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The Replicator in Retrospect

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Abstract. The history and theoretical role of the concept of a "replicator" is discussed, starting with Dawkins' and Hull's classic treatments and working forward. I argue that the replicator concept is still a useful one for evolutionary theory, but it should be revised in some ways. The most important revision is the recognition that not all processes of evolution by natural selection require that something play the role of a replicator.

Key words: interactor, replicator, unit of selection

1. Introduction

David Hull has always headed unerringly for big issues. This drive towards the fundamental is evident in his work on the nature of species, on the most general features of evolution by natural selection, and on the role of social structure in scientific change. Fittingly, given his interest in the social structure of inquiry, Hull has also contributed enormously to the social life and organization of philosophy of biology.

Twenty years ago, Hull's "Individuality and Selection" (1980) introduced his distinction between "replicators and interactors." The distinction has had several roles. One role has been to clear up misunderstandings in the "units of selection" debates. Hull also uses this framework in his selectionist account of scientific change (1988). In the present paper, my main focus will be biological. I will survey Hull's and other central discussions (Section 2), then discuss recent criticisms of the replicator concept (Section 3), and finally (Sections 4 and 5) sketch my own view of how to understand the replicator concept and its role within evolutionary theory.

2. The original role of the replicator/interactor distinction

Let us look first at the debate to which Hull was most directly responding. In *The Selfish Gene* (1976), Richard Dawkins had argued that individual genes must be seen as the units of selection in evolutionary processes within sexual populations. This is primarily because the other possible candidates, notably whole organisms and groups, do not "replicate." Organisms and groups are ephemeral, like clouds in the sky or dust storms in the desert. Only a replicator, which can figure in selective processes over many generations, can be a unit of selection. Dawkins was extending a conception of natural selection initially outlined by G. C. Williams (1966).

One line of reply to Dawkins and Williams, exemplified in Stephen J. Gould's "Caring Groups and Selfish Genes" (1977), was to argue that genes cannot be units of selection because natural selection is not able to "see" (operate on) single genes, only on whole organisms. For natural selection to operate on single genes, organisms would have to be patchworks made up of the separate causal contributions of those genes. And that "patchwork" assumption is incompatible with what we know about gene action and development (see also Mayr 1975).

Lewontin (1970) had argued that natural selection at any level requires variation, heredity and differential fitness. The disagreement between Dawkins and some critics such as Gould can be seen as a matter of people stressing different parts of an agreed-upon recipe for evolution. Dawkins insisted on a stringent conception of what is involved in passing structure across generations. Gould and others required that a "unit of selection" have a certain direct relationship to differential survival and reproduction, a relationship genes usually do not have.

Hull's 1980 and 1981 papers did a lot to clear up this situation. Hull argued that people had been packing into one concept, "unit of selection," criteria associated with two distinct and equally important roles:

> *Replicator*: an entity that passes on its structure largely intact in successive replications.
>
> *Interactor*: an entity that interacts as a cohesive whole with its environment in such a way that this interaction *causes* replication to be differential.

There are Hull's (1988) definitions (p. 408). The 1980 versions use the word "directly" in each definition; a replicator passes on its structure directly, and an interactor interacts with the environment directly (p. 318).

Dawkins also formulated a distinction between "replicators" and "vehicles," intended to clear up some of the same confusions that Hull sought to treat (Dawkins 1982a). Dawkins' view, unlike Hull's, retained the central

place in evolution for replicators. As many remarked, Dawkins' "vehicles" seem subordinate to the replicators; a vehicle "can be regarded as a machine programmed to preserve and propagate the replicators which ride inside it" (Dawkins 1982a, p. 295). Dawkins and Hull also differed over which entities are replicators. For Hull, asexual organisms and sexual organisms in species with low genetic variability can be replicators. At the edge of the concept, even organisms in genetically variable populations can be replicators, if the variation is selectively neutral (Hull 1980, p. 322). Dawkins, on the other hand, denied even that asexual organisms are replicators. Only their genome qualifies (1982b, p. 167).

As Hull said, the question "what is the unit of selection?" is often asked in a confused way. Both the replicator and interactor are relevant "units." This is true even in cases where one entity plays both roles. So for Hull, a "unit of selection" question should always be disambiguated: do you want to know the replicator or the interactor? In familiar cases of natural selection involving whole-animal phenotypes in a polymorphic sexual population, such as industrial melanism in moths, genes are the replicators and whole organisms are interactors. But Hull also stresses that these cases provide a poor model for much of the biological world, where asexuality and complicated mixed systems of reproduction are common.

Hull's definitions take "replication" as a primitive. Dawkins defines replicators as "anything of which copies are made" (1982b, p. 162). Both Dawkins and Hull take copying or replication to be a fairly straightforward concept. Clearly though, the concept of replication they use has two main elements, a *resemblance* between copy and copied, and some suitable *causal* relation linking the copy to the copied. Later I will discuss the details of these relations.

Through the 1980s, most philosophical discussion of units of selection problems was, in effect, discussion of the role of interactor. Only some of this literature explicitly used Hull's framework, but much of the debate can be usefully understood in his terms. In their review of the literature, Sober and Wilson (1994) make only limited use of Hull's distinction. Sober and Wilson express unease with the replicator concept (partly for reasons discussed in section 3 and 4 below). They also think that Hull's interactor concept has not been made precise enough for use in assessing difficult cases where there are rival candidates for the role (p. 539).

Sober and Wilson are right that Hull's formulation of the interactor is hard to apply to problem cases. Along with Lloyd (1988), however, I think the usefulness of Hull's distinction should not be underestimated when sorting through the complexities of the units of selection debates over the past 30 years, especially in the case of group selection.[1]

Philosophical discussions of interactors (whether conducted under that name or not) became embroiled in issues concerning causation and reductionism.[2] If it is acceptable to "average out" all the context-sensitive aspects of a gene's effects, and treat the resulting average as a genuine causal capacity of the gene, then nearly all causal description of evolution can, in principle, be conducted at the gene level. If so, the distinctive role for interactors other than genes in evolutionary explanation is much reduced. But if this averaging distorts the real causal structure, then the reductionist strategy fails. This long discussion of interactors never produced a consensus. Despite the unresolved problems, Hull's replicator/interactor distinction cleared up a good deal of confusion.

Lloyd has also recently carried Hull's diagnostic and therapeutic analysis further (1992, forthcoming). She notes that there has often been another kind of language used about units of selection, language which gestures towards roles other than those of replicator and interactor. Writers often claim that when we have isolated the unit of selection, we have found the entity that a behavior was "for the sake of," or the entity that "benefits" from the presence of some adaptation.

How are these properties linked to those of replication and interaction? Lloyd (1992) isolates two other roles besides Hull's, the roles of *beneficiary* of selection and *owner-of-adaptations*. The beneficiary, for Lloyd, is the entity that "ultimately benefits" from a process of evolution by selection. The owner of an adaptation is described sometimes as the entity which has an adaptation as a part of it, but sometimes as the entity which the adaptation is "for the sake of."

As Lloyd says, intuitions about beneficiaries and owners of adaptations have been important in driving the units of selection debates. For Dawkins, replicators are always the stars in an evolutionary story because replicators are the beneficiaries of selection and, in an ultimate sense, the owners of adaptations. Dawkins allows his description of these roles to become infused with subtle attributions of agency; replicators are the indirect "programmers" of what adaptations do. Dawkins' focus on these roles is the source of a moral and political loading that many have found in his biological writings; this (rather than genetic determinism) is the true source of his description of organisms as "lumbering robots" programmed by their genes (1976, p. 21).

Lloyd is right in her analysis of the role played by intuitions about "beneficiaries" and "owners." But there is a question about the status of these intuitions. I suggest that most of the language of "ownership" and "ultimate benefit" in this context is merely metaphorical; there is no real scientific question about what "owns" an adaptation. In contrast, there are facts of the matter about replication and interaction.

The roles of "owner" and "beneficiary" do have relatives that are not metaphorical. The question of which objects have adapted structures as parts is a factual question, and sometimes Lloyd describes the "owner" role in that way. Do groups ever have adapted structures as parts, where these structures are not parts of any individual organism? Similarly, there is the question of which interactors had their interactions with the environment positively affected by a given trait. Eyes are for enabling organisms to see. That is, whole organisms are the interactors whose dealings with the world have been helped by the presence of eyes, resulting in natural selection favoring eyes. Those are biological facts, and facts which figure in selection-based theories of biological functions.[3] But there is no extra fact about who "owns" the eyes. In her most recent work, Lloyd has switched from talk of "owners" of adaptations to talk of "manifestors" of adaptations. As the manifestor of an adaptation is just the (lowest-level) entity which has the adapted structure as a part, this "manifestor" concept is a good way to filter out the factual from the metaphorical.

In the case of "beneficiaries" the same issue arises. We have facts about which entities had their survival and proliferation furthered by various evolutionary factors and processes. But there are no "ultimate beneficiary" facts beyond these.

3. Recent criticism of the replicator concept

I said that Hull's distinction between replicators and interactors has helped to reduce confusion. But a number of people have suggested that the framework itself is in trouble. In contrast to the earlier focus on interactors, during the 1990s the replicator was the target of suspicion.

Nobody objects to the weakest possible concept of a replicator, in which anything is a replicator if it gets copied, by some mechanism or other. The problem is that genes are often taken to be replicators in a stronger sense, and some think the appeal of the replicator concept in biology derives from covert use of this stronger sense. Genes are often described as "replicating themselves" (Dawkins 1982a, p. 99), or as directing their own replication and the production of whole organisms. A number of writers have argued, roughly, that the standard replicator concept gives genes too much credit, and is part of a "gene-centric" view of life and evolution.

One version of the objection is Lewontin's:

> Nor are genes self-replicating. They cannot make themselves any more than they can make a protein. Genes are made by a complex machinery of proteins that uses the genes as models for more genes. When we

> refer to the genes as self-replicating, we endow them with a mysterious, autonomous power that seems to place them above the ordinary materials of the body. Yet if anything in the world can be said to be self-replicating, it is not the gene, but the entire organism as a complex system (1991, p. 48).

A similar view is expressed by Sober and Wilson; they are uneasy with the replicator concept because "[o]ne implication of the term 'replicator' is that replicators control their own destiny" (1994, p. 538).

In response to these objections, one can argue that the language of "self-replication" is no more than a colorful turn of phrase, not intended to attribute any special causal powers to genes. But a problem lurks here. Suppose we restrict ourselves to the weakest sense of replication – an object is replicated if it participates in causal processes of *any* kind, in which new objects are made that resemble the original. Then as critics have pointed out, a great many things beside genes will be replicators. Human thumbs, for example, will pass this weak test. I will return to this issue below.

An alternative framework to Hull's is also being developed by Jim Griesemer (forthcoming a, b). Griesemer argues for the centrality of a concept of *reproduction*, rather than replication. The key differences are that similarity is not essential to reproduction, and all biological reproduction involves a certain kind of material overlap. In reproduction, part of the material that makes up the parental generation becomes part of the offspring generation as well.

Griesemer's framework is still being developed, but there are reasons to think it will not dislodge the replicator concept. If material overlap is made central, it will be hard to describe the evolution of some kinds of viruses, especially retroviruses, and also oddities like viroids and prions. In these cases, it appears that the traditional concept of replication fits the phenomena well, but it is impossible to describe retroviral generations (for example) as linked by Griesemer-style reproduction, as the viral sequence is copied from RNA to DNA and back to viral RNA, and the viral coat is made afresh in each generation. There is no stage in which the descendant strand is a material part of the parental RNA molecule, let alone a stage in which the descendant is part of the complete virus.

For most organisms, making more of their kind requires growth, and various processes involving material overlap of the kind Griesemer describes. But where there is reproductive machinery, the possibility of parasitism arises. Viruses exploit the opportunity, and sidestep the need for the usual processes found in reproduction.

So it appears that even if reproduction with material overlap is important in some situations, we will also need a broader concept of replication.

A third criticism of the Dawkins/Hull replicator is found in the work of Paul Griffiths and Russell Gray (1994), who are among the proponents of "developmental systems theory" (DST) as a general approach to development and evolution (Oyama 1985/2000; Oyama, Gray and Griffiths, forthcoming). According to Griffiths and Gray, the replicator/interactor distinction is the product of a "dichotomous" view of evolution and development, where the "dichotomy" involves an illegitimate division between two fundamental types of developmental causes, the "genetic" and the "environmental." Griffiths and Gray claim that the standard replicator/interactor distinction is a "projection into evolution" of dichotomous views of development (1994, p. 298).

The developmental systems theorists argue that the only thing which actively replicates or reproduces itself is the entire life cycle. They also argue that the life cycle is the relevant unit for evolutionary theory: "the prime unit of evolution (unit of self-replication) is the developmental process, or life cycle." We should conceive evolution as the "differential replication of developmental processes/life cycles" (Griffiths and Gray 1994, p. 304; see also Gray, forthcoming).

Here I have focused on quotes in which the entire life cycle is picked out as a unit. But Griffiths and Gray's discussions tend to move between the view that only the whole life cycle replicates, and the view that any element or "resource" within the life cycle replicates. Griffiths and Gray do partially resolve the ambiguity: "If we insist that a replicator have the intrinsic causal power to replicate itself, there will only be one replicator, the life cycle. But if we allow the status of 'replicator' to anything that is reliably replicated in development, there will be many replicators" (1994, p. 300). The problem is only partially resolved, however, because Griffiths and Gray's aim is not just to say what replication is, but to revise the replicator concept as it figures in analyses of natural selection. And neither of Griffiths and Gray's candidates are wholly satisfactory for that task.

Suppose initially that DST views the entire life cycle as the unit that replicates and competes in processes of natural selection. This view works for asexual populations, but it runs into a familiar problem with sex. In a sexual population any individual "turn" of a life cycle is initiated by contributions from two parents (and in fact from other sources, as DST stresses). If the population is genetically variable, each individual turn of the life cycle will be unique and unrepeatable in the familiar sense discussed in the earlier "units of selection" literature. An individual turn of a life cycle is a temporary aggregation. Later I will suggest that in some possible cases of Darwinian evolution, there is no persisting "unit" selected over many generations, in the way that Dawkins and Hull require of replicators. Replicators are not essential to evolution. This view could help Griffiths and Gray. But as I interpret them,

Griffiths and Gray are not proposing that replicators are optional; they are substituting developmental systems into the replicator role. In the case of sexual populations it is hard to see how that works.

Perhaps we should use the other sense of "replicator" recognized by DST. In this sense, any part of a life cycle that is reliably reproduced each time the cycle turns is a replicator. But this would add a huge number of things to the category of replicator – not just nests and bird song patterns, which might be good additions, but thumbs, stomachs, leaves and so on. The replicator concept becomes so broad it seems to collapse.

A paper by Sterelny et al. (1996) proposes a view that would solve some of these problems. Sterelny et al. accept that the replicator concept has sometimes been used to attribute genes too many causal powers, but they think the replicator can be salvaged as a useful concept without going over to the DST position. Their proposal is complex, but for present purposes it has three main features. First, they reject the idea that replicators must be causally pre-eminent in development. Second, they use teleo-functional concepts, grounded in natural selection, to understand replication. And thirdly, they argue that a variety of non-genetic biological replicators exist. Later I will outline a view that agrees with the first and the third of these ideas. For now, the important idea is the second one, the suggestion that we understand replication in a teleo-functional way. For this would provide a way to distinguish a special subset of replicators from among the various components of a life cycle that are reliably reproduced in each generation.

Sterelny et al. claim that a replicator has that status in virtue of biological functions. More strongly, they claim that replicators *represent* developmental outcomes, in virtue of these functions, but here I will mostly just discuss functions. Sterelny et al. describe two different origins for these functions. First, natural selection has selected replicators for their contributions to developmental processes (p. 389). Second, replicators are the immediate causal products of mechanisms which have copying as their function, so a replicator is in a sense *supposed* to resemble the structure it was copied from (p. 396).

Here I will not raise the question of whether these claims about functions are sustainable, and I am uncertain about the best way to combine these two kinds of assignment of functions in a single view. My argument is that regardless of those internal details, Sterelny et al. use a criterion for replication that is too strong.

Consider first a requirement that replicators be naturally selected for their contributions to development. So far as I can see, this implies that there can be no replicators that are permanently selectively neutral, and which evolve only by drift. But surely there can be permanently selectively neutral replicators. In this case their second route to the assignment of functions will work better;

even a neutral gene has been copied by evolved copying mechanisms. But this still lays down requirements that seem very strong. A good replicator concept is one which fits well with the explanatory structure of evolutionary theory. In order to have the role of a replicator, must something be produced by mechanisms that have the specific biological function of making copies? I suggest that the biological role of a replicator involves no requirements at all on the functions of a replicator's mechanisms of production. Indeed, one thing evolutionary theory must explain is how special-purpose copying mechanisms evolved from situations in which replication only occurred haphazardly. More strongly, if there is no evolution without replicators, but no replicators without a prior selective history, the view is headed for a regress.[4] Sterelny (in correspondence) avoids the regress by denying that evolution, in its early stages at least, requires replicators. I will discuss evolution without replicators in the next section, though the early stages of evolution are where I think replicators are likely to be particularly important.

4. Revising the role of the replicator

The replicator/interactor distinction seemed to make a positive contribution to the units of selection debates, but has since been criticized. Have we reached a point where the framework is no longer useful?

I suggest that the replicator concept, properly understood, is still useful. But some common ways of thinking about replicators should be changed. In this section I will argue for three revisions to the theoretical role associated with replicators. These revisions are intended to accommodate some of the points made by critics discussed in Section 3. In Section 5, I will outline a positive analysis of replicators. I leave interactors for another day.

Three revisions of the replicator concept are needed. The first is to purge from the concept any suggestion that replicators are, qua replicators, the primary controllers of developmental processes. Officially, of course, everyone acknowledges that a gene can only have effects on an organism with the aid of a mass of other machinery. Dawkins and others are happy to accept, officially, an "interactionist" view about development, in which the causal role of any component of the system is dependent on other components. But the role of the replicator is often described in a way that is at tension with that official position. Here I have in mind three kinds of descriptions:

1. Descriptions using simple but strong causal language, as when it is said that a replicator "produces" or "creates" either interactors or more replicators.
2. Descriptions in which replication is conceived as active *self*-replication.

3. Descriptions using semantic or computational concepts, as when it is said that replicators "program" or "instruct" the processes of development or the creation of an interactor.

To deny that these are good descriptions of the replicator *role* is not to deny that *some* replicators might do these things. Bacteria can more or less self-replicate, given the right circumstances. But the biological role of replicator does not essentially involve those sorts of powers, and individual genes do not have them.

A good deal of uncertainty surrounds the idea of a "genetic program," and the use of semantic concepts to describe genes (Moss 1992; Godfrey-Smith, forthcoming). But however these ideas are understood, a case can be made for severing them from the biological role of replicator. One possibility is that these semantic or computational terms are used to express empirical hypotheses about the mechanisms involved in development. If so, our response should be to ensure that the role of replicator is not tied to any particular mechanisms, to preserve the concept's generality. Another possibility is that they are used in something like Sterelny et al.'s teleo-functional way. That view was criticized above. A third possibility is that they are just metaphors used to gesture towards the role of genes in explaining the order and complexity of developmental processes. But if so, they have proved to be rather misleading metaphors.

So we should purge the replicator concept of any suggestion that a replicator, *qua* replicator, creates, generates or programs the organism. One way to express this is to say that the term "replicator" is in many cases less suitable than "replicatee." I will not change the terminology here, but the term "replicator" should not be understood to contain any contrast with "replicatee."

Is this purging of the replicator concept really needed? Hull's definitions do not contain these problematic descriptions. Dawkins's discussions have always used this sort of language, but maybe we should treat this as colorful talk. Am I just fussing about ellipses and rhetorical excesses?

Hull has indeed been careful about the causal description of replicators, though he has occasionally used language that invites misinterpretation. In his 1980 discussion he said: "Replicators not only replicate themselves but also produce other entities that interact with ever more inclusive environments" (p. 318). And on the same page: "As a replicator it need interact with its environment only to the extent necessary to replicate itself." Admittedly, the first quote might be read, in context, as Hull's description of Dawkins' concept. And more generally, it's likely that Hull was using these terms just for brevity. I expect that Hull will not find the purge I am describing unwelcome.

The case of Dawkins is different; he is not using these terms just for brevity. Dawkins argues that replicators are, in an indirect but significant sense, the ultimate controllers of all of evolution's products. Any such claim is incompatible with the position I am suggesting here.

My second revision of the replicator concept is related to the first. In discussions of complex organisms the replicator concept is sometimes found doing two kinds of jobs. But the replicator concept should, except in simpler cases, only be assigned to one of these two jobs.

The first job, and the one for which the replicator concept is generally not suited, is explaining *the re-creation of biological structure* across generations. The DST critics are right that the key concept for this explanatory task is the life cycle, or total developmental system.

The second job is explaining *the heritability of variation*, in the sense relevant to evolution by natural selection. This second job is the proper one for the replicator concept. Here what is to be explained is not the capacity for one generation to give rise to a new generation of complex living things; what is to be explained is transmission from parent to offspring of differences in the biological structures that the life cycle produces.

In simple cases, such as the evolution of replicating molecules in a primordial soup, the replicator concept can be used in both theoretical roles (to the extent that they are distinct). And bacteria are, more or less, self-replicating entities. In the case of more complex organisms with sex and a life cycle though, the theoretical role of the replicator is more specific. To say this is not to deny that genes, the paradigm replicators, figure in many other biological explanations. But the replicator concept abstracts a particular role that genes have, their role in transmitting variation.

I said replicators are important in explaining the heritability of variation, but exactly what role do they have in the explanation? Is the existence of replicators strictly necessary for evolution by natural selection? I claim that replicators are not strictly necessary; the importance of replicators in evolution here on earth is a contingent matter. This is my third revision of the replicator role.

Hull and Dawkins, as I read them, tend to suggest that replication is an essential feature in any evolutionary process; there always has to be a replicator. Of course, whether replicators are viewed as necessary will depend in part on how one conceives of replication. But I suggest that even on a liberal conception of replication, such as Hull's, replicators are not strictly necessary.

Let us approach this point via Lewontin's formulation of the recipe for Darwinian evolution (1970). Evolution requires a population in which there is variation in phenotype, differential reproduction on the basis of phenotype, and heredity of the traits associated with differential reproduction. Heredity is

conceived as a correlation between parents and offspring. As Lewontin says, it does not matter how the correlation is achieved, so long as it exists.

In Lewontin's 1970 discussion, the term "unit of selection" has a simple and thin sense – the units are just the entities in the population which satisfy his three conditions. These "units" need not be replicators, because in a sexual population there can be a great deal of difference between parent and offspring. Yet if parent and offspring are correlated – if parent and offspring are *more* similar than randomly selected pairs of individuals in that population – then evolution by natural selection can occur.[5] The requirement of heredity in traits affecting fitness is weaker than a requirement that there exist replicators, and heredity is all that is needed for evolution.

A correlation between parent and offspring can, in principle, have any mechanism. Here on earth, these mechanisms generally involve replicating nucleic acid molecules – genes. The existence of this mechanism makes it possible for us also to think of a "population" of competing genes underlying the population of organisms. But the existence of these underlying entities that pass on their structure largely intact is not strictly necessary. I return to this topic at the end of the next section.

5. Replicators and life cycles

In this section I will try to put the pieces from previous sections together, and give a positive analysis of replicators and their role.

Having argued that replicators are not strictly necessary for evolution, we should pause and ask whether a precise definition of replicators is really needed. The replicator concept is, with respect to abstractness, sandwiched in between the concept of a gene and the concept of heredity. Do we need a precise concept in between these two? Is there a general family of mechanisms for heredity, including but not restricted to genes, that it is worth singling out in this way? Perhaps the answer is no. Or perhaps the replicator concept is useful when kept loose and informal, but not when made too exact. ("Replicator" would then be a bit like "tree.") Over-analysis is a perennial peril in philosophy of science. These are possibilities, but in this paper I will continue to explore the idea that it is worth developing a somewhat more precise concept of replication.

The original formulations suggest that both resemblance and causal relations are involved in replication. That seems right. But the best way to proceed is to use those concepts to first give a definition of a *replicate*.

> Y is a *replicate* of X if and only if: (i) X and Y are similar (in some relevant respects), and (ii) X was causally involved in the production of Y in a way responsible for the similarity of Y to X.

Replication is any process by which a replicate is produced. The term "replicator" is often used in a slightly ambiguous way, meaning either (a) something *liable* to generate replicates via a process of replication, or (b) something *actually embedded* in a lineage of replicates. Both concepts are relevant and the ambiguity is usually harmless.[6]

This analysis is neutral on the question of whether material overlap, of the kind stressed by Griesemer, is needed for replication. The definition includes cases where later members of a lineage are completely materially derived from earlier, as in bacterial fission. The definition also includes cases where later members have no material continuity at all with earlier members, as in the case of a photocopied page, or a retrovirus. The replication of DNA falls between these extremes.

This concept of replication is neutral also about how much control a replicator has over the processes of its replication. A replicator must be causally responsible, given the context, for some relevant similarity between the replicator and its replicates. But the replicator need not be entirely causally responsible for producing the replicate. Many other factors can be involved. Photocopying illustrates the distinction: a page on a copier is in no sense able to do the whole work of copying itself, but given the existence of the surrounding machinery, the copied piece of paper is causally involved in the production of the copy in a way responsible for their similarity.

My account concurs with Hull, rather than Dawkins, in classifying whole asexually reproducing organisms as replicators. But the offspring of sexual reproduction involving gametic fusion will never be replicates of a single parent, no matter how low the genetic variation is, because one parent is never responsible for the overall similarity of parent to offspring. (The other parent is not just "surrounding machinery.") One parent can be responsible for replicated parts of the offspring though, as in the case of mitochondria.

So far we have an abstract and flexible concept of replication, which can be sharpened up in specific contexts as the need arises. Following the discussion in earlier sections, let us look again at the role of replicators in relation to development and complicated life cycles.

The life cycle characteristic of a species is a type of causal process which exhibits some variation across its instances. I will use the term "component" for all the objects and structures that reliably recur in each "turn" (generation) of a life cycle. So the components of life cycles include such things as stomachs, thumbs, enzymes, nests, leaves, birdsongs and genes.[7] This is the broader category which I said in Section 3 should not be identified with the category of replicator.

Within this large class, however, we can distinguish between components that merely recur in each turn of the life cycle, and components that are

replicators. First, some structures will be replicated *within* a generation, such as DNA molecules. Then the definition can be applied in a straightforward way. The more difficult problems arise when we try to work out whether the repeated appearance of a structure in *different* generations is due to replication. In those cases, replicators are components of the life cycle which are causally involved in their own recurrence in a way that transmits variation across generations. If a similar component appears in two generations, there is replication if the earlier one was causally involved in the production of the later one in a way responsible for the similarity between them.

So genes are clearly replicators. When we look across generations, the size of a genetic replicator depends on the role of recombination, as Williams and Dawkins argue. Thumbs and leaves are clearly not replicators. In principle, various kinds of non-genetic replicators are possible though – the replicator concept is "extended" in a way that resembles the Sterelny et al. view. Some nest structures may well qualify. The distinction between replicators and non-replicators will not be a sharp one, however. Later I will stress the role of borderline cases.

Importantly, any component of the life cycle will have its replicator status with respect to some changes of state and not others. Which changes of state can be transmitted across generations will depend on other features of the total machinery. We must also deal with the familiar ambiguity that motivates the distinction between locus and allele in genetics. I will use the terms "replicator variable" and "replicator variant" to express the distinction needed. A replicator variable is a structure that has a number of transmissible states; each of these states are replicator variants.

How does my account compare with the discussion of replicators in Maynard Smith and Szathmáry (1995)? They use a basic and very general concept of a replicator, and several more specific concepts as well. Most generally, a replicator is anything that "can arise only if there is a preexisting structure of the same kind in the vicinity" (p. 41). A *hereditary* replicator is one that can exist in several different forms, where these differences are passed on in replication. A *simple* replicator does not have this capacity for heritable variation. They also distinguish *limited* hereditary replicators, which can only exist in a limited range of possible forms, and *indefinite* hereditary replicators, which have an indefinitely large number of replicable states. They claim that continuing and creative evolutionary processes require indefinite hereditary replicators.

Maynard Smith and Szathmáry put these distinctions to impressive use in their book. But I suggest that their basic concept of replication is not well formulated. The criterion of being something that "can arise only if there is a pre-existing structure of the same kind in the vicinity" is too weak in some

cases and too strong in others. Consider any enzyme used in translation. Such an enzyme can only be produced by translation of mRNA, so it can only arise if there are pre-existing enzymes of the same kind in the vicinity. The enzyme qualifies as a replicator in virtue of that fact; the test seems too weak. The Maynard Smith and Szathmáry definition also disqualifies anything from being a replicator, regardless of its *usual* role, if it *could* also be produced without preexisting structure of the same kind being in the vicinity. The test now seems too strong.[8]

At another point Maynard Smith and Szathmáry give a slightly different definition of their basic concept: a replicator is "an entity that only arises by the division or copying of a pre-existing entity" (p. 58). In the case of division, I assume they mean an entity that only arises by the division of a pre-existing entity *of the same kind*. That seems fine, but it only works in cases where material division is involved. In cases where there is no division, we are again left with an undefined concept of "copying." Working out the relevant concept of copying is one of my aims here.

I said earlier that replicators are not strictly essential to inheritance and evolution. For the rest of this section I will fill out this claim, although the question is difficult and the claims I will make are presented cautiously.

The paradigm replicators, genes, persist through the entire life cycles of organisms in which they occur. In the case of bacteria, the DNA in the chromosome materially persists through the generation, and is replicated prior to fission.[9] In the case of organisms like ourselves, genes persist through the life cycle in the form of a lineage of replicated molecules. In both cases, the physical structure characteristic of a replicator variant is found through the whole life cycle, either in a single molecule or in a lineage. This persistence is a contingent matter; heredity would be possible without it. When heredity is not due to a single, definite structure persisting through the life cycle, the replicator concept becomes strained. Ultimately, in complex cases, the concept becomes inapplicable.

I will illustrate the relevant phenomena with a hypothetical example. Hull dislikes fanciful thought experiments, but I hope he will forgive this one, as it illustrates not just the space of possibilities but also some real cases. Imagine there is *reverse translation*, from protein primary structure to nucleic acid sequence, as well as forward translation. Then we can imagine an organism in which the genetic material initially contributed by parents is in the form of DNA, but once the new individual has used these genes to manufacture proteins, the DNA is broken down. (The proteins regulate their own activities during this middle stage.) At the end of the cycle, new genes for the next generation are made by reverse-translating (and reverse-transcribing) from protein to nucleic acid. In this case, any "allele" exists in two physically

different forms through the life cycle – first as nucleic acid base sequence and then as amino acid sequence. Mutations in either form will be passed on.[10]

What are the "replicators" here? The proteins produced are not replicates of the DNA molecules, nor vice versa. This is because they are not similar.[11] But is a stretch of DNA produced at the end of the life cycle, via reverse translation, a replicate of the corresponding DNA that started the life cycle? The answer is yes, so long as the causal link involved in replication can be somewhat indirect (contra Hull's 1980 definition, but not his 1988 one).[12] But if so, the protein molecules produced in one generation are replicates of the proteins in the previous generation as well, so long as the transmission of that amino acid sequence was reliable. So both the DNA and the proteins are replicators in this case? That seems OK. But note that we seem to be double-counting replicators, as each of these "replicators" is dependent on the other; a single variant takes two forms during the cycle. One thing we might say is that a single replicator is realized here as a sequence of materially different entities; we have a "replicator sequence" rather than a single entity acting as replicator. This is already pushing the boundaries of the concept, but perhaps not too far.

The situation changes, however, if the processes involved in the transmission of a variant become sufficiently complicated and causally dispersed. We can imagine further multiplying the physical guises in which a replicator appears during the life cycle, and can imagine a complicated role for other causal factors in modifying the transmission of these variations. As the causal web gets more complicated, it becomes less and less appropriate to try to identify a replicator, where a replicator is a definite entity, or identifiable lineage of related structures, that is responsible for heredity. The more factors that are involved in creating a new Y that is similar to X, and the more places in the network at which dissimilarity could be introduced, the less true it is to say that "X was causally involved in the production of Y in a way *responsible* for the similarity of Y to X," as the definition requires.

As with many tests using causal concepts (and similarity concepts), there is no sharp borderline between cases that pass and cases that fail. In the case of a very complicated life cycle, there will be a great many structures that have *some* causal connection to the recurrence of similar structures in later generations; even thumbs have *some* causal role in the processes by which new thumbs appear, though they fail any reasonable interpretation of my test for being a replicator. Lots of components in a life cycle will contribute in a marginal way to their recurrence, so there will be many marginal replicators. But in the limit of causal complexity, all that can be said is that the entire

causal network in the life cycle somehow manages to transmit variation from generation to generation.

Does any of this matter? Reverse translation does not exist. Is there any reason to think about strange cases in which discrete replicators get lost in a sea of causal complexity? Yes, because aside from the need to explore the space of possibilities, these complicated translations and reverse-translations are ubiquitous in cultural transmission.

With Hull and Dawkins, I accept that there are some genuine cultural replicators. But within culture, the phenomena that make the replicator concept hard to apply are common. Structures recur, but the processes responsible for their recurrence tend to be complicated and dispersed. As a consequence, genuine cultural replicators in human contexts might be rare, or confined to unimportant phenomena involving photocopiers.[13] To admit the existence of some cultural replicators is not to commit to a general explanatory theory explaining cultural change in terms of variation and selection.

Even simpler cultural replicators often exhibit changes of form similar to those in the hypothetical case outlined above. Suppose a bird learns its song from a parent or from other local adult birds. Then the song pattern takes two distinct forms in this process.[14] The young bird acquires its song by picking up sound waves. This results in the formation of neural structures, which persist when the song is not being sung. The song is passed to new birds in the form of sound waves again. We have a causal channel through which the inheritance of variation is possible, but any replicator variant must exist in two physically different forms during the cycle. A mutation at either stage can be passed on. Birdsongs of this kind are not as unproblematic replicators as genes, but they are still good candidates, even though they are of the complicated type illustrated by my hypothetical “reverse-translation” case.

To the extent that cultural transmission involves a lineage of structures, distinct to some extent from the causal sea surrounding them, where earlier members of the lineage can be causally involved in the production of similar later members, in a way causally responsible for the similarity between them, we have replicators. To the extent that no lineage can be isolated because of constant blending, and to the extent that the similarities between cultural products over time result from a network of dispersed and interacting causal factors, in which all the quirks of human preference and flexibility are involved, we do not have replicators. These are reasons to be skeptical about general replicator-based theories of cultural change, of the type advanced by Dawkins (1976), Hull (1988) and Dennett (1995).

I have stressed the space of possible mechanisms for biological inheritance, but let us come back to earth. Replicators might be optional, but

actual living systems make very extensive use of them. Here on earth the most important replicators, DNA molecules, do persist, either materially or in the form of a lineage of copies, through entire life cycles. Maynard Smith and Szathmáry (1995) cautiously give reasons for believing that, given the way chemistry works, only the template replication of nucleic acids has the capacity to enable the reliable transmission of a massive, open-ended range of possible states in a biological system.[15] Perhaps replicator-free biological inheritance is something that could only ever have a limited role in worlds like ours.

6. Conclusion

The abstract concept of a replicator is still useful, but three revisions are needed to the theoretical role associated with replicators. Replication need not be self-replication, the replicator is not a substitute concept for the life cycle, and replicators are not essential to evolution by natural selection. I see the view outlined here as mostly compatible with the goals and orientation of Hull's discussions. The main divergence is on the question of whether all processes of evolution by natural selection require, in principle, that something plays the role of replicator. But whether replicators are strictly essential or not, they are clearly very important here on earth, and Hull, of course, is a philosopher with an unusually close focus on actual events here on earth.

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Notes

[1] In my paper with Lewontin, (1993), Hull's framework was not used because Lewontin was not convinced that Hull's framework is helpful – see Section 3 of the present paper for his objections. In my paper discussing Wimsatt's and Lloyd's views (1992), I used Hull's framework.

[2] The literature is large. See Wimsatt (1980), Sober and Lewontin (1982), Brandon (1982), Sober (1984), Sterelny and Kitcher (1988), and Waters (1991). Some also tried to solve the problem without appealing to doctrines about causation or reduction (Lloyd 1988).
[3] See Buller (1999) for a good collection of papers on natural selection and biological functions.
[4] Mathew Barrett pointed the regress out to me.
[5] See also Sober (1993) who stresses that genes are not required for the kind of parent/offspring correlation relevant to evolution (pp. 9–11). Sober and Wilson (1994) make a similar point, but they incline more towards regarding talk of replicators as a way of redescribing heredity, rather than saying that replicators are one family of mechanisms for heredity. On a related point, I don't think Darwin's ignorance of genetic mechanisms is sufficient to show that replicators are inessential; replicators of some kind might have been a hidden commitment in his theory. Maybe his "gemmules" qualify as replicators, by my definition in section 5.
[6] Hull's and Dawkins's discussions tend to contain this harmless ambiguity; they use present-tense formulations such as "entity that passes on its structure" which can be read both ways.
[7] In this discussion I remain neutral on difficult issues concerning what exactly a life cycle or developmental system contains (see Griffiths and Gray 1994 and Sterelny et al. 1996). I also avoid using the DST term-of-art "resource" for what I call components.
[8] Their view will also disqualify many of the "indirect" cases of replication discussed below, as there need be nothing similar "in the vicinity" in those cases.
[9] I ignore plasmids and conjugation throughout my discussions of bacteria in this paper.
[10] This need not generate a strongly "Lamarckian" situation, as the range of events that produce a transmissible mutation in the amino acid sequence might be limited, and mutations might still be undirected.
[11] Williams (1992, chapter 2) might address this problem by claiming that the physical differences are not important as the two structures "contain the same information." I think this is a restatement rather than a solution of the problems at hand, but owing to space constraints I defer discussion of this (difficult) issue.
[12] Some kinds of simple systems of replicating molecules feature indirect replication, in which A produces B and B then produces A again. For an interesting discussion of a variety of simple mechanisms for replication of molecules, see Rebek (1994).
[13] Theoretically, not practically, unimportant!
[14] See Francis (forthcoming) for a relevant discussion of the transmission of birdsong.
[15] The only similarly powerful system of transmission, they think, is human language.

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