

I have expressed genetic absolutism in terms of a relationship between a complete genome and an associated organism. As such it is compatible with decomposition of a genome into genes which, in turn, individually have absolute effects—though the effects of each gene may be qualified to an arbitrary extent by interaction with every other gene in the genome. However, the possibility of such systematic decomposition into smaller absolute units (so-called *Genetic Atomism*—see for example Dawkins 1989b, p. 271) is not *necessary* to the argument I will present; I will therefore ignore it, thus restricting myself to the strongest form of the argument.

Genetic absolutism has, no doubt, a long pedigree. It was perhaps implicit in the formulation of the very term *gene* for *genetic determinent*. It seems that, with the rediscovery of Mendelian inheritance, there came about an idea or assumption that genes were some kind of biological "atom" analogous to chemical atoms; that, in the same way as the characteristics of chemical compounds were implicit in characteristics of the different elementary atoms, the characteristics of biological "compounds" (organisms) were implicit in characteristics of elementary biological "atoms" or genes (*whatever* genes might ultimately prove to consist in).[7] In these terms, genetic absolutism evidently has roots in a generally reductionist philosophy; but again should not be confused with reductionism *per se*. It should be clear in the discussion which follows that genetic absolutism *per se* can be false without compromising a *general* biological reductionism. In any case, I am not qualified to review the detailed development or ramifications of genetic absolutism—and such detail is not necessary to my purposes. It is enough to recognise that genetic absolutism is, at least, a *coherent* theory of biological organisation, and to then ask where that leads us.

The question at hand is: what implication, if any, would genetic absolutism have for choosing between genomes and organisms as D-actors?

To develop an answer, note first that, if such a reliable and universal mapping existed between genomes and organisms, then we could *theoretically* dispense entirely with everything other than genomes in our analysis or description of the evolution of organisms (including the growth of adaptive complexity)—for everything else can be "calculated" back in at any time. Thus, genomes, in themselves, would be *sufficient* candidates, and, furthermore, would be uniquely distinguished as the *minimal* entities for which this holds. Therefore they would indeed deserve to be preferentially regarded as the "true" D-actors in the biological world.

The fundamental problem with all this is, of course, that, if our modern theories of the processes of molecular biology are to be believed at all, then genetic absolutism is simply false. In fact, I do not know of any contemporary biologist who explicitly, and unambiguously, espouses such a view (although Dawkins, among others, can sometimes *seem* to be doing so—a point I shall return to in due course).

Now it is true that there are some limited aspects of the genotype/phenotype mapping which seem to be more or less universal (at least on Earth)—notably the "genetic code" relating DNA/m-RNA base sequence to the amino acid sequence of protein. But even this is not completely universal (e.g. Dyson 1985, p. 26), and is not in any sense intrinsic or absolute—the coding seems to be have a significant degree of arbitrariness, a result of historical evolutionary contingency. It is now effectively fixed—self-reinforcing as it were—but there seems to be no reasonable sense in which a DNA based genetic material requires, imposes, or causes, this one particular coding to be used. Indeed, Hofstadter (1985, Chapter 27) has provided an extended analysis of the essentially arbitrary nature of this coding.

In any case, universality of coding between DNA/m-RNA and protein is a very far cry indeed from universality (and uniqueness) of the *complete* mapping from DNA to phenotype (and thus to S-value).

We may conclude that genetic absolutism, though it is coherent, and perhaps even initially plausible, has been decisively refuted; it follows that the claim that genomes provide a minimally sufficient (and thus preferred) candidate D-actor must be rejected.

Finally, I may say that the underlying goal of identifying a minimally sufficient D-actor can actually survive this criticism—but only just. While it is true that genomes, on their own, are not sufficient to predict phenotypes (and thus S-values) it does not *necessarily* follow that whole organisms are the

[7] As noted in (McMullin 1992b, Section 3.2), this absolute and atomistic concept of gene was *not* proposed by Mendel himself.

only other choice. That is, we can envisage that, corresponding to any conventional organism, there may be some kind of minimal biochemical machinery which is, admittedly, more than a naked genome, but less than a complete organism, which *would* be capable of developing into the complete organism *in vitro*. We can imagine this all right; but it has by no means been demonstrated, and there is no particular reason to suppose that the required decomposition of organisms, even if it is possible at all, would be systematic (in the way that the genotype/phenotype decomposition is systematic). In any case, it seems to me extremely doubtful that any useful insight could be gained into evolutionary biology by directing attention at these hypothetical quasi-organisms as preferred D-actors, in place of conventional organisms.

5.4 On Genetic Relativism

I now turn to a much modified version of the argument of the previous section, which seeks to recover the preference for genomes over organisms among candidate D-actors, *despite* the fact that genomes do *not* have absolute phenotypic expressions or interpretations.

The argument goes like this. Even though genomes do not have an absolute mapping to organisms, the mapping of any new (mutated) genome is well defined *relative* to the mapping of the parental genome. So, given any starting point—some initial organism(s)—the subsequent evolution of the system *can* be tracked purely in terms of genomes. This is not because the genomes have absolute phenotypic effects; rather, each mutational step from the founder organisms is characterised by a well-defined phenotypic effect *relative* to the phenotypic effects of the immediate ancestor; knowing the original effects, accumulation of these relative effects yields a mapping for an arbitrary descendant genome. This is, in effect, an explicit formulation of my earlier claim that a well defined genotype/phenotype mapping can be established as long as we are willing to accept some localisation in space and time. In effect, we say something like "within this species", or "within this genus", certain genotypes cause certain, consistent, phenotypic effects.

Another way of looking at this is to say that, although genomes in themselves do not permit the prediction of phenotypes (and thus S-value), a genome *augmented* with some extra information does allow such prediction; and this extra information is, in a sense, also "genomic"—it is an ancestry, identifying the particular sequence of genomic *changes* between the given genome and the founder stock (for which the phenotypic expression is presumed known).

This kind of argument is precisely what licenses the use of genomes as D-actors at all; and we may, indeed, say that, in any context where this is done, the genomes (suitably augmented, as described) *are* minimally sufficient D-actors, and should be (mildly) preferred for that reason.

I actually accept this as far as it goes; but I want to emphasise its limitations, and the ease with which this argument can lead into error.

Firstly, this approach clearly does *not* squeeze organisms out of the evolutionary story: since the phenotypic effects of the genomes are only defined relatively, they must ultimately be referred back to some founder stock of *organisms*. There is no question of being able to tell a complete evolutionary story *entirely* without reference to organisms.

Secondly, the approach becomes more and more cumbersome, as genomes become further removed from the founder organisms (relative to which their phenotypic expressions can be ultimately evaluated). As long as we restrict attention to a species or genus there may be some hope of a fairly uniform mapping from genomes to phenotypes; but if we wish to think about the broad sweep of biological evolution, then the mapping from genomes to phenotypes is likely to have altered drastically, and the process of establishing a mapping by tracing back to some remote ancestor whose mapping is assumed known seems, at best, counter intuitive.[8]

Let us recall what our D-actors now are, in effect. They are genomes, *augmented with whatever information is necessary to infer their phenotypic effects* (always, of course, as functions of whatever extra-organismic environmental factors are relevant). We stipulate that this information which is being tagged on the genomes *can* always take the form of a record of genomic changes relative to some ancestor organism—plus a specification of the mapping

[8] The situation is, formally, much more complex that I detail here. For example, it is a commonplace assumption that evolutionary descent is theoretically reversible (e.g. Dawkins 1982a, p. 3), although such reversal is of vanishingly small probability over any significant number of steps (this is known as "Dollo's Law"—Dawkins 1986, p. 94); but this ignores the essential relativity involved in genotypic expression. Specifically, a "reverse mutation" (which restores a prior genomic configuration) cannot be *guaranteed* to restore a prior phenotypic expression. It seems to me that such a guarantee would, in general, be possible *only* under conditions of genetic absolutism. However, such complications serve only to further strengthen my claim that evaluating the phenotypic expression of a given genome by reference to some arbitrarily remote ancestor is conceptually misguided, and virtually impossible to carry through in practice.

for the ancestor organism (this is the force of our assumption that S-creation *necessarily* involves correlated genomic modification). We recognise however that, in simple cases, this "extra" information may be essentially identical for all the genomes under consideration—for example, if they are all only one mutational step away from the founder organism(s). In such cases, we can lapse into thinking of the genomes themselves as being the D-actors—but only temporarily; as further mutations occur, particularly if they accumulate in particular (D-)lineages, we may have to augment and diversify this "extra" information that is associated with each genome.

It is admitted that genomes, augmented in this way, have become (by definition) minimally sufficient D-actors, and preferred candidates for that reason. But we must surely ask whether there is any more concise representation for this extra information which is conceptually tagged on to the genomes—particularly if we are concerned with long term evolutionary change. That is, rather than having to trace back through the relative effects of particular genomic modifications, to some potentially remote ancestor, could we short circuit this process by, as it were, accumulating the changes as we go along? In effect, instead of tagging on a record of genomic descent, can we tag on a record of the "current genotype/phenotype mapping"?

Once the process is viewed in this light, the answer is hopefully fairly clear. There is already an entity which concisely accumulates precisely the information we require; conveniently, it is already securely attached to each genome—it is, of course, the organism itself (or the phenotype, if you prefer)!

We are now back essentially at the same conclusion as at the end of the previous section. There is, conceptually, such a thing as a minimally sufficient D-actor; it is certainly something more than just a genome; it is either less than, or equal to, the complete organism; in fact, it is very close to a *definition* of what we mean by the "organism", and the attempt to systematically distinguish it, in practice, from a complete organism offers no benefits (that I can perceive).

The conclusion from all of this is that, if we seriously wish to pursue a minimally sufficient D-actor, in the solution of P_d, then organisms are the only candidates which are even approximately satisfactory. We can use genomes (or even genes etc.) as D-actors in strictly circumscribed circumstances. However, the severe limitations of these other candidates must be carefully remembered; otherwise, one will slip almost inevitably into a position which is hardly distinguishable from the fallacy of genetic absolutism.

6 Hull: Replicators and Interactors

I now turn to a detailed comparison between the analysis presented in previous sections, and the work of David L. Hull. Hull is a philosopher, who has explicitly taken on the problem of revealing the ontological or metaphysical foundations upon which (biological) Darwinism is built. In what follows, I shall rely on the two papers (Hull 1980; 1981) in which he has presented his ideas at some length.

Hull's analysis is explicitly related to, and draws upon, earlier work by Dawkins. Dawkins, in turn, has integrated some of Hull's ideas back into his own framework. Thus, their two approaches are strongly interconnected. Nonetheless, there are fundamental differences between them, and I therefore consider them separately, even at the risk of some repetition.

To anticipate somewhat, I shall first summarise the interpretation I propose of Hull's work. I shall then attempt to validate this interpretation in detail (for it is by no means obvious).

I take Hull to be concerned *exclusively* with the question of which kinds of biological entities can be validly regarded as D-actors (especially, but perhaps not exclusively, in the solution of P_d), and which (if any) should be preferred; he is *not* concerned with the (important) distinction between the abstract rôles played by D-actors and S-lineages. However, somewhat unfortunately (though consistently with other related literature), he identifies his discussion as part of the debate over "units of selection". As I have already argued, it may be preferable to restrict this particular term (following Dawkins) to entities for whose benefit Darwinian adaptations may be said to exist; as such, the debate over the "units of selection" should then be best regarded as concerned with the relative merits of S-lineages over D-actors (regardless of which biological entities are mooted for realising these rôles)—a question which Hull does not touch upon at all.

In considering the question of which biological entities can or should be regarded as D-actors, I interpret Hull as follows:

- He accepts that (A-)genes and organisms (at least) can be formally equivalent alternative candidates.
- He recognises that a pragmatic choice between these candidates may be made, in particular

cases, by emphasising S-class inheritance (*formation* of coherent S-lineages) relative to S-value differences (*selection* between S-lineages), or *vice versa*.

- He accepts that the former emphasis would yield a preference for regarding genes (or other entities close to that "level" of biological organisation) as D-actors, and the latter would suggest a preference for regarding organisms (etc.) as D-actors.

- But he stresses that, in any case, neither pragmatic emphasis will serve, on its own, to support the understanding or analysis of selection processes, for both S-class inheritance and S-value differences are essential to such processes.

- Thus, neither genes nor organisms (nor, implicitly, other related candidates) have a unique claim to be preferentially regarded as the "true" biological D-actors.

Thus far (on this interpretation) Hull is making essentially the same points as I have argued for in section 5 above—which is to say that, thus far, we are in complete agreement. We disagree only when Hull attempts to go slightly further, and suggests that the rôle played by genes may be especially distinguished from that of organisms, in having a certain "causal" priority; and that, further, this may give a basis for regarding genes as uniquely preferred D-actors. I shall argue that this idea should be interpreted as a version of the claim, based on genetic relativism, for genes (or, better, genomes) being minimally sufficient D-actors; and, as already discussed, I consider *this* idea to be, at best, misleading. I should stress however, that Hull introduces this idea only tentatively, and as a minor qualification of his main claims; the scope of my agreement with him is much greater than that of any disagreement.

It now remains to justify these interpretations of Hull's position.

Hull describes his central idea as being to distinguish between two functions which he says are necessary for (Darwinian) evolution: *replication* and *interaction*. Hull considers this distinction to be of critical importance; he goes as far as to say that "A pervasive ambiguity in the literature on levels of selection can be eliminated by consistently distinguishing between replication and interaction" (Hull 1981, pp. 33–4).

I shall argue that Hull's "replication" denotes essentially the preservation of S-class in S-descent—the necessary condition for the formation of coherent S-lineages; and his "interaction" denotes whatever gives rise to significant differences in S-value between distinct S-lineages—a necessary condition for selection between S-lineages (competition is, of course, also required).

However, as well as distinguishing between these *functions* as such, Hull goes on to distinguish between *entities* capable of discharging these functions—*replicators* and *interactors* respectively. I think this is a dangerous, if not actually erroneous, step. By taking this step Hull certainly implies, and sometimes comes close to explicitly stating, that the two functions can be sensibly thought of as being *independently* discharged by distinct biological entities—which is to say that there exist biological replicators which are not also biological interactors and *vice versa*.

Now Hull actually uses this as a sort of straw man—he wants to argue, in effect, that the *functions* of replication and interaction must go together to support selection. That is, only replicator/interactors can serve as D-actors. I agree with the conclusion of course—"replicator/interactor" could be read almost as a *definition* of my concept of D-actor. But I find the preliminary idea—that the functions of replication and interaction *might* be separated into independent entities—to be a potentially misleading fiction. To repeat: in my view, an S-classification capable of identifying a selection process can only be fully defined by reference *both* to the fact of its being heritable, *and* that the consequent S-lineages of the different S-classes have well defined (and significantly different) S-values (in specified environmental circumstances).

Before examining Hull's analysis proper, I want to establish that he genuinely is talking about candidate D-actors, and not about a contrast between D-actors and S-lineages. As detailed in section 3, these is some potential confusion about this issue—particularly from Dawkins, and Hull does take the term "replicator" from Dawkins after all. Hull's intended position can be seen (fairly) unambiguously in the following passage:

> In order to perform the functions they do, both replicators and interactors must be discrete individuals which come into existence and cease to exist. In this process they produce lineages which change indefinitely through time.
>
> Hull (1981, p. 41)

So: both replicators and interactors are "individuals" with finite lifetimes. I suggest that this must be interpreted, in my terms, as meaning that they are candidates for the rôle of D-actor. In fact,

the point Hull ultimately intends to make is that, not only are they candidates for this rôle, but they are strictly *complementary* candidates—any putative D-actor must exhibit both kinds of function.

Hull adds that, *qua* (D-)actors, both replicators and interactors form lineages. It should be carefully noted that Hull does not distinguish here between lineages in general, and S-lineages in particular (but see also his reference to "spatiotemporal sequences of replicates", discussed below); to put it another way, he does not distinguish between descent in general and S-descent in particular. Hull would presumably consider *all* offspring of any given replicator or interactor as members of the parent's lineage(s). I, on the other hand, consider that there are crucial distinctions to be made. S-lineages are the entities which compete with, and may selectively displace, each other, and they can do this precisely because they do *not* "change through time", in some well specified and relevant sense. If we must speak of something changing or evolving through time at all, then *D-lineages* are arguably the best candidates. In any case, this is a digression here: the point of immediate interest is that Hull intends both replicators and interactors as candidate (D-)actors, and not as lineages (of any sort).

Let me now consider Hull's notion of replication and replicators in more detail. Hull bases his discussion on Dawkins' original ideas, but elaborates as follows:

> ... what really matters in selection processes is, as Dawkins points out, *retention of structure through descent* ... Two atoms of gold can be structurally identical to each other without one being a replicate of the other, or both being replicates of some other atom. Descent is missing. Conversely, a complex organic molecule can be broken down into smaller molecules by rupturing its quarternary bonds, and these molecules broken into even smaller molecules, etc., but the resulting molecules would not form replicates because retention of structure is missing.
>
> For selection to take place, spatiotemporal sequences of replicates are necessary. Similar entities alone won't do; neither will spatiotemporal sequences of entities alone.
>
> Hull (1981, pp. 31–32)

Hull's point here is that, in thinking about selection processes, we must clearly distinguish between relationships of similarity and of descent. The two *need* not go together (Hull gives examples where each could be present without the other), but they might—and of course, the latter is the crucially interesting case because a selective dynamics then becomes possible. This is the case for which Hull reserves the term *replication*, and entities satisfying this condition will be called *replicators*.

What exactly qualifies as "structure"? Hull is not explicit, but I take him to mean any objectively measurable characteristic (or array of such characteristics) of an entity; thus, Hullean replication is a relationship of descent distinguished by the fact that parent and offspring entities are also similar, in that they share certain (presumably, *specified*) measurable characteristics. This is essentially the inheritance condition whereby S-descent was defined, so Hullean replication can be taken as synonymous with S-descent. Hullean replicators will (at least) satisfy this inheritance condition for D-actors, and his "spatiotemporal sequences of replicates" are, precisely, my coherent S-lineages.

Hull raises a question here as to whether replication merely involves *similarity* of structure, or whether strict *identity* is required ("required" in the sense of being able to support a selective dynamics). As quoted above, Hull himself has only stipulated that similarity is required, but he immediately goes on to say:

> Dawkins' exposition is couched, however, not in terms of *similarity* of structure, but in terms of *identity* of structure. *Although nothing much rides on the decision, I find requiring structural identity too strong.* For example, physicists consider two atoms to be atoms of the same element even if they are not structurally identical. Isotopes are allowed. Similarly, biochemists consider cytosome *c* to be a single protein even though extensive variation in its composition is common. Such examples could be multiplied indefinitely.
>
> Hull
> (1981, p. 32, emphasis added)

I agree entirely with Hull's conclusion here—i.e. that we should require only "similarity"—but I should like to be a little more formal. By "similarity" I mean firstly that we restrict attention to some specified set of characteristics rather than requiring similarity of "all" characteristics. Secondly we require "similarity" of these specified characteristics, in the following sense: a partition is defined over the set of all discriminable values for each characteristic; two entities are said to be "similar" with respect

to a characteristic iff their respective values for the characteristic fall into the same class (as defined by the partition). By choosing the set of characteristics, and the partitions on their ranges, we can make this criterion of similarity as loose or as tight as we wish in any particular case. The point is that an entity (or class of entities) is only a replicator *with respect to* some such specific definition of "similarity". There can be no such thing as a replicator "as such" (of and in itself). If we can validly call a real entity a replicator at all, it will be true only under some particular representation(s) of it.[9]

I think that all this is fairly clear, and I conjecture that Hull would endorse it. However, I spell it out at some length because I want to disagree strongly with Hull's associated assertion that "nothing much rides on the decision" (between requiring similarity or identity of structure, as a criterion of "replication"). On my interpretation (and I can see no other feasible one) "identity" of structure must imply identity of *all* objectively measurable characteristics (possibly subject to some "tolerance" on each characteristic—defining such a tolerance satisfactorily would be very problematic, but I will happily allow the possibility since it does not affect the argument at all). Now such a requirement would be infeasibly strong. The "objectively measurable" characteristics of any real (as opposed to formal) entity are never (known to be) even finitely enumerable—never mind actually (known to be) enumerated. So "identity" of "structure" between two real entities is not something we could ever positively establish (though its absence may well be demonstrable). Indeed, strict identity may not, in fact, be possible in the real universe at all—though that must evidently remain a metaphysical question.

To put it in a more tangible way: if we restricted the interpretation of "replication" to require strict identity of structure in this sense, then *none* of the candidate biological entities which Hull (or Dawkins, for that matter) wishes to discuss would qualify! As Hull himself notes, two atoms of the same element can be structurally different—for example, if they are of different isotopes. But nothing in the modern theory of DNA based heredity ensures that isotopic properties of the parental DNA molecules are preserved in the offspring, and, as a matter of empirical fact, such properties are *not* so preserved.[10] On a criterion of strict "identity" we should have to conclude than not even fragments of DNA (embedded in biological organisms)—which are the very paradigmatic cases for both Hull and Dawkins—qualify as "replicators".

Seen in this light, Hull's assertion that "nothing much rides on the decision" cannot be sustained, and was, for me at least, deeply confusing. Furthermore (and *pace* Hull) I cannot accept that Dawkins *has* required strict identity as a criterion for "replication" (not, at least, in the sense of "identity" which I have described—and again, I have been unable to identify a plausible alternative), though I can possibly see how such an impression might arise. I shall return to Dawkins' views in the next section; for now I merely note that, insofar as Dawkins proposes any special criterion for "replication", this is not so much about any notion of a "degree" of similarity between parent and offspring, but rather about the *mechanism* whereby such similarity as may exist is achieved. This is a quite separate issue.

I now turn to Hull's notions of interaction and interactors. Consider first the following points:

> Replication by itself is sufficient for evolution of sorts, but not evolution through natural selection. In addition, certain entities must interact causally with their environments in such a way as to bias their [*sic*] distribution in later generations.
>
> Hull (1980, p. 317)

> If an entity is to function as a replicator, it must have a structure and be able to pass this structure on to successive generations of replicators. As a replicator it need interact with its environment only to the extent necessary to replicate itself.
>
> Hull (1980, p. 318)

The point Hull is making is that, given the way he has defined replication, it only guarantees that spatiotemporal sequences of entities (S-lineages) will form, where the entities are "similar" only according to an essentially arbitrary criterion; it says nothing, and can say nothing, about the dynamic behaviours (and interactions) of these sequences—whether they

[9] This definition of "similarity" is essentially equivalent to the notion of "S-classification", and the condition stated here for regarding an entity as a Hullean replicator is equivalent to the definition of S-descent as a case of descent preserving a particular S-class (McMullin 1992a, Section 5).

[10] The example is not capricious: precisely this kind of substitution has been used in certain experimental investigations of the nature of the DNA molecules in living organisms—see, for example, Hardy (1965, pp. 112–113).

will expand, contract, stabilise, oscillate, etc. Indeed, there is no guarantee (even in a fixed environment) they they will show any dynamic regularities at all, because, so far, we have no constraint or axiom linking the "structure" which is preserved in replication with the "behaviour" of the replicators and/or the sequences of replicators. To put it another way, it is a truism that the characteristics of an entity (which is to say, its "structure" in general) in some sense define its possible behaviours in its world; but there is no guarantee that the characteristics which are relevant to its behaviours (in the natural environments it finds itself in) are the *same* characteristics as are preserved in replication; and if they are not the same—if the "behavioural" structure is not in some degree related to the "replication" structure—then the sequences of replicators, though similar in some more or less abstruse technical sense, will not be related in relevant behavioural respects, and will not exhibit any regular dynamics.

So: if we want entities which will form (S-)lineages exhibiting some kind of dynamic regularities (including, of course, *selection*) then, certainly, they must be replicators, but *replication on its own is (still) not enough.* It matters *what* is being replicated, which is to say which, of the potentially infinite number of objectively measurable characteristics of any entity, are being preserved, and how well. This, of course, is the stage in my own formulation where I introduce the ideas of S-fecundity and S-mortality (then leading on to S-value) of an S-lineage, and stipulate that these should be "reasonably" determinate functions of the S-class (and the environment—the latter possibly including other S-lineages, of course)—see (McMullin 1992a, Section 6.1). Without this correlation between S-class and S-value we cannot possibly have a selective dynamics.

Hull introduces *interaction* for essentially the same purpose:

> When Dawkins [Dawkins 1978a] defines "replicator," he has replicators interacting with their environments in two ways—to produce copies of themselves and to influence their own survival and the survival of their copies.[11] Just as Dawkins coined the term "replicator" for the entities that function in the first process, I [Hull 1981] have suggested "interactor" for the entities that function in the second process . . . Thus the two sorts of entities that function in selection processes can be defined as follows:
>
> *replicator:* an entity that passes on its structure directly in replication.
>
> *interactor:* an entity that directly interacts as a cohesive whole with its environment in such a way that replication is differential.
>
> Hull (1980, p. 318)

(Note carefully the distinction Hull makes here between himself and Dawkins. As Hull describes it, a Dawkinsian replicator combines the functions of *both* a Hullean replicator and a Hullean interactor.)

Now Hull does two things at once here—he distinguishes the *functions* of replication and interaction, and also distinguishes *entities* which perform these functions. I endorse the first step; indeed, as already discussed, I have embedded essentially the same idea in my distinction between the preservation of S-class in (S-)descent, and the correlation of S-class with S-value. But I find Hull's second step—distinguishing *entities* which perform the two functions—to be unhelpful and confusing. It suggests that the two functions are *logically* independent—which is to say that replication need not entail interaction (which I accept), and that interaction need not entail replication (which I reject).

Certainly, as we have already seen, one can envisage something which replicates but does not interact (in Hull's senses)—a "pure" Hullean replicator. But it seems to me very difficult, if not impossible, to imagine something which could interact (still in Hull's technical sense) but not replicate—that is, a pure Hullean interactor. I suggest that interaction *does* necessarily entail or presuppose replication, and that Hullean interactors must be regarded as a *subset* of Hullean replicators (effectively, the subset representing Dawkinsian replicators, or my D-actors—though that will have to be qualified later). This is implicit in the fact that Hull's very definition of interactor refers to "replication". It is explicit in my own formulation of S-lineage selection: we can formulate the inheritance (S-descent) requirement without reference to S-value, but not *vice versa*—because without inheritance there are no S-lineages, and, without S-lineages, the concept of S-value cannot even be formulated.

However, the important issue here is not whether Hull *implies* the existence (or conceptual coherence)

[11] Hull lapses here into the confusion between survival_1 (i.e. of actors) and survival_2 (of lineages) discussed in (McMullin 1992b, Section 5.1.1); but his intention is presumably that (Dawkinsian) replicators influence the S-value of their S-lineages (survival_2)—via S-mortality (mean survival_1) or otherwise. . .

of entities which would be interactors but not replicators, but whether he actually attempts to explicitly *use* such entities. It would only be in the latter case that there would be a substantive (as opposed to merely terminological) difference between us. Unfortunately, there is no straightforward answer to this.

Certainly Hull consistently emphasises the need for *both* replication and interaction in order to have a selective process. Thus, we have the following:

> With the aid of these two technical terms [replicator and interactor], the selection process itself can be defined:
>
> *selection:* a process in which the differential extinction[12] and proliferation of interactors cause the differential perpetuation of the replicators that produced them
>
> Hull (1980, p. 318)

Or again:

> Evolution of sorts could result from replication alone, but evolution through natural selection requires an interplay between replication and interaction. Both processes are necessary. Neither process by itself is sufficient. Omitting reference to replication leaves out the mechanism by which structure is passed from one generation to the next. Omitting reference to the causal mechanisms that bias the distribution of replicators reduces the evolutionary process to the "gavotte of the chromosomes," to use Hamilton's [Hamilton 1975] propitious phrase.
>
> Hull (1980, pp. 319–320)

But even here, the underlying ambiguity of Hull's analysis reasserts itself. What are we make of the statement that "Neither process by itself is sufficient" except that both replication and interaction *can* conceivably occur, each without the other? Which is to say that there could, in principle, be such a thing as an interactor which is not a replicator? But then again consider the following:

> The structure of replicators is differentially perpetuated because of the relative success of the interactors *of which the replicators are part.*
>
> Hull
> (1981, p. 41, emphasis added)

Hull all but stipulates that interactors always "contain" replicators, which would effectively be a recognition that interactors must *be* replicators. The argument would go like this: the characteristics of a "part" of an entity are surely characteristics of the entity itself; so if characteristics of the part are preserved in descent, then characteristics of the entity itself are also preserved in descent, and the entity (the whole interactor) must be a (Hullean) replicator in its own right. The only flaws in this argument would seem to be if Hull envisages that not all interactors have these particular "parts" (in which case the original definition of interactor seems to fail) or replication of these "parts" does not necessarily correspond in any definite way with "replication" (procreation?) of the interactors themselves—but in that case I cannot see how the relative success of the interactors (in "proliferation" and "extinction") can map onto "differential perpetuation" of the embedded replicators.

There *is* one case in which Hull seems to explicitly and unambiguously refer to entities which *are* interactors but *not* also replicators:

> In order to function as replicators, species must exhibit structural characteristics and be able to pass on these characteristics. Species must somehow 'reproduce' themselves as distinct individuals. One of the major reservations which biologists have to species selection is that they do not see how species can make the necessary copies of themselves to permit selection at the level of species. *But even if species cannot function as replicators, they still might be sufficiently cohesive to function as interactors* ... A species, once formed, is not capable of extensive change. Instead, species form lineages, and it is these lineages which evolve ...
>
> Hull
> (1981, p. 40, emphasis added)

In the present context I am not concerned with the details of the argument as to whether "species selection" is or is not a real biological phenomenon; rather I am concerned with the structure of the

[12] Again, we have a possible confusion between notions of survival_1 and survival_2; from the context I presume Hull must mean death of individual interactors, rather than "extinction" (in the sense of termination of an interactor lineage).

argument, and the specific way Hull employs the concepts of replicator and interactor. In outline, Hull recognises that a particular kind of entity ("species") might be absolutely unable to qualify as a replicator because it doesn't even procreate ('reproduce'); but that, notwithstanding this, it might act as an interactor. Unfortunately, after allowing that the entity cannot procreate, Hull immediately goes on to speak of its being a member of a lineage (which may evolve). But without procreation the entities cannot form lineages—which is to say that, while the entities in question may be "interactors" in some colloquial sense, they seemingly cannot be *Hullean* interactors—and it is precisely this latter point which is at issue.

I shall not labour this discussion any further. My point is firstly that I absolutely agree with Hull's distinction between the *functions* of replication and interaction, and secondly that, although I find his further distinction between *entities* (replicators and interactors) to be deeply confusing, I have been unable to establish that it represents any substantive disagreement between us.

So far I have argued that Hull's idea of an entity which performs functions of replication *and* interaction is essentially equivalent to my concept of a (D-)actor. Having distinguished these functions, the substance of Hull's analysis is then to consider how well (or badly) specific biological entities, at different "levels of organisation" qualify as performing them. His conclusion is that the balance between these two functions is clearly different at different levels. Thus, genes can be seen as very directly discharging a function of replication, but their (Hullean) interaction with the environment (and hence the relationship between their structure and S-value) is extremely indirect. Conversely, organisms can be seen as very directly interacting with their environment (their structures are directly related to S-value) but their replication is extremely indirect—as exemplified by the fact that, without actually carrying out breeding experiments, it is extremely difficult to establish whether a given organismic characteristic is heritable ("replicated") or not.

Hull's further claim is that a significant element of disagreement over "units of selection" may be traced to a failure to distinguish the functions of replication and interaction, and a *tacit* emphasis on only one or the other. Thus, if one concentrates on the fact that D-actors *must* replicate, one may strongly advocate the primacy of genes (or, at least, entities at that general level) over organisms; and conversely, if one concentrates on the fact that D-actors *must* interact (in Hull's technical sense), then one may strongly advocate the primacy of organisms over genes (etc.). Hull's point is that this is a mistaken dichotomy—*both* functions are necessary, and both must be discussed in any adequate theory of selection. He makes this point explicitly as follows:

> Genes tend to be entities which pass on their structure most directly, while they interact with ever more global environments with decreasing directness. Other, more inclusive entities interact directly with their environments but tend to pass on their structure, if at all, more and more indirectly. *Both* processes must be performed successfully if evolution by natural selection is to take place. Reasons for choosing one necessary element over the over as *the* unit of selection are hard to come by ...
>
> Hull (1981, pp. 34–35)

This can now be seen to be essentially equivalent to my own discussion of these issues in section 5 above, as originally claimed.

I should emphasise that Hull explicitly accepts that whole organisms (and possibly even more inclusive entities) *can* function as (Hullean) replicators (albeit with a relatively indirect replication process). This is significant because he differs in this regard from Dawkins; Dawkins argues, for various reasons, that organisms *cannot* function as replicators—not, at least, in his sense of that term. I shall consider Dawkins' arguments fully in the next section. For the moment I merely wish to say that I agree entirely with Hull's analysis on this point; indeed, insofar as I come up with a preference among candidate D-actors at all, it is in favour of organisms.

Finally we come to the point where Hull and I may diverge somewhat. I agree totally up to the conclusion of the passage quoted immediately above, where Hull observes that reasons for preferring replication over interaction (or *vice versa*), in judging candidate D-actors, are "hard to come by"; however, Hull does not stop at this conclusion, but continues instead to *seek* some reason for ranking one function over the other:

> ... The best I can do is the following. Everyone agrees that both genes and organisms are individuals, and that genes form lineages by replication. Any change in a gene is reflected immediately and directly in successive replicates of that gene. Because the sort of inheritance attributed (inappropriately) to Lamarck does not occur,

> changes in the phenotype cannot be transmitted directly to the genetic material to be passed on to future generations. Instead, the only influence which changes in the phenotype is which organisms succeed in reproducing themselves and which not [*sic*]. Genes causally produce other genes. They also enter into the causal production of organisms. But the only thing that organisms can do is influence quite indirectly the statistical distribution of genes in future gene pools. In passing from the action of genes to the action of organisms, we proceed from definite gene lineages to amorphous gene pools, from causal connections to relative frequencies. As persuasive as these considerations may (or may not) be, they depend on viewing species as classes rather than lineages, an interpretation which biologists are beginning to question in increasing numbers.
>
> Hull (1981, p. 35)

I have quoted this passage in full, because I find it extremely difficult to interpret.

Thus: it is simply not true that "any change in a gene is reflected immediately and directly in successive replicates of the gene"—the example of a change in isotopic constitution demonstrates this much. Similarly, it is simply not true that, by contrast, "the only thing than organisms can do is influence quite indirectly the statistical distribution of genes in future gene pools"—or at least, it is not true if Hull is making a valid comparison with a similar claim about genes. The point about a gene is not that "any" change to it is replicated, but that at least *some* changes to it are replicated—which is just another way of saying that it is, indeed, a replicator. But, under Hull's own analysis, organisms are also perfectly good replicators, in their own right. Or to put it another way, a change to a gene *is* a change to an organism. Granted, depending on the precise point in the organismic life cycle at which such a change occurs, it may not be fully "expressed" (at the organism "level"—or higher) until one or more subsequent generation(s) have elapsed; but Hull can hardly mean to attach importance to this, since its ultimate significance is on the working through of selection, and thus it has exactly the same relationship to both genes and organisms—it determines the fate of their respective S-lineages.

Hull seems to see some crucial difference in the "causal" rôle of genes and organisms—a theme that will be taken up again by Dawkins, as we shall see—but it is difficult to know what he could be getting at. He seems to say that genes have a causal rôle in the production of organisms, but not *vice versa*; but, of course, he cannot really intend that (not at least with a common sense interpretation of "causation") for it would be tantamount to my genetic absolutism again, which is embryological nonsense. On the contrary, organisms play a very major "causal" rôle in the production of their offspring (including the production of their offspring's genes). If Hull's case is to rest on "causation", some further clarification of his intended usage of the concept would be required.

Despite these difficulties of interpretation, I do think that Hull has a substantive point to make—and that the key to this lies in his final remark, when he raises the question of whether "species" should be viewed as classes or lineages. In the light of this final remark, I will attempt to reformulate Hull's argument in a more explicit form.

Suppose species are well defined classes (in Hull's sense of "class", which is to say that membership is spatiotemporally unrestricted). This suggests the possibility (at least) that, for each such species, there exists a (spatiotemporally unrestricted) mapping from genotype to phenotype. If this is so, then, providing organisms can be successfully classified as to their species membership, then all evolutionary processes can be (minimally) expressed purely in terms of genes (or, at least, genomes), without any explicit reference to organisms at all (since the Hullean interaction effects, up to and including the establishment of S-values, can be inferred from knowledge of the genotype, the species, and the environment). This yields a basis (within each such species) for regarding genomes as minimally sufficient, and therefore uniquely qualified, D-actors.

This, of course, is just the position I considered and rejected in my own analysis. It may well be satisfactory if it is limited, exclusively, to phyletic evolution—but even then only if no significant evolutionary changes in "embryology" are occurring. It is flawed as a general view of biological evolution, because, on any view of what constitutes a "species", we want to be able to deal with evolutionary establishment of species; whereas, if our models are restricted to work in terms only of genomes *within* a species (which is precisely the hypothesis under discussion), then they are preempted from addressing such establishment.

In anticipating this discussion previously, I said that Hull presents this particular argument only tentatively, so that the scope of the disagreement between our analyses is small. I can now say that even this may be an overstatement of our differ-

ences. As Hull himself makes clear, the argument he presents only goes through *if* species are viewed as classes (rather than somewhat arbitrarily delimited organism lineages). I can actually agree with that position—as far as it goes; but I then go on to make a strong assertion that species should *not* be regarded as classes, and that the argument therefore fails (and is, in fact, pernicious and misleading). By contrast, as far as I can see, Hull does not commit himself one way or the other on how "species" should be interpreted, and thus leaves the argument hanging. So I should say, in conclusion, that any lingering disagreement I have with Hull seems to be very small indeed, and may actually be non-existent.

7 Dawkins: Replicators and Vehicles

Unlike Hull, Dawkins is primarily an evolutionary biologist rather than a philosopher, and has not generally regarded himself as being concerned with an abstract, metaphysical, or ontological analysis of Darwinism. However, as Hull (1981, p. 30) puts it, "Although he is likely to be shocked, if not offended at being told so, Dawkins [Dawkins 1976; 1978a] has made an important contribution to the metaphysics of evolution". Indeed, Dawkins himself has explicitly made the claim that Darwinism (in a suitably abstract formulation) captures principles which should be applicable to all *possible* forms of life:

> When astronauts voyage to distant planets and look for life, they can expect to find creatures too strange and unearthly for us to imagine. But is there anything that must be true of all life, wherever it is found, and whatever the basis of its chemistry? If forms of life exist whose chemistry is based on silicon rather than carbon, or ammonia rather than water, if creatures are discovered that boil to death at -100 degrees centigrade, if a form of life is found that is not based on chemistry at all but on electronic reverberating circuits, will there still be any general principle that is true of all life? Obviously I do not know but, if I had to bet, I would put my money on one fundamental principle. *This is the law that all life evolves by the differential survival of replicating entities.*
>
> Dawkins (1976, pp. 205–206, emphasis added)

The reference here to "electronic reverberating circuits" strongly suggests that Dawkins' claim for the generality of (abstract) Darwinian processes extends *mutatis mutandis* to any attempt to realise "artificial" life inside some kind of computational system. The general concept of the *necessary* universality of Darwinian principles has since been elaborated in more detail by Dawkins (1983).

It is clear then that Dawkins's views are directly relevant to the stated objectives of this essay (and, indeed, the others in this series); unfortunately, as has been anticipated somewhat in previous sections, these views are not at all simple or clear-cut. In this section I shall present my own detailed reinterpretation and critique.

I shall first attempt to justify my claim that Dawkins' doctrine of the "Selfish Gene" is *essentially* equivalent to my argument for the "Selfish S-lineage". I consider this to be the important and valid core of Dawkins' ideas. Secondly I consider Dawkins' arguments for genic selectionism—for the idea that genes (in the sense of fragments of DNA in terrestrial organisms) are preferred candidates for the rôle of D-actor (in the solution of P_d). Dawkins offers two general kinds of arguments—one applying only in the case of sexual reproduction (with recombination of the genetic material) and the other applying generally. I shall claim that both of these are flawed, though in very distinct ways. However, I shall finally conclude that, once the question of genic selectionism is separated from the question of actors versus (S-)lineages—a separation which Dawkins himself does not recognise or admit—then it becomes very doubtful whether Dawkins would really wish to pursue the arguments for genic selectionism *in isolation* at all.

I should stress at this point that Dawkins does recognise that there are two separate questions at issue; but his particular decomposition is, in my view, mistaken. Consider the following statement of his position:

> ... 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 vehi-

> cles 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.
>
> Dawkins (1982b, p. 46)

In the light of the previous discussion, I would rephrase this as follows (throughout, "survival" should be read as survival$_2$, or survival of *lineages*):

> I shall develop a distinction between *S-lineage survival* and *D-actor 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 *S-lineages.* L-genes (for example) are S-lineages; A-genes, organisms and groups of organisms are not S-lineages, they are (at best) D-actors which compose S-lineages. D-actor selection is the process by which some D-actors are more successful that other D-actors in ensuring the survival of their S-lineages. The controversy about group selection versus individual selection is a controversy about the rival claims of two suggested kinds of D-actor. 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 an D-actor *at all,* or an S-lineage.

Read in *this* way, there would seem to be no disagreement between us at all. This appearance of agreement arises because, in the passage quoted, Dawkins comes very close to using "vehicle" in my sense of "D-actor" and "replicator" (or "gene" for that matter) in my sense of "S-lineage". Unfortunately, such an interpretation seems not to be consistent with the bulk of Dawkins' writings. The difference between us is exemplified by the fact that, according to Dawkins, biological organisms *cannot* qualify as Dawkinsian replicators; whereas I argue that they certainly *can* qualify as D-actors. The task then, is to establish precisely the scope both of our agreement and of our disagreement.

7.1 What *is* a Selfish Gene?

I must now try to justify the claim that the idea of the selfish S-lineage is (at the least) compatible with Dawkins' analysis—that Dawkins holds, firstly, that the unit of selection must be a lineage rather than an actor and, secondly, that the particular and unique kind of lineage required is what I have termed an S-lineage.

I have already argued that Dawkins uses "replicator" in both of the distinct senses I have identified—A-replicator and L-replicator. I can now elaborate this claim in more detailed and formal terms; I claim that:

1. A Dawkinsian L-replicator is a lineage whose members are Dawkinsian A-replicators, though he refers to both these distinct kinds of entities simply as "replicators".

2. When Dawkins is discussing the identity of the unit of selection—particularly in phrases like *The Selfish Gene* or *The Selfish Replicator*—he exclusively uses "gene" or "replicator" in the sense of L-replicator.

3. Dawkins' A-replicators are *essentially* (but not exactly) equivalent to my D-actors. In particular, when their offspring are "identical" in Dawkins' terms, they are procreating as D-actors (preserving S-class) in my terms, and such offspring are S-offspring, members of S-lineage(s). These, and only these, lineages are what Dawkins' recognises as L-replicators or the units of selection.

I shall not argue the first point: were this not the case, Dawkins' ambiguity of usage between A-replicator and L-replicator (already documented in section 3 above) could hardly arise.

Taking the second point, consider the following sample quotations:

> Genes, like diamonds, are forever, but not quite in the same way as diamonds. It is an individual diamond crystal that lasts, as an unaltered pattern of atoms. DNA molecules don't have that kind of permanence. The life of any one physical DNA molecule is quite short—perhaps a matter of months, certainly not more than one lifetime. But a DNA molecule could theoretically live on in the form of *copies* of itself for a hundred million years.
>
> Dawkins (1976, p. 37)

> What is the selfish gene? It is not just one single physical bit of DNA . . . it is *all replicas* of a particular bit of DNA, distributed throughout the world.
>
> Dawkins (1976, p. 95)

> The reason a replicator is interesting to Darwinians is that it is potentially immortal, or at least very long-lived in the form of copies. A successful replicator is one that succeeds in lasting, *in the form of copies,* for a very long time measured in *generations* . . .
>
> Dawkins (1982a, pp. 87–88, emphasis added)

> I have lavished much rhetoric, or irresponsibly purple prose if you prefer, on expounding the view that 'the unit of selection' . . . must be a unit that is potentially immortal [Dawkins 1976, chapter 3] a point which I learned from Williams [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.
>
> Dawkins (1982b, p. 48)

I think these clearly show that Dawkins intends gene/replicator as a lineage rather than an actor in this context, i.e. when he is speaking of 'the unit of selection'. It is important at this point to reiterate a critical difference between Dawkins and Hull. This is that Hull normally uses "replicator" (*and* "interactor", for that matter) in the sense of an actor, whereas Dawkins uses "replicator" in the sense of a lineage—at least when he is referring to the "unit of selection". Thus their two viewpoints on the unit of selection are not merely different, but incommensurable—they are answering different questions, using different concepts; which makes it somewhat unfortunate, to say the least, that they both elect to employ a single token (*replicator*) for their different purposes. This point must be emphasised because it has not been recognised by Hull, and Dawkins has actively suggested that the opposite is the case—that they are both presenting similar answers to the same question (Dawkins 1982a, pp. 82–83).

But to return to the issue at hand: the fact that Dawkins intends *gene*, or *replicator*, as a lineage rather than an actor still leaves open the question of whether he means *S-lineage* as such (and not any other kind of lineage). This, in turn, depends on the third claim made above, that when Dawkins uses replicator in the sense of A-replicator, he has in mind the notion of descent with *similarity* (S-descent); that is, only offspring which are similar, in some particular way(s), to the parent(s) will "count" as offspring at all.

Consider then Dawkins' basic definitions:

> I define a *replicator* as anything in the universe of which copies are made. Examples are a DNA molecule, and a sheet of paper that is xeroxed . . .
>
> An *active replicator* is any replicator whose nature has some influence over its probability of being copied. For example a DNA molecule, via protein synthesis, exerts phenotypic effects which influence whether it is copied: this is what natural selection is all about. A *passive replicator* is a replicator whose nature has no influence over its probability of being copied. A xeroxed sheet of paper at first sight seems to be an example, but some might argue that its nature *does* influence whether it is copied, and therefore that it is active: humans are more likely to xerox some sheets of paper than others, because of what is written on them, and these copies are, in their turn, relatively likely to be copied again. A section of DNA that is never transcribed might be a genuine example of a passive replicator . . .
>
> Dawkins (1982a, p. 83)

> No copying process is infallible. It is no part of the definition of a replicator that its copies must all be perfect. It is fundamental to the idea of a replicator that when a mistake or 'mutation' does occur it is passed on to future copies: the mutation brings into existence a new kind of replicator which 'breeds true' until there is a further mutation. When a sheet of paper is xeroxed, a blemish may appear on the copy which was not present on the original. If the xerox copy itself is now copied [the] blemish is incorporated into the second copy (which may also introduce a new

> blemish of its own). The important principle is that in a chain of replicators errors are cumulative.
>
> Dawkins (1982a, p. 85)

There are a number of points to be made here.

Firstly it should be clear that, throughout *this* discussion, Dawkins is referring to A-replicators and not L-replicators.

Secondly I should mention that, in addition to distinguishing active and passive A-replicators, Dawkins also distinguishes between "germ-line" and "dead-end" A-replicators. I take the latter distinction to be an attempt to deal with the fact that multicellular organisms (for example) have multiple genomes (etc.), which complicates the numerical relationship between genomes and (organismic) S-sizes. Related difficulties can arise in various other contexts also—social insect colonies for example. I chose to deal with this kind of problem via the distinction between c-genomes and o-genomes. Obviously I have a (mild) preference for my own approach; but I do not think there is a fundamental disagreement here, and I shall not pursue this particular issue.

Thirdly, while Dawkins is not too explicit about exactly what "copying" involves, it clearly includes *at least* the notions of descent and similarity—which are essentially the defining notions for my S-descent, and for Hull's "replication". Indeed, Dawkins explicitly and favourably quotes Hull's distinction between similarity and descent (as also quoted in section 6 above), and the need for both to be present (Dawkins 1982a, p. 84).

Fourthly, in distinguishing active and passive A-replicators Dawkins introduces something essentially equivalent to Hull's distinction between replication and interaction. That is, *all* Dawkinsian A-replicators are Hullean replicators; *active* Dawkinsian A-replicators additionally qualify as Hullean interactors; *passive* Dawkinsian A-replicators qualify as Hullean replicators but not as Hullean interactors (and are thus a class of entity for which Hull has no explicit name).

As already discussed, Hulls' approach implies the possible existence of interactors which are not (Hullean) replicators—i.e. entities which realise (Hullean) interaction but not (Hullean) replication. I said that I found this implied concept incoherent, and Hull's presentation confusing as a result. Dawkins' approach, on the other hand, is explicitly stated as a *partition* of the class of Dawkinsian A-replicators, and avoids any analogue of the Hullean interactor-but-not-replicator; one does not even try to imagine an entity which, in Dawkins' terminology, would be *active* but not a (Dawkinsian) A-replicator. I therefore prefer Dawkins' formulation. On the other hand, as Dawkins himself stipulates, it is only active A-replicators which are of interest in discussing Darwinian evolution, so the distinction is perhaps not all that important anyway. In my own formulation of the (D-)actor, this distinction is implicit in my discussion of the requirement that (for selection to occur) S-class must be "predictive" of S-value; but, like Hull, I do not introduce a separate term to explicitly distinguish such S-classes (McMullin 1992a, esp. Sections 6.1 and 6.4).

I may say that I consider my formulation to be marginally superior to Dawkins' insofar as I explicitly note that S-value depends on *both* S-mortality or S-fecundity, whereas Dawkins' "influence over its probability of being copied" seems to explicitly allow only for S-fecundity effects; but having said that, Dawkins' formulation could be easily augmented to take account of this—I do not suggest that its omission is a substantive defect.

I should note here that Dawkins himself adopts a very different view of the relationship between his concepts and Hull's. He has not given any extended or detailed analysis, as far as I am aware, but he seems to consider that their two versions of "replicator" are equivalent, and he explicitly equates Hull's "interactor" with a kind of entity he now terms a *vehicle* (Dawkins 1982a, p. 100). I will consider the Dawkinsian "vehicle" concept in somewhat more detail below; for the moment it is sufficient to record my disagreement with Dawkins on these points. I shall argue that there is a subtle, but important, distinction between their respective notions of (A-)replicator; and also between Hull's "interactor" and Dawkins' "vehicle". Dawkins' equation of the latter two seems to be based on the fact that Hull recognises organisms as more or less prototypical cases of interactors, while Dawkins recognises them as prototypical vehicles. That this interpretation is too shallow is revealed, for example, by the fact that Hull argues at length that organisms *also* qualify as replicators in his terms, whereas Dawkins argues (at even more length) that organisms definitely are *not* replicators in his terms.

However, let us return to the immediate question—which is the relationship between Dawkins' A-replicators and my D-actors. Dawkins explicitly recognises the idea of an A-replicator giving rise to a copy which is different from the parent in a way which is, subsequently, heritable. That is, Dawkinsian A-replicators incorporate a distinction which is clearly related to my distinction between

S-descent (preservation of specified parental characteristics in the offspring) and S-creation (origination of new characteristics, not present in the parent(s), which may be preserved in subsequent offspring).

Neither Dawkins nor Hull make the distinction between S-descent and S-creation quite as sharply as I do; but that they have an equivalent distinction in mind is apparent from their discussions of the "degree of similarity" between parent and offspring required for an entity to qualify as a replicator (under either of their interpretations). Hull requires that parent and offspring be similar enough "to respond similarly to similar selection pressures" (Hull 1980, p. 321); Dawkins expresses himself in essentially the same terms (Dawkins 1982a, p. 89). Both Hull (implicitly) and Dawkins (explicitly) refer to the following comment from Williams, as a source for their views:

> In evolutionary theory, a gene could be defined 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.
>
> Williams (1966, p. 25)

I should emphasise that I accept this general position, which envisages that both heritability and selection pressure may, in general, form continua, and that the differentiation between S-descent and S-creation may not be easy to definitively establish in any particular context (subject to some caveats: see, in particular, my discussion of "digital" inheritance, McMullin 1992a, Section 6.5). My point is that, while the two may be hard to distinguish *methodologically*, the distinction is *conceptually* a fundamental one—S-descent and S-creation play entirely different rôles in the Darwinian solution of P_d, rôles which must not be confused. It is for this reason that, in contrast to Hull and Dawkins, I have made this distinction so explicit, even to the point of introducing an additional technical terminology to reflect it.

For strictly *Darwinian* evolution I have stipulated that S-creation must be "unjustified", in the sense of not involving anticipatory models of resulting S-value (this is my distinction between "actors" in general, and "D(arwinian-)actors" in particular). Dawkins has not discussed this particular issue in terms of his abstract A-replicator concept; however, he has dealt with it in his more general evolutionary writings, as I have discussed elsewhere (McMullin 1992b, Section 3.4). I shall not repeat that discussion, but simply adopt the implication that, here again, Dawkinsian A-replicators are compatible with my notion of D-actors.

The conclusion from all these arguments is that Dawkinsian active A-replicators have all the required properties to qualify as D-actors in my terms; specifically:

- Their offspring are generally related to the parent(s) by both descent and similarity.
- The similarity in question is precisely such as to allow selection between (similarity-)lineages to work itself through.
- There is a possibility of offspring which incorporate new characteristics, not possessed by any of its parent(s), but which are subsequently heritable, thus founding new (similarity-)lineages which may participate in selection.
- This origination of new, heritable, characteristics is, in some cases at least, unjustified.

I claim, furthermore, that these properties, shared by D-actors and Dawkinsian A-replicators, are the *only* properties required for Darwinian evolution to take place. In particular, this establishes that when Dawkinsian A-replicators produce "perfect" offspring, they are behaving as D-actors, forming S-lineages, between which competitive elimination (etc.) may occur. This, precisely, is the logic to identifying (L-)replicators as the "units of selection", and this validates my claim that Dawkins' "Selfish Replicator" (or "Selfish gene" for that matter) is equivalent to my "selfish S-lineage".

Having established that Dawkins' abstract analysis of selection (and Darwinian evolution generally) is at least compatible with my analysis in terms of D-actors, I now finally claim that the latter is somewhat preferable because of its more explicit treatment of the following points:

- Both S-fecundity and S-mortality must be correlated with S-class (not just S-fecundity as implied in Dawkins' version).
- S-fecundity and S-mortality are exclusively characteristics of S-lineages not of D-actors (this is confusingly ambiguous in Dawkins' version).
- The essential requirement is that the selection dynamics (especially complete elimination of one S-lineage by another) be effectively *deterministic* (once the initial S-lineages are given); at best, this is only implicit in Dawkins' treatment.

- S-descent and S-creation play quite different and distinctive rôles.
- S-creation must be unjustified with respect to S-value.

7.2 Rival D-actors?

In contrast to "replicator", Dawkins does not provide a detailed discussion of his abstract notion of "vehicle". This is because he ultimately considers the concept to be of limited value. However, it is a convenient tool for our present purposes. Roughly speaking, Dawkins identifies as vehicles any entities which, for whatever reason, are not Dawkinsian A-replicators in themselves, but which "contain" A-replicators, and which work toward the propagation (replication) of those contained A-replicators. Dawkins stipulates that, in the biological world, organisms must be regarded as vehicles and not A-replicators. My criticism will be directed at this latter claim, rather than at the general or abstract notion of the Dawkinsian vehicle itself.

To outline the structure of my discussion, I must first note that a significant point was left dangling in the previous section. I commented, variously, that the Dawkinsian A-replicator was "essentially" (*not* exactly) equivalent to my D-actor, and that there is a "subtle, but important" distinction between the Dawkinsian A-replicator and the Hullean replicator. Briefly, my position is this: all Dawkinsian A-replicators qualify as D-actors (and, indeed, as Hullean replicators)—but not *vice versa*.

I defer, for the moment, the examination of exactly how Dawkinsian A-replicators and D-actors differ: the important point, at this stage, is that not all D-actors will qualify as Dawkinsian A-replicators. Quite generally in fact, Dawkins' distinction between A-replicators and vehicles may be regarded as a partition of my class of D-actors; that is, it will turn out that only certain D-actors qualify as Dawkinsian A-replicators, and all other D-actors should be regarded, by default, as Dawkinsian vehicles.

It follows that the question of the nature or rôle of biological organisms in selection processes can be divided into two subsidiary questions:

1. Do organisms qualify as D-actors? My answer is a simple *Yes*. Dawkins, on the other hand, distinguishes the cases of purely asexual organisms, and organisms engaging in sexual reproduction (with meiosis); he appears to accept that the former would qualify as D-actors, but rejects the latter.
2. Do organisms qualify as Dawkinsian A-replicators? Dawkins and I are agreed that the answer to this is *No*. Specifically, although I argue that all organisms qualify as D-actors, I accept that the Dawkinsian A-replicator is a more restrictive class than that of D-actor, and that organisms do not qualify as belonging to this more restrictive class. *However,* Dawkins and I differ fundamentally on the implications which may be drawn from this fact. Dawkins effectively claims that the distinction between D-actors in general, and Dawkinsian A-replicators in particular, is such that the latter should be preferentially regarded as the "true" D-actors in selection processes—at least as compared to formally equivalent candidates which are *not* Dawkinsian A-replicators. I shall argue that the distinction Dawkins identifies cannot bear the theoretical weight he places on it; that it does not give a basis for rejecting organisms as candidate D-actors; and that, on the contrary, the general issues Dawkins raises may yield a mild *preference* for organisms as D-actors (over, say, genomes or genes etc.).

I shall consider these questions in turn, in the following sections.

7.2.1 Does sex *really* matter?

> In sexually reproducing species, the individual is too large and too temporary a genetic unit to qualify as a significant unit of natural selection. The group of individuals is an even larger unit. Genetically speaking, individuals and groups are like clouds in the sky or dust-storms in the desert. They are temporary aggregations or federations. They are not stable through evolutionary time...
>
> Chromosomes too are shuffled into oblivion, like hands of cards soon after they are dealt. But the cards themselves survive the shuffling. The cards are the genes. The genes are not destroyed by crossing-over, they merely change partners and march on. Of course they march on. That is their business. They are the replicators and we are their survival machines. When we have served our purpose we are cast aside. But genes are denizens of geological time: genes are forever.
>
> Dawkins (1976, pp. 36–37)

Dawkins' argument for the inadmissibility of organisms (and, indeed, genomes and chromosomes too) even as *candidate* D-actors, in sexually reproducing species, seems straightforward enough at first sight.

We first accept that there is significant standing genetic heterogeneity in natural populations (e.g. Ayala 1978). It follows that, with overwhelming probability, every organism will be genetically unique. If we S-classify simply on this basis, then no coherent S-lineages can form (since no offspring will match the S-class of either parent, so no S-descent will occur), and selection cannot take place. If, however, we consider, instead, relatively short fragments of DNA, occupying some identified locus, then (S-classifying them in the same way) there will now be a "high" probability that they will bear offspring of the same S-class; so coherent S-lineages can form, and selection becomes possible. Therefore, under conditions of sexual reproduction, nothing more inclusive than a "short" fragment of DNA (not chromosomes, not genomes, and definitely not organisms) can function as a D-actor.

Despite the seeming clarity of the argument we should already be deeply suspicious. For example, my reference to fragments of DNA "occupying some identified locus" is immediately problematic—for such a locus can, at best, only be defined *relative* to a complete genome—yet genomes supposedly have no more stability than "dust storms in the desert", and thus can hardly form a context for identifying the putative "immortals". Nonetheless, clearly identifying *exactly* what is wrong with Dawkins' argument is quite tricky. I shall critically consider a number of attempted refutations.

Hull has considered the possibility that Dawkins may be overstating the degree of standing genetic heterogeneity present in natural populations—at least in the cases of most evolutionary interest. He cites a number of biologists as arguing that speciation, in particular, usually occurs among sexually reproducing organisms when a very few become isolated from the main body of their species; he then continues:

> The effects of such a rapid reduction in population size are numerous and fundamental. For example, in most populations, several different alleles exist at most loci. One pregnant female, to mention the most extreme case, is unlikely to express much of the genetic heterogeneity of her population in her offspring. The ensuing inbreeding characteristic of such small populations is likely to increase homozygosity even further. It may well be true that the genomes of sexually reproducing organisms are 'torn to smithereens' at meiosis in large, genetically heterogenous populations, but according to the model of speciation by 'genetic revolutions' currently so popular, all that is going on in such large populations is the haphazard fluctuation of allele frequencies. When it really matters, when new species are arising, sexually reproducing organisms converge on functioning as replicators.
>
> Hull (1981, p. 36)

I find this argument unconvincing, as presented. It is no doubt true that the genetic variability represented by a single pregnant female will generally be significantly *less* than for the population as a whole; it is also true that heterogeneity is likely to fall further immediately following isolation, essentially due to the significant effects of genetic drift in small populations (Hull's "inbreeding characteristic"); but this is a long way from establishing that genetic variability in the isolated population will be reduced to just a single genetic locus—which would seem to be what is required by Hull's argument. Hull does not seem not to allow for the possibility that, even though the variability present in a small subpopulation may be small *relative* to the population as a whole, it could still be quite large in *absolute* terms; and that, further, the variability required to make Dawkins' argument go through is not very large in absolute terms anyway, due to the combinatorial explosion which is implicit in it. That is, it does not require variability at very many loci to mean that, with overwhelming probability, a genome will be disrupted by recombination.

To be specific, Ayala (1978) has estimated that in the case of humans, for example, the genetic variability present even in a single (diploid) individual (which would be a marginally *more* restrictive case that Hull's pregnant, diploid, female, which is effectively a "population" of somewhat more than a single genetic individual) is still such as to allow for something of the order of $10^{2,000}$ (!) genetic variations by recombination. The probability of "perfect" replication of a genome would be roughly of the order of the reciprocal of this, and is obviously negligable. I doubt that, as a general principle, inbreeding (which would presumably be transient, as the population expands?) would materially affect this calculation. If Dawkins' argument works at all (and Hull makes his point explicitly in the context of *accepting* "Dawkins' general analysis of replicators"),

then it seems that it would still work *even* in the case of very small populations. This idea of Hull's is interesting, but ultimately cannot go through as it stands.

A quite different, but closely related, argument could be mounted based on the theory of *punctuated equilibrium* of Eldredge & Gould (1972). This is the general idea that most species evolve little through most of their existence; that evolutionary change in concentrated in small, relatively short, bursts, associated with speciation (i.e. the establishment of a new lineage reproductively isolated from the predecessors). So far, this is similar to Hull's discussion. However, Eldredge and Gould have gone further than this, and have argued that speciation events *might* be "essentially random" with respect to long term evolutionary changes (Gould & Eldredge 1977), an idea they attribute (perhaps misleadingly) to Sewall Wright. If this is the case, then the decisive selection events in evolutionary history are not between lineages *within* an interbreeding population (or between genes in a shared gene pool) *at all*; rather they take place between lineages which are *reproductively isolated* from each other—separate species in effect. Now, if this is the case, then, by definition, the relevant genetic differences are not getting rearranged or recombined in sexual reproduction—for the two species are reproductively isolated. In effect we are back to the logic of asexual reproduction and Dawkins' argument is simply not applicable. However, I should point out that, as far as I am aware, neither Eldredge nor Gould have explicitly applied the theory of punctuated equilibrium in the particular way in which I use it here.

I consider that this argument is valid as far as it goes; but its scope is explicitly limited to selection between reproductively isolated lineages. It represents not so much a refutation as an avoidance of Dawkins' argument.

A separate, but related, argument has been proposed by Hull. The idea is that there may well be "genetic variability" at many loci, but this does not mean that most, or even any, of this is significant for selection. Thus, in discussing sexual reproduction between genetically heterogenous organisms, Hull states that "Dawkins would argue that only those segments of the genetic material which remained undisturbed can count as replicators, while I see no reason not to consider the organisms themselves replicators if the parents and offspring are sufficiently similar to each other" (Hull 1981, p. 34); this is in the context of an earlier interpretation of "similar enough" as meaning to "react similarly to similar selection pressures" (Hull 1981, pp. 32–33). Elsewhere, Hull makes the more explicit claim that "Much of the genetic heterogeneity present in populations has little or no phenotypic effect", (Hull 1980, p. 321). Thus (though Hull does not spell this out) it may well be that there is, at any given time, at most *one* genetic locus at which the variability is correlated with significant differences in S-value of the S-lineages that are labelled by the gene at that locus.

We can distinguish two sub-cases here. There may be genetic variability which, though empirically detectable, simply has no significant effect at all, in the normal environment of the D-actor. If it has no effect at all, it clearly has no selectively significant effect. An alteration of a codon to another synonymous codon (i.e. such that the cistron still codes for the same protein) would seem to be a possible candidate for this category. The second possibility is that the genetic variability has some effect, but this effect is either neutral or balanced with respect to selection. For example, a balanced genetic polymorphism (such as the male/female dimorphism) represents genetic variability in the population, but there is no on-going "selection" (in the sense of displacement of one S-lineage by another) going on. Neglecting the possibility of non-linear interactions between the dynamics of S-lineages anchored on different genetic loci (i.e. neglecting the possibilities of linkage and/or epistasis)—for the moment at least—this variability will not be affected by selection at any other locus, but will remain (statistically) constant. It is thus not significant from the point of view of S-classification, and can be factored out.

In both cases, the conclusion is that if, for whatever reason, selection is only occurring with respect to a single genetic locus at any given time, then standing genetic "variability" at other loci is irrelevant (to S-classification) and cannot be used as a basis for arguing that whole genomes, or more inclusive entities, are not "replicated".

In effect, Hull here introduces the idea that S-classification need not respect *all* "genetic" differences (i.e. all differences of DNA base sequence), but can (indeed must) be limited to recognising only those differences which identify *selectively* distinguished S-lineages. In this way it becomes much more plausible that the significant or relevant genetic variability may be limited to a single locus—in which case Dawkins' argument from disruption through recombination no longer applies.

Again, however, while this refutation is valid as far as it goes, it does not seem to go far enough. It *still* succeeds only by avoiding rather than confronting Dawkins' argument. We would

prefer a refutation which works even in the case of concurrent selection at multiple loci—the case where Dawkins' argument would seem to be at its strongest. We must now face up squarely to this case.

By hypothesis then, we suppose that there is selectively significant genetic variability in the population, at a number of loci. If we attempt to consider genomes (or organisms) as D-actors, S-classifying them on the basis of *all* these loci, then, due to the fragmenting effect of meiosis, every genome (and organism) will, with overwhelming probability, be in a unique S-class. Thus, S-lineages cannot form, and selection cannot arise. Contrariwise, if we consider individual genes as D-actors they will not be disrupted by meiosis, S-lineages can form, and we can see selection in action. This is the thrust of Dawkins' argument that *only* genes can qualify as D-actors in this case.

The fundamental defect in Dawkins' argument can now be identified: it is the (implicit) assumption that, if genomes or organisms are to be considered as candidate D-actors, they must be S-classified on the basis of *all* (variable) genetic loci. But there is no compelling reason why S-classification should be restricted in this way. This point has already been analysed at length in section 5.1 above, and the analysis will not be repeated here. In brief, the relevant point is that we can (for example) identify genome (or organism) S-classes relative to just a single locus; indeed, this is precisely the typical practice of evolutionary biologists. Genome (or organism) S-lineages identified in this way will necessarily show essentially the selective dynamics which Dawkins assumes for gene S-lineages. Granted, the genome (or organism) S-lineages anchored on distinct genetic loci will intersect in ways which the gene S-lineages do not, which *may* make the state descriptions in terms of genomes (or organisms) somewhat more complicated; but it doesn't seem to me that this has any deep-seated significance.

Dawkins has not, as far as I am aware, expressly recognised, much less disputed, this point of view. I therefore consider that, (*pro tem*) it offers a decisive refutation of this particular argument of Dawkins'; indeed, it may be said to subsume the valid core of all the other attempted refutations considered above.

This establishes the general equivalence or interchangeability of gene, genome, and organism (for example) as candidate D-actors. However, before finally closing this discussion I should now like to push one step further and argue that, in fact, organisms may be *preferable* candidates (under the assumption of sexual reproduction). This argument has already been outlined in section 5 above. My purpose here is to specifically review how this issue has been dealt with by Dawkins himself, and by his critics.

Consider first the following criticism offered by Gould:

> No matter how much power Dawkins wishes to assign to genes, there is one thing that 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. A gene is a bit of DNA hidden within a cell. Selection views bodies. It favours some bodies because they are stronger, better insulated, earlier in their sexual maturation, fiercer in combat, or more beautiful to behold.
>
> Gould (1980, Essay 8, p. 76)

Gould goes on to assert that Dawkins' view could (only) work if there were some kind of "one-to-one" mapping between genes and tangible, external, bodily manifestations; and of course, there is no such mapping.

Dawkins has taken the opportunity of a new edition of *The Selfish Gene* to respond to this and similar criticism by quoting, at length, from the original edition, his repeated rejection of any such "genetic atomism", (Dawkins 1989b, pp. 271-2). Dawkins argues, correctly in my view, that interaction between genetic loci in their correlation with phenotypic effects does not, *in itself,* undermine his analysis in the slightest.

I suggest that Gould actually had the correct idea here, but it unfortunately got fatally sidetracked. It is true that, *if* there is a one-to-one mapping between genetic loci and selectively significant phenotypic effects, it is quite possible (likely even) that Dawkins' genes-eye view can be validated. *But the converse does not follow.* The fact (as biological fact it seems to be) that there is no such one-to-one mapping does not, of itself, mean that the genes-eye view is at all problematic.

I suggest that the crucial question here is precisely that of *non-linear* interaction between the S-size dynamics of S-lineages anchored on distinct genetic loci. It seems to me that this is only loosely related, if at all, to the question of whether the mapping between genetic loci and phenotypic traits is one-to-one, and that Gould was mistaken in compounding the two. It is quite possible to envisage very complex interactions between genes, but which still combine linearly in their effects on S-value. In

such cases (and only such cases) it is precisely true that the outcome of selection can be represented in terms of selective forces acting *directly* (and I mean this as literally as is possible in the context) on individual genetic loci—*pace* Gould.

I think that this point—that the genes-eye is perfectly compatible with arbitrarily "complicated" interaction effects, *provided* that they do not give rise to non-linearity in the coupling between S-lineages anchored on different loci—has been central to at least some of Dawkins' analysis. In particular, it is implicit in his *initial* formulation of an analogy for natural selection based on the selection of a rowing crew:

> One oarsman on his own cannot win the Oxford and Cambridge boat race. He needs eight colleagues. Each one is a specialist who always sits in a particular part of the boat—bow or stroke or cox etc. Rowing the boat is a cooperative venture, but some men are nevertheless better at it than others. Suppose a coach has to choose his ideal crew from a pool of candidates, some specializing as cox, and so on. Suppose that he makes his selection as follows. Every day he puts together three new trial crews, by random shuffling of the candidates for each position, and he makes the three crews race against each other. After some weeks of this it will start to emerge that the winning boat often tends to contain the same individual men. These are marked up as good oarsmen. Other individuals seem consistently to be found in slower crews, and these are eventually rejected. But even an outstandingly good oarsman might sometimes be a member of a slow crew, either because of the inferiority of the other members, or because of bad luck—say a strong adverse wind. It is only *on average* that the best men tend to be in the winning boat.
>
> Dawkins (1976, p. 40)

While Dawkins does not explicitly refer to "linearity" (or even "additive" effects) here, I suggest that this particular formulation is clearly based on an assumption that, in fact, the performance of a crew is linearly related to characteristics of its members.

Let us stipulate then that, in the absence of non-linearity, the genes-eye view is perfectly satisfactory (and may even offer some formal simplification relative to viewing genomes or organisms as the D-actors, at least for diploid or polyploid organisms). The question is to assess the significance of non-linearity for the dynamics of selection, under sexual reproduction.

As with most questions dealing with non-linear dynamical systems, there are no simple or universal answers, though we may be able to make progress by considering special cases. As far as I am aware, there are essentially just two relevant special cases which have been considered in the literature, though they are not all that easy to characterise precisely.

I consider first the case introduced by Dawkins, via an extension of his rowing analogy:

> Suppose it is important in a really successful crew that the rowers should coordinate their activities by means of speech. Suppose further that, in the pool of oarsmen at the coach's disposal, some speak only English and some speak only German. The English are not consistently better or worse rowers than the Germans. But because of the importance of communication, a mixed crew will tend to win fewer races that either a pure English crew or a pure German crew.
>
> The coach does not realize this. All he does is shuffle his men around, giving credit points to individuals in winning boats, marking down individuals in losing boats. Now if the pool available to him just happens to be dominated by Englishmen it follows that any German who gets into a boat is likely to cause it to lose, because communications break down. Conversely, if the pool happened to be dominated by Germans, an Englishman would tend to cause any boat in which he found himself to lose. What will emerge as the overall best crew will be one of the two stable states—pure English or pure German, but not mixed.
>
> Dawkins (1976, p. 91)

Again, Dawkins does not explicitly refer to linearity here; indeed, he generally phrases his discussion of this issue in terms closer to *frequency dependent selection*. This is presumably because he expresses and justifies his conclusions by reference to Maynard Smith's *Evolutionary Game Theory*, which is, indeed, typically introduced in just these terms (Maynard Smith 1989, Chapter 7). My understanding of the situation is that, under sexual reproduction, non-linear interactions between the dynamics of S-lineages anchored on distinct loci can give rise

to a form of frequency dependent selection; and that this is, in fact, the implicit mechanism in Dawkins' discussion of the issue; in particular, it is the mechanism in the mixed-language rowing crew analogy quoted above. I think it may be better to explicitly identify the rôle of non-linear interactions (between loci) in this context, as frequency dependent "selection" *can* also arise in the context of a single genetic locus.

In any case, in the particularly simple circumstances described by Dawkins above, the outcome is relatively clear—even though the mechanism, and the detailed dynamics, may be rather complicated. Dawkins attempts to generalise this lesson as follows:

> The interesting question is what makes a gene good. As a first approximation I said that what makes a gene good is the ability to build efficient survival machines—bodies. We must now amend that statement. The gene pool will become an *evolutionarily stable set* of genes, defined as a gene pool that cannot be invaded by any new gene. Most new genes that arise, either by mutation or reassortment or immigration, are quickly penalized by natural selection: the evolutionarily stable set is restored. Occasionally a new gene does succeed in invading the set: it succeeds in spreading through the gene pool. There is a transitional period of instability, terminating in a new evolutionarily stable set—a little bit of evolution has occurred. ... Progressive evolution may be not so much a steady upward climb as a series of discrete steps from stable plateau to stable plateau.
>
> Dawkins (1976, pp. 92–93)

This is obviously not a formal or quantitative analysis; but, informally, Dawkins' suggestion seems to be that, in some cases at least, non-linearity can have the effect of stabilising the presence of certain genetic combinations in the population—which is to say, preventing the kind of concurrent, gene-level, selective displacements *at multiple loci* that might otherwise be expected. It is thus difficult (if not impossible) for concurrent selection (at least in the sense of displacement) to occur at multiple loci; but it still leaves open the possibility for selective displacement to occur at any *single* locus at one time, coupled with *sequential* displacements at different loci. *This* kind of single locus selection dynamics (possibly associated with a shift in the dynamic equilibrium at other loci) can operate in essentially the same manner as in the absence of non-linear interactions.

But if this is truly a fair interpretation of Dawkins' analysis, then it undermines precisely his strongest case. The positive argument for regarding genes as D-actors (under sexual reproduction) seemed to be that it could provide a somewhat simpler formal description of concurrent selection at multiple loci. If the selection events of long term significance (i.e. selective displacements) at distinct loci are constrained by non-linearity to be sequential rather than concurrent, then this argument loses most if not all of its force. For selection going on at only a single locus, the genes-eye view offers little, if any, formal simplification over a view based on genomes or organisms; in fact, in these circumstances, the latter viewpoints will not even be marred by the need to track intersecting S-lineages anchored on different loci. Moreover, the distinctive S-value associated with a particular gene, at a particular locus, will (as always) only be manifest by taking account of the particular genetic background prevalent in the population—which is to say by looking at whole genomes or organisms.

Notwithstanding all this, Dawkins still states a preference for the genes-eye view; but his argument now seems to become much weaker, or even obscure. Thus, in a final version of the rowing analogy, he could conclude only that he found it more "parsimonious" to think of the coach selecting at the level of the independent candidates rather than whole crews (Dawkins 1976, p. 92). On the face of it this is a substantive retreat from his original claim that *only* genes could qualify as D-actors, under sexual reproduction.

Even this is not the final conclusion however. If I have interpreted him correctly, Dawkins has suggested one possible view of the significance of this kind of non-linearity for the phyletic evolution of sexual populations—namely that it may force selective displacements at multiple loci to occur sequentially rather than concurrently; but it is by no means clear that this is the only possibility. Wright's Shifting Balance theory (e.g. Wright 1982) provides another, quite different, possibility. Roughly speaking, Wright suggests the possibility that genome or organism S-lineages, distinguished from each other (S-classified) by differences at *multiple* genetic loci, may selectively displace each other—and, indeed, that this may be the predominant process in phyletic evolution. While such selection processes *could* be formally modelled at a gene level, such a description would necessarily *obscure* the selection process,

relative to a description at the genome or organism level. That is, under shifting balance, genomes or organisms actually provide a significantly *simpler* (and thus preferable) formal description of selection, as compared to a gene level description.[13]

Wright has presented essentially this criticism of genic selectionism, as a specific response to Dawkins, in his paper *Genic and Organismic Selectionism* (Wright 1980). Dawkins is certainly aware of this paper, having cited it in, for example, (Dawkins 1982a) and (Dawkins 1983). The former is worth quoting at some length:

> I think this [Wright 1980] is a valuable paper, even though its ostensible purpose is to attack the view that 'with respect to natural selection ... it is the gene, not the individual or group, that is the unit'. Wright concludes that 'The likelyhood of organismic, instead of merely genic, selection goes far toward meeting one of the most serious objections to the theory of natural selection encountered by Darwin.' He attributes the 'genic selection' view to Williams, Maynard Smith and me, and traces it back to R.A.Fisher, I think correctly. All of which must lead him to be somewhat bemused by the following accolade from Medawar [Medawar 1981]: 'The most important single innovation in the modern synthesis was however the new conception that a population that was deemed to undergo evolution could best be thought of as a population of fundamental replicating units—of genes—rather than as a population of individual animals or of cells. Sewall Wright ... was a principal innovator in this new way of thinking...'
>
> Dawkins (1982a, pp. 238–239)

At face value, this passage seems to dismiss Wright's attack on genic selectionism simply on the basis that a third party (Medawar) has identified Wright as one of the originators of genic selectionism in the first place; which is to suggest that Medawar knows what Wright thinks better than Wright himself. This is not an argument, and Dawkins cannot mean to rely on it.

[13]It should be emphasised (as Wright himself has done) that the shifting balance process is *not* a form of "group selectionism", as normally construed; and arguments against group selectionism are not *ipso facto* arguments against shifting balance. For a more detailed discussion of this point see, for example, Maynard Smith (1989, Chapter 9).

Dawkins goes on:

> In the rest of this chapter, I hope to show that the version of 'genic selectionism' that can be attacked as naively atomistic and reductionistic is a straw man; that it is not the view that I am advocating; and that if genes are correctly understood as being selected *for their capacity to cooperate* with other genes in the gene-pool, we arrive at a theory of genic selection which Wright and Mayr will recognize as fully compatible with their own views. Not only compatible but, I would claim, a truer and clearer expression of their views.
>
> Dawkins (1982a, p. 239)

Dawkins indicates here that he is going to rebut, or at least disarm, arguments (against genic selectionism) presented by both Wright and Mayr. Unfortunately, that is the last explicit reference to Wright which appears in the chapter (or the book, for that matter)—Dawkins' subsequent treatment deals explicitly only with points raised by Mayr. I shall not consider any details of the latter, for I have no strong disagreement with Dawkins on those issues (indeed, Dawkins' own claim is that he himself has no substantive disagreement with Mayr). The significant point is that the structure of Dawkins' presentation here seems positively misleading. Although he has promised to confront Wright's argument, he never does so—or, at least, not *explicitly*. One must suppose that Dawkins intends his comments on Mayr to also apply *mutatis mutandis* to Wright, but I am not convinced that this can be made to work.

More specifically, Dawkins makes no mention of the shifting balance process *per se*, whereas it is central to Wright's criticism. The point which Dawkins emphasises repeatedly is that genes should be understood as being selected for their "capacity to cooperate"—but this seems to mean, in each particular case, cooperation with whichever genes *already* dominate the gene pool. If this is a fair interpretation, then it misses Wright's claim that, under shifting balance, there may be evolutionarily significant selection events which simply cannot be decomposed into such separate "selection" of individual (sexually segregating) genes—in the sense that genes at distinct loci are either selected *as* a co-dependent set (in competition with alternative, allelomorphic, co-dependent sets) or not at all. If I have grasped it correctly, Wright's point is that, with a suitable population structure, this can be an effective

process *despite* the ongoing disruption of the co-dependent set through recombination. By contrast, Dawkins seems to hold that such concurrent selection of a co-dependent set can arise only if disruption through recombination can be neglected—which is to say, "only if the genes ... were linked tightly in a supergene" (Dawkins 1982a, p. 244). This, of course, would effectively mean a return to the gene's eye view, except now in terms of a "supergene". Dawkins seems to be implicitly denying that the shifting balance process can be effective—but without giving specific arguments as to why not.

In terms of the rowing analogy again, shifting balance envisages a situation somewhat like the following. Suppose that, for whatever reason, a mixed crew is always better if there are more English rowers in it, but that an all German crew will beat an all English crew. Suppose, further, that the initially selected crew is all English. Now all perturbations of this, short of forming an all German crew, will make it worse, and in Dawkins' view of evolution it would seem that an all German crew could never become established; and even if it were, it would be immediately broken up. Wright's point is that, if we made the model more biologically plausible by imagining that a population of such crews existed, all initially dominated by English speakers, but geographically dispersed, with limited migration (of whole crews), and that they "reproduced" with "recombination", then a statistical fluctuation might allow a small, but viable, subpopulation of all German crews to become established in some local geographical area; this would then be able to expand and selectively eliminate the all English crews. This is a very rough and qualitative argument, and I have omitted to specify many details which would be necessary to make it work; but if, as Wright suggests, some such outcome is possible at all, it seems that it simply cannot be interpreted in terms of individual crew members being selected (for their "ability to cooperate" or otherwise).

To conclude finally on the relevance of sex to the candidacy of D-actors: under sexual reproduction with meiosis, individual genes *may* qualify as candidate D-actors; in certain circumstances they may even be preferred candidates; however, in all the cases considered, genomes (and organisms) are viable alternative candidates; and in some cases (notably under Wright's shifting balance process) genomes (and organisms) are *much* preferable candidates. Dawkins' simple claim that, as a logical consequence of meiosis, genes should be decisively favoured as candidate D-actors, must be rejected.

7.2.2 Causal Arrows...

In the previous section I considered whether genomes (and organisms) could, in general, be regarded as satisfactory candidate D-actors. I now come to a superficially similar, but actually quite different question, which is whether organisms can qualify as Dawkinsian replicators. Dawkins' answer to this question is 'No'. I shall actually agree with this, which necessarily involves finally identifying the distinction between my *D-actor* and Dawkins' *replicator*. It should then become clear that the substantive question is not whether organisms can qualify as Dawkinsian replicators as such, but whether Dawkinsian replicators play a peculiarly distinguished evolutionary rôle, which cannot be played by D-actors in general.

First I must explain the distinction between D-actors and Dawkinsian replicators. Briefly, both involve a combination of similarity and descent; but Dawkinsian A-replicators have an *additional* property not necessarily exhibited by my D-actors (or Hullean replicators for that matter). This additional property is a constraint on the *mechanism* whereby similarity is achieved (i.e. on how S-class is preserved in S-descent): namely that this mechanism must be one of "copying".

Now, it is not all that easy to formalise exactly what is meant by a "copying" process, and I shall not even try to do so here. For the moment, let me simply stipulate that genes (or even whole genomes, in the case of asexual organisms) are, indeed, "copied" during biological reproduction, whereas organisms (or "phenotypes" generally) are not.

This qualification on the mechanism of S-descent underlies Dawkins' concept of "vehicle". In effect, both Dawkinsian A-replicators *and* Dawkinsian vehicles qualify as D-actors in my terms—which is to say that they are alternative candidates for the rôle of D-actor in discussing selection processes.[14] Dawkins concludes, in fact, that his A-replicators should be preferred; whereas I conclude that, if anything, his vehicles should be preferred. But we are both agreed that genes (and perhaps genomes) are Dawkinsian replicators, whereas organisms are Dawkinsian vehicles.

It should be noted at this stage that Dawkins' notion of the abstract *replicator* has, itself, evolved somewhat.

In his earliest explicit consideration of rival can-

[14] Note, again, that this distinction is not related to the relative merits of S-lineages versus D-actors as the "unit of selection", but is purely about the relative merits of two candidate D-actors.

didates for the rôle of A-replicators Dawkins accepted that *certain* organisms could play this part (Dawkins 1978a, p. 69).[15] At this point, while he had defined replication in terms of copying, he was placing no particular stress on this fact; in effect, his replicator was still more or less synonymous with my D-actor, requiring only similarity and descent to characterise it.

However, Dawkins subsequently changed his mind, deciding that nothing more inclusive than the "genome" could ever operate as an A-replicator (Dawkins 1982b, p. 50). This applied to organisms as such (whether sexual or asexual), and generally to any more inclusive entities. It was at this point that he identified *copying* as an *essential* ingredient of replication, and started to claim a unique evolutionary rôle for replicators in this more restricted sense (i.e. restricted relative to D-actor, or Hullean replicator). It is precisely his basis for this step which is the target for critical discussion here.

Dawkins has summarised his argument as follows:

> To regard an organism as a replicator, even an asexual organism like a female stick insect, is tantamount to a violation of the 'central dogma' of the non-inheritance of acquired characteristics. A stick insect looks like a replicator, in that we may lay out a sequence consisting of daughter, granddaughter, great-granddaughter, etc., in which each appears to be a replica of the preceding one in the series. But suppose a flaw or blemish appears somewhere in the chain, say a stick insect is unfortunate enough to lose a leg. The blemish may last for the whole of her lifetime, but it is not passed on to the next link in the chain. Errors that affect stick insects but not their genes are not perpetuated. Now lay out a parallel series consisting of daughter's genome, granddaughter's genome, great-granddaughter's genome, etc. If a blemish appears somewhere along *this* series it will be passed on to all subsequent links in the chain, because in each generation there are causal arrows leading from genes from body. But there is no causal arrow leading from body to genes. No part of the stick insect's phenotype is a replicator. Nor is her body as a whole.
>
> Dawkins (1982a, p. 97)

The reference here to "causal arrows" should presumably not be taken too literally. It is manifestly the case that there *are* "causal" relationships directed from organisms to their genomes, as evidenced (for example) by the fact that a naked genome, in the absence of some minimal set of phenotypic products (including at least the enzymes associated with its replication), cannot actually replicate. But let us accept Dawkins' substantive point: there exist possible modifications of genomes which, if they arise, will be preserved in offspring, whereas this is not the case for modifications which do not affect genomes. To put it another way (a way which does not rely on causal "arrows"), it is true that non-genetic modification may result in *some* modification of the offspring—but such resulting modification of the offspring will not be the *same* modification as that originally applied to the parent. The *only* kind of modification which can persist through generations is a modification including, or anchored upon, a genetic modification.[16]

Accepting all this, the question which arises is what significance should we attribute to these facts. Dawkins' view clearly is that entities which have this property—that (specified) modifications to them will be preserved in subsequent generations—play some unique evolutionary rôle, which cannot be played by any entity lacking this property. But what *is* this unique rôle? Dawkins does not, unfortunately, spell it out explicitly, and we must therefore be satisfied with attempting more or less plausible inferences.

The first possibility is this: Dawkins may be claiming simply that any D-actor must incorporate what is, in effect, some kind of "information storage" mechanism (whose information content is duplicated

[15] Specifically: those organisms employing asexual reproduction. This restriction to asexual reproduction was to take account of the argument relating to fragmentation of the genome at meiosis, detailed in the previous section. On my analysis, such a restriction is now seen as an unnecessary distraction.

[16] Actually, it seems to me that even this is probably not *strictly* correct. If it is accepted that the mapping from genotype to phenotype is, in some respects, *arbitrary*, then, by implication, there exist alternative, self-consistent, phenotypes which could viably correspond to any given genotype. It follows that a "modification" of any one of these into any other would be a purely phenotypic modification which would be preserved in subsequent generations. However, the "modification" in question now amounts to a thorough rebuilding of the existing phenotype, and could not conceivably arise by a process of unjustified variation (compare the more detailed discussion in Hofstadter 1985, Chapter 27). Thus, I happily stipulate that the only modifications which need be practically considered in a properly Darwinian evolutionary process are just those which Dawkins calls genetic.

in reproduction) in order to "stabilise" the preservation of S-class in S-descent. This information storage mechanism is then, precisely, what Dawkins calls a *replicator* (I should note that there seems no intrinsic reason which this mechanism should be "neatly" separable from the rest of the putatively "reproducing" entity). This is an interesting claim, which I am inclined to accept. In fact, I believe that the credit for this insight, and its important ramifications for the possibility of a Darwinian growth of "complexity", is properly due to John von Neumann (1966); but that is not a discussion which I wish to pursue in the current context. For my present purposes, in trying to contrast different candidate D-actors, it still amounts simply to focusing on the requirement that S-class be preserved in S-descent, while more or less ignoring the requirement that S-class be predictive of S-value (in given environmental conditions). As discussed in section 5.2 above, this might sometimes yield a pragmatic preference for a gene-level choice of D-actor, but it is, at best, a weak preference, contingent on the particular circumstances. In any case, this pragmatic interpretation of Dawkins' position is not actually sustainable. In his general development of the replicator concept, he has stipulated that it is "active" replicators which are of evolutionary significance—and "active" replicators are precisely entities whose characteristics are not just preserved in reproduction but are predictive of S-value also.

The second possible interpretation is that Dawkins means to propose what I have earlier called *genetic absolutism*. That is the view that there is an absolute mapping from genotypes to phenotypes. If this were so, then, since we grant that all evolutionarily significant changes *include* genetic changes, the entire evolutionary story can be told in terms of genomes alone—the phenotypic implications can be "calculated" back in at any time, if desired. I repeat that this does not involve any claim that the genotype-phenotype mapping be deterministic (or "atomistic" for that matter).

I do not believe that Dawkins really means to advocate genetic absolutism; but there is no doubt that he implies something very close to this in much of his writings, and he could easily be (mis-?)understood in this way. Consider the following passages, for example:

> We are survival machines—robot vehicles blindly programmed to preserve the selfish molecules known as genes.
>
> Dawkins (1976, p. ix)

> What weird engines of self-preservation would the millenia bring forth? Four thousand million years on, what was to be the fate of the ancient replicators? They did not die out, for they are past masters of the survival arts. But do not look for them floating loose in the sea; they gave up that cavalier freedom long ago. Now they swarm in huge colonies, safe inside gigantic lumbering robots, sealed off from the outside world, communicating with it by tortuous indirect routes, *manipulating it by remote control.* They are in you and in me; they created us, body and mind; and their preservation is the ultimate rationale for our existence. They have come a long way, those replicators. Now they go by the name of genes, and we are their survival machines.
>
> Dawkins (1976, p. 21, emphasis added)

> The genes too *control* the behaviour of their survival machines, not directly with their fingers on puppet strings, but indirectly like the computer programmer.
>
> Dawkins (1976, p. 56, emphasis added)

This last quotation is particularly suggestive. It is excerpted from a longer discussion, in which Dawkins compares genes (or, more generally, replicators) to computer programs, saying *inter alia* that a program "is not fussy which physical computer it uses to act out its skills" (Dawkins 1976, p. 55). This is, of course, true as far as it goes, but could easily be read as meaning that computer programs have *intrinsic* or absolute interpretations, which are merely realised by plugging them into *any* (arbitrary) computer. This would be nonsense—programs have well defined meanings or behaviours only relative to a specified computer (or class of computers). In terms of the analogy to genes, we should imagine instead the possibility of programmed computers which, in executing their programs, actually cause the construction of new computers, and also equip such offspring computers with a copy of the parental program.[17] The important point is that, following a "mutation", the offspring computer may differ from the parental computer, so that even though the offspring has (more or less) the "same" program (in

[17] Again, this idea was essentially pioneered by von Neumann (1966).

some kind of *informational* or *syntactic* sense), its *function* may be arbitrarily different. Thus, an evolutionary story expressed solely in terms of the lineage of computer *programs* would definitely not allow the "phenotypic" details to be calculated back in.[18]

I should emphasise here that Dawkins consistently recognises that individual (sexually segregating) genes do not have absolute interpretations or meanings; unfortunately, in doing so, he can seem to imply that, by contrast, complete genomes, in given (extra-organismic) environmental conditions *do* have absolute interpretations. Thus, we have the following, for example:

> ... the relationship between a gene and its phenotypic effect is not an intrinsic property of the gene, but a property of the forward developmental consequences of the gene when interacting with the consequences of many other genes and many external factors.
>
> Dawkins (1982a, p. 176)

Dawkins explicitly states that the effect of any one gene is contingent on the other genes in the genome, and on "external" (presumably extra-*organismic*?) factors, thus implying, by omission, that it does *not* depend on any non-genetic, but internal (organismic) factors; which is to say, he again implies some form of genetic absolutism.

Dawkins most definite commitment to something very like genetic absolutism appears in his book *The Blind Watchmaker*, where it implicitly underlies his so-called *biomorph* computer model of evolution:

> There is another mathematical space filled, not with nine-gened biomorphs but with flesh and blood animals made of millions of cells, each containing tens of thousands of genes. This is not biomorph space but real genetic space. The actual animals that have ever lived on Earth are a tiny subset of the theoretical animals that *could* exist. These real animals are the products of very small number of evolutionary trajectories through genetic space. The vast majority of theoretical trajectories through animal space give rise to impossible monsters. Real animals are dotted around here and there among the hypothetical monsters, each perched in its own unique place in genetic hyperspace. Each real animal is surrounded by a little cluster of neighbours, most of whom have never existed, but a few of whom are its ancestors, its descendants and its cousins.
>
> Dawkins (1986, p. 73)

Dawkins' clear implication here is that there is some universal mapping from genomes to "animals" (phenotypes). He is perhaps even more explicit in a more or less parenthetical discussion in *The Extended Phenotype*, where he essentially claims that if a genetic change is reversed the phenotypic consequences will necessarily be reversed also (Dawkins 1982a, pp. 3–4); but, as already commented upon in section 5.4, this conclusion would seem to be warranted only under an assumption of genetic absolutism.

In any case, my objective here has simply been to establish that Dawkins can reasonably be interpreted as espousing genetic absolutism. The arguments *against* such a view have already been adequately rehearsed in section 5.3 and need not be repeated here. The more interesting question is whether there is any basis for supposing that Dawkins should *not* be interpreted in this way. As it happens, there is at least fragmentary evidence to this effect. Thus, in Dawkins' more recent discussion of his *biomorph* model, we find the following:

> ... we need to make a preliminary distinction between two kinds of mutation: ordinary changes within an existing genetic system, and changes to the genetic system itself. Ordinary changes within an existing genetic system are the standard mutations that may or may not be selected in normal evolution within a species. One allele is replaced by an alternative allele at the same locus, as in the famous case of industrial melanism where a gene for blackness spread through moth populations in industrial areas (see any biology textbook). This is how all normal evolutionary change happens. But it is an inescapable fact that different species, to a greater or lesser extent, have different genetic systems from one another, even if this only means that they have different numbers of chromosomes. "The same locus," when we are talking about an elephant and a human, may not even be a meaningful thing to say. Humans and elephants employ basically the same *kind* of genetic system, but they don't have

[18] It should also, incidentally, be clear from this discussion that a phenotype cannot be considered merely as part of the "environment" of the genotype.

> the same genetic system. They have different numbers of chromosomes and you can't make a locus-for-locus mapping between them like you can between two individual humans. Yet humans and elephants undoubtedly have a common ancestor. Therefore, during their evolutionary divergence, there must have been changes to the genetic systems, as well as changes within the genetic systems. These changes to genetic systems must have been, at least in one sense, major changes, changes of a different order from the normal allele substitutions that go on within a genetic system.
>
> Dawkins (1989a, p. 217)

Dawkins is here coming very close to renouncing genetic absolutism, which I naturally agree with. However, I should note that I disagree with Dawkins on several substantive details.

Thus, while I agree that humans and elephants almost certainly have different "genetic systems", I disagree that this can be reliably inferred merely from the fact that there have different numbers of chromosomes (by analogy, computer programs may differ widely—length, organisation and number of subprograms etc.— and yet be still valid, or functional, programs for the same computer). Indeed, attempting to distinguish "genetic systems" by appeal to purely genetic differences (such as chromosome number) is essentially to relapse back into genetic absolutism.

There again, while agreeing with Dawkins that there must have been evolutionary changes to "genetic systems" I reject his suggestion that genetic changes might be neatly divided into those which affect this "genetic system" and those which do not. I think Dawkins has, in this case, been badly misled by the simplifications inherent in his computer models. In the latter it is easy (even convenient) for the "genetic system" to be a distinguishable subsystem of his actors, "coded for" by a definite fragment of the genome. But there is no reason to suppose that real organisms incorporate any such neat subdivisions. Instead, given the prevalence of pleiotropic effects, I conjecture that many (perhaps even most) genetic modifications have *both* kinds of effect identified by Dawkins ("changes within an existing genetic system" and "changes to the genetic system itself"). If this conjecture is even close to the truth then Dawkins' attempted distinction between "normal" evolutionary steps, and steps "of a different order" is mistaken and misleading. What Dawkins would regard as "normal" evolutionary steps could actually have the automatic side-effect of changing ("incrementally" or otherwise) the "genetic system".

I have elaborated my detailed disagreement with this passage of Dawkins' for a definite reason. Taking the passage as it stands, the implication is that, in discussing biological evolution, we need to track both genetic changes, as such, and the "genetic system" which is appropriate to any given genome. Dawkins implies that this "genetic system" can be thought of as constant (read "absolute") for the purposes of considering most ("normal") evolutionary change; and that it can be distinguished as a subsystem of the organism (I again ignore the counterproductive idea that the "genetic system" could itself be inferred from the genome). If Dawkins grants even this much, he would surely admit that a "minimal sufficient" candidate D-actor must consist of a genome plus its genetic system (as opposed to a genome on its own). To my knowledge, Dawkins has not recognised this implication. But, in any case, if my further qualifications are valid—that the "genetic system" is neither constant over long periods of evolutionary time, nor a neatly distinguishable subsystem in itself—then one will be forced to recognise essentially the whole *organism* as being the best available approximation to a minimally sufficient D-actor.

The thrust of this last argument has been to suggest that, if Dawkins does *not* embrace genetic absolutism, then the only possible, remaining, interpretation of his position would seem to be some form of genetic relativism. But the above analysis, derived from Dawkins' own admissions about the maleability of "genetic systems", essentially retraces the rejection of genetic relativism already presented in section 5.4. I conclude that Dawkins' concept of replicator, *insofar as it differs from my concept of D-actor,* is a misleading distraction, and should be dispensed with.

7.2.3 A Resolution?

There is, however, one final comment worth making here. While I have gone to some length to trace Dawkins' arguments for some kind of preeminence of the genetic D-actor, it seems to me that Dawkins himself is ultimately ambivalent about this. This may be illustrated by the following two highly suggestive quotations:

> It is the incidence of phenotypes that we are interested in explaining, not the incidence of molecular configurations of DNA. And if any reader thinks that last remark

> contradicts my basic thesis, I must have failed to make my basic thesis clear.
>
> Dawkins (1982a, p. 154)

> 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.
>
> Dawkins (1982b, pp. 59–60, emphasis added)

If this last passage is read in the light of a rejection of genetic absolutism, then it identifies a Dawkinsian replicator *plus* some attached "suite of characters", as the preferred candidate for the rôle of D-actor. On *this* interpretation, Dawkins and I would finally speak with one voice.

8 Conclusion

This has been a long and technical essay. In conclusion I should like to try to distil out the central point more concisely.

We have two apparently complementary and exhaustive ways of looking at the evolutionary rôles of genes and organisms:

1: Organisms subserve Genes. This is Genic Selectionism in its purest form. It holds that genes have no "function" other than to ensure their propagation into future generations—i.e. to replicate—and organisms are merely tools or "vehicles" constructed and maintained by the genes to facilitate this process. The most explicit and provocative proponent of this view has been Richard Dawkins—hence the extended discussion devoted to his ideas in this essay.

2: Genes subserve Organisms. This is Organismic Selectionism in its purest form. It holds that organisms have no function other than to ensure their propagation into future generations, and genes are merely a tool (an information storage mechanism) constructed and maintained by the organisms to facilitate this process.

My claim is that this is ultimately a false and sterile dichotomy; that both claims are better replaced by the following synthesis:

3: Organisms and Genes both subserve S-lineages. That is, neither genes nor organisms have functions in themselves other than to facilitate the success of S-lineage(s) of which they are members, in competition with other S-lineage(s). This is the point of saying that the S-lineage is the unit of selection.

Both genes and organisms may qualify as more or less satisfactory D-actors, depending on the precise circumstances. But *if* we wish to identify the *minimal sufficient* D-actor, then genes (and genomes) definitely do not qualify, and organisms are probably the uniquely best approximation which is typically available.

Does this actually make any difference? Well, quite apart from any aesthetic appeal of this formulation, I think that the forthright, detailed, and comprehensive, rejection of Genic Selectionism, which I have attempted here, is a necessary prerequisite for the *satisfactory* realisation of artificial Darwinism. For Genic Selectionism is strongly linked to Genetic Absolutism; and Genetic Absolutism reduces the Darwinian evolution of adaptive complexity to a trajectory through a predetermined genetic vector space. By contrast, real Darwinian growth of adaptive complexity (and, by implication, any worthwhile artificial growth of adaptive complexity) is just not that simple.

In closing, there is one last crucial point to be made. I have argued elsewhere that Organismic Darwinism (where organisms are treated as D-actors) offers a more or less satisfactory solution to the problem of the phylogenetic growth of organismic complexity (McMullin 1992b). I have argued here that, to the extent that that previous analysis was correct, then it may, in some cases at least, be equally satisfactory, and essentially equivalent, to regard certain other entities, especially genes or genomes, as D-actors—although I have argued that the organism level is still the most generally satisfactory choice. But I must emphasise now that I do not pretend that the organismic view offers, in any sense, a complete or final explanatory framework for all growth of complexity in the biological world. In particular, even in (McMullin 1992b) I acknowledged that quite distinct new kinds of D-actors *could* emerge, which would *not* be formally equivalent to organismic D-actors; and that the distinct D-systems which result might interact in significant ways, which would render any explanation

phrased in terms of only a single kind of such D-actor quite inadequate; and, indeed, that this *may* already have been a significant phenomenon in biological evolution to date. I have in mind here the kind of hierarchical Darwinian theory described, for example, by Gould (1982). In terms of such an hierarchical theory, my purpose has been restricted to the attempted clarification of Darwinian theory within *one* hierarchical level; but I do not imply, and do not suppose, that such a single level theory exhausts the scope of Darwinism.

This is an internal Technical Report; as with the previous essays in the series, I rely on the informality of that medium to excuse the rough edges remaining. In any case, I would greatly appreciate comments and criticism of any sort.

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This series of essays form part of an ongoing attack on the problem of realising the spontaneous growth of Artificial Knowledge by Darwinian (or any other!) means. In this pursuit, I have benefited greatly from discussions with colleagues, particularly Noel Murphy in DCU, and John Kelly of University College Dublin. Paul McKevitt also made useful comments on earlier drafts of this material. I am indebted to the School of Electronic Engineering in DCU (particularly through the agency of its Head, Charles McCorkell) for continuing encouragement, not to mention material support. All errors remain, of course, my own responsibility.

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