On Meme-Gene Coevolution

Larry Bull, Owen Holland[#,*] and Susan Blackmore[+]

Faculty of Computer Studies and Mathematics

[#]Faculty of Engineering

[+]Faculty of Applied Sciences

University of the West of England

Bristol BS16 1QY, U.K.

larry@ics.uwe.ac.uk

[*]Microsystems Laboratory

Department of Electrical Engineering

California Institute of Technology (136-93)

Pasadena, CA 91125, USA

Abstract

In this paper we examine the effects of the emergence of a new replicator, memes, on the evolution of a pre-existing replicator, genes. Using a version of the *NKCS* model we examine the effects of increasing the rate of meme evolution in relation to the rate of gene evolution, for various degrees of interdependence between the two replicators. That is, the effects of memes' (suggested) more rapid rate of evolution in comparison to that of genes is investigated using a tunable model of coevolution. It is found that, for almost any degree of interdependence between the two replicators, as the rate of meme evolution increases, a phase transition-like dynamic occurs under which memes have a significantly detrimental effect on the evolution of genes, quickly resulting in the cessation of effective gene evolution. Conversely, the memes experience a sharp increase in benefit from increasing their rate of evolution. We then examine the effects of enabling genes to reduce the percentage of gene-detrimental evolutionary steps taken by memes. Here a critical region emerges as the comparative rate of meme evolution increases, such that if genes cannot effectively select memes a high percentage of the time, they suffer from meme evolution as if they had almost no selective capability.

1. Introduction

Dawkins [1976] coined the term "meme" to describe information copied from person to person by imitation. Memes vary, are selected, and are inherited (i.e. copied by imitation); they therefore fit the evolutionary algorithm and can be considered as a replicator [Dennett 1995]. Most theories of human evolution, including sociobiological theories, assume that natural selection has operated on only one replicator, the genes. However, humans are adept at imitation almost from birth [Meltzoff 1996] and therefore live in an environment pervaded by memes. We should therefore consider human evolution to be a product of two replicators - memes and genes.

Blackmore [1999a] has argued that competition between these replicators is responsible for both the evolution of language and the enormous increase in brain size in humans. In a process termed "memetic driving" the most successful memes force the genes to create machinery that is especially good at copying those memes. The process works as follows: competition between memes results in some memes becoming more successful and being widely imitated, while others fail. These successful memes will include those that have been copied because they are useful (such as new skills and technologies) and some that are useless or even harmful. People who are best at copying the most successful memes acquire advantages in terms of status and survival, as well as being preferentially selected as mates. Therefore genes that facilitate the imitation of those particular memes are favoured and increase in the gene pool. One simple application of this general argument is to brain size. If we assume that having a larger brain makes imitation easier, memetic driving will favour genes for larger brains. On this theory the human brain expanded rapidly in size for memetic, not purely genetic, reasons.

The same process can account for such uniquely human abilities as the enjoyment of music. As soon as early humans began imitating sounds, memes based on tunes and rythms could spread. Some of these sounds would evoke emotional reactions, act as signals of status, or have other effects that improved their chances of being copied. People who could copy the most successful sounds would be at an advantage and so the genes that made them good at copying those sounds would spread. Musical ability would generally increase in the population, allowing more memes to spread and increasing the speed of memetic evolution. In this way our musical abilities have been shaped by a long history of coevolution between the memes that were copied and the machinery that copied them.

As the process continues, the memes evolve faster and faster, outstripping the genes' ability to change. When this happens we should expect traits that are adaptive to the memes but maladaptive to the genes to spread. In this situation the genes would benefit if they could select out maladaptive memes. We arrive at a conception of the human brain as evolved not only to spread memes, but to select which memes to replicate. In this sense it is comparable with the immune system, being evolved to select between desirable and undesirable replicators [Blackmore 1999b].

There have been several previous coevolutionary theories (e.g. [Durham 1991], [Lumsden & Wilson 1981]). Cavalli-Sforza and Feldman [1981] developed a mathematical model of gene-culture evolution but, in spite of showing how maladaptive traits could survive (that is, maladaptive to the genes), they ultimately assumed, as Lumsden and Wilson put it, that "the genes hold culture on a leash". Boyd and Richerson [1985] developed a mathematical model to explore the conditions under which social learning (including imitation) would be favoured, and have shown that dual-replicator models can account for group selection processes [1990] but they have not considered memetic driving. Best [in press] has used a simple simulation environment in which evolving agents learn by imitation, and shown that imitation is (like individual learning) able to guide genetic evolution towards an adaptive goal. However, when genes and culture pursued diametrically opposed goals, he found that culture was no match for genetic evolution with individual learning.

None of these models considers two issues that are important to Blackmore's coevolutionary model; the relative speed of the two replicators, and whether one replicator can exert an explicit selection pressure on the other. These issues are addressed here using versions of the *NKCS* model [Kauffman 1993].

We examine the effects of different rates of evolution between the two replicators. With low dependence between the memes and genes, it is found that varying the rate of meme evolution with respect to the rate of gene evolution has no significant effect on either replicator. That is, both are able to evolve successfully within their respective attribute spaces. However, with a slightly larger degree of interdependence, increasing the rate of meme evolution generates a phase transition-like phenomenon under which meme evolution (rapidly) benefits significantly and gene evolution degrades to a random walk; for any significant amount of inter-replicator dependence, large benefits/losses can be experienced by altering the difference in evolution rates between them.

We then extend the model to examine the effects of genes actively selecting memes such that meme evolution is restricted, by varying degrees, in the extent to which it may have detrimental effects on the genes. It is found that, for significant meme-gene interdependence, the benefits to genes of increasing their selective pressure over memes increases. It is further shown that, as the relative rate of meme evolution increases, significant benefits to genes are only obtained under high selective pressure.

The paper is arranged as follows: the next section describes the *NKCS* model. Section 3 presents results from its use to examine the different relative rates of meme and gene evolution. Section 4 introduces and examines the version of the model in which genes exert some explicit control over meme evolution. Finally, our findings are discussed.

2. The *NKCS* Model

Kauffman [1993] introduced the *NKCS* model to allow the systematic study of various aspects of natural evolution between interacting species. However, the model is abstract, and so can be applied to the study of any collection of interacting replicators, e.g. memes and genes. In the basic model a given population is represented by a single set of *N* replicators, each with two possible states, i.e. a population is assumed to be converged to all individuals having the same value for each constituent replicator. The fitness of an individual depends upon the contribution made by each replicator, each of which depends upon *K* other replicators (random) within the individual (epistasis).

Increasing *K*, with respect to *N*, increases the epistatic linkage, increasing the ruggedness of the fitness landscape of a population by increasing the number of fitness peaks, increasing the steepness of the sides of the peaks and

decreasing their typical heights. That is, as the dependence between an individual's aspects increases, the attribute

Figure 1: Showing an example *NKCS* model. (a) shows each replicator depends on one other locally and on one in the other population. Therefore there are eight possible configurations, each of which is assigned a random fitness as shown in (b). Each replicator of each population has such a table created for it. Total fitness of a given (converged) population of replicators is the averaged sum of these values (c).

space becomes increasingly complex. Each individual replicator is also said to depend upon C replicators (random) in the other populations with which it interacts. Hence, the adaptive moves made by one population of replicators may alter the fitness landscapes of its (S) partners; altering C, with respect to N, changes the extent to which adaptive moves by each individual deforms the landscapes of its partnering populations. As C increases, mean evolutionary fitness drops and the time taken to reach an equilibrium point increases, where the fitness level of the equilibrium decreases.

The model assumes all inter-population (C) and intra-population (K) interactions are so complex that it is only appropriate to assign random values (Gaussian distribution) to their effects on fitness. Therefore for each of the possible $K+C$ interactions, for each given replicator, a table of $2^{(K+C+1)}$ fitness contributions is created, with all entries in the range 0.0 to 1.0, such that there is one fitness for each possible combination of replicators. The fitness contribution of each replicator within a population is found from its individual table. These contributions are then summed and normalised by N to give the actual fitness of the individual (Figure 1).

Kauffman considered converged populations of species and random hill-climbing to evolve each in turn, i.e. each species/population uses the current context of the others to determine whether a random alteration to its configuration represents progress. That is, from a given configuration, a population randomly alters one replicator's state and calculates the resulting fitness. If the new fitness is greater than the population's current fitness, in the current environment, the population adopts the new configuration (see [Kauffman 1993] for full details of the
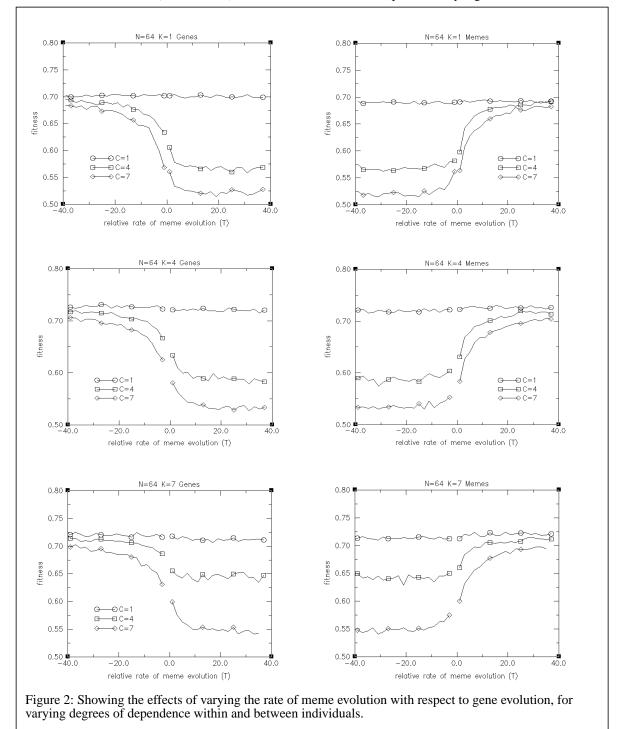
model).

3. An *NKCS* Model of Meme-Gene Coevolution

In this paper we consider the coevolutionary progress of two populations of replicators - memes and genes (S=1). Each population consists of individuals of sixty-four replicators (N=64), with varying degrees of inter- and intra-population dependence. We also introduce a new parameter, T, which determines how many generations of meme evolution occur per generation of gene evolution. In this way we are able to examine the effects of the emergence of memes upon genes (low T) and the effects of increasing their respective rate (> T). Thus in the standard *NKCS* model it is always the case that T=1.

Results were obtained by generating a random landscape for the given set of parameters (K, C, T) and recording the resultant fitness of each population after five thousand *gene* generations. For each generated landscape, ten runs were carried-out, such that the results are the averages of a hundred runs per set of parameters. Note that in the figure negative values for T imply reciprocal meme rates, e.g. T=-5 means five generations of gene evolution per generation of meme evolution.

Figure 2 presents the initial results and shows that, for all K, once there is significant dependence between the memes and genes (roughly C>2), memes can benefit significantly from increasing their rate of evolution with respect to that of genes. Conversely, increasing the rate of meme evolution can have significantly detrimental effects on gene evolution. It can be seen that at around T= -30, i.e. thirty generations of gene evolution per meme generation, the memes begin to have a detrimental effect on the genes. This effect increasingly degrades gene evolution until around T=10, by which time gene evolution effectively stops, with higher C genes performing about

as well as a random walk (fitness = 0.5). However, memes do not experience any significant benefits from an

Figure 2: Showing the effects of varying the rate of meme evolution with respect to gene evolution, for varying degrees of dependence within and between individuals.

increase in their rate of evolution until around T=-10. The benefits then increase until around T=30, after which no further benefits are seen. That is, a sharp transition occurs across a range of forty rates which results in the effective cessation of gene evolution and greatly improved meme evolution. Note that the size of the benefit/detriment in fitness increases with increasing C.

As stated above, as C increases, the amount of epistatic coupling between the fitness landscapes of the populations

of replicators increases. The effect of this coupling is to cause movement in the partners' landscapes, which increases the difficulty experienced by evolution in locating high-fitness optima. By increasing their rate of evolution, memes are able to reduce the effects on themselves of this fitness landscape movement since the increase in rate reduces the number of possible evolutionary advances/changes which can be made by the genes before the memes have to "re-adapt" to them. That is, the greater *T*, the more (temporarily) stationary the memes' fitness landscape appears to them. Similarly, for the genes, as the memes are able to make more and more evolutionary advances before the genes have the chance to re-adapt, the more the genes experience the deleterious effects of the coupling between the landscapes.
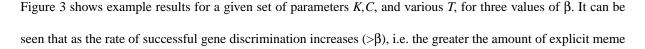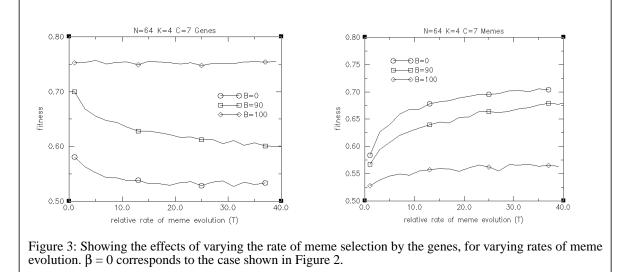
4. Brains as Immune Systems

It has been noted that, since memes have the potential to be maladaptive to genetic evolution, they can be viewed as analogous to viruses (e.g. [Dawkins 1993][Brodie 1996] - see [Bura 1994] for simulations in which lethal memes emerge and persist). In the above model the genes had no explicit control over meme evolution; memes were seen as completely autonomous replicators. However it has recently been suggested that, to some degree, genes may actively "select" memes, stopping them from evolving in gene-detrimental directions [Blackmore 1999b]. This can be seen as akin to the situation in which a set of genes' immune system must attempt to respond to the presence of a fast evolving virus such as HIV.

We have extended the model described in the previous section to allow genes to explicitly exert a selective pressure over the memes' evolutionary process. Now, with a probability represented by a percentage rate β, each time the meme population finds a new advantageous configuration via mutation, it does not move to that configuration if it is detrimental to the current configuration of the gene population. Hence, in the previous model the memes were always able to move to a meme-beneficial configuration ($\beta = 0$). Using this model we are able to examine the effects of altering the number of gene-harmful meme mutations which propagate, i.e. the effects of varying the genes' ability to successfully discriminate between memes.

Results were obtained by using the same random landscapes from the previous section, with given sets of parameters (*K, C, T*), recording the resultant fitness of each population after five thousand gene generations, for

varying β. Again, ten runs on each of the landscapes were carried out such that the results are the averages of a hundred runs per set of parameters. Note that we only consider positive values of *T* here, but the same trends are found for lower rates of meme evolution (not shown).

Figure 3 shows example results for a given set of parameters *K,C*, and various *T*, for three values of β. It can be seen that as the rate of successful gene discrimination increases (>β), i.e. the greater the amount of explicit meme

Figure 3: Showing the effects of varying the rate of meme selection by the genes, for varying rates of meme evolution. β = 0 corresponds to the case shown in Figure 2.
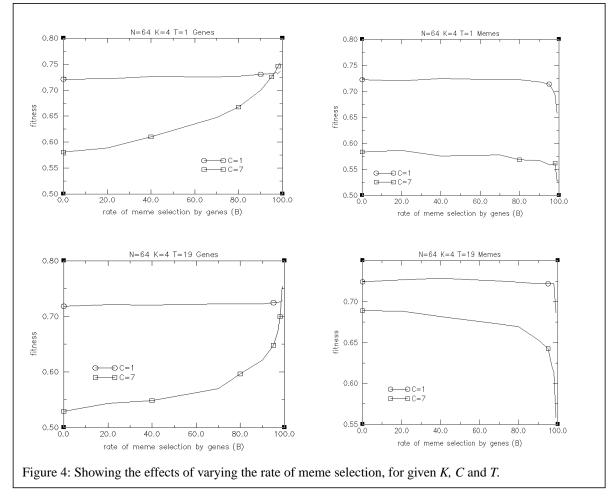
selection by the genes, the greater the benefits to the genes. At the highest rates the effects found in the previous version of the model are lost and the genes evolve to fitness levels similar to those found for very low *C*. Conversely, the memes do less well as β increases, as expected. This has been found to be the case whenever there is significant dependence between the replicators. Figure 4 confirms this result, showing the resultant fitnesses after five thousand gene generations for the range of possible β, and given values of *K, C* and two values of *T*.

It is clear that, when there is high dependence between memes and genes, more meme mutations will be produced which will have an effect on the genes [Kauffman 1993, p.250]. Thus a greater number of gene-detrimental mutations can arise, and hence the genes will increasingly benefit from restricting meme evolution. As the relative rate of meme evolution increases, the number of meme mutations increases, again increasing the probability of gene-detrimental mutations occurring; increasing *T* is akin to increasing *C* here. However, meme evolution effectively stops under high β since fewer and fewer evolutionary steps become possible.

From Figure 4 it can also be seen that, as the relative rate of meme evolution increases, the benefits to genes of

increasing β are lower for medium β. Figure 5 shows this result explicitly by comparing the results from runs with

three values of *T* over a range of β. It can be seen that, whilst in all cases being able to successfully discriminate

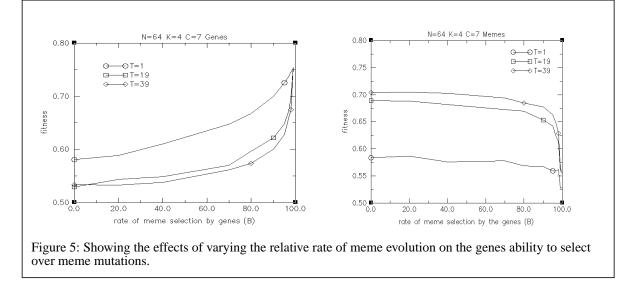between memes a high percentage of the time is very beneficial to genes, this benefit increases more sharply with

Figure 4: Showing the effects of varying the rate of meme selection, for given *K, C* and *T*.

increasing β under high T; for high *T* the benefits of high β decrease more rapidly than for low *T*. Note also that the

rate of change in this behaviour does not alter linearly since there is a far greater change between *T*=1 and *T*=19,

than between *T*=19 and *T*=39. Thus as the relative rate of meme evolution increases, genes being able to exert an

explicit selection pressure on meme evolution over seventy percent of the time gives the genes a sustained benefit.

5. Conclusions

For some time it has been recognised that, within complex social environments, particularly those involving humans, a form of cultural replicator exists. Such a replicator must therefore coevolve with genes, the existing replicators. This coevolution will be influenced by the degree of coupling between the replicators and also by their relative rates of replication.

In this paper we have presented results from a version of the *NKCS* model of meme-gene coevolution. It has been found that, for most degrees of dependence between the two replicators, regardless of the dependence within the populations, a phase transition-like dynamic occurs as the relative rate of replication is varied. Within our model,

Figure 5: Showing the effects of varying the relative rate of meme evolution on the genes ability to select over meme mutations.

until the rate of meme evolution is a thirtieth that of genes, genes remain unaffected by their presence. From then on, until the memes evolve ten times faster than the genes, the genes experience increasingly negative effects from the presence of the memes, *and thereafter are unable to evolve effectively.* Conversely, the memes do not experience any benefit from increasing their rate of evolution until it is around a tenth that of the genes. From then on, until they evolve thirty times faster than the genes, they experience increasing benefit from increasing their rate of evolution. Thereafter they suffer no beneficial or detrimental effects from any increase. The symmetry here is due to the symmetry in the rest of the model. Varying *N*, e.g. *N*=128, was found to have little effect (not shown).

The effects of genes being able to exert a selective pressure, of varying degrees, over memes have also been examined within the model. It has been found that, under conditions of significant meme-gene interdependence, a critical amount of pressure exists with increasing meme replication rate in which genes experience large benefits,

11

and meme evolution falters. If it is assumed that for larger amounts of selection pressure to be exerted by genes, bigger brains are required [Blackmore 1999b], then this result adds to a memetic explanation of the origin of humans' unexpectedly large brains [Blackmore 1999a].

The concept of phenotypic plasticity, i.e. gene-based lifetime learning of phenotypic traits, whether individual or through imitation - the Baldwin effect [Baldwin 1896] - has not been examined here. That is, in this model, genes are not able to respond structurally during their life-time/evaluation to the existence of memes capable of faster evolution than themselves ($T>1$). We suggest that such phenotypic plasticity has the potential to be of benefit to genes, but note that results here show genes suffer most significantly from the presence of memes whilst their rate of evolution is still greater than that of memes, i.e. *before* lifetime learning could have any significant effect. Once the rate of meme evolution is greater than that of genes, phenotypic plasticity could perhaps allow genes to track meme evolution, such that all effects of the difference in rates would be lost (assuming equivalent effective rates of learning and meme evolution). However, it is known that too much learning can disrupt the Baldwin effect due to the reduction in selection pressure [Maynard Smith 1993]; it is therefore possible that for faster rates of meme evolution, equivalent life time phenotypic plasticity may in itself have detrimental effects on gene evolution. Genes which further evolve imitation to keep track of rapidly evolving memes have the potential to do themselves more harm in the long run than if they used individual learning; genes producing learning by imitation not only creates the necessary conditions for memes to emerge, which may have detrimental effects on gene evolution, but its continued use has the potential to makes things worse ! We are currently extending the models presented here to include phenotypic plasticity.

References

Baldwin, J. M. (1896) A New Factor in Evolution. *American Naturalist* 30: 441-451.

Best, M. (in press) How Culture Can Guide Evolution. *Adaptive Behaviour.*

Blackmore, S. (1999a) *The Meme Machine*. Oxford University Press.

Blackmore, S. (1999b) Can the Memes Get Off the Leash ? Presented at *Do Memes Account for Culture ?* King's College Research Centre Conference, King's College, Cambridge 4th-5th July 1999.

Boyd, R. & Richerson, P.J. (1985) *Culture and the Evolutionary Process*. University of Chicago Press.

Boyd, R. & Richerson, P.J. (1990) Group Selection Among Alternative Evolutionary Stable Strategies. *Journal of Theoretical Biology*, 145:331-342.

Brodie, R. (1996) *Virus of the Mind: The New Science of the Meme*. Integral Press.

Bura, S. (1994) MINIMEME: Of Life and Death in the Nosphere. In D. Cliff, P. Husbands, J-A. Meyer & S. Wilson (eds.) *From Animls to Animats 3.* MIT Press, pp479-486.

Cavalli-Sforza, L.L. & Feldman, M.W. (1981) *Cultural Transmission and Evolution: A Quantative Approach*. Princeton University Approach.

Dawkins, R. (1976) *The Selfish Gene*. Oxford University Press.

Dawkins, R. (1993) Mind Viruses. In B. Dahlbohm (ed.) *Dennet and his Critics: Demystifying Mind*, Blackwell, pp13-27.

Dennett, D. (1995) *Darwin's Dangerous Idea*. Penguin.

Durham, W.H. (1991) *Coevolution: Genes, Culture and Human Diversity*. Stanford University Press.

Kauffman, S. A. (1993) *The Origins of Order*. Oxford University Press.

Lumsden, C.J & Wilson, E.O. (1981) *Genes, Mind and Culture*. Harvard University Press.

Maynard Smith, J. (1993), *The Theory of Evolution*, Cambridge University Press.

Meltzoff, A.N. (1996) The Human Infant as Imitative Generalist: A 20-year progress report on infant imtation with implications for comparative psychology. In C.M. Heyes & B.G. Galef (eds) *Social Learning in Animals: The Roots of Culture*. Academic Press, pp347-370.