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The Extended Replicator

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Abstract. This paper evaluates and criticises the developmental systems conception of evolution and develops instead an extension of the "gene's eye" conception of evolution. We argue (i) Dawkins' attempt to segregate developmental and evolutionary issues about genes is unsatisfactory. On plausible views of development it is arbitrary to single out genes as the units of selection. (ii) The genotype does not carry information about the phenotype in any way that distinguishes the role of the genes in development from that of other factors. (iii) There is no simple and general causal criterion which distinguishes the role of genes in development and evolution. (iv) There is, however, an important sense in which genes but not every other developmental factor represent the phenotype. (v) The idea that genes represent features of the phenotype forces us to recognise that genes are not the only, or almost the only, replicators. Many mechanisms of replication are involved in both development and evolution. (vi) A conception of evolutionary history which recognises both genetic and non-genetic replicators, lineages of replicators and interactors has advantages over both the radical rejection of the replicator/interactor distinction and the conservative restriction of replication to genetic replication.

Key words: development, developmental systems, gene, genetic information, evolution, information, inheritance, interactor, Lamarck, Meme, replicator, selection, unit of selection, vehicle, Weismann.

1. Introduction

Our purpose in this paper is to evaluate a conception of evolution in general, and the units of selection in particular, that have been articulated by a group that we shall refer to as Developmental Systems Theorists.[1] So we first outline

their distinctive "take" on evolution and the units of selection, and contrast it with three other perspectives. We then compare and contrast our views of genes and replicators with that of Developmental Systems Theory. In resisting their view that genes play no distinctive informational role in inheritance, it becomes clear that though the genes play a very special role in development, they are not alone in playing this role. We argue that despite its insights Developmental Systems Theory has serious problems. Moreover, its insights can be captured by a less radical take on the units of selection problem. We think that Dawkins (1982) and Hull (1981, 1988) were right to distinguish between replication and interaction, but we think they underestimate the range of biological replication. Finally, we speculate on that extended range, and suggest that there are good reasons for thinking that Bateson's famous reductio of the replicator is no reductio at all.

1.1 *Four evolutionary mindsets*

We begin by offering a low-resolution picture of the logical geography. We see four very general ways of characterising evolution.

(1) The Received View takes evolution to be the result of competition between individual organisms varying in fitness by virtue of heritable characteristics. Opinions within this camp differ on the relative importance of selection and drift, and on how to link historical accident with phylogenetic and developmental constraints. But evolution shapes populations by acting on individual organisms in virtue of their traits.

(2) The Gene's Eye sees evolution as the result of competition between genes that differ primarily in their capacities to affect interactors. These interactors' differential reproduction results in differences in the depth and bushiness of germline lineages.

(3) The Extended Replicator hijacks these conceptual tools without any commitment to the idea that genes are the sole, or even the predominant, replicator. The Gene's Eye fades into this conception as we recognise more and more nongenetic replicators. In this debate Dawkins is an important but equivocal figure. We think this paper is a development of one of the threads of his work, for it has never been part of his official definition of "replicator" that only genes are replicators. The other category he recognises are memes, elements of culture, especially human culture. So one picture that descends from his work is that change results from two rather distinct evolutionary processes. Biological evolution works out the fate of competing lineages of genes. Social evolution does the same for competing lineages of memes. Instead, we think a single evolutionary process determines the fate of lineages of replicators of many kinds, by virtue of the differential success of their associated interactors and extended phenotypic effects. This

concept is certainly consistent with Dawkins' basic conceptual structure, but is discordant with at least some aspects of his actual practice, which has emphasised the gene to the exclusion of other replicators. (Dawkins 1976, 1982 and 1989.)

(4) Finally, there is the Developmental Systems conception of evolution. This conception is hard to characterise precisely, but the following elements seem diagnostic. (i) In the cycle from one developmental system to its successor, no element of the developmental matrix plays any privileged causal role. (ii) There is no theoretically significant distinction between internal and external factors. All are necessary, and though they are necessary in different ways, none of these different ways are special. Genes are but one important element of the developmental matrix. For some particular purposes, a focus on genes is indeed appropriate, but they have no general or overarching privilege. (iii) Defenders of this family of conceptions are sceptical about the idea of the transmission of information through inheritance mechanisms, and the associated metaphor of the genetic program. (iv) For them, the developmental system as a whole is the unit of selection. It is those that are rebuilt from cycle to cycle. Evolution, on this view, is the result of competition between lineages of developmental systems. Their view is radical in part because they conceive of the developmental matrix, the resources that are reduplicated from developmental cycle to developmental cycle, very broadly, including elements that are on most conceptions part of the environment.

Some versions of Developmental Systems Theory – for example, Griffiths and Gray 1994 – take the replicator to be the set of processes through which the developmental system is built, rather than the system itself. We do not think this distinction important in this context, though in some contexts it is. For example, in defending the idea of a gene's extended phenotype, Dawkins is concerned to emphasise the fact that adaptive phenotypic effects do not necessarily come bundled into discrete organisms. Here, the emphasis on process rather than object makes a point: the adaptation is the process through which, say, a chick manipulates its parent's behaviour. But when the "object" is the developmental system as a whole – all the entities and their relations that go into constructing an organism – we do not see that this is a distinction that makes a difference.

1.2 *Common ground*

Three important ideas about evolution are common ground between our view of evolution and that of the Developmental Systems Theorists. Everyone agrees that the genome of a developing organism is not sufficient for the development of any of its characteristics. Even so, many evolutionists think the genome's causal role in the development is privileged. In commenting on

an earlier version of this paper, David Hull expressed a common intuition: with the exception of cultural transmission in a few groups of animals, genes are the only, or almost the only, cause of structure. Developmental Systems Theorists deny this. We think they are right to make the stronger claim that the gene plays no privileged causal role in the development of phenotype from genotype.

Second, one way to think of inheritance is to see it as a causal bridge from phenotype to phenotype. On the received picture, that bridge proceeds exclusively through the DNA. The only pathway of inheritance, the only bridge between the generations over which information flows from phenotype to phenotype, is the DNA in the gametes. In both our view and that of the Developmental Systems Theorists this picture is not just an idealisation; it is seriously mistaken.

Third, Developmental System's Theorists do not exile the gene in order to embrace the organism. The Gene's Eye, Extended Replicator and Developmental System's conceptions of evolution agree in not conceptualising evolution *only* as a history of organisms in competition. The scope of evolution is richer, and weirder, than lineages of organisms. So arguing against "Organismism", the idea that "the organism is the subject and object of evolution" (Lewontin 1985), is not the aim of this paper.

1.3 *Development and replication*

Many critics of the Gene's Eye have emphasised the complexity of development and the many: many relation between genotype and phenotype. Those worries are not our worries. We are not claiming development is so complex that genes are not replicators. Rather, our view is that there are other replicators beside genes. Nor is Developmental Systems Theory just a rehash of the many arguments from the failure of bean-bag genetics. It has a new take on the units of selection problem. Developmental Systems Theorists want to emphasise the interdependence of developmental and evolutionary theory. This interdependence is contested. In, for example, his 1982, Dawkins has defended combining a particulate view of the evolutionary role of the gene with an interactionist view of their developmental role. Though genes interact in development, they have independent evolutionary trajectories. So an argument is needed to forge a link between development and interaction. We think there is such an argument. But it has not been very clear, so our best reconstruction of it is as follows:

Step 1. If we consider a lineage of organisms from developmental cycle to developmental cycle, we will see that on each cycle there are many repetitions of important elements of the previous cycle. Genes, cellular machinery of various kinds, morphological and physiological traits, behaviours, and social

structures are reliably rebuilt, cycle by cycle. There are many constancies maintained through these lineages, constancies that permit selection to be cumulative.

Step 2. The developmental process through which each cycle repeats is fabulously complex with the effect of each element depending on the effects of many others. Even so, we think genes would be the beneficiaries of adaptation and hence the unit of replication, if genes controlled, directed or were the organising centre of development. For genes would then still have a privileged role in development. It would not be arbitrary to think of them as *the* replicators.

Step 3. The notion of genetic information and its relatives cannot be made good in a way that singles out the genes as having particular significance. Genes predict phenotypic characters only in the same sense that environmental factors predict them.

Step 4. No causal selection scheme picks out genes, either in the development of one phenotype in the lineage, nor in the recreation of that developmental factor in the next link in the lineage. In particular:

- directness does not single out genes. The processes through which genes are copied and through which they produce their phenotypic effects are highly indirect.
- causal asymmetry does not pick out the genes. It is true that every other factor in the cycle from link to link in the lineage depends on the capacity of (germline) genes to produce copies of themselves. But the capacity of germline genes to replicate equally depends on the reliable reproduction of a host of these other factors.
- causal responsibility for variance does not distinguish the role of the gene. Genes can be selected in virtue of their effects. For example, relativised to a normalised set of background conditions, the substitution of one gene for another may yield a boldly striped organism. So that gene is the "gene for bold stripes". But the same symmetrical comparison between variants, relativised to a normalised background, gives us incubation temperatures for traits, cellular chemicals for traits, and so on as causally responsible for variance.
- fidelity does not pick out the genes. Genes are not the only factors that re-appear with great reliability. Moreover, there is no fidelity threshold on replication.
- causal importance does not pick out the genes either. Gene-environment interactions are too messy and ill-behaved for us to say genes are the most important causes of some traits whereas the environment is the most important cause of others.

Step 5. They conclude that nothing singles out genes as special. Hence genes are not the replicators; whole developmental systems are.

In this paper, we propose getting off this inference bus at step 3 (Smith thinks one might be able to get off at step 4, too; see his 1994). The way we do this commits us to the view that genes plus a fair range of other elements in the developmental matrix are replicators. Against the Gene's Eye conception of evolution we argue that the genes play no unique informational role. We argue against Developmental Systems Theorists that the replicator/interactor distinction remains of value, that it captures all that their alternative captures and that the Developmental System's conception of evolution suffers from problems it has yet to resolve.

2. Developmental systems theory

In this section we briefly characterise Developmental Systems Theory and our reservations about it. We think serious problems remain unresolved and that its important insights can be incorporated within a more traditional biological ontology. Moreover, that ontology continues to have independent motivation.

According to Developmental Systems Theory, genes are but one element of a developmental matrix which ranges from genes through proteins in the maternal cytoplasm to exposure to rank order in the local primate population. The whole set of elements, and in particular the relations between them, form a complex whole which is the unit of evolution and selection. Of course, in different systems different resources will play different roles. But there is no privileged gene-role; nor even a gene-role that some nongenes also play. The developmental system as a whole is the only replicator, and evolution is the differential success of lineages of replicators. The full range of developmental resources is the complex system replicated in development.

2.1 *Holism*

A *prima facie* problem for this conception of evolution is its apparent commitment to holism. Everything causally connects to everything else. Even so, we can understand something without understanding everything. So if developmental systems include everything causally relevant to development, they are too ill-defined to be a coherent active unit; they are too diffuse to be the objects of selection. Is Elvis Presley part of our developmental systems by virtue of his role in the development of our musical sensibilities? No biologically meaningful unit includes both Dickison and Presley. The intermeshing of causal connections, and transitivity of causation, will import the

same problem for any other organism. As Gray and Griffiths 1994 realise, the defence of the developmental systems conception of evolution requires distinctions amongst the factors causally relevant in development. This distinction cannot depend on any *simple* measure of causal relevance; for there is none that will distinguish the role of Elvis from the role of early nutrition. But some causal influences are going to be part of the developmental system that made us, and others are not, on pain of all developmental systems reducing to one. That would be holism run amok.

2.2 *Boundary and other problems*

The transitivity of causation is implicated in several problems, not just one. Elvis highlights the boundary problem. What events and processes go into the developmental system, given that not everything causally relevant can do so? A "cycle problem" brings into focus the lineage. Evolution, and most especially the evolution of adaptive complexity, depends on cumulative selection, and hence on lineages of similar organisms or organism surrogates. That problem is not difficult for any theory that privileges the organism, for the lineage is just a sequence of organisms related by descent. Nor is it a particularly serious problem for those who think of evolution as a struggle between lineages of genes. But one of the ideas that unites the developmental systems theorists with others sceptical of the Received View is their refusal to define basic evolutionary processes by appeal to just one of the contingent products of evolution, the organism. The developmental system relies on the stable generation by generation reproduction of developmental resources. But developmental system generations are not to be identified with organism generations. So when do generations begin and end: do we count from bird to bird, egg to egg, or nesting hole to nesting hole? Cycles of developmental resources are not necessarily in sync. For birds that breed for more than one season, the nest cycle is shorter than the bird cycle. Other resources cycle slower than organism generations (e.g. the social group, many parasites' hosts). The cycle length of symbionts need not be the same. So on inspection, the developmental system replicating itself generation by generation seems perhaps a congerie of associated and perhaps co-evolving but still independent lineages; more a guerilla band than a regular battalion. There is a related problem with counting lineages. Is a ring of Mullerian mimics one developmental system or many? Is an ant-plant mutualism a single developmental system or several?

To the best of our knowledge, Gray and Griffiths' 1993 and 1994 are the only explicit attempts to solve these problems. We do not intend to offer a point by point discussion of their line of argument, for it is not our view that these problems are intractable. But we do think they are difficult, and aim

to show that through a sketch of their treatment of the "boundary" problem. They argue that we must:

> distinguish... developmental outcomes which have evolutionary explanations from those that do not. The interactions that produce outcomes with evolutionary explanations are part of the developmental system. There is an evolutionary explanation of the fact that the authors... have a thumb on each hand.... The thumb is an evolved trait. But the fact that one of us has a scar on his left hand has no such explanation. The scar is an individual trait (we are referring of course to the trait of having a scar just thus and so, not the general ability to scar). The resources that produce the thumbs are part of the developmental system. Some of those that produced the scar... are not. (Griffiths and Gray: 1994, p. 286)

Obviously, there is indeed a difference between the thumb and the scar. Though all of Griffith's parents had thumbs, there is no reason to believe that they had scars on them. But phenotypic plasticity suggests that a reliance on parent/offspring similarity would draw the distinction between individual and evolved traits in the wrong place. The lyrebird's song is unique to each bird, for they are famous mimics, and pick up all manner of extraneous sounds, including those of humans, their animals and machines (Reilly 1991). Yet this does not seem to be an "individual" trait in the same sense that a scarred hand is. Moreover, there is a sense in which the scar has an evolutionary explanation: scarring events are "historically associated with" the human lineage. There is an evolutionary component of any individual scar construction. So we have our doubts about the robustness of their distinction.

3. Extending the replicator

In this section, we argue that genes do play a very special role in development. But we also argue that genes are not the only developmental resources that play that special role. Some developmental factors do not just cause similarities between one developmental cycle and its successor. They have the form they do because they cause those similarities. These are the replicators. We do not distinguish them on the grounds that there are more important than other factors in the developmental process; rather, we distinguish them because they are adapted to play the role they do in development.

3.1 *Farewell to genetic traits*

Genes are not the primary cause of phenotypic traits. Perhaps, though, they represent genetic traits or carry information about them. Such seems to be

the idea of those who have followed Mayr's lead in speaking of the genome as a program that directs development. But even the idea of genetically programmed traits is in trouble. For information is typically understood as dependence. A signal carries information about a source to the extent that characteristics of the signal co-vary with features of the source. So the genome carries information about a phenotype just so long as features of the genome co-vary with phenotypic characters. Phenotypic plasticity means that this co-variation is far from perfect. It improves, of course, if we hold the environment fixed. Aspects of the genome will co-vary well with traits *in an environment*. But as Johnston (1988) and Smith (1992) emphasise, a similar dependence holds between the environment and the developing phenotype. If we hold the genome fixed, there will be co-variation between environment and phenotype, and hence features of the environment will carry phenotypic information. The human genome carries information about, say, human skeletal structure. But so does the nutritional, biochemical and cellular environment of the foetus. The link between genome and developed system – holding environmental factors constant – is not unique. A network of necessary environmental factors – holding genetic background constant – correlates equally well with the developed system. So that network carries information about development in just the same sense that the genome does.

Moreover, the genome quite frequently correlates better not with the designed outcome of development but with various dysfunctional outcomes. Sterelny and Kitcher (1988) unpacked "a gene for X" by appeal to the gene's role in normal total environment. But Griffiths and Gray (1994) point out that this idea does not work. Most acorns rot, so acorn genomes correlate better with rotting than with growth. So if we are to talk of information, we should talk of the acorn genome carrying information about how to rot rather than grow, for it correlates *better* with rotting than growing.

We doubt that there is a quick fix for this problem. Of course, this example would collapse if there were a "gene for growing", a gene complex which had a better than 50% chance of growing. But it is most unlikely that there is any such gene complex. Of course, some gene complexes have a better chance of growing than others. But if information just is correlation, and all gene complexes correlate better with rotting than growing, then they carry the information about how to rot, not how to grow. No doubt we can take a more fine-grained view of oak environments, for there will be some circumstances, "microenvironments" (see Brandon 1990), in which some gene complexes will have a better than even chance of growing. But then it's the pair of gene complex and microenvironment which correlate with growing and hence which carries the information, not the gene complex itself.

Thus, any view which identifies the genome as a representation of the phenotype must be consistent with the interaction of the genome with the other factors of the developmental matrix. In virtue of this interaction, any element of the developmental matrix correlates with the developed phenotype if we hold constant the rest. No factor correlates with the developed phenotype unless we hold constant the rest. We can indeed speak of "the gene for red eyes". For relativised to a normalised set of background conditions, the substitution of that gene for a rival yields a red-eyed organism. But precisely the same comparison between variants, relativised to a normalised background, shows we can speak of incubation temperatures for traits, cellular chemicals for traits, and so on. For example, phenotypic plasticity in plants is common, and often manifested in a fine-tuned adaptive response to the specifics of a particular environment (Sultan 1987). Environmental variant, relativised to constant genetic background, predicts phenotypic variant.

The elements of the developmental matrix interact in ways that make it impossible to regard the environment as the mere trigger of a genetically caused process. The Buckeye butterfly (*Precis coenia*) shifts colour seasonally in ways that on first inspection fit the paradigm of a genetic process with an environmental trigger, and hence the conception of the genome as control centre. As the season advances, the colour pattern of emerging butterflies shifts from a tan ventral hindwing (WILDTYPE) in the Spring to a reddish hindwing (ROSA) in the autumn. This is not just an environmentally induced change, for it is easy to breed strains that express the reddish morph under all conditions. But nor is it a genetic subroutine with an environmental trigger, for butterflies of most genotypes can be induced to emerge as red morphs. There is no single environmental trigger: there are multiple ways to induce red shift in the population: both low temperatures or short daylengths. Most importantly of all, environmental influences interact: an inductive temperature under one daylength is not inductive under another and vice versa. So the causal structure of red shift in the population cannot be analysed as an almost universal genetic program with redundancy built into the environmental triggering (Smith 1993b).

The genome is interdependent not just with the external environment. Parents contribute much more than genetic material to the developing organism. Gametes are not just packets of DNA. Even the sperm is more than a mere packet of DNA. Centrioles organise the axis of genetic segregation by migrating in cell division to opposite poles of the cell to serve as anchors for the filaments attaching to the chromosomes. They thus play an indispensable role in ensuring that the daughter cells get an equal number of chromosomes. Yet centrioles are transmitted parallel to the genetic material in the gametes. They are not built by gametic DNA (Glover, Gonzales and Raff 1993).

For genes to become active in the construction of proteins, the coding sequences must be read out of these sequences to build the exons that code for proteins. This machinery is not just a causally necessary conduit – a more or less noisy channel – through which genetic information passes. Some sequences are ambiguous, so different exons can be constructed from the same transmitted sequences. Which exon is built, and hence which protein is coded for, depends on the cellular environment (Fogle 1990). Cellular machinery does not just play a role in allowing coding DNA to be read; it affects what is read.

3.2 *Symmetry, information and representation*

Genes and other factors are thus interdependent in development. Consequently, genes do not correlate with developmental outcomes in any way that distinguishes them in the developmental system. Nevertheless, we think the genome does *represent* developmental outcomes. For representation depends not on correlation but function. The plans of a building are not the primary cause of a building or of its features. A plan may correlate better with graft, waste and overspending than with the actual traits of a building. Despite failures of correlation, and despite correlation without representation, plans represent buildings because that is their function. Some elements of the developmental matrix – the replicators – represent phenotypes in virtue of their functions.

The genome is one of the *designed mechanisms* in virtue of which phenotypes and genotypes duplicate themselves. Adaptation is seen in the proofreading and repair mechanisms of the genes but not only there. For example, many arthropods are linked in obligatory symbiosis with micro-organisms on which they depend for growth, micro-organisms which are transmitted in the egg. This mechanism can be very precise. For example, in one species of aphid *Colophina arma* the micro-organisms are not transmitted in those eggs designed to be dwarf males or sterile female soldiers, for in these morphs no growth spurt is required (Morgan and Baumann 1994). This idea of a designed copying mechanism is the key to understanding the privileged role of the replicators in the total developmental matrix. Some parent-offspring similarities result from elements of the developmental matrix than have been selected to produce those similarities. Replicators exist because of those selection histories, and that distinguishes their role in development.

These functional differences are reflected in counterfactual differences within the developmental matrix. Thus both the replicators and the environmental factors correlate equally well with normal development. But there is an important asymmetry. In our view, the genes are not the only replicators, but let us for the moment focus on them. Consider a facultatively

desert-adapted shrub; a shrub whose leaf structure and shape reduces water loss if grown in arid environments. Both aridity and the shrub's genome are necessary for that shrub's adaptive response to the environment. But that genome only exists because of the causal path (in that environment) from genome to desert-resistant shrub. By contrast, the aridity of the environment exists independently of the causal path (in that genetic environment) from arid conditions to desert adapted shrub. One element of the developmental matrix exists only because of its role in the production of the plant lineage phenotype. That is why it has the function of producing that phenotype, and hence why it represents that phenotype.

A similar asymmetry lurks in the second problem. Acorn genomes correlate better with rotting than growing. But if all acorns rotted, there would be no development trajectory from acorn to worm food. In contrast, if all acorns germinated and grew, there would still be a developmental trajectory from acorn to oak (and savage sapling mortality). So the acorn-wormfood correlation depends on the correlation between acorn and oak, but not vice versa. One developmental path depends on the other, and hence we can regard the acorn to oak link as privileged despite its rarity. That is why it is legitimate to talk of the acorn carrying information about the tree.[2] There is not just covariation between signal and source; the genes have the biofunction of guiding phenotypic development.[3]

Our account of representation involves an important difference between our own program and that of Dawkins. He has argued that because genes are the *beneficiaries* of adaptation, they are not *for anything*; they are not themselves adapted (Lloyd 1992). They have no teleofunctions, they just are. If that were right, a mutation would not be a mistake, only a change. And if a phenotype developed abnormally because of that change, we could not say that the gene was failing to do what it is supposed to do. Only things with functions can malfunction. So Dawkins' conception seems to rule out this option of holding that the genes are privileged in development through their role of representing the proper outcome of development.

We disagree with Dawkins in two ways. First, the roles of replicator and interactor are not exclusive. On this, we line up with Hull 1981. He points out that genes are interactors, and are adapted for those roles. Even "neutral" genes have effects at the cellular and subcellular level, effects in virtue of which they are copied.[4] Outlaw genes carry adaptations for their own replication which subvert others' prospects; for a recent example, see Werren 1991. The dual role of genes both as replicators and as bearers of adaptation is even clearer in genes which do have phenotypic effects. As a consequence of its structural and relational properties, a gene can have the function of telling the developmental program how to build haemoglobin

molecules – for that function derives from its evolutionary history (Millikan 1989). Those structural and relational properties are properties of the genes in that lineage – they are there because they have often enough resulted in those genes initiating developmental sequences that lead to normal haemoglobin, resulting often enough in the replication of that gene. Hence, there has been a malfunction if disruption of the gene's structural and relational properties leads to the formation of nonstandard haemoglobin. The gene is not doing what it is supposed to. It is not doing what its ancestors have done in the past that ensured their replication.

Second, even if genes and other replicators were not themselves the bearers of adaptation, they are the products of copying mechanisms. That is, there are mechanisms which have the function of ensuring cycle to cycle similarities; of copying the replicators. So there is an error when they mistranscribe. If there are mechanisms in a bird lineage which are there because they have ensured (often enough) nest site fidelity, a copying error has taken place when a bird returns to the wrong site. The same is true of a genetic mutation: that counts as an error because a copying device has malfunctioned. Hence we can speak of misinformation in the replicator to replicator cycle.

3.3 *The varieties of replication*

We think this is the right way analysis of genetic information. But *genetic* bridges across phenotypes are not the only mechanisms of inheritance. This role of the genome is distinctive but not unique. There are a range of mechanisms through which the similarity between successive developmental systems in a lineage is maintained, and maintained as a consequence of design. Extra-genetic causal and informational transmission is not an odd footnote; it is central to the development of the phenotype. Our conception of the replicator is expansive but not promiscuous. Not every reliably re-occurring factor is a replicator. The human hand is not a replicator. The hand's biofunction is economic, not developmental. Replicators are devices with developmental biofunctions. These of course include DNA in the gametes but also a good deal else. Examples of the rest include: Kakapo track-and-bowl systems, nest site imprinting and other mechanisms of habitat stability; song learning, food preferences and other traditional examples of cultural transmission in animals; gut micro-organism transmission in food and other micro-organism symbionts which parents are adapted to transplant to offspring; and centrioles and the other causally active non-genetic structures that accompany genetic material in the gamete. Thus, Clayton and Harvey (1993) have illustrated the heritability of nest structure. Keller and Ross (1993) have documented the cultural transmission of queen morphology in fire ants. Goodwin

(1989) and Wagner (1988) detail examples of nongenetic transmission of cell structure.

Some of these mechanisms have been entirely ignored by those who focus on genetic replication. Some have been relegated to the exceptional category of cultural transmission. Still others have been taken to be just side effects of the genes. In our view they are all routes across the generations that exist, in part, in virtue of their role in ensuring parent-offspring similarities.

Indeed, we think there is a case for claiming that extending our census of the replicator makes the notion of information less mysterious. Platypus DNA bears information about future platypuses and their burrows, but only in the rather subtle sense that platypus DNA correlates by design with platypus traits. So replicators carry two sorts of information. First, they carry information about the interactions in virtue of which a new generation of replicators are constructed. Secondly, they carry information about that next generation of replicators. Nongenetic replicators may carry this second breed of information in a more direct way than genetic replicators. The construction of gene to gene links in a lineage is complex and indirect. Nongenetic replicators may bear information to rebuild similarities across generations in a more direct way: they act as templates in the construction of a new developmental system. For example, Moss argues that intercellular structures act as templates in cell division (Moss 1992, p. 345). If young platypuses tend to copy their natal burrow so that changes in a burrow are transmitted to the next burrow generation, platypus burrows may be templates for future burrows. Platypus burrows also carry information of the first kind. Obviously, they provide generalised support for platypus development. That may be all some genetic replicators do. But burrows may also be specific causes of interaction: for example, if natal burrow influences platypus choice of size, site and materials for future burrow building. (On platypus biology, see Grant 1989)

Other replicators thus play the same basic role in development as genes. Nor is there is any quantitative criterion that singles them out. Two obvious candidates are directness and accuracy. Hull used to defend the idea that genetic replication is particularly direct and that directness mattered (Hull 1981). Griesemer (forthcoming) has argued to the contrary, emphasising the extraordinary complexity of DNA replication. We think he is right, but like him think it is still more important to see that directness is of no evolutionary importance. It does not matter whether replicators are organisms, traits, genes, or developmental systems. The number of steps in the process through which one replicator makes another is of itself of no evolutionary significance. (Smith 1994). Indeed, DNA replication is highly indirect, we conjecture, to ensure high fidelity replication.

Fidelity is of evolutionary significance. But we doubt that fidelity distinguishes genetic from other forms of replication. Other inheritance mechanisms have not been sufficiently conceived of as inheritance mechanisms for their fidelity to have been calibrated. But the problems are not just empirical. Replication could not require the reproduction of every property of the original in the copy. Only numerically identical objects share all their properties, so then we would have one replicator, not original and copy. Replication – even perfect replication – requires only the reproduction in the copy of the *relevant* parental properties. So not every change from parental song is a failure to replicate that song. Some variations are neutral. We need to understand the message before we can count variations from it. But *comparing* replication fidelity across different media is harder yet. Even when we have a sense of the message, we may not have a common currency for comparing the replication accuracy of gut micro-organisms from parent to offspring with the replication of nest site preference or DNA. We do not think information theory will help here for we cannot use an information theoretic notion of accuracy without a principled conception of the range of possibilities at that source. But what is the space of possible bird songs? Or is the space not of possible bird songs, but of possible mechanisms of species recognition or devices of territorial display?

In his 1982, Dawkins appealed to fidelity to argue that asexual organisms are not replicators (p. 97). An aphid that loses one of its legs will still give birth to six-legged offspring. If it changes, it does not pass the change on, hence is no replicator. The idea is that *a certain sort* of high-fi is required for replication. Some copying errors are permitted; some aspects of a replicator's structure may not make it to the next link in the lineage. But any *change* in a replicator must make it to the next link. This criterion backfires against genetic replication. Many changes in the germline genes are not passed on. The point of the proofreading and repair mechanisms is to avoid the transmission of changes. So if genes are replicators, some changes in replicators need not be passed on; those censored by the proofreading and repair mechanisms. But then we can see the production of a six-legged aphid from its eventually five-legged forebear as a triumph of the *aphid's* proof-reading and correction mechanisms. In any case, Dawkins' hi-fi criterion will not cull out all our candidates for nongenetic replication. A change in a bird's gut fauna may well be passed on to its descendants. The extraordinary concordance between an aphid clade and some of its symbionts (Morgan and Baumann 1994) shows such changes have been passed on, as the aphids and their fellow travellers speciated together.

Copy fidelity is relevant to evolutionary concerns. But there is no fidelity threshold that all replicators must meet. For there is a relationship between

fidelity and the strength of selection. If copying is very error ridden, and selection is weak, then noise can swamp selection, and cumulative selection will be unable to build complexly adapted interactors. But stronger selective regimes can drive evolution in less perfect replication regimes (Wimsatt 1981).

So our conception of replication and replicators must enable us to identify the links in a lineages of replicators exposed by their associated interactors to cumulative selection. This conception requires perfect fidelity of neither replicator nor interactor. Indeed, the interactors associated with successive links in a lineage of replicators can vary quite widely, as adaptive plasticity, variation within a population, sexual dimorphism, and the alternation of generations in plant lineages illustrate. There is no general reason deriving from the importance of very high copying fidelity for supposing that genes are the only, or almost the only, replicators. The issue is empirical. Are other mechanisms of inheritance from interactor to interactor so noisy that cumulative selection on them is not possible? We doubt it.

Experience suggests that our extension of the roll of replicators will be seen as a rejection of Weismann in favour of Lamarck. It is important to short-circuit this misconception. Weismann and his successors have shown that one apparently possible mechanism of evolutionary change is not actually possible. An organism's phenotype can change and change in a way that alters its genotype: a mouse can shift its residence to a leaky nuclear reactor. But an organism's changes cannot restructure its genotype so that its descendants manifest the changed trait. A mouse that acquires the ability to exploit a new food source cannot transmit that ability to its descendants via changes in its genome. The discovery of this constraint on inheritance is of great significance. But the discovery is a discovery of a constraint on a specific mechanism of inheritance, not a constraint on any mechanism of inheritance. Moreover, Weismann did not show that the only inheritance mechanism is genetic; few deny that social learning is an inheritance mechanism. Nor did he discover that nongenetic inheritance is subject to the same constraint. Nothing in this paper is inconsistent with Weismann's constraint on genetic inheritance. Nothing we say is inconsistent with what Weismann actually discovered rather than with what he is occasionally imagined to have discovered.

There are important distinctions amongst the elements of the developmental system. Amongst the factors that influence development, some but not all, are part of a copying and interaction cycle. Garbage cans are part of the developmental matrix of many suburban-adapted Australian possums, but possum behaviour does not result in a flow of new cans. Only some elements of the developmental matrix are adapted for their role in development. The

explanation for their existence and nature is that earlier copies played a similar role in the development of similar phenotypic systems. Platypus burrows exist in their contemporary forms because earlier copies played a similar role in the development of burrowing platypuses. That is not true of the relationship between the paradise parrot and the termite mounds they nested in. Termite mounds continue; sadly, the paradise parrot does not.

4. Replicators and interactors

Most of the original motivations for the replicator/interactor conception are really arguments against the Received View rather than arguments for a particular heterodoxy. But not all: one interesting and important line of thought supports the replicator-interactor conception of evolution.

4.1 *Organismism*

Hull and Dawkins use the Gene's Eye conception of evolution to release the grip of the organism on our biological imagination. Hull emphasises botanical and other examples that undercut the idea that the organism is well-defined and readily identifiable (1988, chapter 11). In his hands, this line of thought emphasises the atypicality of the "paradigm organism": the multicellular animal. So conceived, organisms are rare, special, aberrant. Most of life does not come in packages like that. Protozoa, colonial organisms, many plants and their clones do not fit. Evolutionary theory should not be conceptualised by appeal to examples that are atypical of life's evolution.

Hull reinforces these intuitive considerations by a more formal argument. The natural kinds of biology are those that play a distinctive role in biological theory. There is nothing, Hull argues, that all and only genes, organisms, groups or species do. Nothing of evolutionary interest is true of, for example, all and only organisms. The natural kinds are replicators, interactors and lineages. Genes are paradigm replicators, but not quite the only ones: in asexual or genetically homogeneous populations (e.g., cheetahs) much larger units – chromosomes, genomes or even the organism itself may qualify. Organisms are the paradigm interactors but not the only ones.

Dawkins' arguments for the extended phenotype (1982 and 1989) are another version of this idea. Selection does not see the naked replicator. Genes are selected in virtue of their phenotypic effects. But the effects in question need not be effects expressed in the body in which the replicated gene is situated. Genes have extended phenotypes. Some of their jointly constructed adaptations are aspects of the organisms in which they ride, and through

which they replicate. But some are not. Dawkins emphasises adaptation-at-a-distance: adaptations that aid the replication of genes, but which are not adaptations of the body in which the genes live. Adaptation-at-a-distance enables us to see the evolutionary identity of nest building of the caddis fly with shell secretion of molluscs. Though the caddis house is not a trait of the organism, it is just as much an adaptation, and an aspect of the caddis genes' active replication. The same perspective shift enables us to see the altered behaviour of a parasite's host as an adaptation of the parasite's genes.

This line of thought is permissive rather than compelling: the Received View remains viable. It is possible to insist that the adaptation in question is the secretion altering the behaviour of the host. But there does seem to be reason for singling out one link in this chain. The effects on the hosts' behaviour is the most salient link. The adaptive effect of the parasite's genes is the effect on the host's behaviour.

We like these arguments. But they are neutral amongst those positions which reject the Received View. Consider, for example, a robin feeding a robin chick and it feeding a cuckoo. On the Received View, one comes out as adaptive behaviour; the other, maladaptive. But the alternatives capture a commonality. Developmental Systems Theorists see both behaviours as stable results of typical packages of developmental interactants which tend to recreate the conditions for their own reproduction. In both the robin-robin and the robin-cuckoo systems, the boundaries of the organism are of no special significance. With this the defenders of the Gene's Eye agree: both behaviours express extended phenotypic effects. The feeding genes are in the chicks. The robin is being manipulated by outside genes in both cases, but we overlook robin chick manipulation because we are often blind to conflicts of interest between parent and offspring. The Extended Replicator, in this instance, takes over the Gene's Eye explanation. For us though, it is an open question whether the genes are the only replicators in these interactions with extended phenotypic effects. In the manipulation of the robin, other replicators may play a role. The physical surrounds – the nest structure – may be an essential feature of the chick's manipulations. So the nest may be collaborating with the robin chick (and cheated by the cuckoo chick) to ensure a new generation of nests (see 5.1). There may be egg-line but not DNA factors essential to the survival of the cuckoo's egg, or the development of the chick's manipulative skills. In sum, the heterodox will describe these cases in somewhat different languages, but they all see something that the Received View misses.

4.2 *Comparing heterodox perspectives*

Dawkins 1982 points to phenomena that can be explained from the perspective of the Received View, but which can be much more easily grasped from that

of the Gene's Eye. For example, it is much easier to see the problem posed by sexual reproduction from the gene's perspective. How can it benefit a gene to collaborate in a system of replication in which its chances of being replicated are only 50%? The *Extended Phenotype* is full of examples of the value of these perspective shifts. One particularly striking example is that of the organism itself. From the perspective of the germline cells, the construction of the body is an enormous investment of resources that might instead be directed directly to replication. Why is it worth it? Why is this investment not inevitably subverted by cell-line rebellion? (Buss 1987) A perspective shift reveals these questions. We see no help here from the Developmental System perspective. On that view, neither the existence of organisms nor of sexual reproduction seem particularly problematic. No doubt it is possible to formulate these problems in that language, but they are not "in your face".

So one important argument for the Gene's Eye is not undermined by the more radical perspective. Since we take the genes to be replicators, we inherit this advantage. But why prefer our picture to that of Dawkins? There is a formal argument: any decent definition of replication applies to lots of nongenes, for there are many inheritance mechanisms, not just one or two. There is an historical argument: genes evolved from earlier extinct replicators (Cairns-Smith 1982). We think there may also be a heuristic argument: seeing certain processes as replication opens up new questions and poses interesting problems. We cannot claim proof here: it remains to be seen whether that perspective generates fruitful new work. But we think it may, while blocking none of the heuristic advantages of Dawkins' perspective shift.

Consider, for example, sexual reproduction. Sexuality surely says something about differences amongst developmental agents. Genetic recombination within a restricted group produces controlled phenotypic diversity. If other developmental factors are capable of playing the same role, we would expect to see reproductive systems that exploit other factors to generate but control diversity. So we expect to find asexual lineages which generate controlled phenotypic diversity by varying the developmental matrix through migration, or through varying the time or season of reproduction. And we expect to find sexual lineages where controlled diversity is generated not just by recombination but by other adaptations. Our hunch is that there are other elements exploited to generate diversity and that these will be adapted to play a similar role to germline genes. If we turn out to be right, then our conception would be shown to have the same heuristic advantages over the Gene's Eye that it has over the Received View. Moreover, it would decisively rebut Sober's claim (e.g., Sober 1990) that if gene selectionist claims are about replication they are trivial, merely telling us what we already know.

4.3 *What is copying?*

We need an account of replication which does not prejudge the count of replicators, nor understate the complexities of replication. We do not see complexity as problematic, for an e-mail copy of this paper is very indirectly produced, and depends essentially on many elements additional to the Word document. Yet it is a copy for all that. So without attempting an explicit definition, we propose that the following elements are part of the biologically interesting notion of replication.

If B is a copy of A:

(i) A plays a causal role in the production of B

(ii) B carries information about A in virtue of being relevantly similar to A. This similarity is often functional: B has the same, or similar, functional capacities to A. Indeed, we should probably think of "copy" as a three-term relation: B is a copy of A with respect to C, where C is often some function of A.

(iii) B respects the xerox condition: B is a potential input to a process of the same type that produced it.

(iv) Copying is a teleological notion. For B to be a copy of A it must be the output of a process whose biofunction is to conserve function. On this view, the mere similarity of B to A does not suffice for B to be a copy of A. A fossil of a leaf is not a copy of a leaf. B must be meant to be similar to A; that similarity is why those mechanisms exist. Copying is a process with the function of producing from one token another which is relevantly similar.

We can thus see why genes can be copies without downplaying the complexity of this process. Nor need we suppose that genes, nor any other replicator, are "self-replicating".

5. Against lifeism?

There are many mechanisms of inheritance. In this final section, we wish to explore the possibility of a dramatic extension of inheritance notions by examining recent rhetoric in evolutionary biology. One strand of the anti-adaptationist literature, and one strand of vicariance biogeography have talked of life and the environment evolving together. We take seriously the idea that elements of the environment evolve.

5.1 *The selfish burrow*

Nesting burrows are replicators. The causal relations between burrows and burrowers is like that between genes and their interactors. No gene makes an organism. But variance explains variance: a variable oystercatcher may be black rather than pied because it has one gene complex rather than another, even though no gene complex makes a colour pattern. Similarly, a variation in a burrow can cause a variation in a burrower: a particular penguin chick may be healthy and safe because its burrow has one site rather than another, even though no burrow features make penguin flesh.

Second, burrows are part of a copying and interaction cycle. They exist in the forms they do because of their role in this cycle. We think this is important. Griffiths has urged against our extension of the concept of replicator that we underplay the significance of relationships and their reproduction. So he points out:

> the evolution of the hermit crab-shell relationship is interesting. Something is being replicated, and it isn't the shell. Is it the shell-using behaviour? Maybe, but that would hardly explain the evolutionary dynamics of the population documented by Gould which uses fossil shells and is now going extinct (personal communication).

We read a quite different moral out of this example. The crab-fossil shell relationship is not a replicator, *precisely because* the hermit crab is unable to influence the availability of a critical resource in the next generation. There are no mechanisms in this developmental matrix which have the biofunction of shelter-making. That is part of the rather sad evolutionary dynamics here. So while this relationship is of evolutionary interest, it is not copied, though shell hunting and occupying behaviour may be. The hermit crab snail shell relationship is thus quite unlike that between penguins and their burrows.

Burrows bear information about burrowers and the next burrow generation. For burrows interact with their guests in ways that result in mutual changes. They and their guests coevolve. Chance changes in burrow copies can proliferate. A chance favourable burrow copying at a new and superior site – for example, one less liable to flood – may result in a bushy lineage of similarly changed burrows. So they are replicators in the sense closest to Dawkins' heart: a change in a burrow is sometimes copied through to the next generation.

These ideas emerge most clearly from a consideration of Bateson's challenge to the *Selfish Gene*. Bateson (1978) pointed out that one consequence of Dawkins' conception is that nests are replicators, and that a bird is but a nest's way of making another nest. Dawkins rejected this idea on the grounds that variation is not transmitted. Whatever the merits of the Selfish Nest as

an evolutionary hypothesis, it cannot be rejected on those grounds. First, because Dawkins appeals here to the same criterion used to exclude asexual organisms as replicators; a criterion unsatisfactory on other grounds. Second, it is not in general true. Environmentally altered patterns in cilia are inherited through fission (Majerus and Hurst 1993). Variation in both nesting materials and nest siting can be transmitted (Dickison 1992; Gray 1992). Perhaps even variations in builders can be: some New Zealand petrel nests are inhabited by both petrels who build them and tuatara who live in and maintain them all year around: this change in occupancy pattern has become typical of that local deme of burrows, and is now part of local burrowgenesis. Naturally, as in all new fields, delicate empirical questions remain. Tuatara are known to eat nestling petrels from time to time. So it's a question for future research whether this impact on the burrow-building population so adversely affects burrow fecundity as to outweigh their positive effect on longevity. If so, we would have to regard tuatara as burrow-parasites.

If nests are replicators, they are clearly active replicators. Their properties of insulation, durability, protection and cost of construction quite obviously influence the probability of their being reproduced. They form lineages. A nest plays a role in the construction and protection of builders who disperse to produce a new and relevantly similar nest. There is a flow of information linking nest generations through the builders. Nests and burrows are adapted for the growth of burrow builders and nest-makers. Those interactors carry the information through which the nest is replicated.

Bateson was right: sometimes, perhaps always, nests meet Dawkins' definition of a replicator, and they meet ours too. Nests are produced by mechanisms whose biofunction is to reproduce this critical developmental resource. But there is no reason to suppose this is a reductio of these accounts of replication; Dawkins here betrayed a rare moment of timidity.

5.2 *Selfish burrows or selfish burrowing genes?*

Of course, a Gene's Eye conservative may deny that the burrow is the replicator here. They might claim that the real replicator is the penguin gene or meme for burrowing on (say) predator-free Kapati Island rather than the predator-infested Wellington foreshores.

Redescription is possible but not appropriate. First, it is certainly possible for the burrow lineage to grow bushy without a burrowing meme that grows bushy. The successful colony can grow as the result of greater than average burrow success, even if burrow residents are no more likely to reburrow there next season. The increase in burrowers together with the differential survival of Kapiti burrows might explain a bushier burrow lineage there: its growth does not imply a change in the genetics or psychology of its

denizens. Moreover, the considerations which favour Dawkins' conception of an extended phenotype reapply here. With the usual caveats about costs, changes in the developmental mechanisms that make no difference to the caddis house itself are selectively neutral; only variations that vary house design matter. Hence, it is reasonable to focus on caddis houses rather than caddis larvae house building. Similarly, changes in the way burrows are built that do not result in variation amongst burrows are not relevant to burrow evolution. It's the burrow, not the particular form of the burrowing meme that matters to burrow evolution.

Second, we can accept that burrow genes and burrowing memes are replicators without denying that burrows are replicators. There probably will be a site tradition handed on amongst burrow denizens, so as the burrow lineage grows bushy this one will grow bushy with it. The Extended Replicator will often allow a two-way view. From the perspective of the burrow, the burrower is an interactor whose differential reproduction differentially replicates the burrow. Equally, from the perspective of the burrowing genes and memes the burrow is an interactor – part of the interaction process whose differential reproduction promotes a longer and bushier burrowing gene. The same will be true of more conventional mutualisms; for example those between ants and plants. From the perspective of each the other is a interactor: adapted to the environment in such a way that its success vis-a-vis its competitors results in the differential replication of its colleague.

There is nothing mysterious about this perspective in which the one entity acts both as replicator and interactor; both the beneficiary of adaptation, and the bearer of adaptation (Lloyd 1992). The Gene's Eye view is itself committed to a restricted form of this pluralism. Even if we were to restrict our focus entirely to genetic replication, not all of a gene's adaptations need be adaptations for that gene. Sometimes they are: because they ensure its replication, various molecular and cellular relations of a gene will count as adaptations of it for it. But from the perspective of other genes, these can be interactors carrying their adaptations for other genes' benefit. For example, consider a repressor gene. For other genes, it's an interactor carrying adaptations for their benefit. The characteristics it has to ensure high-fidelity replication benefit them too. They may well themselves have characteristics which make its replication and repression more effective. (Moss 1992). So some of their characters count as adaptations they bear partially for it. It benefits from their characters; they benefit from it. Now consider an ex-outlaw gene now permanently turned off, and hence part of junk DNA. It still gets replicated, so some of its structures and relations ensure that, and hence are adaptations for it. But some of those relations ensure it is inactive. Though these are adaptations of the outlaw gene, they are not adaptations for it but

for other genes. They are extended phenotypic effects of, and adaptations for, repressor genes.

Third, there is no reason to privilege the causal dependence of the selfish burrow on selfish genes and memes. Of course, it is true that there is only a causal chain from burrow to burrow because there are causal chains from burrow genes to burrow genes. The burrows can replicate only if burrow genes replicate. But this dependence is symmetrical: holding causal background constant, burrow genes replicate only if burrows replicate. If the chain of burrows were to fail, the chain of burrow genes fails too. Nor can this symmetry be broken by tracking back into history. Each replicator has been part of the evolutionary history of the other, and we are certainly in no position to claim that any current burrow gene or meme lineage is deeper rooted in history than the burrow lineages.

For those who think this view must be a joke, at best, we offer the following two cautions. First, evolution produced the paradigms of the living, so evolution cannot be restricted to lineages of entities that we would now count as living. Evolutionary change in lineages of nonliving or quasi-living entities must be possible. Second, it does not follow that these evolutionary processes have the same power as those operating in the living world. It is sometimes said that one gets evolution under natural selection whenever there is heritability, variation and differential fitness amongst the variants. Perhaps so, but if selection is to explain major adaptation it must be cumulative. Innovation is the result of a long sequence of selective episodes rather than one. Cumulative selection requires much tighter conditions. So even if nests and burrows evolve, it may be that their evolutionary dynamics and possibilities are more like those of the long-disappeared lineages of proto-prokaryotic cells rather those that have lead to complex adaptation.

6. Conclusion

In sum, the radicals' insistence of the seamless nature of developmental interaction is important and they are right to deny to the gene any exclusive role in development or evolution. But we think both the replicator-interactor distinction, and some of the reasons for making it, survive their critique. However, rebutting the Developmental Systems Theorist's critique extends our conception of the cast of replicators, and hence should shift our perspective from that of the Gene's Eye to that of a still more raucous and motley crowd of squabbling replicators.[5]

Notes

[1] See paradigmatically: Oyama 1985; Gray 1992; Gray and Griffiths 1994; Moss 1992. Oyama prefers to speak of "Evolutionary Developmental Systems". In her view, we are interested in a species of a larger genus of views and interests. This group draws on other figures: see especially Bateson 1976, 1983 and 1991. Johnston 1987 and 1988. Griesemer forthcoming and Smith 1992 and 1993a defend some of the their distinctive negative theses but not their positive ones.

[2] Refugees from the philosophy of mind will recognise here an adaptation of Fodor on mental content; see for example his 1990, chapter 4. Ruth Millikan 1991 pointed out that these asymmetries must be given a teleological twist.

[3] It's a consequence of our view that the informational role of the gene is to carry information about the parental phenotype to the developing phenotype. It would follow that the source is the interactor/replicator system of generation 0, the signal is the gamete, and receiver is the interactor/replicator system of generation 1.

[4] We disagree on whether neutral genes are interactors. For a neutral gene to be an interactor, it must have effects which makes its replication differential with respect to actual or past rivals. Sterelny thinks that the relational properties of neutral genes can give them replication advantages; for example, they can hitch-hike. Smith and Dickison think that this trivialises the concept of an interactor. On both views, a mutation in a neutral gene counts as a mistake in the second sense: a copying mechanism has not operated as designed. In Sterelny's view, a neutral gene can fail to do as it is designed to do in the first sense as well: if some disruption of its structural and relational properties block its replication.

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References

Bateson, P.: 1976, 'Specificity and the Origins of Behavior', *Advances in the Study of Behavior* **6**, 1–20.

Bateson, P.: 1978, 'Review of The Selfish Gene', *Animal Behaviour* **26**, 316–318.

Bateson, P.: 1983, 'Genes, Environment and the Development of Behaviour', in Slater, P. and Halliday, T. (eds.), *Animal Behaviour: Genes, Development and Learning*, Blackwell.

Bateson, P.: 1991, 'Are There Principles of Behavioural Development?', in Bateson, P. (ed.), *The Development and Integration of Behaviour*, Cambridge University Press, Cambridge, pp. 19–39.

Brandon, R.: 1990, *Adaptation and Environment*, Princeton University Press, Princeton.

Buss, L.: 1987, *The Evolution of Individuality*, Princeton University Press, Princeton.

Cairns-Smith, G.: 1982, *Genetic Takeover and the Mineral Origins of Life*, Cambridge University Press, Cambridge.

Clayton, D. and Harvey, P.: 1993, 'Hanging Nests on a Phylogenetic Tree', *Current Biology* **3**, 882–883.

Dawkins, R.: 1976, *The Selfish Gene* (second edition 1989), Oxford University Press, Oxford.

Dawkins, R.: 1982, *The Extended Phenotype*, Oxford University Press, Oxford.

Dickison, M.: 1992, 'The Death of the Organism or The Selfish Nest', *Paper to AAP(NZ)*.

Fodor, J.: 1990, *A Theory of Content and Other Essays*, MIT Press, Cambridge.

Fogle, T.: 1990, 'Are Genes Units of Inheritance', *Biology and Philosophy* **5**, 349–372.

Glover, D., Gonzales, C. and Raff, J.: 1993, 'The Centrosome', *Scientific American* **268**, 32–39.

Goodwin, B.: 1989, 'Unicellular Morphogenesis', in Stein, W.D. and Bonner, F. (eds.), *Cell Shape*, Academic Press, New York.

Grant, T.: 1989, *The Platypus: A Unique Mammal*, New South Wales University Press, Kensington, Sydney.

Gray, R.: 1992, 'The Death of the Gene', in Griffiths, P. (ed.), *Trees of Life: Essays in the Philosophy of Biology*, Kluwer, Dordrecht, pp. 165–210.

Griesemer, J.: 'The Informational Gene and the Substantial Body: On the Generalization of Evolutionary Theory By Abstraction', forthcoming in Cartwright, N. and Jones, M. (eds.), *Varieties of Idealisation*, Poznan Studies in the Philosophy of the Sciences and the Humanities, Amsterdam.

Griffiths, P. and Gray, R.: 1993, 'Individuating Developmental Systems', *Paper to the International Society for the History, Philosophy and Social Studies of Biology*, Boston.

Griffiths, P. and Gray, R.: 1994, 'Developmental Systems and Evolutionary Explanation', *Journal of Philosophy* **91**, 277–304.

Hull, D.: 1984, 'Units of Evolution: A Metaphysical Essay', in Jensen, R. and Harre, R. (eds.), *The Philosophy of Evolution*, Harvester, Brighton; reprinted in Brandon, R. and Burian, R. (eds.), *Genes, Organisms Populations*, MIT Press 1984.

Hull, D.: 1988, *Science as a Process*, Chicago University Press, Chicago.

Johnston, T.: 1987, 'The Persistence of Dichotomies in the Study of Behavioural Development', *Developmental Review* **7**, 149–182.

Johnston, T.: 1988, 'Developmental Explanation and The Ontogeny of Birdsong: Nature/Nuture Redux', *Behavioral and Brain Sciences* **11**, 617–663.

Keller, L. and Ross, G.: 1993, 'Phenotypic Plasticity and 'Cultural Transmission' in the Fire Ant *Solenopsis Invicta*', *Behavioural Ecology and Sociobiology* **33**, 121–129.

Lewontin, R.: 1985, 'The Organism as the Subject and Object of Evolution', in Levins, Richards and Lewontin, Richard (eds.), *The Dialectical Biologist*, Harvard University Press.

Lloyd, E.: 1992, 'Unit of Selection', in Fox Keller, E. and Lloyd, E. (eds.), *Keywords In Evolutionary Biology*, Harvard University Press, Cambridge.

Majerus, M. and Hurst, G.: 1993, 'Weird Genetics? Evolution and Nonmendelian Genes', *Trends in Ecology and Evolution* **8**, 310–311.

Millikan, R.: 1989, 'In Defense of Proper Functions', *Philosophy of Science* **56**, 288–302.

Millikan, R.: 1991, 'Speaking Up for Darwin', in Loewer, B. and Rey, G. (eds.), *Meaning in Mind: Fodor and his Critics*, Blackwell.

Morgan, N. and Baumann, P.: 1994, 'Phylogenetics of Cytoplasmically Inherited Microorganisms of Arthropods', *Trends in Ecology and Evolution* **9**, 15–20.

Moss, L.: 1992, 'A Kernel of Truth? On the Reality of the Genetic Program', in Hull, D., Forbes, M. and Okruhlik, K. (eds.), *Proceedings of the Philosophy of Science Association*, volume one, pp. 335–348.

Oyama, S.: 1985, *The Ontogeny of Information: Developmental Systems and Evolution*, Cambridge University Press, Cambridge.

Reilly, P.: 1981, *The Lyrebird*, New South Wales University Press, Kensington, Sydney.

Smith, K.: 1992, 'The New Problem of Genetics: A Response to Gifford', *Biology and Philosophy* **7**, 331–348.

Smith, K.: 1993a, 'Neo-Rationalism Versus Neo-Darwinism: Integrating Development and Evolution', *Biology and Philosophy* **7**, 431–452.

Smith, K.: 1993b, 'The Effects of Temperature and Daylength on the Rosa Polyphenism in the Buckeye Butterfly, *Precis coenia* (Lepidoptera: Nymphalidae)', *Journal of Research on the Lepidoptera* **30**, 225–236.

Smith, K.: 1994, *The Emperor's New Genes: The Role of the Genome in Development and Evolution*, Ph.D Dissertation, Duke University, Durham, NC, USA.

Sober, E.: 1990, 'The Poverty of Pluralism: A Reply to Sterelny and Kitcher', *Journal of Philosophy* **87**, 151–158.

Sterelny, K. and Kitcher, P.: 1988, 'The Return of the Gene', *Journal of Philosophy* **85**, 339–361.

Sultan, S.: 1987, 'Evolutionary Implications of Phenotypic Plasticity in Plants', in Hecht, M., Watson, B. and Prance, G. (eds.), *Evolutionary Biology* **21**, 127–178.

Wagner, G.P.: 1988, 'The Vexing Role of Replicators in Evolution', *Biology and Philosophy* **3**, 322–336.

Werren, J.H.: 1991, 'The Paternal Sex Ratio Chromosome of *Nasonia*', *American Naturalist* **137**, 392–402.

Wimsatt, W.: 1991, 'The Units of Selection and the Structure of the Multi-Level Genome', in Asquith, P. and Giere. R. (eds.), *Proceedings of the PSA 1980*, Volume 2, Philosophy of Science Association, East Lansing.