

Evolution, Learning, and Culture: Computational Metaphors for Adaptive Algorithms

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Abstract.

Potential interactions between connectionist learning systems and algorithms modeled after evolutionary adaptation are becoming of increasing interest. In a recent short and elegant paper Hinton and Nowlan extend a version of Holland's genetic algorithm (GA) to consider ways in which the evolution of species and the learning of individuals might interact [17]. Their model is valuable both because it provides insight into potential interactions between the *natural* processes of evolution and learning and as a potential bridge between the *artificial* questions of efficient and effective machine learning using the GA and connectionist networks. This paper begins by describing the GA and Hinton and Nowlan's simulation. We then analyze their model, use this analysis to explain its nontrivial dynamical behaviors, and consider the sensitivity of the simulation to several key parameters.

Our next step is to interpose a third adaptive system — culture — between the learning of individuals and the evolution of populations. Culture accumulates the “wisdom” of individuals' learning beyond the lifetime of any one individual but adapts more responsively than the pace of evolution allows. We describe a series of experiments in which the most minimal notion of culture has been added to the Hinton and Nowlan model, and we use this experience to comment on the functional value of culture, and similarities between and interactions among these three classes of adaptive systems.

1. Introduction

It is interesting to note that two of the most promising approaches to the design of algorithms for machine learning can trace their origin to the study of

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naturally occurring systems. Connectionist algorithms, since their cybernetic beginnings, have often had a real concern with how neurons in biological nervous systems allow organisms to adapt to their environment. Similarly, models of simulated evolution generally and the genetic algorithm (GA) in particular are attempts to mimic the way species become adapted to their respective environmental niches. Both classes of algorithms have since been developed so that their correspondence to the original natural phenomena is often only metaphoric, but as we attempt to compare and possibly combine these techniques it makes sense to start with a basic understanding of the interaction between the natural processes of evolution and learning.

In order to adopt a perspective that allows one to capture the GA and connectionism within a single view it is necessary to back up, far off-stage, and each of the characters consequently becomes quite small. That is, in an attempt to capture interactions among enormously complicated phenomena — evolution, learning — the models of each of these constituents must necessarily become extremely simplified. There are certainly other comprehensive models of learning in cognitive systems than the connectionist account (e.g., SOAR [23]). Even within connectionist models of learning there is great diversity, and most connectionist systems are extremely complicated in their own right. The same is true of various models of evolution, and the complexity of various versions of the GA. But our concern here is not with the the validity of connectionism as a model of learning or the GA as a model of evolution. A central premise of this work is that *connectionism and the GA represent two very adequate models for learning and evolution, respectively*. We propose to take these two models as canonical in order that we might begin to explore *interactions* between these two forms of adaptive search.

Merging these models, and especially making sense of the hybrid's behavior, is difficult. In a very short and elegant paper in these pages, Hinton and Nowlan describe a simulation suggesting “how learning can guide evolution” [17]. In order to achieve this goal, their model incorporates an impoverished notion of learning into the GA. The result of this simplification is a simulation in which the *interactions* between the GA and individuals' learning are appropriately accentuated. The work reported here builds squarely on this beginning.

The question of “how learning can guide evolution” is critically important because it is concerned with how the results of one adaptive search (individuals' learning) can be capitalized on by another (the evolution of a population). When one considers the natural correlates of these adaptive algorithms, it is not at all clear what the medium for this exchange of information might be; the *time scales* of individual learning and a species' evolution is wildly different. Yet, at least in the case of the human species, it does seem as if the learning accomplished over a lifetime has become coupled with the process of evolution. Whether this perception is accurate or not, we will argue that it is appropriate to consider *culture* as a third, intermediate adaptive system used by societies, between the learning used by individuals

and the evolution used by populations. Preliminary experiments suggest the addition of “cultural artifacts” helps mediate between the regularities found by evolution (over many generations) and the learning accomplished by an individual (within a single lifetime).

The rest of this report is divided into six sections.¹ We begin by presenting the basic features of the GA and Hinton and Nowlan’s model, then make several comments about important assumptions built into these prerequisites. Section 3 presents our analysis of the model, and this provides the basis for our characterization of the model’s behavior in section 4. Section 5 considers the sensitivity of the simulation to some of its key parameters. In section 6 we introduce a very crude notion of *culture*, as an additional “channel” through which knowledge acquired by one individual might be passed to other members of the species. The paper concludes with a discussion of some of the issues raised by these experiments and potential directions for further research.

2. Preliminaries

This research builds squarely on two previous bodies of work, the genetic algorithm (GA) and Hinton and Nowlan’s model. This section will begin by presenting the central features of the GA, then present Hinton and Nowlan’s model and the basic results of their simulations. Our preliminaries will conclude with several comments about important features of this model.

2.1 The genetic algorithm

The GA has been investigated by John Holland [18] and students of his for almost twenty years now, with a marked increase in interest within the last few years [14,15,26]. The interested reader is advised to begin a more thorough introduction to these algorithms with the excellent new text by Goldberg [12].

Attempts to simulate evolutionary search date back as far as the first attempts to simulate neural networks [11]. The basic construction is to consider a population of individuals that each represent a potential solution to a problem. Then, if the relative success of each individual on this problem is considered this individual’s fitness, this number can be used to select the most fit individuals to produce similar but not identical offspring for the next generation. By iterating this process, the population efficiently samples the space of potential individuals and eventually converges on the most fit.

More specifically, consider a population of N individuals x_i , each represented by a chromosomal string of L allele values. An initial population is constructed at random; call this generation g_0 . Each individual is evaluated by some arbitrary environment function that returns the fitnesses $\mu(x_i) \in \mathfrak{R}$ of each individual in g_0 . The evolutionary algorithm then performs two operations. First, its selection algorithm uses the N fitness measures to determine

¹Portions of this report appeared in an early report [3].

how many offspring each member of g_0 contributes to g_1 . Second, some set of genetic operators are applied to these offspring to make them different from their parents. The resulting population is now g_1 , these individuals are again evaluated, and the cycle repeats itself. The iteration is terminated by some measure suggesting that the population has converged.

A critical distinction among simulated evolutionary algorithms is with respect to their genetic operators. Often the only genetic operator used is mutation: some number of alleles in the parent are arbitrarily changed in the child. This amounts to a *random* search around the most successful individuals of the previous generation, and is therefore not very powerful. The central feature of Holland's GA is its use of an additional crossover operator modeled on the biologically veracious operation of genetic recombination: during sexual reproduction segments from each of the parents' chromosomes are combined to form the offspring's. The GA's crossover operation picks two points $1 \leq m, n \leq L$ at random and builds the offspring's bit string by taking all bits between m and n from one parent and the remaining bits from the other parent. For example, if $L = 10$, $m = 2$, $n = 6$:

Parent(1):	1111111111	Offspring(1):	1100001111
Parent(2):	0000000000	Offspring(2):	0011110000

The appeal of the GA is due both to empirical studies that show the crossover operator works extremely well on real, hard problems, and also to the "schemata" analysis Holland has provided to show why this is the case.

One key property of the GA is that it works on a population of (binary) bit strings with absolutely no knowledge of the semantics associated with these bits. Its only contact with the environment is the global fitness measure associated with the entire string. This is considered an advantage of the algorithm because it ensures that the GA's success is not related to the semantics of any particular problem. This is not to say that the GA works on all problems equally well, only that these differences can be attributed to the underlying search spaces rather than the semantics of the problem domain [4].

2.2 The Hinton and Nowlan model

Attempts to integrate the learned behaviors of individuals into their genetic consequence on a species have dogged the theory of evolution since Darwin. Every high school biology student knows that Lamarck's theory — that modifications of the phenotype over the course of a lifetime can become *directly* incorporated into the genotype — depends impossibly on mechanisms of "reverse transcription" for which there is almost no evidence. Less well known is a hypothesis first attributed to Baldwin [2], but later expressed by others [30], regarding an *indirect* effect the experiences of an exploratory organism can

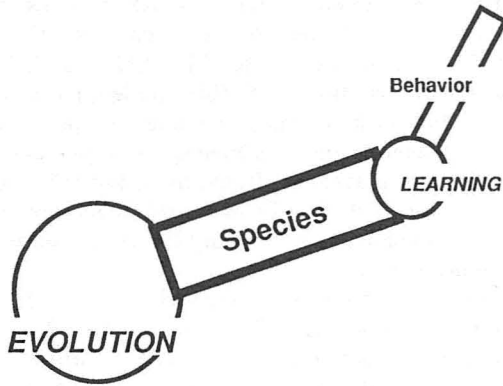


Figure 1: "Baldwin's arm."

have on its species' genetic search. John Maynard Smith summarizes the hypothesis:

Even if we accept [that individuals' learning cannot alter information in the gene], it is still possible for individual learning to facilitate evolution. If individuals vary genetically in their capacity to learn, or to adapt developmentally, then those most able to adapt will leave most descendants, and the genes responsible will increase in fitness. In a fixed environment, when the best thing to learn remains constant, this can lead to the genetic determination of a character that, in earlier generations, had to be acquired afresh each generation [28].

One useful way to graphically portray the effect is shown in figure 1. Evolution can be viewed as a process for moving a "phenotype limb" (corresponding to a species or gene pool) in order to better adapt it to an environment. At least some organisms are capable of generating a range of behaviors, and these can be viewed as a second, "behavior limb" being moved by the "joint" of learning. To simplify, imagine that being fit means being able to reach a point in space. The simple but profound idea of the Baldwin effect is that since it is sufficient for the two-limbed, coupled arm of phenotype+learned behavior to be able to reach that point, an improved capacity for learning increases genetic fitness.

Hinton and Nowlan have developed a computational model that "demonstrates the magnitude" of the Baldwin effect [17]. As connectionists they assume a neural network as the basis for learning and use evolution to search for a neural wiring that allows an individual to learn effectively.

More specifically, they imagine trying to construct a neural network of L potential connections. An individual is considered successful if and only if they have all of these connections correctly specified. Thus there is exactly one right answer out of the 2^L possible combinations. By convention we will imagine that this right answer is the string $111\dots 1$. It is important to recognize what a difficult “environment” this problem poses; it is sometimes called a “needle-in-a-haystack” or “impulse function” problem. Not only is there only one correct answer out of a huge space of possible solutions, but there is also *no information* available from any of the other solutions about where the correct answer might be. In such problems there exists no better way to search than by exhaustively sampling the entire space. In particular, the GA would fare no better.

Individuals are represented by a ternary string $x_i \in (0\ 1\ ?)^L$. A 1 specifies that a connection is present, a 0 that it is absent, and a ? a “learnable” connection: one that can be set either open or closed during the “lifetime” of the individual. An individual’s life experience amounts to repeated attempts to set these learnable connections correctly. In the interest of parsimony, their model of learning is extremely austere. Each individual is given a fixed number G of completely random guesses for the settings of their ? values. They are also given the ability to recognize the fact that they have found the correct setting.

For evolution, Hinton and Nowlan use a fairly standard version of the GA. The fitness μ associated with each individual x_i is a function of the number of guesses g that individual made before finding the correct settings:

$$\mu(x_i(g)) = 1 + \frac{(L-1)(G-g)}{G} \quad (2.1)$$

This function provides the key advantage that allows the GA to work. Hinton and Nowlan describe it in terms of the “shoulders” that have been added to the impulse fitness function of figure 1:

With learning, there is a zone of increased fitness around the spike ...[corresponding] to genotypes which allow the correct combination of potential connections to be learned.

There is still only one correct answer, but now there is also a basin of attraction around this needle that the GA can use to moves its search toward that needle. Another way to say the same thing is that for realistically large L the chance of finding the needle, 2^{-L} , is vanishingly small; without learning this is what an evolutionary search must do. But with learning, evolution is required only to find a solution that is within the basin of attraction of the needle, and this is much more likely.

For their simulations, Hinton and Nowlan picked the parameters carefully. With $L = 20$, if (on average) half of the alleles are learnable ?s, there are $2^{10} = 1024$ combinations to try and an individual given 1000 guesses stands a very good chance of finding the correct one. It is no coincidence, then, that Hinton and Nowlan began with a population consisting of 50% ?s, and

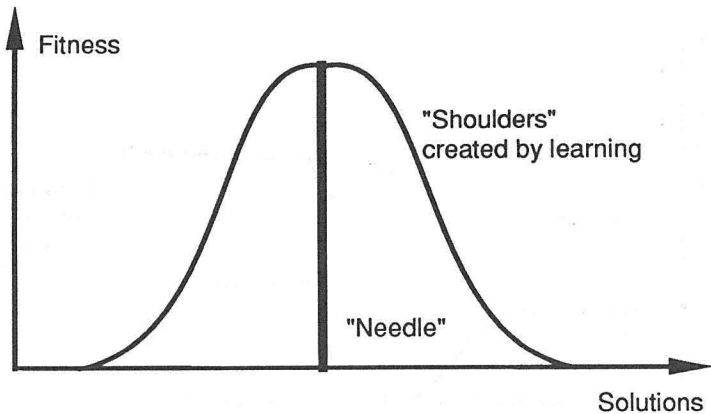


Figure 2: Creating an adaptive basin.

25% 0s and 1s, and gave each individual $G = 1000$ guesses. They also used a population of 1000 individuals, a crossover rate of 1.0,² and no mutation [24].

One property the GA shares with connectionist systems is the notion of “distributed representation.” That is, just as “concepts” in connectionist networks are represented diffusely by a pattern of activity across many nodes, so too are solutions found by the GA represented across individuals of a population. Thus, the simulation variables to be monitored are population-wide characteristics rather than features of any individual. The major dependent variables considered by Hinton and Nowlan are the population-wide distributions of correct, incorrect, and undefined alleles.

Our own work began with a simple replication of the Hinton and Nowlan results, as shown in figure 3.³ Here their three major variables (the allele ratios of undefined, correct and incorrect alleles) are plotted as a function of generation. These basic results will be analyzed in some detail in sections 3 and 4 and then extended in sections 5 and 6.

2.3 Discussion of the Hinton and Nowlan model

The Hinton and Nowlan model is a very fertile platform from which to investigate questions about the interaction between evolutionary and learn-

²A crossover rate of 1.0 means that, on average, each individual will participate in 1.0 crossover operations.

³Grefenstetdt has developed a useful simulation facility for GA investigation called Genesis [13], and it was used as a basis for the simulations to be presented here.

Base - Allele ratios

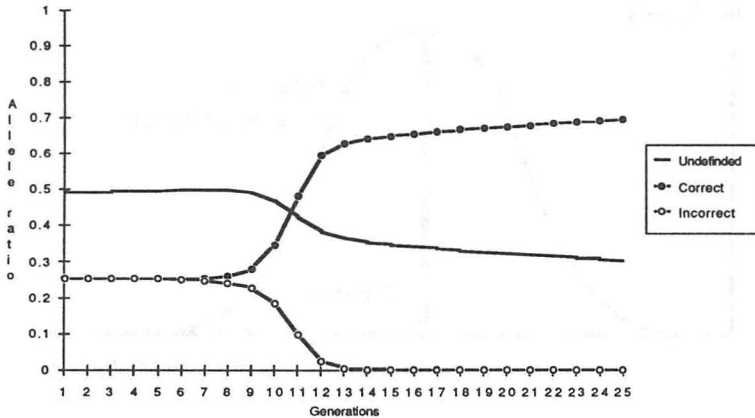


Figure 3: Hinton and Nowlan results.

ing adaptive mechanisms. It does incorporate some important assumptions, however, and it is worth making these explicit.

First, a great deal of the simulation's behavior depends critically on the fitness function of equation 2.1:

$$\mu(x_i(g)) = 1 + \frac{(L-1)(G-g)}{G}$$

The central feature is that it is a monotonically decreasing function of the number of guesses required to find the needle. Hinton and Nowlan also chose to scale this quantity by the ratio of chromosome length to the total number of guesses allowed. This scaling is reasonable but also less critical to the simulation's behavior.

Equating learning with a series of statistically independent guesses is obviously quite inadequate. In fact, by "learning" we typically mean almost exactly that subsequent behaviors of an individual are *dependent* on his or her previous experiences. Thus Hinton and Nowlan's guessing model is properly considered an analytically tractable lower bound on the performance we should expect of a realistic (e.g., connectionist) learning algorithm. In their words, "Thus, using a more sophisticated learning procedure only strengthens the argument for the importance of the Baldwin effect."

It may seem suspect to assume that an individual is capable of recognizing when they have found the correct solution (needle). Hinton and Nowlan provide the following rationale:

This recognition ability . . . is required to make learning effective. . . . Thus it is possible that some properties of an organism which are currently genetically specified were once behavioral goals of the organism's ancestors.

This argument sounds almost Lamarckian and perhaps even absurd when in the context of an individual knowing when its own neural wiring is correct. However, simply giving an individual the ability to recognize when it has found a good thing is not so far-fetched.

A more unrealistic assumption (acknowledged by Hinton and Nowlan) is that the correspondence between phenotypic feature (i.e., neural connection) and genetic position is one-to-one. This is a great simplification. In actuality, there are immensely complicated developmental processes that mediate between genotypic description and phenotypic expression; a better understanding of this relationship is a critical issue for further work in this area.

Another troubling aspect of the simulation is that it ignores the real semantics of neural networks: there is an obvious asymmetry between genetically specified and learned connections. Virtually all connectionist learning algorithms allow connections to come to have zero weight, making them act as if the connection was not there. Thus an existing connection (1) can learn to have zero weight; an absent connection (0) cannot ever become nonzero. We should expect this bias to be exploited by any hybrid adaptive system that combines evolutionary and (connectionist) learning subsystems.

Hinton and Nowlan make another strong and important claim:

. . . each learning trial can be almost as helpful to the learning search as the production and evaluation of a whole new organism.

Thus they view each learning trial as a “cheap” version of actually producing an individual. Our conception of the relation between these two adaptive subsystems is considerably more complex, as we will discuss in section 6.

3. Analysis of the Hinton and Nowlan model

3.1 Definitions

We begin by defining some of the major variables of the simulation. Let:

- L = Length of individual
- G = Maximum number of guesses allowed each individual
- N = Population size
- p = Fraction of population's alleles that are 1
- q = Fraction of population's alleles that are ?
- r = Fraction of population's alleles that are 0

Note that p , q , and r are defined in terms of the total population's genetic distribution. In many cases, however, this is also a good estimate of *individuals'* allele distributions, for example the probability that an individual allele is 1 is also p . Also, we will on occasion use a subscript to indicate generation; for example, p_0 is the fraction of 1s in the initial population.

Unless noted otherwise, these parameters are set as in the Hinton and Nowlan simulations:

$$\begin{aligned} L &= 20 \\ G &= 1000 \\ N &= 1000 \\ p_0 &= 0.25 \\ q_0 &= 0.50 \\ r_0 &= 0.25 \end{aligned} \tag{3.1}$$

Also, recall that Hinton and Nowlan use a “needle-in-a-haystack,” impulse function environment; i.e., there is exactly one value in the domain with a nonzero fitness associated with it. By convention and without loss of generality, we will assume this value to be the string 1111...1.

We will call an individual without any 0s a Potential Winner, since only such individuals are capable of using some combination of 1s and ?s to find the needle. A Winner is defined to be a Potential Winner that actually guesses the correct setting of its learnable alleles; a Loser contains at least one 0. We will refer to the set of all Winners in the population as the Nobility and the rest of the population as Commoners.⁴

We begin with the simple observation that if an individual has Q learnable (?) alleles, the probability of guessing their correct values is 2^{-Q} . Let us define:

$$c \equiv 2^{-Q}$$

To be a Winner, an individual must first be a Potential Winner and second guess the learnable (?) alleles correctly:

$$\begin{aligned} \text{Pr}(\text{Winner}) &= \text{Pr}(\text{no 0s}) \text{Pr}(\text{guessed correctly}) \\ &= (1-r)^L [1 - (1-c)^G] \end{aligned} \tag{3.2}$$

Using the parameter values given above, $\text{Pr}(\text{Winner}) = 0.00198$, and we should therefore expect to find about 2 Winners in an average population of 1000. Our simulations agree with this estimate (see section 5).

⁴This terminology is admittedly colorful and perhaps even offensive, but as you trudge through the symbology and simulation data, it helps to breath some life into it all.

3.2 Expected fitness

It is important to know the expected fitness of an individual under Hinton and Nowlan's "random guessing" fitness function:

$$\begin{aligned} \mu(g) &= 1 + \frac{(L-1)(G-g)}{G} \\ &= L - (L-1)\frac{g}{G} \end{aligned} \tag{3.3}$$

We begin by finding the expected number of guesses g required to pick the learnable (?) alleles for an individual, and this can be derived in two different ways.

First, we can begin with the straightforward derivation of $E(g)$ by summing over the possibilities that the first correct guess comes on exactly the k th trial is

$$E(g) = \sum_{k=1}^{\infty} k(1-c)^{k-1}c$$

However, because our function stops guessing after G attempts, this series must be truncated:

$$E(g) = \left(\sum_{k=1}^G k(1-c)^{k-1}c \right) + G(1-c)^G \tag{3.4}$$

Equation 3.4 is a geometric series. Beginning with the standard identity:

$$\sum_{i=0}^n x^i = \frac{1-x^{n+1}}{1-x}$$

differentiation then gives:

$$\sum_{i=0}^n ix^{i-1} = \frac{1+nx^{n+1}-(n+1)x^n}{(1-x)^2}$$

Then substituting $x = (1-c)$ and $n = G$ into the identity and replacing the series in equation 3.4 with this expression gives:

$$\begin{aligned} E(g) &= \frac{1+G(1-c)^{G+1}-(G+1)(1-c)^G}{c} + G(1-c)^G \\ &= \frac{1-(1-c)^G}{c} \end{aligned} \tag{3.5}$$

We can arrive at equation 3.4 in a more elegant fashion by using Wald's Lemma [5]. This lemma relates the expected payoff $E(S_N)$ of a run of inde-

pendent, identically distributed trials, each with expected payoff p , using an arbitrary stopping criterion N with expected value $E(N)$:

$$E(S_N) = pE(N)$$

In our situation, “success” simply means guessing the Q learnable values correctly and so $p = c$. Our strategy N stops either when we have guessed correctly or guessed G times without success. Our payoff will therefore be one unless we were unable to guess the correct combination in G trials, hence:

$$E(S_N) = 1 - (1 - c)^G$$

Solving for $E(N)$, which is exactly the same as the $E(g)$ we desire, we again get equation 3.4.

Note that if we are allowed many guesses, $E(g) \gg c$ and we are almost certain to succeed; conversely, if there are many ?s, $E(g) \ll c$ and we will almost certainly fail. Thus we are most interested in the case when $E(g) \sim G$. Alternatively, if we define:

$$\lambda \equiv Gc$$

we are interested in the case $\lambda \sim 1$. In this case, we can approximate $1 - c \approx e^{-c}$ and equation 3.4 becomes

$$E(g) \approx \begin{cases} G & \text{for } \lambda \sim 0 \\ \frac{1-e^{-\lambda}}{\lambda} G & \text{for } \lambda \sim 1 \\ 1/c & \text{for large } \lambda \end{cases} \quad (3.6)$$

We are now able to substitute $E(g)$ into equation 3 to obtain the expected fitness of an individual:

$$\begin{aligned} E(\mu) &= L - \frac{L-1}{\lambda} (1 - (1-c)^G) \\ &\approx L - \frac{L-1}{\lambda} (1 - e^{-\lambda}) \end{aligned} \quad (3.7)$$

Figure 4 approximates the derivative of this function with respect to g using differences. This graph makes two important points that will be echoed later. First, it shows how little selective pressure there is to replace ?s with 1s in a string of almost all ?s (see section 5.2). Second, it shows how little selective advantage there is to replacing the last few ?s (see section 4).

3.3 Asymptotic behavior

Consider the situation late in the simulation, when the entire population is composed of only 1s and ?s. Let us assume that all but one member of the population has exactly \hat{Q} ?s, and that this one individual x^* is a bit more fit, with exactly n less, $Q^* = \hat{Q} - n$. We will ask just how likely it is that this more fit individual will get to make more than one copy of itself in the

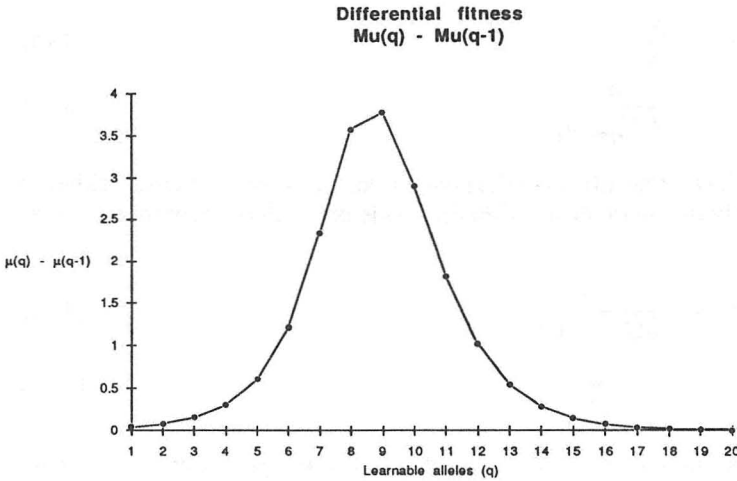


Figure 4: Differential $E(\mu)$.

next generation, thereby increasing the population’s ratio of 1s, since this is just the process of gradual replacement underlying the system’s asymptotic behavior.

Recall (equation 3.6) that when λ is large, we can approximate $E(g) \approx 1/c$. Some algebra then gives us the difference:

$$E(g^*) - E(\hat{g}) = 2^{\hat{Q}}(2^{-n} - 1)$$

Substituting these values into equation 3.2, a bit more algebra gives us the fitness advantage the loner x^* enjoys over the rest of the population. We will define this difference as α :

$$\alpha = \mu^* - \hat{\mu} \tag{3.8}$$

$$= \frac{L-1}{\lambda}(1 - 2^{-n}) \tag{3.9}$$

In order to relate the individuals’ fitness μ to the number of copies made of each individual, ν , we must consider the details of the genetic algorithm’s “selection” procedure. Following Baker [1], we can imagine each individual being allocated a slice of a wheel proportional to its relative fitness and then uniformly selecting from this wheel N times to pick our new population.

Thus:

$$E(\nu^*) = \frac{\mu^*}{\bar{\mu}} \quad (3.10)$$

$$= \frac{\mu^*}{\frac{1}{N} \sum_{j=1}^N \mu_j} \quad (3.11)$$

where $\bar{\mu}$ is the average fitness of the population. In the case we are considering (with exactly one more fit individual), this is particularly easy to compute:

$$E(\nu^*) = \frac{\hat{\mu} + \alpha}{\frac{1}{N}(N\hat{\mu} + \alpha)} \quad (3.12)$$

$$= \frac{1 + \frac{\alpha}{\hat{\mu}}}{1 + \frac{\alpha}{N\hat{\mu}}} \quad (3.13)$$

Consider the quantity $\alpha/\hat{\mu}$, the ratio of the unique individual's fitness advantage to the fitness of the rest of the population. This ratio will always be small and since $\alpha/N\hat{\mu}$ is even smaller, we can legitimately approximate this using the first two terms of the Taylor series expansion:

$$E(\nu^*) \approx (1 + \frac{\alpha}{\hat{\mu}})(1 - \frac{\alpha}{N\hat{\mu}}) \quad (3.14)$$

$$= 1 + \frac{\alpha}{\hat{\mu}} - \frac{1}{N}(\frac{\alpha}{\hat{\mu}} + \frac{\alpha^2}{\hat{\mu}^2}) \quad (3.15)$$

Thus the number of copies we can expect to make of a slightly more fit individual when the entire population is quite fit is only a bit greater than one. For example, if we use the parameters of equation 2.1 and consider the case when $\hat{Q} = 6$ (see figure 4) and the more fit individual has only one less ? ($n = 1$), we find that $\alpha/\hat{\mu} = 0.0324$; the more fit individual has only slightly better than a 3% chance of making any more copies of himself in the next generation than anyone else. Even if a very lucky individual happens to get *two* less ? ($n = 2$), his or her chances improve only to about 4.8%. Further, selection in the GA is *discrete*; even this slight advantage is only in the *expected* value of $E(\nu^*)$.

4. Explaining the model's behavior

Armed with the analytic tools of the previous section, we are in a position to explain the simulation results presented in figure 5. This figure shows the same three allele ratio curves of figure 3, but has been overlaid with the average fitness of the population and delimited into what we will argue are four significantly different phases. Beginning with the population composed

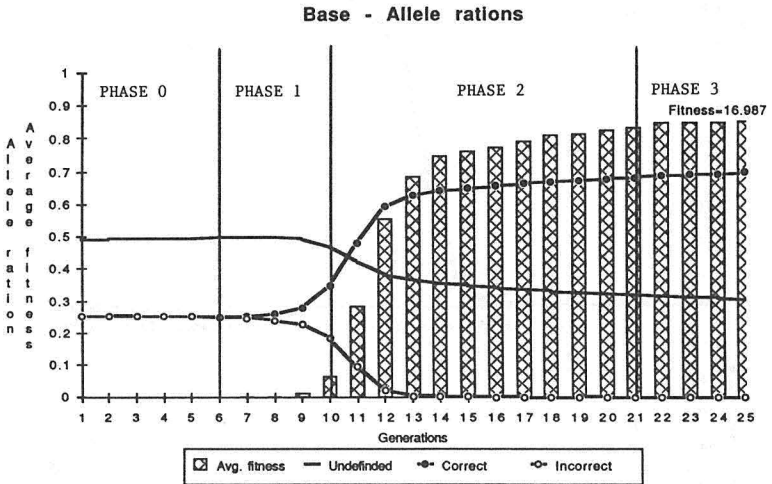


Figure 5: Four phases.

of 50% ?s and 25% 0s and 1s, the population can be viewed as passing through four different phases.

For a surprisingly long time, which we will call Phase 0, the allele ratios of the population remained almost unchanged. One possible explanation for this apparent equilibrium is that no individual has yet guessed the correct solution. But this is not the case. The previous section shows that with the parameters used we should expect approximately two Winners per generation, and figure 6a shows this to be the case; the important first generations of this curve have been expanded in figure 6b.

A small number of Winners are being produced each generation, but they have minimal effect on the population. The problem is that, because the GA models sexual recombination, offspring of the Winner will also have significant genetic material from some Commoner and hence are almost certain to be Losers themselves. However, these Winners do have the subtle effect of enriching the population-wide ratios of 1s and ?s slightly. The reason is simply that Winners, composed entirely of 1s and ?s, are much more fit than average and hence make more copies of themselves. This constant, albeit small, enrichment of 1s and ?s provided by Winners guarantees that the number of Winners steadily increases.

John Maynard Smith has made a similar observation about the Hinton and Nowlan simulation [28]. He notes that in a population without any learning capability (i.e., with only 1s and 0s), asexual reproduction can be

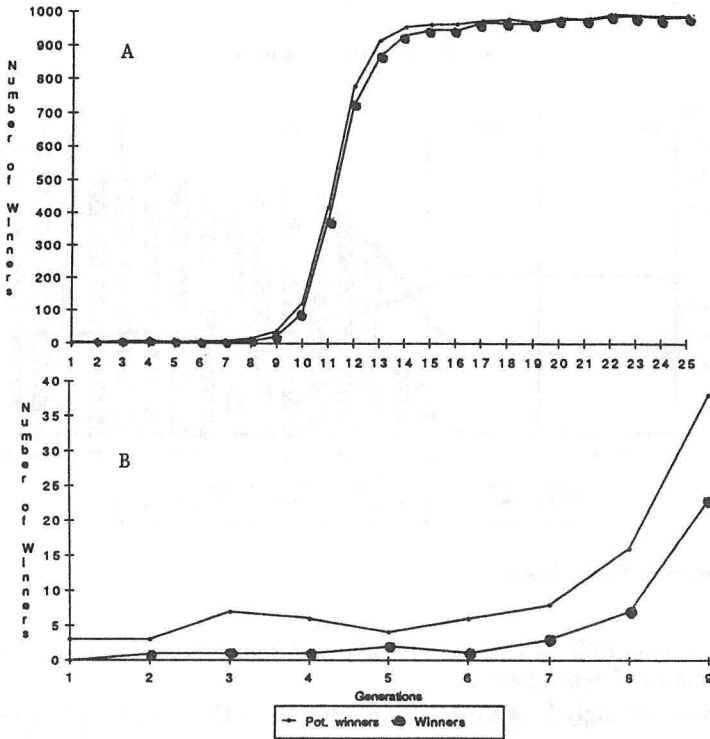


Figure 6: Winners.

expected to find the solution more quickly than with *sexual* reproduction and crossover. It will take both methods a very long time⁵ to find the needle, but once found asexual reproduction will reproduce this solution with more fidelity than sexual reproduction.

During Phase 0, it is extremely unlikely that two Winners will “find one another” and produce winning offspring. Phase 1 begins when two Winners find one another, forming offspring that are themselves Potential Winners. When this happens, Winners begin to beget Potential Winners and a strong positive feedback cycle is entered in which Losers are steadily replaced with Potential Winners. Note that the only difference between a Loser and a Potential Winner is that all 0s have been replaced by a 1s. Hence, during Phase 1 the ratio of 0s goes down, the ratio of 1s goes up and the ratio of

⁵There are 2^L combinations to guess, so if the size of the population is N , it will take $2^L/N$ generations.

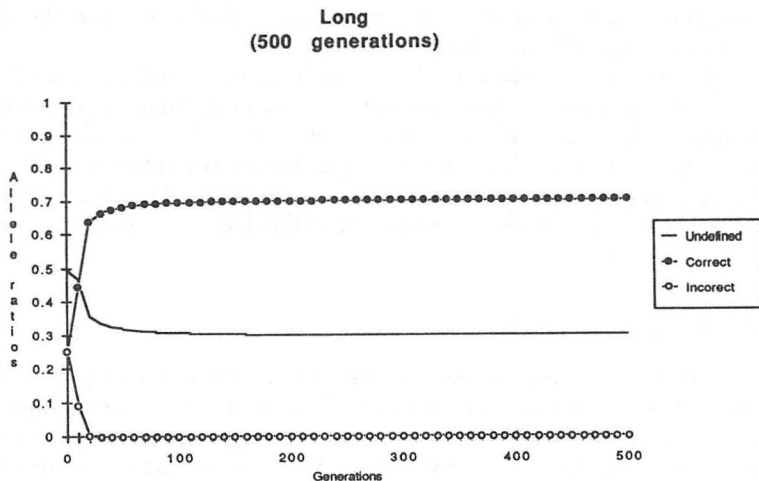


Figure 7: Long run.

?s remains almost unchanged. Notice also that the average fitness of the population changes little during Phase 1.

Phase 2 continues this same basic trend, but begins to also apply pressure against guessed alleles (?) in favor of genetically specified correct ones (1). In terms of the fitness function (see equation 3.2) an individual is always better off not having to guess, and so there is a constant selective pressure against ?s. In other words, as the 0s of Losers are replaced to create Potential Winners, the ?s in these Potential Winners are almost immediately replaced to make them more likely to be actual Winners. Thus it is during Phase 2 that the average fitness of the population increases most markedly.

However, as Hinton and Nowlan correctly observe, this advantage diminishes as the number of ?s in a Potential Winner becomes small. The analysis of section 3 shows how fitness depends on the number of ?s, and in particular how little adaptive advantage there is to replace the last few ?s with 1s. Ultimately, in Phase 3 this effect results in an almost steady-state population composed of approximately 30% ?s and 70% 1s. While these two curves are in fact asymptotically approaching 1.0 and 0.0, respectively, progress is extremely slow. In fact, figure 7 shows that these levels remain virtually constant out to 500 generations.

The reasons the asymptotic convergence of the population has been effectively stalled can be found in the analysis of the last section. As shown in figure 4, there is very little selective advantage to replacing the last few ?s. More concretely, the asymptotic analysis of section 3.3 showed that, late in Phase 3, a single more fit individual has only a slightly higher probability

of producing even one additional offspring. In short, the additional fitness achieved by replacing a 1 with a ? in Phase 3 is so small that the probability of producing more than an average number of offspring and thereby increasing the ratio of 1s is infinitesimal.

The picture we have, then, is of a dynamical process with first a slight but constant movement toward a critical point (during Phase 0), an almost instantaneous “ignition” (when Nobilepersons first find one another) that within a few generations changes the composition of the entire population, first by replacing incorrect alleles with correct ones (Phase 1) and then by replacing learnable alleles with correct ones as well (Phase 2) at an increasingly slow rate (Phase 3).

5. Parameter sensitivity

One elegant aspect of Hinton and Nowlan’s simulation is the way in which their parameters are delicately balanced against one another. For example G — the number of guesses allotted — was chosen with regard to the average number of ?s in an individual (see section 3). A second phase of our own research perturbed some of the major variables of the simulation and found it to be surprisingly robust. Section 5.1 considers stochastic variability and a question we believed to be related, the size of the population. These experiments also substantiate the predictions of section 3.3 concerning asymptotic behavior of the model. The sensitivity to allele ratios in the initial population is considered in the next section. Finally, we investigate the effect of *mutation* on the model. Hinton and Nowlan did not include mutation in their experiments, and so it is something of a misnomer to investigate it under the rubric of “parameter sensitivity.” However, mutation is a very standard part of most GA implementations and easily incorporated into the model. Section 5.3 shows it to have nontrivial effects.

5.1 Stochastic variation and population size

The GA, let alone the individuals’ guessing procedure, depends heavily on stochastic procedures so we must be suspicious of any one run. For this reason a series of runs were performed that differed only in their initial random number seed. Figure 8a shows the average and standard deviation of population fitness (a typical statistic) of five separate runs. These statistics show that while the initial and ultimate behaviors of the simulation are quite consistent, there is significant variation during the middle generations. However, figure 8b shows that this variation is of a particular form, viz., the generation in which Phase 1 begins. Recall that this event depends on two Winners finding one another out of a large population of other potential mates, and while the increasing levels of 1s and ?s during Phase 0 guarantees it will occur sometime, the generation of the actual occurrence is highly variable.

We hypothesized that increasing population size (N) would, because of the Law of Large Numbers, decrease this variability, and a number of simu-

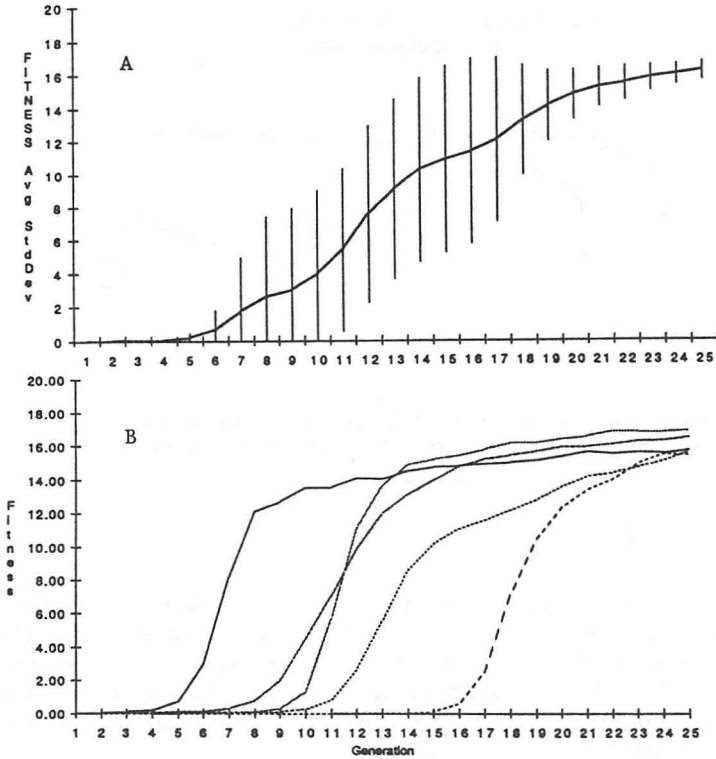


Figure 8: Stochastic variation.

lations were run varying this parameter. In fact, stochastic variation did not appear to vary with changing population size. The most significant effect of varying population size was that smaller populations consistently moved more quickly through all three phases than larger ones (see figure 9). This behavior can be attributed to the fitness “scaling” strategy used in the Hinton and Nowlan (and our) model: even the most fit individual can make at most L copies of itself. Since this represents a much larger fraction of small populations, Winners can come to dominate the population that much more quickly.

5.2 Initial ratios

We can first limit the range of possible initial population compositions using qualitative arguments. The central point of the Hinton and Nowlan paper is that, without learnable (?) alleles, pure evolutionary search is blind and ex-

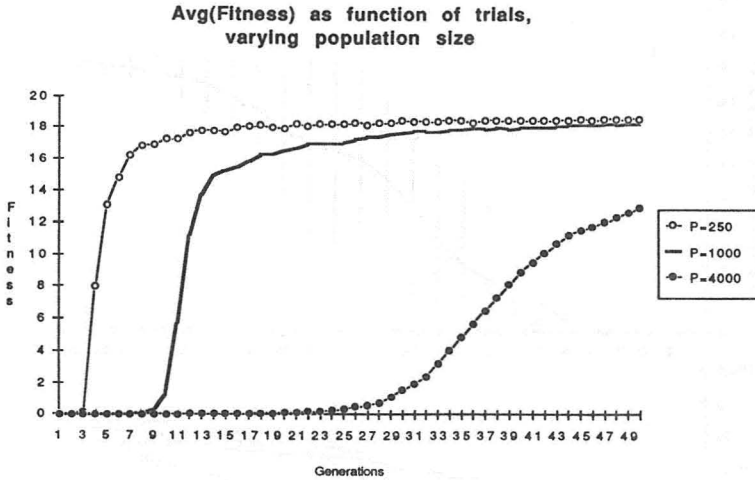


Figure 9: Population size.

ceedingly slow. Hence, q_0 must be significant. With the analysis of section 3.2 we can make a more refined statement; figure 4 shows that an individual has little additional fitness until he (or she!) contains about 7 ?s, which suggests q_0 must be about 35%. Similarly, an individual is completely unfit if it contain even one incorrect 0 allele, so r_0 large simply delays the time before, by chance, Potential Winners are created.

With these considerations in mind, a wide range of initial populations were explored. Figure 10 shows a typical variation. In this run the initial ratio of ?s to 1s was maintained at 2:1 (46% to 23%), but a higher ratio of incorrect 0s than correct 1s was used (31% vs. 25%). The result was to significantly slow down the population's ability to converge on the correct solution. There is a long phase (which might be viewed as a Phase -1, coming before the Phase 0 mentioned above) during which 0s are steadily replaced with 1s. However, once the number of Winners produced in a generation is sufficient to ensure the positive feedback of Phase 1, this population behaves the same as the original one.

5.2.1 Poor man's diploid

Experiments with varying initial population ratios produced another curious result that helps to make an important point about representation and the GA. Figure 11 shows a population that was begun with 50% ?s and 50% 1s and no 0s. Yet by Generation 2 almost 8% of the population's alleles are 0s. If the mutation operator were in effect (see section 5.3 below), this could be

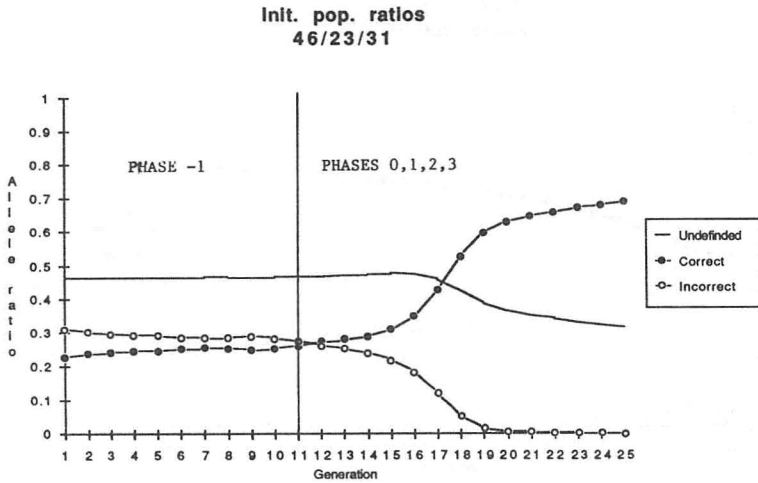


Figure 10: Varying initial population.

expected to introduce new alleles in this manner but this run was without mutation, but without mutation how is this possible?!

In order to explain this effect, recall the binary encoding of the three allele values described in section 2.1: It obviously takes at least two bits to represent the three allele values 0, 1, and ?. In our simulations the encoding was

Allele	Bits
0	10
1	11
?	0*

with star (*) indicating “don’t care”; i.e., this bit can be either 0 or 1. Given the random procedures for creating the initial population we should expect about half the ?s to be of each type; call the two varieties ?₀ and ?₁.

Now consider what happens when crossover is performed between two parents in our initial population of only 1s and ?s. If one parent contains a ? of the ?₀ variety and the other parent contains a 1, it is possible to produce an offspring with a 0 allele by performing the crossover at the point between the two bits of the allele. Despite the fact that the population begins without any 0s the genetic material for producing this allele value is still present due to the redundancy of the encoding. It would obviously be possible to modify the crossover operator so that it operated only between alleles rather than between bits, but this is again against the “semantics-free” spirit of the GA (see section 2.1).

NoZero: 50/50
Allele rations

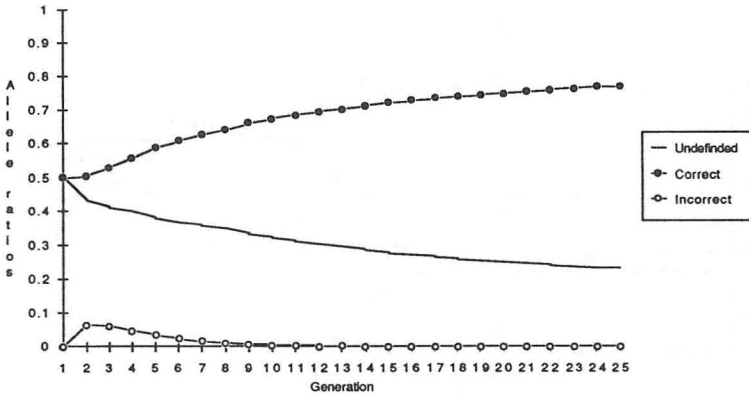


Figure 11: Initially no Losers.

This is more than a curiosity. It suggests an aspect of the GA's representation that might be called "Poor Man's Diploid." The ability of diploid chromosomes to maintain *recessive* alleles while typically expressing the *dominant* allele is a central component of population genetics. This mechanism allows a species to maintain an alternative solution should the environment (again) demand it. In true diploid species, however, the cost paid is the *representational redundancy* of having two full copies of each allele.

Almost all versions⁶ of the GA (including this one) use a single, haploid chromosome, but these experiments show how any encoding redundancy can have a similar effect, even in haploid species. As soon as there is any spare representational capacity (e.g., the *-bit in this case), it is potentially available to hold information that is not of immediate functional consequence.

From another perspective, this effect helps to deflate a strict "adaptationist" position that sometimes argues that any and all sustained genetic variation is a direct reflection of increased fitness. This is true only if the representation is minimal. Otherwise, unused representational capacity is available for other purposes, such as maintaining alternative solutions, but also including use by other adaptive systems (see section 7.1).

⁶Grosso's thesis investigated diploid populations along with other, more biologically plausible variations of the GA [16].

5.3 Mutation

One important simplification of the Hinton and Nowlan model of evolution is the absence of any mutation effects. “Mutation” is sometimes used to refer to any and all modifications to the genotype passed from parent to child. In the GA, however, mutation is used to describe only *random* modifications. These play a much less important role than the principled modifications generated by the crossover operator (see section 2.1), so that mutation is properly considered a “background” operator in the GA. Nevertheless, mutation has been shown to play an important role in the GA, viz. preventing “fixation” [10]. That is, mutation provides the only mechanism for reintroducing allele values that may, by chance, have become removed from an entire population. As a result, without mutation it is possible for a population to converge prematurely on a suboptimal solution. On the other hand, it is also true that if the mutation rate is too high, this random operator effectively stalls progress of the GA by breaking up the schemata processed by crossover.

Mutation rates are typically defined in terms of the probability of changing *bits*, but when the alleles are not binary this definition is somewhat problematic. For one thing, it allows “silent” mutations (i.e., not changing the allele value) if the *-bit is the one that happens to change. Alternatively, we could modify the mutation operator to ensure that all modifications changed allele values.⁷ However, this seems against the “semantics-free” spirit of the GA, and so the simulations presented here simply treat the individuals as bit strings of length $2L$.

A number of simulations were run with varying mutation rates; some of these results are shown in figures 12. In some respects, these curves are in agreement with intuition. Small mutation rates (10^{-5}) have little effect,⁸ intermediate mutation rates (10^{-3}) slow down the genetic search, and very high mutation rates (0.2) disrupt the search entirely. However, notice that during the early Phases 0 and 1 an intermediate mutation rate (10^{-2}) has about the same effect as that of no mutation whatsoever, but then slows down the final convergence of Phase 3. Thus there is a nonmonotonic relation between mutation rate and population convergence.

In order to explain these effects, we must notice that mutation has two counterbalancing effects in the early phases. The first effect is to “clobber” members of the Nobility. That is, an individual whose parents passed on Potentially Winning genetic material is ruined if any of it is mutated. The second effect is to increase the variance of the Commoners. With very small mutation rates, neither of these two effects is achieved, and the result is the standard simulation. With intermediate mutation rates, the fragile Winners are likely to be ruined, but the variance of the Commoner population is

⁷This may become necessary to accurately capture real genetic phenomena such as “back mutation,” where the probability of mutating from allele X to allele Y is *not* equal to the probability of the inverse change.

⁸To be concrete, a mutation rate of 10^{-5} means that with a population of 1000 individuals, with each individual of length 20 and with two bits/allele, there is only a 40% chance that *anyone* in the population will be changed at all.

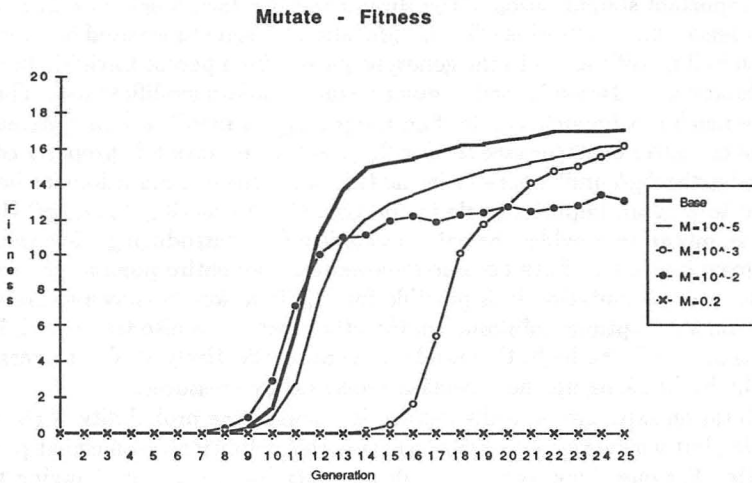


Figure 12: Mutation.

not changed dramatically. As the mutation rate is increased, no additional harm can be done to the already-ruined Nobility, but Commoners stand a better and better chance of being mutated into Potential Winners. Thus, intermediate mutation rates provide a new source of Winners. Finally, if the mutation rate is made extremely high, the ability of crossover to pass on solutions to subsequent generations is undermined.

Later, in Phase 3, the dominant effect of mutation is to introduce 0s into a population that would otherwise be composed entirely of Potential Winners. It is obvious that this will slow the population's final convergence.

6. Incorporating culture into the model

Hinton and Nowlan's original model and our extensions to it point to subtle ways in which the learning of individuals and the evolution of species of such individuals can be coupled to form effective, hybrid adaptive systems. But when we consider the human species and the rate at which evolution has shaped it as compared to the rate at which individual humans learn, the mismatch (perhaps 12 orders of magnitude) is quite striking. Further, it seems that, at least in the case of the human species, additional adaptive processes have developed to allow the learning experiences of one individual to become useful to other individuals much more quickly and directly. *Culture* can be viewed as a third, interposed adaptive system that allows the hard-won knowledge learned by an individual to improve the evolutionary fit-

ness of other conspecifics (i.e., members of the same species) via nongenetic informational pathways.

Our basic model can be extended to investigate some of the issues raised when the constructs of culture, evolution, and learning are juxtaposed in this way. Obviously, such a model will be macroscopic to the extreme, just as the genetic algorithm is a gross simplification of evolution and random guessing a gross simplification of learning. However, when the object of investigation is the interaction among three such extraordinarily complicated systems, such radical simplification is necessary.

6.1 Dual inheritance

Boyd and Richerson have developed a “dual inheritance” model encompassing both evolution and culture that emphasizes the two ways, genetic and nongenetic, in which conspecifics can pass adaptively useful information [6]. Following a suggestion of Hutchins, we inject this notion of culture into the Hinton and Nowlan model as directly as possible: If and when a parent becomes a Winner, they confer upon their offspring a cultural advantage (CA) that gives these offspring a better than average chance of guessing the correct value of the learnable alleles. So, for example, if $CA = 0.1$,⁹ the offspring of a Winner has a probability of 60/40 chance of guessing each of its learnable alleles correctly, rather than its normal (culturally disadvantaged) 50/50 chances. The rationale is that parents have some ability to “imprint” their offspring by skewing the allocation of random trials somewhat. A slightly more elaborate notion will be considered in the next section, and the more general issues of modeling culture are considered in section 7.

Figure 13 shows the results of this modification using $CA = 0.1$. A CA allows a population to find and then converge to a solution more quickly, simply because more effective guessing broadens the shoulders of the search space around the correct answer still further. Just as learning creates a basin of attraction around the solution needle, information (in this case passed extra-genetically) that makes the learning/guessing process more effective broadens this basin still further.

The second observation, however, is that a CA removes some of the pressure to achieve optimum performance; i.e., while the population converges more quickly, the solution it converges to is not as good as without CA. The reason can be seen in figure 14, comparing the allele ratios with and without CA. The most striking difference is that the maximum levels of ?s and 1s have been reversed. The population with CA reaches a ratio of about 80% ?s and 20% 1s before beginning its slow descent through Phase 3. Because an individual is now more likely to guess learnable alleles (?s) correctly (because of the CA imparted by its parent), a much larger number of ?s can be tolerated and the selective pressure against them is much reduced.

The pressure is not entirely removed, however, as the slight slope on these two lines indicates. It is still always advantageous to replace a ? with a 1,

⁹This value was used in the simulations described below unless noted otherwise.

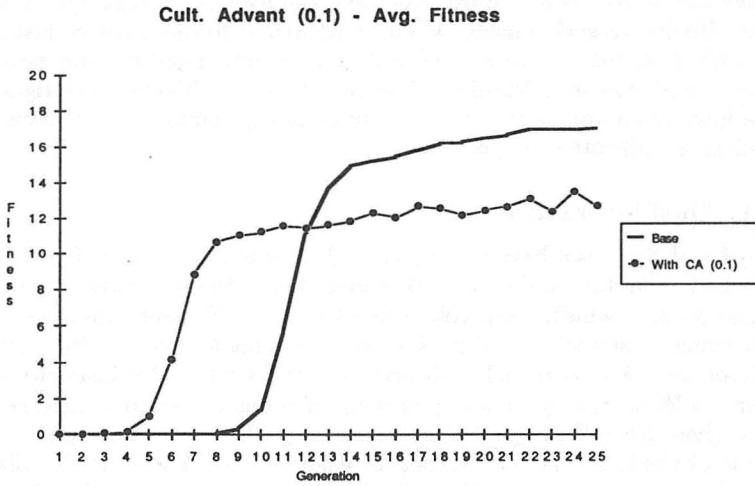


Figure 13: Effect of CA on fitness.

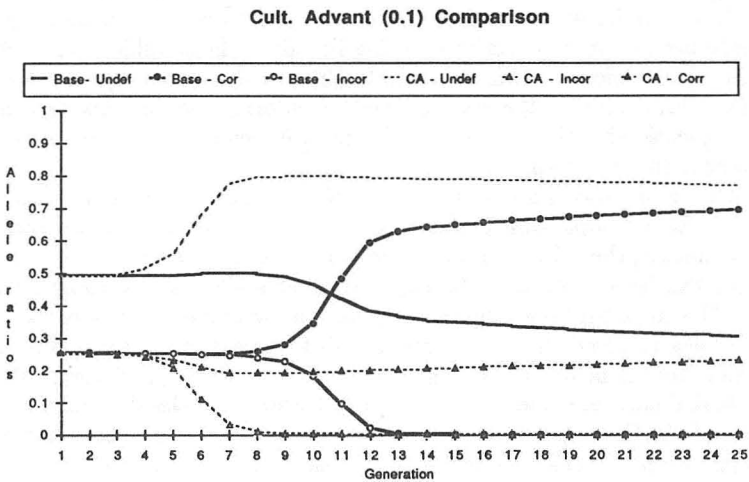


Figure 14: Allele ratios with CA.

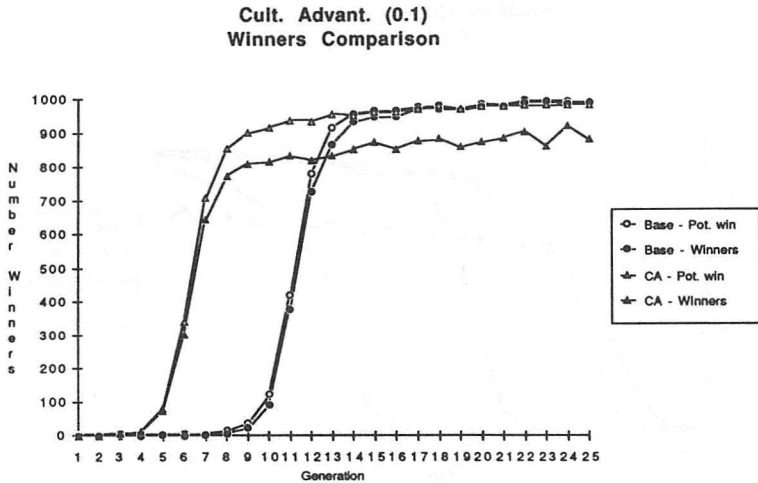


Figure 15: Winners with CA.

since knowing for sure is always better than even effective guessing. Figure 15 shows the number of Winners and Potential Winners, both with CA and without. It is interesting because it shows that even though a population with CA does not produce as many actual Winners, it does produce just the same number of Potential Winners. CA can therefore be viewed as making the population something of an “underachiever.” CA is useful for initially discovering the solution, but it also removes the selective pressure required to push the population to its optimal performance with all 1s.

It turns out that this diffidence can be corrected by introducing a very low mutation rate. Figure 16 shows that a combination of cultural advantage and mutation produces a population that converges even more quickly than with cultural advantage alone, and finds an equally good optimum. Again, CA facilitates the initial discovery of the solution “needle,” and mutation then increases the pressure against ?s by increasing the probability that they will be replaced by 1s.

6.2 Plasticity

Allowing some information to be passed extragenetically from generation to generation allows a population to maintain a higher ratio of learnable versus genetically specified alleles, for longer. Why might such “plasticity” be advantageous? One reason is that learnable alleles can be modified in the face of a changing environment, while genetically fixed ones cannot. A series of simulations were performed to illustrate this point. At fixed intervals

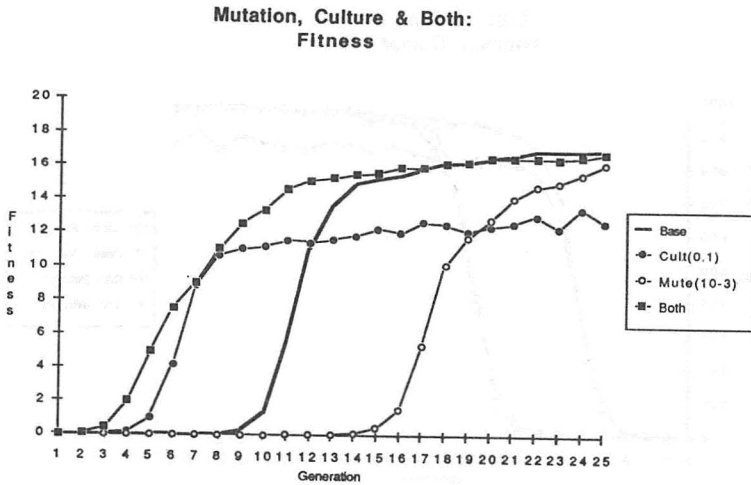


Figure 16: Interaction between CA and mutation.

(25 generations in this simulation) several of the bits of the needle (i.e., correct answer) were changed; this can be viewed as moving the needle in the search space slightly. Figure 17 shows the response of various populations to this sort of modification. First, a standard population, without mutation or cultural advantage, is unable to track these environmental changes. Without some mechanism to reintroduce lost alleles, the population converges on the early solution but is then unable to search any other portion of the space; this “premature convergence” effect is well known in the genetic algorithm literature [9].

Mutation is an extremely robust mechanism for allowing a population to back out from (what has become) a maladaptive solution. When the new solution is relatively close to the old one, the simple random-walk strategy of mutation has a good chance at success. But as these curves show, random changes are not terribly intelligent and so this can be a slow process.

Cultural advantage was successfully able to respond to the first of the environmental changes (at generation 25), exactly because it had learnable versus genetically fixed alleles at these positions. This was the expected result. Notice, however, that the population with CA was not able to respond to the environmental change at generation 50. While cultural advantage allows individuals to have a higher ratio of η s, some positions are still genetically fixed, including (it so happens) one of those involved in the environmental change at generation 50. A population with CA will remain more plastic but once an allele becomes genetically fixed it is no better off in the face of environmental change than one without. The best solution, as shown by the

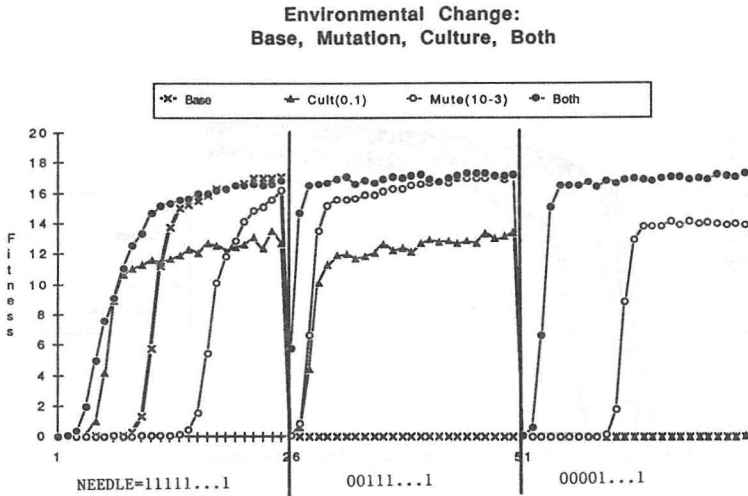


Figure 17: Changing environment.

fourth curve in the figure, is the combination of both mutation and cultural advantage. This gives the population the responsiveness of culture with the robustness of mutation. Together, these two mechanisms allow a population to track environmental change very well.¹⁰

6.3 Broadcast vs. lineage models

The “dual inheritance” notion of culture broadens the range of possible channels through which the experience acquired by one individual might be conveyed to another. Rather than requiring that all information be encoded genetically, we have allowed some useful (albeit unspecified) information to be passed from parent to child extra-genetically. But now that the transmission of information has been cleaved from a strictly genetic basis, culture can also allow for a broadened range of recipients for this information than strictly biological offspring. Rather than conveying the cultural advantage only via lineage, we can tell it to other, genetically unrelated members of the population. In other (more metaphoric) words, when we find the secret of the needle, we don’t have to tell (only) our own children; we can tell the kids down the street. In fact, we could “broadcast” the CA to as many as

¹⁰Following a suggestion of Phillip Kitcher, it would be interesting to explore how quickly the environment can change and still have the population able to track it. Similarly, variation of the size of environmental changes (the number of bits altered) can be expected to be a critical parameter.

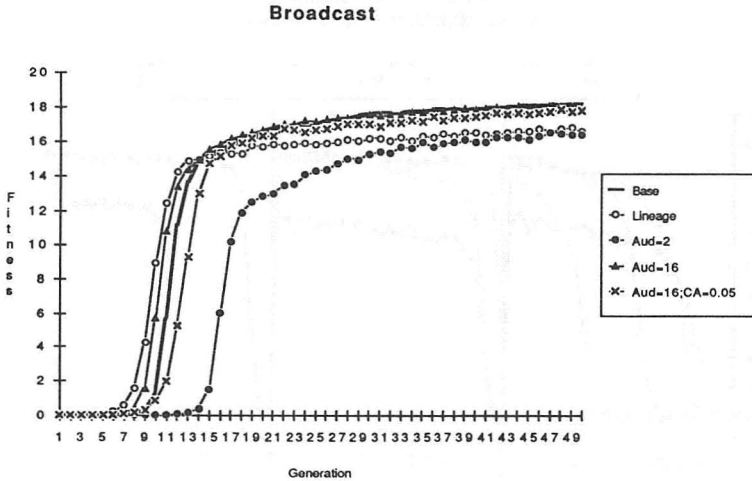


Figure 18: Broadcast CA.

we like! We might expect this use of a powerful new communication ability to improve the ability of a population to converge quickly.

Another set of simulations were run investigating the broadcast of CA to arbitrarily selected members of the population. More specifically, once the needle is found by a Winner, CA is passed on to an audience of B randomly selected members of the next generation. Typical results are summarized in figure 18. This figure compares a standard run (with no CA), a population with CA passed in the “lineage” fashion discussed above, two runs in which the CA is passed from a Winner to either $B = 2$ or $B = 16$ randomly selected members of the next generation, and finally one in which again $B = 16$ but the CA has been reduced by half, $CA = 0.05$.

There are several interesting things to note here. First, broadcasting the CA can be more effective than passing it to just biological offspring, but only if a fairly large audience is allowed to benefit. Comparing the lineage and $B = 2$ results, we note that the latter does significantly worse; giving the CA to two, randomly selected members of the next generation is less effective than conveying it to biologically related ones.¹¹ The reason is that it does little good to convey CA to an offspring that is genetically unfit to receive it. The probability that a randomly selected offspring will be a Loser is much higher than that of a biological offspring of a Winner. In other words, much of the wisdom of broadcast CA falls on “deaf ears.”

¹¹This comparison is a bit simplified, since in the lineage model the number of biological offspring to which the CA may be conveyed can be more, or less, than two.

But if the CA is broadcast to a sufficiently large population, as in the $B = 16$ curve, this form of communication can become more effective. In the extreme, if the CA is made available by a Winner to the entire population of the next generation this becomes a clear advantage over the lineage model. However, it is also reasonable to expect the “fidelity” of the broadcast message (whatever it might be) to erode as the size of the audience increases. We can model this as simply a smaller CA, and this condition is shown in the final curve of the figure.

In summary, biological evolution proceeds by conveying partial solutions from parents to children via genetic material. Introducing a cultural construct allows us to consider both the effect of allowing a parent to pass extra-genetic information to his or her own children, as well as the effect of passing this information to conspecifics that are not biological offspring.

7. Discussion

7.1 Modeling culture

Our current experiments have only begun to address the phenomena of culture. They treat culture simply as an extra-genetic channel through which information about successful search can be passed from one generation to the next. Because our model of individual lives is so crude (viz., a number of guesses for the correct settings of plastic alleles), culture takes on a similarly crude form (an increased probability of success at this guessing game). Still, the exercise has been useful in that it has helped to define a set of issues for future work.

In our model, culture has been reduced to a single real number, reflecting a “cultural advantage” in the otherwise random process of searching for a solution. It is conferred by an individual that has found the correct solution to some members of the next generation. Even in this crude model we can distinguish two separate issues that seem common to any model of culture, the *artifact* (object of the transmission) and the *audience* (recipients of the transmission).

In our model, the artifact is a single real number. Note that this is a particularly uniform, global notion of culture and certainly wrong. Exactly the same artifact is used throughout the entire society/population, and uniformly at each bit position. Thus all the critical issues of *language* — how successful individuals describe their solution, how others understand this description — are ignored in favor of a single variable capturing the effectiveness of any and all extra-genetic information flow.

We have experimented with two different notions of audience. The first, “lineage” model has only biological offspring of successful individuals receiving cultural advantage. Our second, “broadcast” model comes closer to

capturing culture's ability to communicate information to arbitrary, nonbiologically related elements of the population.¹²

Another key distinction has to do with the "age" of the audience. In the simulations reported here, each generation is disjoint in time from previous and future ones.¹³ Similarly, the audience for a successful individual's cultural advantage is always restricted to members of the next generation only. This was useful for investigating "dual transmission" questions of *inter*-generational genetic and cultural information. However, it is also possible to consider *intra*-generational communication of cultural information. It is difficult to imagine the value of intra-generational communication within the current model, but the next section suggests a role for this kind of "symbiotic" search. In fact, it seems likely that these two types of cultural transmission will play profoundly different roles in an account of evolution, culture, and learning; this observation dates back to Vygotsky [29].

Finally, it is important to note the way in which our ability to model culture is constrained by our model of individual learning. Our individuals spent their (bleak!) lives making a set of random, disconnected guesses. In this simple model, the only obvious way culture can enrich these individuals' lives is by making them better guessers. However, as we allow more sophisticated learning mechanisms we are given the opportunity to pass more interesting, useful artifacts. *Hutchin's "citizens" provide an excellent example [22]*. His individuals use several PDP networks for learning and are given the opportunity to learn both from direct experience (with the environment) and "mediated" experience (with artifacts left by others). If we are interested in the central questions of culture and language, especially intra-generational communication, we must expect that our models of individual lives and learning will contain more complexity of this sort.

7.2 Functional consequence of culture

An immediate benefit of even the crudest attempts to integrate culture into a model with learning and evolution is that it provides concrete proposals for exactly why culture is valuable. Anthropology typically takes as axiomatic that culture is important and then proceeds to catalog and explicate various cultural activities. However, this leaves a gap as to *why* culture is important that threatens to undermine any theoretic understanding of culture or how it relates to other aspects of individual behavior in a physical environment. By casting the problem as one of adaptive search by a species, we can motivate culture in very functional terms of improved performance.

Our model illustrates two clear advantages for culture. First, populations with cultural advantage are able to converge on a solution significantly *faster*

¹²This model could be refined further with a spatial distribution of the population that would limit the broadcast of information to "near" neighbors.

¹³Genesis has a very convenient parameter, "generation gap," controlling the overlap between generations. Simulations investigating interactions between this parameter and cultural variables are planned.

than those without. In our model, culture helps individuals guess better than chance, and anything that makes an individual better at guessing improves their fitness.

The second advantage of culture suggested by our model is the additional robustness it provides a population in the face of environmental change. An improved ability to guess caused the population to devote more genetic material to learnable vs. genetically specified alleles. This additional plasticity allowed the population to track environmental changes, through changed cultural information, rather than being trapped into obsolete, genetically fixed solutions.

We can envision several other functional advantages for culture that go beyond our current model and are necessarily more speculative. These require *intra*-generational communication among peers within the timespan of a single generation (in addition to the *inter*-generational communication now used) and attempt to capture a notion of *symbiotic* search.

The most straightforward extension of our current model is suggested by the critical event of the two Winners finding one another and igniting the population into Phase 1 (see section 4). Currently, mates are selected at random from the entire population and this event is therefore highly variable (though guaranteed) and slower than it might be. If some mechanism could cause Winners to “find one another” more quickly, this would almost certainly expedite the population’s search. There has been significant investigation of “restricted mating” strategies with the GA literature (e.g., [9]) and this seems very relevant, but we envision culturally-based mechanisms. For example, if individuals were simply allowed to communicate some reduced and perhaps errorful description of themselves to potential mates¹⁴ and similarities in these descriptions were used to bias mate selection, Winners would be much more likely to find one another. This observation is consistent with the critical role attributed to mate selection in the models of “niche construction” of modern theoretical biology [25].

A second construct assumes a more complex, “conditional” environment of several different, mutually exclusive regions, with individuals thrown at random into some one of these regions. The problem facing the species, then, is to allow specialization with respect to each of the conditions, despite the fact that an individual does not know “at birth” the environment in which it will find itself. Culture could play a critical role here in allowing adaptive *specialization* without the genetic *speciation* that irreversibly partitions the population. We imagine a cultural mechanism that helps to match the genetic abilities of a new offspring with the range of environmental conditions to be solved.

Another possible role we see for culture in symbiotic search is when the solution to be found requires the distinct abilities of more than one individual. For example, imagine that the “needle” being found by our current search had twice as many bits. Imagine further that we add only a single

¹⁴Something like a “Personals” column!

bit to the length of current individuals, with its semantics being whether the individual was “good at” the first half of the solution or the second. Finding the needle would now be much harder, requiring not only the construction of two individuals specialized at the “jobs” of the first and second halves of the needle but also that these two specialists be able to communicate at least enough to find one another and work together. This construction is admittedly contrived but it helps to illustrate yet another important role for culture: the solution of problems more complex than those solvable by individuals working alone.

In summary, we see several clear, functional motivations for what culture can add to an adaptive system using evolution and learning. Culture helps to expedite the search process by disseminating information about success more quickly than genetic mechanisms alone. Second, the extra-genetic informational channel provided by culture helps to keep the genetic description more plastic, a characteristic that can be critical as the species’ environment changes. Culture may also help compatible mates to find one another, to allow specialization to environmental niches despite indeterminism as to the niche in which an individual will find itself, and allow groups of individuals to collectively solve complex problems that would be beyond their isolated efforts. Our current simulations provide some evidence as to the efficacy of the first two of these, while only suggesting the last.

7.3 Adaptive algorithms

It is possible to view the interacting processes of evolution, learning, and culture as adaptive algorithms that search huge spaces (environments) for potential solutions. In this view, our criterion for success changes from accurate modeling to efficient computation.¹⁵ In this section we outline some of the basic features of evolution, learning, and culture as adaptive algorithms.

One obvious approach is to view evolution, learning, and culture as three distinct and competitive types of adaptive algorithm, looking for their relative advantages and disadvantages. It certainly appears, for example, that the *local search* performed by most gradient descent connectionist learning algorithms makes it appropriate for much different adaptive problems than the kind of *global sampling* performed by something like the GA.

Alternatively, we can view evolution, learning, and culture as components of a *single* adaptive system. Figure 19 presents a view of how the three systems might interact, as joints of a single movable arm that each help the ultimate “fingers” reach a desirable point; this is obviously an elaboration of the picture of Baldwin’s arm of figure 1. This image is meant as evocative metaphor, but it does help to make several observations. The first is the mutually constraining relationships among evolution, learning, and culture. Culture must be built upon the results of evolution, while individuals’ learn-

¹⁵In the background, of course, is the “Holy Grail” of cognitive science: correct models of naturally occurring phenomena and efficient artificial computations will turn out to be two faces of the same solution.

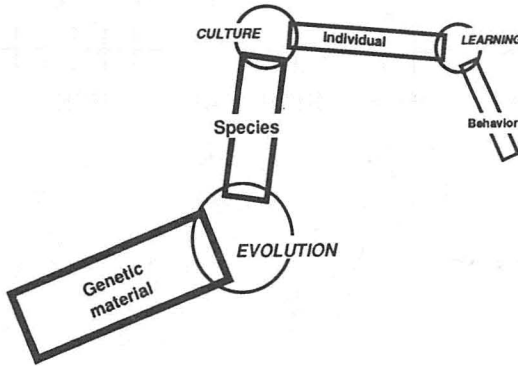


Figure 19: Robot arm.

ing depends both on the artifacts made available to them by culture and the genetic material given them by evolution. Second, this image helps to capture the way in which the plastic systems (“joints”) of evolution, culture and learning mediate between the more rigid constraints (“bones”) of extant genetic material, species, and artifacts. Finally, the relative size of the three limbs is meant to indicate their relative inertias. That is, the process of evolution is much slower and cumbersome than the process of cultural development, which in turn is slower than the process of individual learning.

It is this notion, that the three adaptive systems are each attempting to capture environmental regularities of different time scales, that is most persuasive. Referring to interactions among a slightly different set of adaptive subsystems, Simon has made a similar comment [27]:

What is invariant in adaptive systems will depend on the time intervals during which we observe them. There are at least three time scales of relevance to such systems, corresponding to three different forms of adaptation. On the shortest time scale intelligent — hence adaptive — systems continually change their behavior in the course of solving each problem situation they encounter. . . . On a somewhat longer time scale, intelligent systems make adaptations that are preserved and remain available for meeting new situations successfully. . . . On the longest time scale, intelligent systems evolve.

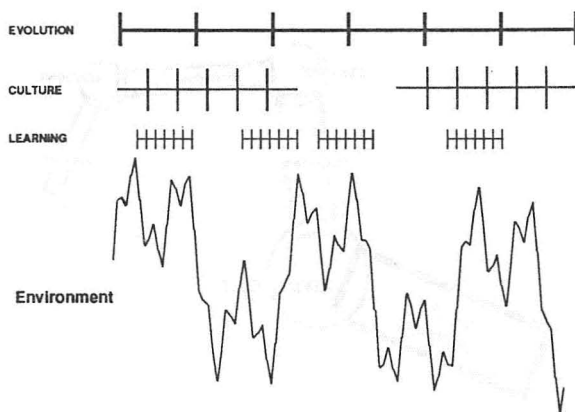


Figure 20: Environmental wave.

Thus, the relative inertia of each of these adaptive systems is appropriate to the time scale of the environmental variations it is tracking. In particular, the learning trials of individuals are *not* commensurate with the evolution of individuals, as Hinton and Nowlan seem to suggest (see section 2.3). Evolution is responsive to the most glacial of changes, culture to more rapid changes, and learning to those changes in the environment that can be observed within a lifetime. Of course there is nothing special about these three particular rates of environmental change except that we have identified adaptive systems associated with each. A whole continuum of rates of environmental change are constantly in operation. The resulting picture (see figure 20) is of the environment as a constantly changing waveform, with lower frequency components being tracked by evolution, intermediate frequencies by culture, and the highest frequencies being tracked by learning.

Notice also that the “granularity” of the representations used by each adaptive system is proportional to the time scale within which it operates. Genetics provides an alphabet of appropriate “building blocks” [19] for experimenting with phenotypic variations and measuring the gross fitness of the results, culture develops systems of artifacts that convey useful solutions across generations, and neural networks capture reliable correlations among perceptual and motoric phenomena experienced by an individual. This last, most high-resolution representation of an individual’s experience with its environment is subjected to enormous “data reduction” as it enters into the slower adaptive systems of culture (e.g., if the individual writes an autobiography) or evolution (e.g., perturbs the gene pool frequencies). Conversely,

the granularity of the environmental space experienced by individuals is much smaller than the cumulative experience of entire societies, which is in turn much smaller than the cumulative experience of entire species. In each case, the adaptive system depends on a *repository* for its accumulated experience that is appropriate to the time scale of its regularities and the mechanisms available for modifying this representation.

These are only a sample of the large set of important issues that arise when we consider any one adaptive system (e.g., learning, culture, evolution) as components of a single, grand adaptive system. We hope to investigate these in terms of the time scales in which they find invariants, the information structures used to represent regularities, and the way regularities discovered by one adaptive system serve to constrain the search of the other systems. Another intriguing possibility is that the environmental regularities are “self-similar” across time scales, i.e., *fractal*. If true, this would allow *self-similar adaptive* techniques to be exploited at several levels within the cognitive system. An adaptive mechanism that worked at evolutionary time scales might actually be useful at the level of seconds, just because the regularities they are respectively tracking have self-similar structure selections. For example, so-called “selectionist” generalizations of biological evolution of species have also been used to account for the creativity of individuals [8] and the development of cultural processes like science [7,20]. The argument is that while the “substrates” on which evolution, creativity, and science work are of course wildly different, approximately the same selectionist adaptive algorithm is effective in all these cases. Certainly this must be considered only speculation at the moment, but if true it would help to explain the fact that the apparently disparate phenomena of learning, evolution, and culture are all of central concern to modern cognitive science.

Acknowledgments

This investigation began with a catalytic afternoon’s conversation with Chris Langton and Ed Hutchins, and I benefited from a number of subsequent conversations with Ed. I would also like to thank Janòs Komlóš for his help in the analysis of the Hinton and Nowlan model, and Craig Mautner and Don Norman for careful readings of earlier drafts.

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