Computational Explorations of the Baldwin Effect

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1 Abstract

The Baldwin Effect [2] (B.E.) is a very plausible, but unproven, biological theory concerning the power of learning to accelerate evolution. Simple computational models in the 1980's gave the first *constructive proof* of its potential existence, and subsequent work in evolutionary computation has shown the practical, computational, advantages of hybrid evolution-learning systems. However, the basic theory, particularly it's second phase (involving genetic assimilation of acquired characteristics) is difficult to reconcile in systems controlled by neural networks. This paper gives a brief overview of early B.E. research, describes one of our earlier projects involving B.E. and trilaterally adaptive systems, and sketches our current focus on the investigation of B.E. in trilaterally-adaptive neural networks.

2 The Effects of Learning upon Evolution

One of the first proposals that learning could accelerate evolution was Jean-Baptiste Lamarck's (1744-1829) *inheritance of acquired characteristics*, wherein physical and mental changes incurred during one's lifetime could be passed on directly to offspring. Contemporary knowledge of the germ-soma distinction permits a recasting of Lamarckism in modern Neo-Darwinian terms, as depicted in Figure 1. Thus, the theory entails a reverse transcription of the modified phenotype back into the genotype, a process that is fully realizable and often useful in evolutionary algorithms, but biologically unrealistic except in a few rare cases.

In 1896, James Baldwin postulated an indirect mechanism for the eventual inheritance of acquired characteristics [2]. This *Baldwin Effect* involves two stages. In phase I (Figure 2), assume a set of genotypes spread uniformly about a suboptimal region, D1, of the fitness landscape. If phenotypes have plasticity, then each can essentially perform local search in the fitness landscape (as shown by the circles with horizontal arrows), and a rough estimate of phenotypic fitness will be the time-averaged landscape locations of the phenotype. Clearly, those phenotypes lying near the base of the optimal peak will have better opportunities to learn their way to higher fitness. Hence, they will have a selective advantage, and the population distribution will move from D1 to D2. Basically, learning smoothes the

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Figure 1: Neo-Darwinian Interpretation of Lamarckian Inheritance of Acquired Characteristics: During their lifetimes, phenotypes (circles) can improve and thus climb the fitness landscape away from their innate position and toward the optimal phenotype, P^{*}. Any phenotypic improvements are then reverse transcribed into the genome (changing from filled to open square) prior to reproduction.

fitness landscape and enhances selective pressure such that the population moves toward the optimal phenotype, denoted by P^* on the phenotype axis.

To move, and not merely redistribute, the genotype pool, evolution relies on genetic operators (mutation, crossover, inversion, etc.). If the genotype and phenotype space are well correlated [12], then genetics can initiate the emergence of innately optimal phenotypes, *natural born* P^*s , and, in general, lead to a flattening of distribution D2 into D3 (Figure 3). Additionally, if learning has a cost, as it normally does [12], then the P^* learners will pay it but the natural-born P*s will not, thus giving the latter a selective advantage and moving the population distribution from D3 to D4, where the learned phenotype, P*, becomes fully innate.

Thus, in the Baldwin Effect, learning accelerates evolution; and then, if the fitness landscape is static, evolution obviates learning via genetic assimilation.

3 Simulating the Baldwin Effect

Since the Baldwin Effect involves both learning and evolution, it is extremely hard to test in controlled biological settings. Although some lab animals (typically insects) can be bred quickly, they exhibit much less learning than vertebrates and mammals, which, in turn, have longer maturation and reproductive cycles. It is no wonder that little progress had been made on the phenomena since Baldwin's original paper in the late 19th century.

However, in the mid 1980's, Hinton and Nowlan performed a diabolically simple (yet elegant) set of simulations to illustrate the essence of the Baldwin Effect [7]. They used a standard genetic algorithm to evolve ternary vectors, with each gene have a value of 0, 1 or * (star, the wildcard). The ultimate goal of GA search was to find a particular binary string (of 0's and 1's but no wildcards). Since there was just ONE particular goal string, and since no partial-credit was given for evolved



Figure 2: The Baldwin Effect Phase I: All phenotypes have the ability to learn, but only those near the base of the peak can achieve fitness increases over the innate value. This selective advantage moves the genotype/phenotype distribution from D1 to D2, hence closer to the optimal phenotype, P*. As depicted by the dotted curve, learning effectively smoothes the fitness landscape.

bit strings that were close, but not exactly correct, this was termed a *needle-in-the-haystack* problem, upon which evolution achieves no better performance than random search.

However, their model incorporated learning via the wildcard genes. Essentially, any gene that was 0 or 1 was *fixed*. If any such gene did not match the goal string, then fitness was zero, regardless of the number of *correct* genes. But a wildcard gene opened the possibility for local search within the subspace defined by the fixed genes, where this search simply involved random guesses of 0's and 1's for the wildcard spots. If, in the course of k out of a maximum M random guesses (of values for each of the wildcards), the target string was found, then the original genome received a non-zero fitness, that declined from the maximum fitness as $\frac{k}{M}$ increased. In general, a genome that was close to the target (and did not include any incorrect fixed genes) could be randomly adjusted in less than M attempts to achieve the target, and thus would receive some partial credit. Thus, this guessing (a very primitive analog of learning) provided a selective advantage to genomes that were within striking distance of the target string (and without incorrect fixed genes), and the population gradually moved toward the target. Wildcard genes were an important part of the early phases of evolutionary search, and their cardinality gave a quantitative measure of the amount of learning.

The Hinton and Nowlan experiments showed a clear increase in wildcards (i.e. learning) during the early phases of evolution, thus mimicking phase I of the Baldwin Effect. However, since learning included a cost (due to the inverse effect of $\frac{k}{M}$ upon fitness), there was selective pressure to replace wildcards with (correct) fixed bits, which, in turn, led to a decrease in the average number of wildcards in the population; but all the while, average fitness kept increasing, illustrating phase II of the Baldwin Effect.

This classic simulation showed that early learning helped guide evolution toward



Figure 3: The Baldwin Effect Phase II: Genetic operations spread the population distribution from D2 to D3, producing individuals with hard-wired optimal phenotypes, P^{*}. Due to the cost of learning, these *natural-born* P^{*s} have a selective advantage over the *learned* P^{*s} , and the distribution moves from D3 to D4.

a difficult goal, but as the population approached the target, the flexible portions of the genotype/phenotype became hard-wired to the correct values, thus jettisoning the (costly) learning capabilities.

4 Development and the Baldwin Effect

There are two critical preconditions to phase II of the Baldwin Effect: a) learning must have a cost (in terms of delayed maturation and reproduction, vulnerable periods of juvenile ignorance, or many others outlined in [12]), and b) there is a strong correlation between genotype and phenotype space [12]. Without this correlation, the changes accomplished by phenotypic plasticity have little chance of eventually becoming encoded in the genome via chance genetic operations; or, if they do, they may happen to random individuals as opposed to those *poised and waiting* nearby in genotype space.

Unfortunately, nature promises no such correlation, particularly when the phenotype space encompasses mental activities. The transition from genome to fully-functioning brain is extremely intricate, even in very simple animals. In short, the developmental process that governs the transition from genome to embryo to juvenile poses serious, possibly insurmountable, problems for the Baldwin Effect. Development converts a linear string of DNA into a 3-dimensional organism by a fascinating process, but one that severely confounds the genotype-phenotype mapping.

However, correlations do exist between the spatial locations of a) homeobox genes on the chromosomes of all animals from drosophila to humans, and b) body parts (such as an insect's thoracic and abdominal segments) [17]. In fact, some homeobox gene locations even correlate with regions of the hindbrain, which is the evolutionarily most primitive brain region. However, each neural module consists of thousands or millions of neurons whose heterogeneous activation patterns determine the animal's overt behavior. No single pattern or group of similar patterns, hence no single behavior, maps directly to a gene. Hence, learned behaviors have no obvious possibilities for assimilation into the genome.

To illustrate the absurdity of any strong cognitive Baldwin Effect, consider the human brain, which consists of roughly 10^{11} neurons and 10^{14} synapses, the properties of which are largely believed to realize the knowledge contents of any brain. This knowledge would be difficult to directly encode in the human genome, which consists of a mere 30-40 thousand genes.

Despite this major impediment, mental Baldwinism could be rescued via an acceptable refinement: although learning makes very specific behavioral changes, there could be corresponding genetic changes for that same *general type* of behavior. Thus, while a chess player learns specific opening moves, general board formations and effective move sequences, these are manifest in synaptic changes to localized parts of brain regions such as the basal ganglia. Then, the coarse genetic correlate might be the modification of a few genes whose phenotypic consequences are slight changes in postnatal concentrations of particular neurotransmitters and neuroreceptors across the *entire* basal ganglia. Thus, the child may have innate talent for activities requiring good sequence memory, although no immediate provess at chess. Similarly, the words of human language have no direct genetic correlates, but the ability to acquire language may have genetic components [14]. In fact, Pinker and Bloom claim that the Baldwin Effect is instrumental in the emergence of both language and Chomsky's proposed language acquisition devices [4]. Interestingly enough, simple ALife experiments reveal an evolving predisposition toward language ([13, 3]).

Unlike Lamarckianism [11], Baldwinism remains a viable biological possibility. However, regardless of biological blessing or condemnation, both Lamarckianism and Baldwinism are useful tools for evolutionary computation [9, 1, 5], since they supplement evolution with learning to improve overall search. Furthermore, evolutionary algorithms have increasingly exploited *indirect* genomic representations, which are converted to phenotypes by an (often complex) developmental process. These are particularly useful for difficult search problems such as circuit design [10]. So, even if biological research eventually shows that development so dramatically corrupts the genotype-phenotype mapping as to soundly dismiss the Baldwin Effect, there will remain a need to understand the interactions between development and the Baldwin Effect in *artificial* adaptive systems.

5 The TRIDAP System

To synthetically explore the effects of development on the Baldwin Effect, one needs a system with three adaptive components: evolution, learning and development (where the latter may not be adaptive in silico though it is in nature). Summarized in Figure 4, the Trilaterally Adaptive System (TRIDAP) [6] combines the standard genetic algorithm (GA) [8] with simple developmental and learning procedures.

Each GA chromosome encodes both the rules for a Turing Machine (TM) and an initial tape. Both the TM and tape have sizes and contents determined by evolution, with these sizes encoded in the genome as well. So the initial tape may be innately long and complex, or short and simple. Similarly, the TM may contain many rules or few. The goal is to evolve a good combination of initial tape and developmental recipe such that the fully-developed tape matches a target bit string, with or without

the help of learning.

Development in TRIDAP consists of running the TM on the initial tape, which often results in significant changes (e.g. overwrites and/or insertions of symbols). The developed tape then plays the same role as a genotype in Hinton and Nowlan's system: it consists of 0's, 1's and wildcards, where the latter serve as learning (i.e. guessing) sites. As described in [6], the fitness measures used in TRIDAP are similar to those used by Hinton and Nowlan, with special modifications to speed up the evaluation of long strings.



Figure 4: Overview of the TRIDAP system, which combines evolution, development, and learning in the abstract problem domain of binary-string search.

Blueprints versus Recipes in TRIDAP

As discussed earlier, development confounds the Baldwin Effect by greatly complicating the genotype-phenotype mapping, such that a learned change to the phenotype becomes nearly impossible to reverse engineer into the genotype. So phase II of the Baldwin Effect, genetic assimilation, can suffer under developmentally-dominated mappings. However, oddly enough, this does not preclude development from assisting a learning-driven evolutionary process. In fact, in many of our simulations [6], development is a necessary condition for both evolutionary progress and the Baldwin Effect. Many of these simulations exhibit a clear interaction between two strategies: *blueprint* and *recipe*. The former involves a lengthy initial tape and very little TM development, so the genome directly encodes the phenotype. Conversely, the recipe strategy employs development to grow the phenotype from a very short initial tape. Both strategies can exploit learning equally, since both can produce phenotypes with many wildcards.

To rephrase the problem of developmental Baldwinism in these terms: a recipe strategy has difficulty encoding learned patterns back into the recipe itself. A blueprint strategy has no such difficulty, since the phenotype and the initial tape of the genotype correlate so well. For blueprints, phase II of the Baldwin Effect involves simply replacing some wildcards with the appropriate 0's and 1's. This evolutionary instantiation process can continue until the complete target pattern is encoded in the initial tape. Learning simply buys evolutionary time for the genome, while it gradually instantiates the wildcards. But while wildcards are relatively safe, wrong instantiations are fatal, given the fitness functions above. So instantiation proceeds very slowly, with many failed attempts going extinct.

Blueprints and recipes compete for dominance of the genome, with the amount of repetition in the target patterns often brokering the tradeoffs between winner-takeall and cooperative outcomes. While simple repetitive patterns are more easily found by developmental recipes, a small increase in sub-pattern complexity opens the door for blueprinting. Within a particular target-pattern-size category (e.g. 20-cells, 40 cells, etc.) all patterns are equally easy/difficult to generate via blueprinting. So as subpattern complexity increases in repetitive targets, blueprinting takes over only because development has greater difficulty designing string generators. It still finds them, but at a slower rate than does blueprinting, so natural selection favors the latter.

Perhaps the most interesting results are those of the random 40-cell patterns, where neither strategy has any clear competitive advantages. However, development gives evolution a small, but very significant, start, and cooperation with blueprinting then finds good results, with respect to the fitness function and the high number of learning trials. Basically, development provides scaffolding for the progressive expansion of a blueprint. Without these simple repetitive recipes, evolution simply cannot guess enough correct bits.

For example, consider a random 40-cell pattern:

$11001001101000101110110100000011000010 \tag{1}$

Intuitively, neither blueprinting nor development should have a chance of finding this pattern. However, the combination can often get close. As shown in Figure 5, the early solutions convert a simple initial tape into a large wildcard string. Although far from the target, these have a non-zero probability of learning the target, so the fitness function f_{phn} gives them a small positive value - just enough to bias evolution in the proper direction. Then, over the course of a few hundred generations, the initial tape fills up with non-wildcard cells while maintaining at least one wildcard, which serves as the seed for the TM to grow the central wildcard region. These, along with many of the other simulations from [6] show a clear Baldwinian progression as learning initially aids search but then gradually abates as more bits become assimilated. Development thus helps get genotypes in the ballpark, where the Baldwin Effect can then take over. The final solutions are then a combination of many hard-wired bits (i.e. partial blueprints) and developmental recipes, for producing a segment of wildcards of sufficiently short length to enable learning to complete the search for the needle-in-the-haystack random pattern. The combination of development and blueprinting thus facilitates a complete Baldwin Effect in domains that are either too large for blueprinting alone or too intricate for development alone.



Figure 5: A sequence of best-of-generation phenotypes from ascending, but non-contiguous generations for the 40-cell random target 1100100110100010111011010100000011000010.Wildcards are represented by \star , and arrows denote a developmental process from the initial TM tape on the left to the phenotypic tape on the right.

Since living organisms are complex in terms of both component cardinality and intricacy, it seems fair to generalize from this example and speculate that development in the biological world could indeed enhance the Baldwin Effect via a cooperative arrangement between genes that control general wide-scale properties of embryogenesis and those that have a more direct link to spatially localized phenotypic traits. In evolutionary computation, this cooperation might also be exploitable in problem domains where a) solutions are complex but house intermittent structure, and b) a hybrid recipe-blueprint genome is feasible, with evolution governing their relative contributions.

6 Development and the Baldwin Effect in Neural Networks

Hinton and Nowlan [7] draw the rough analogy between their evolved bit strings and neural-network connection patterns, and thus we, by association, could claim that TRIDAP is evolving neural-net-like structures. But anyone even vaguely familiar with the field recognizes the huge gap between this abstract problem domain and that of designing fully-functional, learning, neural networks.

Furthermore, our TRIDAP work gives no indication that genetic assimilation (i.e. phase II of the Baldwin Effect) can occur *back across* a developmental scheme, since development **can** so severely decorrelate the genotype-phenotype mapping. Still, we have no proof that it **always** scrambles the mapping. As mentioned earlier,

there are numerous correlations between homeobox genes and the longitudinal axis of vertebrate bodies **and brains**.

So to further explore the issues of development and the Baldwin Effect with respect to cognitive evolution, we must move beyond simple bit-string phenotypes and experiment with more brain-like representations, such as neural networks.

Figure 6 portrays the most common characterizations of development and learning in neural networks, based both on contemporary neuroscientific evidence and practical issues of artificial neural network deployment. Here, development involves the generation and linking of neurons, while learning consists solely of the fine-tuning of synaptic strengths; and the two processes occur in lock step, with development ending before learning begins. Furthermore, development is largely controlled by the innate genome (a.k.a nature), while the environment (a.k.a nurture) governs learning.



Figure 6: Illustration of the basic lock-step model of development and learning in neural networks, where neurogenesis and connectivity constitute development, while synaptic tuning embodies learning. The genome and the environment have monotonically decreasing and increasing roles, respectively (depicted by arrow size), during maturation.

This model causes significant problems for the Baldwin Effect. After all, how do the results of learning (i.e. local synaptic change) become assimilated into the genome, which primarily controls neurogenesis and connectivity - both at a relatively coarse level?

This lock-step framework does admit one opening for the reverse engineering of synaptic change into the genome. As shown in Figure 7, if some of the synaptic strengths between neurons in regions A and B are modified during learning, this can be roughly approximated by genetically-controlled connectivity changes in later generations. With more (or less) connections between A and B in the future, there are more (or less) opportunities for synaptic enhancement via Hebbian learning. Since standard Hebbian learning (via long-term potentiation) relies on correlated firing (between neurons in regions A and B in this case), there is a greater chance of such correlation (and thus a promising starting-point for synaptic strengthening) when more synapses (of even weak efficacy) link areas A and B.



Figure 7: Simple illustration of the reverse engineering of a synaptic change (during learning) into a similar (though certainly not identical) developmental process, which should facilitate a similar learning change, though possibly requiring less time and environmental influence.

Another interesting angle on development and the Baldwin Effect stems from evidence that the lock-step model, though correct to a rough approximation, neglects new neurobiological evidence of temporal overlap between neurogenesis, synaptogenesis, and synaptic tuning.

For example, many studies (summarized in [15]) find high levels of long-term potentiation (LTP) and long-term depression (LTD) - both forms of synaptic tuning - during development. In fact, the rates of LTP and LTD (i.e. learning rates) are actually very high during development and much lower during adult life.

In addition, recent work by Shors [16] reveals that a) neurogenesis occurs throughout life, particularly in the dentate gyrus (DG) of the hippocampus, but b) those neurons only hook up to other neurons (and ultimately survive) if the organism subsequently performs cognitively-challenging tasks.

This new evidence motivates a reinterpretation of the Baldwin Effect in neural networks. Instead of viewing the second phase as one of converting synaptic-strength changes (i.e. classic learning) into genomic codes for controlling neurogenesis and synaptogenesis (i.e. classic development) - which represents the reverse encoding of the results of one process into two dramatically different processes - we have an alternative that involves a quantitative, rather than a qualitative, conversion.

To wit, the second (assimilation) phase of the Baldwin Effect may only involve a change in the *rates* of neurogenesis, synaptogenesis and LTP/LTD across an organism's life stages, as shown in Figure 8. Under the view that adaptive changes in later life are predominantly governed by the environment, not the genome, a Baldwinian modification could simply be to move more of that adaptive change into earlier life stages, where genomic control may dominate. For example, when the biochemical bases for LTP and LTD arose in evolution, both processes may have been very active throughout life, requiring constant environmental signaling to tune neural circuitry. However, over many (thousands of) generations, genomic changes could have arisen such that the early stages of development utilized neurogenesis, synaptogenesis and high LTP/LTD to form much of this circuitry with a minimum of environmental influence. Similarly, the rates of neurogenesis and synaptogenesis could have originally been much less variable throughout life, but evolution has gradually found genomes coding for an acceleration of these processes in early development; and thus, more of these activities became governed by genomic rather than environmental factors.



Figure 8: An alternate phase II of the Baldwin Effect in which the *rates* of neurogenesis, synaptogenesis and LTP/LTD (learning) change over the course of evolution such that each process is more active earlier in life. However, within a life stage, the differences between the three processes fit the traditional view: neuro- and synaptogenesis dominate early life, while learning accounts for most neural change in adults.

We are currently exploring models of evolving neural networks in which the classic developmental and learning processes are interleaved, with the genome controlling the rates of each throughout the different life stages. We hope that this will provide further insights into the neurocognitive aspects of the Baldwin Effect.

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