Replication, Information and Complexity

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Draft of 10 July 2006

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ABSTRACT

This paper considers the development and refinement of the replicator concept. We find that the established definition of replication in terms of the triple conditions of causality, similarity and information transfer is too inclusive. We draw inspiration from the literature on self-reproducing automata to strengthen the notion of information transfer in replication processes. Essentially, we find that replication must have the potential to enhance complexity, which in turn requires that developmental instructions are part of the information that is transmitted in replication. In addition to the triple conditions of causality, similarity and information transfer, we adopt a fourth condition that defines a replicator as a *conditional generative mechanism*, a material entity that embodies mechanisms that can turn input signals from an environment into developmental instructions. Demonstrating the usefulness of the replicator concept in the social domain, we identify habits and other social replicators that satisfy all of the four proposed conditions for a possible replicator.

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1. Introduction

In recent years, considerable progress has been made in refining the abstract definitions of the core Darwinian concepts of variation, inheritance and selection. In part, this work has been powered by the realisation that Darwinian principles do not apply to biological organisms alone, but to the evolution of a broad class of phenomena, perhaps including ideas, memes, technologies or social institutions.[1] If Darwinian principles have a general application to complex systems involving populations of varied and causally interacting entities, then we are obliged to specify these principles clearly in abstract terms.

This essay focuses on the issues of inheritance and replication. Since it was coined by Richard Dawkins (1976), the replicator concept has been clarified (Hull, 1988; Sterelny *et al.*, 1996; Godfrey-Smith, 2000; Sperber, 2000; Aunger, 2002; Nanay, 2002). However, efforts in this direction are made more complicated partly by the famous 'evo-devo' dispute concerning the relative roles of development, selection and genotypic transmission (Baguñà and Garcia-Fernàndez, 2003; Gilbert *et al.*, 1996; Stadler *et al.*, 2001; Wimsatt, 1999). Furthermore, recent formulations of 'developmental systems theory' amount to attempts to generalize Darwinian principles in very broad terms that sideline or exclude the replicator concept (Griesemer, 1994, 1999; Griffiths and Gray, 1994, 1997; Wimsatt, 1999).[2]

Theorists emphasising the developmental side of the evolutionary process point out that inherited information itself cannot provide a complete description of the emerging phenotype and consequently development depends crucially on the particular context. Hence heredity and development interact in a way that cannot give overwhelming priority to the genotype. James Griesemer (1994) thus roots the concept of inheritance in whole development lineages, rather than in genotype-to-genotype transmission.

Partly because of its origination from Dawkins, the replicator idea has been linked with overly 'gene centred' accounts of the generalized Darwinian evolutionary process. Paul Griffiths and Russell Gray (1997, p. 473) explain that developmental systems theory 'takes to its logical conclusion the slow unravelling of the idea that genes are the sole evolutionary replicators.' In particular, some biologists have 'drawn attention to the large class of structures which are inherited parallel with the genes and play an essential role in

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[1] See for example Dawkins (1983), Hull (1988), and Dennett (1995). Hodgson and Knudsen (2006) discuss the philosophical basis for Darwinian concepts in the social sciences and Hodgson (2005) for a brief history of attempts to generalize Darwinism.

[2] Wimsatt (1999) rejects the replicator-interactor distinction but retains the genotype-phenotype concepts. This is somewhat inconsistent if we regard the former distinction as a generalization of the latter (Brandon, 1996).

development at the cellular level. … These intra cellular elements of the developmental matrix are essential for the replication of DNA and are not themselves constructed on the basis of DNA sequences.' As Eva Jablonka and Eörs Szathmáry (1995) argue, these parallel structures constitute an additional 'epigenetic inheritance system'. Pushing this still further, developmental systems theory shifts the focus onto the entire 'developmental system' rather than the replicator and its 'vehicle' (Dawkins, 1976) or 'interactor' (Hull, 1988).

We are not biologists – we are social scientists with some philosophical training. For some time we have been examining the possibility of explaining sociocultural evolution in terms of generalized Darwinian principles. Darwin (1859, 1871) himself hinted at this possibility in his brief discussions of the evolution of language and ethical principles. Other social scientists developed the idea more than a century ago (Bagehot, 1872; Ritchie, 1896; Veblen, 1899; Keller, 1915) but research in this area fell out of favour after the First World War. It was revived through the efforts of Donald T. Campbell (1965) and others. Since then, Robert Boyd and Peter Richerson (1985), and William Durham (1991) have considered culture as an inheritance mechanism, and like many others have adopted the Dawkins terminology of 'meme' to describe the unit of cultural replication. However, this recent literature largely bypasses the question of the detailed mechanisms of cultural replication or transmission (Wimsatt, 1999) and even remains ambivalent concerning whether 'memes' are genotypes or phenotypes, replicators or interactors (Blackmore, 1999). By contrast, our aim is to explore in more detail the social and psychological mechanisms of sociocultural evolution, and place these in a generalized Darwinian framework.

It may seem that we would need the broader theoretical framework of developmental systems theory to accommodate the perplexing complications of sociocultural evolution. Once we examine the mechanisms of social or cultural transmission then it becomes clear that whatever a 'meme' is, it is very different from a gene. Once the loose treatment of the units of cultural evolution as ideas or behaviours is probed to find the transmission mechanisms involved, then we are presented with multiple possible substrates, from brain patterns to symbols and artefacts. Nothing in social culture remotely corresponds to the DNA code. As Stephen Jay Gould (1996) and William Wimsatt (1999) elaborate, there are further radical dissimilarities. For example, in the sociocultural domain there are complex patterns of 'parental' inheritance and the absence of clear boundaries between species. For Gould these are reasons to abandon the project of finding communalities between biological and sociocultural evolution. For Wimsatt they mean that evolutionary concepts have to be pushed to a high level of generality, after abandoning the distinction between replicators and interactors.

In contrast, we propose that the distinction between replicator and interactor can and should be retained in the sociocultural domain. Of course, if our proposition proved to be unfounded or unworkable, then we would be obliged either to follow in the steps of Wimsatt and others and adopt a more abstract framework, or to abandon entirely the notion of communalities between the biological and sociocultural evolution.

Part of our rationale for retaining the distinction between replicator and interactor devolves from the understanding that Darwinian evolution is essentially about interacting and replicating *populations* of (varied and developing) entities rather than *singular* 'self organizing' or 'developing' systems. Once this focus on populations is established, then we need to understand the sources of both similarity and variation between elements, and to place this in the context of both the development of individual entities and the evolution of entire populations. We regard the replicator and interactor concepts as essential to this task. As

Robert Brandon (1996, p. 125) argues, the distinction between replicators and interactors 'is best seen as a generalization of the traditional genotype-phenotype distinction'. Hence, without the replicator and interactor, the distinction between genotype and phenotype similarly dissolves. Blind to genotypes, we can then only address phenotypical characteristics and behaviour. While population-level developments are emphasised, the micro-mechanics of individual adaptation and development are relatively neglected. We loose the boundaries between the processes at the individual level and those in the population as a whole.

Our alternative strategy here builds on other recent work on the nature and definitions of replicators and replication. We also pay more attention to the question of levels of replication and selection than some other contributions in this area. Although the possibility of multiple levels of selection has long been mooted by biologists and is well established in the philosophy of biology (Brandon, 1996; Keller, 1999; Michod, 1999; Sober and Wilson, 1998), it is sometimes sidestepped in discussions of replicators and interactors.

Nevertheless, it is especially pertinent in this context, because what might emerge as an interactor at one level might conceivably act as a replicator at another (higher) level. For instance, some prominent accounts view human individuals as biological interactors and their genotypes as biological replicators. The admission of an additional and cultural level of transmission leads Boyd and Richerson (1985) and Durham (1991) to treat individual ideas, dispositions or preferences as replicating units in that 'higher' domain. It is only one further step to consider social groups, structures or institutions as plausible interactors or units of selection (Ritchie, 1896; Veblen, 1899; Keller, 1915; Nelson and Winter, 1982; Henrich, 2004; Hodgson and Knudsen, 2004b). If this is a valid move, then the (human individual) interactors at the biological level become the repositories of replicator-like features (dispositions or preferences) at the sociocultural level. Consequently, the question of multiple levels of selection has to be moved higher up the agenda.

Failure to do this might exacerbate the problems identified by Kim Sterelny *et al.* (1996); they argue that developmental systems theory lacks adequate definition of the boundaries of the units in the evolving population. We propose that some such boundaries must exist in an evolutionary system that is capable of retaining relatively successful adaptations and generating increasing complexity.

A second feature of our strategy is to make a link with the literature in mathematics and computer science on self-reproducing automata (Neumann, 1966; Sipper, 1998; Freitas and Merkle, 2004). Well over half a century has passed since John von Neumann developed his theory of self-reproducing automata, yet the discourse on replication in the philosophy of biology has made little attention to his work. Yet this would seem to be an important, not only because it provides theoretical illumination but also it offers an additional context in which any generalized Darwinian theory must fit.

The remainder of this paper is organized as follows. Section two considers the replicator concept and important contributions to its development and refinement. We find that the widely used definition of replication in terms of the triple conditions of causality, similarity and information transfer is too loose and inclusive. These criteria are necessary but insufficient in defining replication. In particular, we propose that the notion of information transfer must be further clarified. Section three draws from the literature on self-reproducing automata to strengthen the notion of information transfer in replication processes. Essentially, we find that replication must have the potential to enhance complexity, which in turn requires that developmental instructions are part of the information that is transmitted in replication.

Section four redefines the replicator concept on the basis of our proposed clarification of the notion of information transfer. In addition to the triple conditions of causality, similarity and information transfer, we adopt a new condition that defines a replicator as a *conditional generative mechanism*, a material entity that embodies mechanisms that can turn input signals from an environment into developmental instructions. Section five considers in more detail the conjecture that replication must have the potential to enhance complexity. This is a critical part of our argument justifying our proposed additional condition for replication. Section six demonstrates the usefulness of the replicator concept in the social domain by identifying habits and other social replicators that satisfy all of the four proposed conditions for a possible replicator. Section seven concludes the paper.

2. Replicators and Replication

The replicator concept famously originates in the work of Richard Dawkins (1976). He described replicators as having longevity, fecundity and fidelity, but his definition requires further refinement. Dawkins identified both genes as 'memes' as replicators, but the term 'meme' was so broad that it encompasses ideas, practices, writings, paintings, artefacts, pop tunes and social institutions. There is ongoing controversy on what a meme is, and little progress in identifying the equivalent to the 'genetics' of memetic replication.[3]

David Hull (1988, p. 408) defines a replicator as 'an entity that passes on its structure largely intact in successive replications.' A key question here is what structures are significant and why. Again the definition requires refinement. And if a meme is a replicator then what structure is passed on?

One of the first attempts to define a replicator is by Sterelny *et al.* (1996). They propose that if B is a copy of A, and B is produced through a process of replication, then 'A plays a causal role in the production of B' and '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' (p. 396). Their definition of replication thus emphasizes the key points of causal implication, similarity and information transfer. These three elements are central to most subsequent definitions of replication.

There are other aspects of their definition that we need to go into here. We simply observe that all the elements of their definition, taken together, are used to admit a very wide class of replicators. Indeed, Sterelny *et al.* argue that non-organisms such as a bird nests and animal burrows also qualify as replicators. But the causal role that one nest or burrow plays in the production of another is highly limited. It exists only insofar as the attempt to produce one nest or burrow plays a crucial role, through learning or whatever, in the development of the organism's capacity to produce another. And the first attempt would obviously have no preceding copy. If nests and burrows are to be admitted as replicators, then their causality condition has to be interpreted in very weak terms. The causal links between such replicators are merely that each is a practice model for its successors. We suggest that this is an inadequate account of the causality condition: it has to be coupled with stipulations that make the causality connection more meaningful. Below we limit their 'extended replicator' to a broad class of entities that excludes nests, burrows, and most human tools and artefacts.

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[3] In adopting the replicator concept, we need not follow Dawkins's (rhetorical) account of the replicator as a selfish agent.

Dan Sperber (2000, p. 169) also argues that replication involves elements of causation, similarity, and information transfer, and specifies the 'minimal conditions' for replication: 'For B to be a replication of A,

(1) B must be caused by A (together with background conditions),

(2) B must be similar in relevant respects to A, and

(3) The process that generates B must obtain the information that makes B similar to A from A. … B must inherit from A the properties that make it relevantly similar to A.'

Sperber then argues that many cases of so-called memetic replication are not true replication according to this definition, principally because the third condition is violated. Hence the 'grand project of memetics … is misguided' (p. 173).

In another refinement of the replicator concept, Peter Godfrey-Smith (2000, p. 405) also emphasizes that replication involves 'two main elements, a *resemblance* between copy and copied, and some suitable *causal* relation linking the copy to the copied.' He is also sceptical of versions of cultural evolution based on the meme. For Godfrey-Smith (2000, p. 413): 'The … job of explaining *the heritability of variation,* in the sense relevant to evolution by natural selection … is the proper one for the replicator concept.' Godfrey-Smith then (2000, pp. 414-15) constructs the following definitions:

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.

Notably, Godfrey-Smith's definition requires similarity 'in some relevant respects', but does not specify what is 'relevant'.

Robert Aunger (2002) refines Sperber's (2000) definition of replication, to add a fourth condition: 'duplication'. By this he means that during the replication process, one entity gives rise to two (or more). According to Aunger, replication is a special type of inheritance where duplication is involved.

On the basis of these four criteria, Aunger argues at length that the salvation of the memetics project lies in his notion of 'the electric meme', referring to neurons and electrochemical connections in the brain. Aunger regards a meme as essentially the state of a node in a neuronal network capable of generating a copy of itself in either the same or a different neuronal network, without being destroyed in the process. Acts of communication between people lead to neural nodes replicating their state from one brain to another.

The original meme concept referred typically to ideas, not to material entities or structures, without enough consideration of the material substrate of the 'information' in the meme or of the physical mechanisms of replication. Aunger's dramatic reworking of the meme concept overcomes these limitations. However, he ends up with something that may be highly unpalatable to meme enthusiasts. The original notion of memes as replicating ideas is essentially abandoned.

If an idea is communicated from one person to another, then there is no guarantee that the sub-structures of neural states relating to the communicated idea, in the brains of the receiver and the sender will be similar. The idea may take hold in the brain of the receiver on the basis of an entirely different sub-structure of neural states. The idea is communicated, but there is no necessary or likely replication of neural structures. By driving the meme concept into the neuron, Aunger moves away from the communication and cultural transmission of

identifiable ideas, which memetics originally attempted to address. Instead he focuses on the replication of neural states. Aunger insists that replication involves similarity and locates it in the neural domain. Any necessary similarity at the level of ideas is abandoned. Aunger's radical refinement of memetics may well procreate viruses of doubt that eventually undermine the whole memetics project.

While Aunger's argument is illuminating we are unconvinced that his fourth condition 'duplication' is necessary to, or useful in, refining the concept of replication. For us, the critical issue in understanding the process of replication is the nature and function of the structure that is passed on, including any information that it might hold. It is not whether the replicator is lucky or unlucky in surviving the process of replication, leading or not to the coexistence of multiple copies. We concentrate on the key issues of structure and information that are central to replication. The three remaining and aforementioned conditions of causality, similarity and information transfer are necessary but insufficient for this task.

Why are they insufficient? A problem is that the criteria involved are rather weak and cover a broad class of copying processes. The triple conditions of causality, similarity and information transfer would also apply to technologies such as photocopying, and the copying of data on magnetic media or on compact disks. The condition of similarity pertains in all these cases. The causality condition is also satisfied because the original is causally implicated in the production of the copy, in the weak sense that without the original the copy could not exist. Sperber (2000) upholds that the 'information transfer' condition is satisfied in such cases because the 'information' in the copy originates from the original.

Although these criteria cover a broad range of copying processes, Sperber nevertheless claims that some repeated behaviours do not satisfy them. He considers the example of contagious laughter spreading through a group. Although one person's laughter may trigger the laughter of another, the laughter itself is 'not copied' because the second person does not imitate the laughter of the first. Instead 'there is a biological disposition to laughter that gets activated and fine tuned through encounters with the laughter of others. … The motor program for laughing was already fully present in him, and what the laughter of others does is just activate it' (Sperber, 2000, p. 168). He thus claims that the 'information transfer' condition is unsatisfied in this case.

Sperber argues that, by contrast, if a second sound recorder were switched on to record the sounds of a first, then this would be a case of true replication satisfying all three of his conditions. Similar remarks would seem to apply to the photocopying of documents. However, while these examples are different from the contagious spread of laughter through a group, the difference seems mainly of degree. The triggering of patterns of laughter may involve a signal that is much simpler than the detailed information copied by sound recorder or photocopier, but the difference lies in the amount and the kind of information transferred. In no case is the transferred information sufficient itself to produce the copy. In none of these cases is the copying mechanism itself copied.

What if the intensity and style of the initial laughter triggers mirth of a similar intensity and style in others? Then the initial laughter would be carrying information that was critical in forming the character of the copied laughter. Sperber himself admits the possibility of laughter being 'fine tuned' through encounters with others. In which case the initial laughter becomes more than a mere trigger: it carries significant information that is copied.

Furthermore, Sperber's own rephrasing of his third condition as 'B must inherit from A the properties that make it relevantly similar to A' can be interpreted in terms that exclude not only his example of laughter, but also the examples of copying with sound recorders or

photocopiers. It all depends what we mean by 'properties'. In his discussion of laughter, Sperber focuses on the capacity to produce the behaviour. The disposition to laugh is not replicated, but neither is the capacity to make copies with a sound recorder or photocopier. These machines already have the capacity to make copies. Hence, if these 'properties' include the capacity to produce a copy, as well as the information in the copy, then none of these examples satisfy the third condition of information transfer, because some of these properties are not inherited in the copying process.

Consequently, although Sperber's third condition is important and takes us in the right direction, it is insufficiently precise. It depends crucially on what is meant by 'information' and what properties have to be inherited to constitute true replication. We find hints on how to solve these problems in our discussion of self-reproducing automata in the next section.

3. Inspiration from Self-Reproducing Automata

In a series of lectures, posthumously edited and published as a book, John von Neumann (1966) considered the conditions under which automata could be built that would be capable of producing copies of themselves. For him, the copying process had also to be capable of generating novel outcomes and additional complexity. He did not propose that evolution necessarily leads to complexity, but instead he was interested in the conditions under which further complexity could be generated. Von Neumann (1966, p. 80) distinguished between the copy of entities that synthesized to produce higher degrees of complexity, and the copying of entities that reduced overall complexity:

> There is thus this completely decisive property of complexity, that there exists a critical size below which the process of synthesis is degenerative, but above which the phenomenon of synthesis, if properly arranged, can become explosive, in other words, where synthesis of automata can proceed in such a manner that each automaton will produce other automata which are more complex and of higher potentialities than itself.

Following von Neumann, we propose that replication should be confined to cases where increasing complexity is possible. This suggests the refinement of the concept of replication, so that a population of replicators must eventually be capable of producing more complex copies. This would seem to exclude copying with sound recorders or photocopiers, or the copying of nests and burrows. Such copies have no more potential to enhance complexity than their predecessors. To satisfy the criterion of increasing potential complexity, interacting and replicating copies must eventually be capable of producing novel additional components, or performing novel operations in response to new environmental conditions and input signals.

Von Neumann (1966) examined in detail the properties that a self-reproducing automaton must possess to make it capable of producing other automata. These properties include instructions that describe the structure and processes of an automaton, a copying unit that is capable of reading and copying instructions into a new automaton and translating them as directions for a production unit, a production unit that builds a new automaton, and some mechanism of coordination between these elements. His basic research on this topic has proved important for robotics and computer science (Sipper, 1998).

However, much caution is required when translating these concepts to the biological or social domain. Biological replication does not fit exactly the abstract properties of self-reproducing automata outlined by von Neumann. In particular, as developmental systems theorists emphasize, genes themselves do not carry enough 'information' to describe or

generate a new organism. Although the genes play a crucial role, the development of the organism depends additionally not only on environmental stimuli, but also on cellular structures inherited alongside the genes. Consequently, neither genes nor organisms constitute a complete 'production unit' for new organisms.

Given that the information transferred in the replication process can never be sufficient to generate a copy, and always requires an additional informational input, it is nevertheless important to recognize that some vital generative information is involved. The problem is to identify the character and extent of this generative information that is necessary for ongoing replication that can enhance complexity.

Two inspirations that we draw from the theory of self-reproducing automata are that replication must have the potential to enhance complexity, and developmental instructions are part of the information that is transmitted on replication. Essentially, a replicator is a material entity, placed in an environment, and responsive to environmental stimuli or signals. There is at least one signal that can cause a non-degenerative response from the replicator. Such a response consists of further instructions or signals to the interactor, which guide its development. It is non-degenerative in the sense that it leads to outcomes that are conducive to the survival of the replicator and the information it carries.

For example, the DNA 'code' determines the constitution of the protein molecules in the organism. It instructs the processes of cell-formation by governing the production of amino acids and proteins, subject to the circumstances and external conditions involved.

4. The Replicator Redefined

On the basis of the above discussion we adopt an additional proposition in the definition of a replicator:

1. **Conditional generative mechanisms**: The replicator is a material entity that embodies construction mechanisms that can be energized by input signals, containing information about a particular environment. These mechanisms generate further instructions from the replicator to the interactor, to guide its development. (External influences that produce outcomes generally unfavourable to the survival of the replicator or interactor are not described as input signals but as destructive forces.)

This first proposition adds to the established three definitional features for a replicator, namely causal implication, similarity and information transfer. We refine these as follows:

2. **Causal implication**: The source must be causally involved in the production of the copy, at least in the sense that without the source the particular copy would not be created.

3. **Similarity**: The copy must also possess the capacity to replicate and the conditional generative mechanisms in the copy must be similar to those in the source.

4. **Information transfer**: The process that generates the copy must obtain the information and conditional generative mechanisms that make the copy similar to its source from that same source.

Note how the condition of causal implication is clarified. The similarity condition is enhanced by the specification that similarity must apply to the conditional generative mechanisms. A related refinement is made to the information transfer condition. Together these conditions

preserve the spirit of Hull's (1988, p. 408) early definition of a replicator as 'an entity that passes on its structure'.

One of the problems in defining the replicator concept, and of specifying a general Darwinian evolutionary process, is understanding what exactly 'information' means. Our specification helps to fill this gap. The concept of information here does not necessarily carry interpretations or meanings in the same way as the information communicated by humans. It is information in a cruder sense of a code or signal, as stored and manipulated by computers and present in the DNA. For Claude Shannon and Warren Weaver (1949), in their seminal account of information theory, a message has 'information content' when its receipt causes some action. For us, the 'information' involved consists of signals with the potential to trigger generative mechanisms that guide the production of further replicators or the development of interactors.

Hence 'information' in our definition is not ideas; ideas necessarily involve meanings and (once again) these are absent from the general specification of replicators or replication. Furthermore, our first condition insists on the materiality of the replicator, hence ideas as such are not replicators. More appropriately, ideas may be regarded as *emergent expressions* of mental habits or dispositions, which may qualify as replicators, as shown below.

Our first condition is also sufficient to exclude nests, burrows and photocopies from the set of possible replicators. None of these entities is capable of receiving and emitting signals that lead to the development of the interactor. However, given that their survival is dependent on specific environmental conditions, then changes in those conditions can lead to changes in their state. Nests, burrows and photocopies can be destroyed by (say) water or fire. To exclude such destructive influences, the sentence in parenthesis in the first condition establishes that destructive or degenerative environmental influences do not count as signals. Having made this exclusion, nests, burrows and photocopies have no conditional mechanisms to guide the development of the interactor. They cannot enhance complexity through replication and hence they are not replicators.

5. Replication and Complexity

Whether or not evolution generally gives rise to increases in complexity has attracted much interest and debate for two centuries (Adami, 2002; Adami et al., 2000; Bennett, 1995; Gould, 1977; McShea, 1996; Saunders and Ho, 1976, 1981). The jury is still hung on the verdict. Some endorse the proposition that evolution enhances complexity in the biological domain, others suggest the evidence is inconclusive, and yet others reject the proposition.

The disagreement concerns not only the possibility or existence of a positive trend of (biological) complexity caused by evolution, but also what definitions and measures of complexity to use when such claims are assessed (Adami, 2002; Adami et al., 2000). The complexity concept is widely used, often without precise definition. Christoph Adami's (2002) recent useful review of the many definitions and measures of complexity concluded that most of these have drawbacks of either a conceptual or a practical nature when used to capture the complexity of evolving entities.

Consistent with information theory, Adami (2002) finds that the essence of *complexity for an evolving entity is the amount of information that it stores, in its genome, about the environment in which it evolves*. As a promising intuitive measure, he defines the physical complexity of a sequence of symbols:

The physical complexity of a sequence refers to the amount of information that is stored in that sequence about a particular environment. For a genome, this environment is the one in which it replicates and in which its host lives, a concept roughly equivalent to what we call a niche. Information … is always about something. Consequently, a sequence may embody information about one environment (niche) while being essentially random with respect to another. This makes the measure relative, or conditional on the environment, and it is precisely this feature that brings a number of important observations that are incompatible with a universal increase in complexity in line with a law of increasing physical complexity. (Adami, 2002, p. 1087)

This definition conceptualises complexity as information relative, or conditional on a particular environment within which the entity evolves. It further implies that information is obtained from input signals that are actually recorded in a sequence stored in a replicator (genome), which in turn requires a distinction between replicators and interactors. These aspects of physical complexity are all captured by our proposition that a conditional generative mechanism is a defining feature of replicators in the sense that the replicator is a material entity that embodies mechanisms that can be energized by input signals (containing information about a particular environment).

Following Adami we conceptualize complexity in terms of the information that a replicator stores about its environment. Consider first a specific environment E and the features required of a notional replicator to maximise interactor fitness in this environment, captured by a binary string of length L (the number of binary bits used to describe the replicator). The entropy value of these notional fitness maximisers is H_{max}. Population level complexity is the difference between H_{max} and the entropy of the actual population of replicators operating within this environment, also described in terms of binary strings of length L.

To determine the entropy of the actual population of replicators, each bit is addressed in turn. For the population as a whole, the actual frequency of the (binary) fitness-maximising value at locus i is p_i (where $0 \leq p_i \leq 1$). The entropy of a population of replicators X is denoted by $H(X)$. The measure of physical complexity C of a population of replicating entities is the information that the replicator sequences X contain about the environment E:

$$C = H_{max} - H(X) = L + \Sigma p_i \log p_i \tag{1}$$

Our argument for the introduction of conditional generative mechanisms in the definition of a replicator is based in part on the conjecture that the capacity to increase complexity depends critically on the existence and replication of a conditional generative mechanism. We can illustrate this by considering three types of error.[4] Namely there is reading error (concerning input signals), developmental error (concerning the development of interactor traits from the replicator) and copying error (from a replicator to its offspring). A weaker surmise is that copying errors among a population of replicators are generally more destructive to complexity than reading and developmental errors. A stronger proposition is that the capacity to increase complexity depends critically and especially on replication with low copying errors. We offer some arguments below in favour of the weaker surmise, and we refer to some simulation work that we have done that illustrates the stronger proposition.

Consider a process of repeated replication with input signals, the development of interactors and the copying of replicators, as in figure 1. This process involves a population of replicators

[4] Prior research on self-replicating automata (Molofsky, 1994; Wolfram, 1984, 2002) has typically studied emergent properties when applying one particular determining rule without such errors.

that develop traits that are clustered around a well-defined peak in a fixed fitness landscape, relating to a fixed environment. (Changing environments and variable or 'dancing' fitness landscapes are excluded for simplicity, not because they would necessarily undermine our argument.) In this context, both reading errors and developmental errors have a similar type of effect: they cause the population to disperse more around its original position in the fitness landscape. However, if copying errors are zero, and the probabilities of reading and developmental errors remain constant, then the trend of overall dispersion will not increase through time as repeated replication occurs. The effects of reading and developmental errors are *not* cumulative. Information content is preserved through the faithful copying of the replicator.
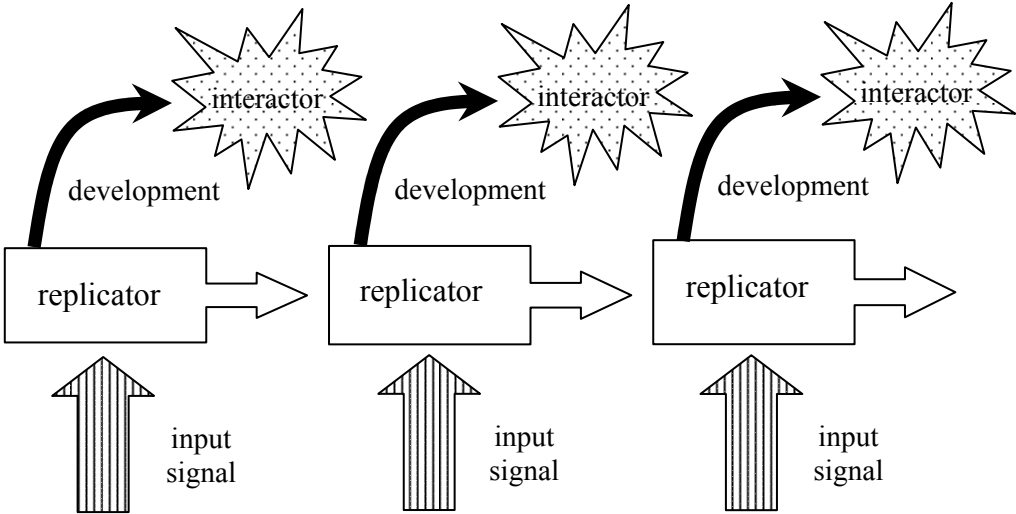

Figure 1: Replication with Input Signals and Interactors

Let us re-start the process but assume that the probability of a copying error is positive. The population of replicators is again clustered around a fitness peak in a fixed fitness landscape. If a replicator is close to the fitness peak, then any copying error is likely to move it away from that peak. There will be no reliable mechanism to tie it to its previous position. A process of drift will occur and the overall dispersion in the population will increase through time. Hence, by contrast, the effects of copying error are cumulative. The entropy $H(X)$ of the population will increase and overall complexity will decline as a result.[5]

This demonstration that copying error is especially destructive to complexity illustrates the special importance of the first and third of our four features of a replicator, concerning the existence and relatively faithful copying of a conditional generative mechanism. We fully

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[5] Note that the introduction of a 'Lamarckian' possibility has a similarly cumulative effect. Lamarckism upholds that the acquired characteristics of the interactor may effect the replicator, leading to the inheritance of these acquired characteristics in future generations. In biology, such Lamarckian inheritance is widely ruled out because of the Weismann barrier. Although social evolution is widely described as Lamarckian, we have challenged the meaning and relevance of the label (Hodgson and Knudsen, forthcoming). Nevertheless, if it did exist, Lamarckian inheritance would have the same cumulative destructive effect on the information stored in the replicator.

acknowledge that the above argument depends on rather restrictive assumptions. Nevertheless, it underlines the importance of copying fidelity and the cumulatively destructive effect on complexity of copying error.

We also admit that destructive forces, which reduce the chances of survival of some replicators, can undermine complexity in a population unless there is a sufficient number of fitter survivors. The occurrence of disasters and (near or complete) extinctions may reverse any trend towards greater complexity. Our argument is based on the *potential* to increase complexity, and not necessarily its empirical manifestation through time.

In order to substantiate the thought experiments relating to our proposition that the capacity to increase complexity depends critically on replication with low copying errors, we conducted a number of simulation experiments based on von Neumann's model of self-reproducing automata (Neumann, 1966). We briefly report the main results, but do not have the space here to elaborate on the details (Hodgson and Knudsen, unpublished).

We used the classical one dimensional (1D) linear automata, specified by binary values in a line of cells, because it is well understood and offers a simple test bed for studying replication. At each time step, there is replication of a rule (an information sequence comprising the replicator) and a subsequent process of development where the replicator is energized by input signals. The outcome of the development process is a new individual entity whose trait value depends on the way the replicator reads and codes input signals.

Compared with prior research, we extended the use of the 1D automaton in a number of ways. We considered the possibility of reading error, copying error, and the role of selection pressure. We found that even low levels of copying error can thwart the enhancement of complexity, and this is undermined still further as the level of copying error is increased. However, selection pressure can diminish the destructive effects of copying error. Overall, we showed that only if replicators comply adequately with all four of our definitional conditions, then complexity will increase towards some maximal level.

Our results are redolent of Adami's (2002, p. 1089) description of evolution as 'a process that increases the amount of information a population harbors about its niche'. According to Adami, natural selection is the only mechanism necessary to guarantee such an increase in complexity in a constant unchanging world. Natural selection can be viewed as an instantiation of Maxwell's demon 'a filter, a kind of semipermeable membrane that lets information flow into the genome, but prevents it from flowing out.' Our results support and further qualify Adami's claim. Selection, defined as differential replication caused by interaction, leads to an enduring increase in complexity only if replicators are conditional generative mechanisms in addition to satisfying the other three conditions of causality, similarity and information transfer. Our definition of replicators as conditional generative mechanisms ensures that the information that flows into the replicators, even if input signals are noisy, is not detrimental to the survival of the replicator.

6. Habits and Other Social Replicators

As noted above, attempts to extend Darwinian principles to social or cultural evolution are as old as Darwinism itself. However, detailed discussions of the units and mechanisms of social or cultural replication are rare. It is important to make progress on this front if Darwinian theories of social or cultural replication are to develop further.

Richard Dawkins (1976) famously coined the term 'meme' to describe the unit of cultural replication and to resonate with 'gene'.[6] A danger here is that in making the social unit of replication seem like that at the biological and genetic level, too much correspondence may be suggested between the mechanisms of genetic and social evolution. The literature on memetics suffers from conceptual confusion and casual descriptions of 'information' or 'ideas' as the basis of the meme. The enthusiasm for 'memes' and 'memetics' far outstrips the achieved degree of clarity and consensus concerning such core categories. A meme has been variously described as a unit of cultural imitation (Dawkins, 1976), a unit of information residing in a brain (Dawkins, 1982), units of culturally transmitted instructions (Dennett, 1995), an influential and replicable unit of information in the mind (Brodie, 1996), actively contagious ideas (Lynch, 1996), or behavioral instructions stored in brains and passed on by imitation (Blackmore, 1999).

In the memetics literature the nature of ideas and the causal mechanisms by which ideas lead to phenotypic behavior are rarely spelt out. It is generally assumed that one leads to the other. Related reservations were spelt out by John Maynard Smith (1995, p. 47) who expressed 'uneasiness with the notion of memes … because we do not know the rules whereby they are transmitted. A science of population genetics is possible because the laws of transmission – Mendel's laws – are known ... no comparable science of memetics is as yet possible.' If the term 'meme' is retained at all, as a general term for replicators in the social and cultural domain, then its manifestations and mechanisms have to be defined.

Instead we have proposed that habits are elemental social replicators in the social world (Hodgson, 2003; Hodgson and Knudsen, 2004a, 2004b). A habit is a disposition to engage in previously adopted or acquired behavior; triggered by an appropriate stimulus. Habits are formed through repetition of behavior or thought. They are influenced by prior activity and have durable, self-sustaining qualities. Habits are the basis of both reflective and nonreflective behavior. Crucially, we may have habits that lie unused for a long time. Habits are submerged repertoires of potential behavior that can be triggered by an appropriate stimulus or context.

Habits fall broadly into two types: observable habits of behavior and unobservable habits of thought. We briefly address the mechanisms of replication of each type in turn. Unlike the replication of DNA or computer viruses, habits of behavior do not directly make copies of themselves. Instead they replicate indirectly, by means of their behavioral expressions. They can impel behavior that is consciously or unconsciously followed by others, as a result of incentive or imitation. Eventually, the copied behavior becomes rooted in the habits of the follower, thus transmitting from individual to individual an imperfect copy of each habit by an indirect route.

The replication of observable habits of behavior satisfies all the aforementioned criteria for replication, including those formulated by other authors. Both the original habit and its copy embody a *conditional generative mechanism* as outlined in our first condition. The acquired habit is both energized conditionally on the receipt of environmental signals and plays a role in the development of the interactor, i.e. the individual with the habit. Furthermore, the habit of behavior in one person *causes* behavior that is copied, at least in the sense that the copy depends on the source, and leads to similar habits being acquired. The acquired habit of

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[6] Although the very similar term 'mneme' was coined much earlier. See Semon (1904) and Maeterlink (1927).

behavior is *similar* to the first with respect to the behavior it might promote under specific conditions. Some kind of tacit or other *information* is transferred in the process.[7]

Habit-forming behavior can be molded by incentives or constraints. In many cases, such as language or traffic conventions, because others are acting in a particular way, we can have powerful incentives to behave accordingly. In doing so, we too build up habits associated with these behaviors. The behaviors are reproduced and also the habits giving rise to them are replicated.

Habit replication also often relies on imitation. Imitation need not be fully conscious, and it will also involve some 'tacit learning' (Polanyi, 1967; Reber, 1993; Knudsen, 2002a). Imitation can occur because the propensity to imitate is instinctive, and this imitation instinct has itself evolved for efficacious reasons among social creatures (Boyd and Richerson, 1985; Simon, 1990; Tomasello, 2000). However, if imitation is more than mimicry, then the rules and understandings associated with it also have to be transmitted. There are grounds to consider a partially instinctive propensity to imitate as a force behind the replication of habits.

We now turn to the replication of habits of thought, which are unobservable by others and thus cannot emerge through behavioral imitation. We define a mental model broadly as a construction of the mind that relates to a particular context and enables inference in that context. We propose that habits of thought consist of mental models that enable conscious deliberation and manipulation of situations that are specific to a particular situation or context. We need to consider that thoughts can be limited by conventions or constraints that somehow bear upon mental activity. Actors form mental models, and they adapt and use their mental model as a guide as they proceed through a particular cultural context (Johnson-Laird, 1981). As habits, mental models are formed by repetition of particular associations and patterns of thought. When agents experience common external constraints or (physical or social regularities), under specific conditions they may develop similar mental models that will direct conscious deliberation towards a particular object in a particular class of situations. Different people that face similar environmental regularities will experience similar mental models that may be caused by different patterns of neuronal activity (Kurthen, 2001). Accordingly, similarities in habits of thought and mental models emerge when enduring similarities in external constraints or conventions exist.

Members of a species are similar regarding their physical construction, sensory organs, nervous system and brain function. These similarities in physical construction provide common inborn constraints that give rise to similarity in the mental experience of the external, physical world; similarities in physical construction become causes as well as evolutionary consequences of similarities in experience. Hence the possibility arises that two human beings will experience similar mental phenomena when confronted with the same external object (Edelman, 1989; Kubovy and Epstein, 2001).

Furthermore, agents can develop similar mental models to exploit common social conventions or constraints. Cultural circumstances or social institutions may vary from society to society, but members of each society have to adapt to them. An evolutionary explanation of the replication of habits of thought or other mental phenomena requires and explanation of the evolution of the social institutions that provide common conventions or

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[7] Similarity in neural connection design, even with similar genes and environment, is very unlikely (Edelman, 1989). Hence the underlying neuronal configurations supporting similar habits are likely to differ between agents. Understanding the replication of habits in terms of similar neural structures appears to be inappropriate.

constraints. Common conventions or constraints are a necessary but insufficient requirement for the replication of habits of thought to take place.

However, if two agents in a common context independently produce similar mental models, then no replication has taken place because no mental model is causally implicated in the emergence of the other. Replication of mental models would require that one agent could somehow access the mental model of another. This raises the question of how crucial information in the replication process is communicated, using a common language.

In psychology and neuroscience, the acquisition of a particular language has been described as a process that initially establishes triadic correspondences of mental models, objects and behaviors that become habits of thought or mental models common to the members of a society (Karmiloff and Karmiloff-Smith, 2001; Tomasello, 2000). The learning of a particular language results in the development of an elaborate correspondence between mental phenomena, behaviors and properties of the physical and social world.

With language the replication of unobservable thoughts can be explained. Through a shared language, a person can access the mental model of another. This transmission of mental models is improved by close interaction that enables error-correction. By gestures and questions, agents establish joint attention that increases the accuracy of transmission of mental models and establishes mutual understandings. Note here that language is an important link in this causal chain: without a common shared language replication is unlikely to occur.

Unobservable habits of thought satisfy all four of our conditions for a possible replicator. Habits of thought and mental models constitute *conditional generative mechanisms* that are essential to a replicator. They are energized conditionally on the receipt of external signals and play a role in the development of the individual. Given the existence of a shared common language as well as common extralinguistic points of reference, the habit of thought in one person *causes* a mental model that is transmitted and can lead to a similar habit of thought being acquired. The acquired habit of thought is *similar* to the first with respect to the mental model it might promote under specific conditions. Some kind of *information* regarding a perceived property of the world is transferred in the process.

Like any replicator, habits do not stand alone. Genes themselves depend on the biochemical substrate of an organism. Similarly, habits cannot exist apart from the human organisms in which they reside. They are formed and stored in the individual human nervous system. But habits differ from genes in their mechanism of replication, and habits do not have anything like the potential durability and copying fidelity of the gene. In social evolution there are additional mechanisms weed out or alter aberrant habits. Mechanisms of social conformity are particularly important (Henrich and Boyd, 2001).

Having established (observable and unobservable) habits as elemental replicators in the social world, we have the building blocks to understand other social replicators. Consider for example routines, in the technical sense of the term employed by evolutionary economists (Nelson and Winter, 1982). A consensus now exists in this literature that that routines relate to groups or organizations, whereas habits relate to individuals (Cohen *et al.*, 1996; Dosi *et al.*, 2000). Individuals have habits; groups have routines. Routines are the organizational analogue of habits.

A routine is here defined as a generative structure or capacity within an organization. *Routines are organizational dispositions to energize conditional patterns of behavior within an organized group of individuals, involving sequential responses to cues.* This qualifies as a conditional generative mechanism and upholds the routine as a social replicator.

Routines depend upon a structured group of habituated individuals, where many of these habits depend upon procedural memory. The behavioral cues by some members of a structured assembly of habituated individuals triggers specific habits in others. Hence various individual habits sustain each other in an interlocking structure of reciprocating individual behaviors. Partly because of procedural memory, organizations can have important additional properties and capacities that are not possessed by individuals, taken severally. The organization provides the social and physical environment that is necessary to enable specific activities, cue individual habits and deploy individual memories.

If one person leaves the organization and is replaced by another, then the new recruit may have to learn the habits that are required to maintain specific routines. Just as the human body has a life in addition to its constituent cells, the organization thus has a life in addition to its members. Generally, the organizational whole is greater than the sum of the properties its individual members, taken severally. The additional properties of the whole stem from the structured relations and causal interactions between the individuals involved.[8]

Other possible social replicators would include social customs and rituals. By understanding their preservation and replication in terms of shared similar habits of thought or behavior, their status as replicators might be similarly established. But we have not got the space to go into the details here.

7. Conclusions

In this paper we have reviewed the literature on replication and proposed an important refinement of the replicator concept. Inspired by the work of von Neumann on self-reproducing automata, we have established the concept of a materially-grounded *conditional generative mechanism* and argued that it plays a key role in the replication process. This mechanism is an essential part of the 'information' that is stored in the replicator and copied through replication. It also informs and guides the development of the interactor.

Also following von Neumann, we associate replication with the *potential* to increase complexity, without assuming that evolution necessarily embodies such a trend. Arguments are provided above to show that copying error is much more destructive to complexity than other forms of error, particularly in environmental interactions or individual development.

We have integrated the conditional generative mechanism in a four-clause definition of the replicator. By this definition, genes clearly qualify as replicators. However, other suggestions, including bird nests, burrows, and photocopiers do not qualify because they lack a conditional generative mechanism, and their replication does not have the potential to increase complexity. Hence our definition has important discriminating powers in the biological domain.

Turning to social and cultural evolution, our definition also has important implications. As widely acknowledged, the literature on memes suffers from vagueness and ambiguity concerning its definition and nature. If memes are simply ideas, then our materiality condition rules them out as replicators. Ideas do not embody construction mechanisms that can be energized by input signals. Instead, we regard ideas as emergent expressions of mental habits

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[8] This is a central proposition in the emergentist tradition of philosophy and social theory (Blitz, 1992; Kontopoulos, 1993; Hodgson, 2004; Wiessman, 2000).

or dispositions that in turn qualify as replicators. This overcomes the dualism and separation of the ideal and material worlds, by grounding ideas upon habits, as in pragmatist philosophy (Joas, 1993; Diggins, 1994; Putnam, 1995; Hodgson, 2004).

Using more precise concepts than the meme, we have shown that both (individual) habits and (organizational) routines qualify as replicators by our four criteria. By pointing to their mechanisms of replication we have begun to address the detailed 'genetics' of social replicators that has been missing from discussions of memes.[9]

Finally, we comment on another important implication of our approach. We have underlined the importance of copying fidelity in information transmission from one replicator to another. Upon this the preservation and potential enhancement of complexity depends. A case can be made in the biological world that the Weismann barrier – limiting any interference by the interactor with the information in the replicator – has evolved to deal with this problem (Maynard Smith and Szathmáry, 1995). A big question for us is whether there is an equivalent Weismann barrier in the social domain. Genetic or culturally transmitted mechanisms of conformism may be important in sustaining such a barrier. This is among several research questions prompted by our refined definition of the replicator.

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[9] Another paper (Hodgson and Knudsen, unpublished) looks at these issues concerning social replication in more detail.

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