Dominance and Development

ABSTRACT

Dominance is the preferential expression of certain gene alleles at heterogeneous diploid loci during development. We employ genetic algorithm simulations over binary diploid individuals to observe average fitness and genetic diversity of a population under different models of dominance. We vary the extent to which dominant alleles are aligned with those that optimize fitness. Well-aligned dominance enhances genetic diversity by allowing recessive alleles to persist without negative impact on average fitness. We consider a simple adaptive dominance model as a means of automatically generating well-aligned dominance. Finally, we add developmental learning to the acquisition of a dominance pattern and compare its impact to that predicted by the Baldwin Effect.

Keywords
dominance, development, Baldwin Effect, learning

1. INTRODUCTION

Dominance has been a notion in the theory of evolution since first being identified by Mendel in his classic study of pea populations 140 years ago [13]. Mendel noted that almost all phenotypes exhibited a single value for a pure hybrid trait, yet the hidden trait reappeared in about one-fourth of offspring of those hybrids. Mendel proposed this was due to the non-uniform expression of gene alleles at a heterogeneous locus, reflecting dominance of the preferred allele over other possible alleles.

We define dominance to be the preferential expression of certain gene alleles at heterogeneous diploid loci during phenotype development. The preferred alleles are referred to as dominant alleles and the others as recessive alleles. The expected or observed role of dominance in evolution has sometimes been used as its definition. Dominance has been defined to be the masking at the level of the phenotype of recessive alleles by dominant alleles [4]. While fairly neutral with respect to the role of dominance, this definition still assigns an action to dominance, i.e., the masking of certain alleles during phenotype development. Dominance also has been defined as a “form of phenotypic robustness to mutation” [3]. This clearly is not a definition from first principles [17], but instead reflects a prescribed role for dominance in evolution.

One approach to the study of dominance would be to consider natural populations and observe impacts related to dominance. However, it is difficult to compare situations with and without active dominance over time in nature. We currently don't have the means to turn dominance on and off and observe the resultant differences in population dynamics. The masking and robustness definitions of dominance given above reflect this limitation, being essentially intragenerational effects of dominance. Artificial evolution, on the other hand, provides us with methodologies for manipulating and controlling processes active in evolution. Artificial evolution studies evolution in an abstract setting, eliminating many of the complexities of environments, individuals, and genetic representations in the hope of uncovering impacts of various processes on population dynamics. It sits between the fields of biological evolution and applied evolutionary computation with its results having implications for both. Research in artificial evolution adopts a theoretical or experimental approach. Theoretical approaches view evolution as search through abstract mathematical landscapes defined as fitness functions and establish theorems regarding how search proceeds in these differing landscapes. Experimental approaches to artificial evolution select particular representations for genotypes, forms of fitness functions, and values for probabilistic parameters and generate instances of population dynamics through simulation.

We investigate the impacts of dominance on the evolution of diploid individuals using simulations based in genetic algorithms. We observe different effects of dominance depending upon how well it is aligned with an environment, i.e., the extent to which dominant alleles optimize fitness. We then consider how well-aligned dominance can arise in evolution. We define a simple model of dominance acquisition that is consistent with the view that information regarding the control of gene expression is transmitted along with gene alleles during reproduction. Finally, we consider a model of dominance acquisition that includes an element of developmental learning.

1.1 A Model of Diploid Evolution

Genetic information about an individual is contained in one or more genotypes. A genotype is a sequence of gene loci, each gene having a value chosen from a set of possible
values called alleles. In our abstract model, each gene locus of a binary genotype takes on one of two possible alleles, selected from the set \{0, 1\}. A diploid binary individual maintains two binary genotypes, one inherited from each of its two parents in the previous generation. This can be contrasted with a haploid binary individual, which holds only a single binary genotype inherited from a single parent or as a recombination of genotypes from two parents in the previous generation. From two binary genotypes, a diploid binary individual develops a binary phenotype, represented as a sequence of \{0, 1\} values.

We simulate the processes of evolution by a generational genetic algorithm, defined as follows: [6] [14]:

Abstract Diploid Evolution(pSize, gSize, f, m)
{p = GenerateInitialPopulation(pSize, gSize);
  Develop(p);
  Evaluate(p, f);
  until(done())
    {p = GenerateNextPopulation(p, m);
     Develop(p);
     Evaluate(p, f);
    }
}

A current population \(p\) consists of a set of binary diploid individuals. We use a fixed-size population model; each successive generation of \(p\) will have the same number \(p\)Size of individuals. The process GenerateInitialPopulation(pSize, genSize) creates a set of binary diploid individuals of size \(p\)Size, each individual consisting of two genotypes containing \(g\)Size alleles; each allele value is selected with equal probability from the set \{0, 1\}. The function done() returns true if a desired halting condition is met, e.g., some number of generations have been generated or some number of successive generations of the population do not differ significantly.

Each of the capitalized processes can be implemented in a number of different ways, which differences serve as the bases for experiments reported here. Our experiments will focus on differences in the development process. The process Develop(p) acts on each individual in population \(p\), creating a phenotype from its two genotypes, perhaps in response to interaction with an environment. In our model, a phenotype is the same size as the genotypes and represents which of the two possible alleles at each gene locus is expressed in the developed individual. Under UniformDevelopment, for each diploid gene location at which two different alleles occur (i.e., a heterozygote), one of the two alleles is chosen with equal probability to be expressed; if the two alleles at a location are the same (i.e., a homozygote), that value is chosen.

Under DominanceDevelopment, each individual maintains a dominance pattern to be used during development. In our model of dominance, a dominance pattern is the same length as the two genotypes with each location having three possible values \{0, 1, \ast\}. During phenotype development, if the dominance pattern location has value 0 or 1, then that value is chosen for the phenotype if that allele is present at that location in at least one of the two genotypes, otherwise the other value must be chosen. If the value at a location of the dominance pattern is \ast, meaning no dominant allele has been chosen, then an allele from one of the two genotypes is chosen at random, as in UniformDevelopment.

We represent an environment by a binary fitness function, which is an arbitrary, functional mapping from a binary phenotype to a real value in the range 0.0 to 1.0. The value of a fitness function when applied to an individual’s phenotype indicates the individual’s degree of adaptation to the environment, i.e., the higher the value the better adapted is the individual. The process Evaluate(p, f) applies a binary fitness function \(f\) to the phenotype of each individual in population \(p\) and associates the resulting fitness value with the individual.

The process GenerateNextPopulation(p, m) creates a new generation of population \(p\), as follows:

GenerateNextPopulation(p, m)
{parents = SelectParents(p);
  p = Recombine(parents);
  return Mutate(p, m);
}

The process SelectParents(p) is where notions of natural selection and selective pressure are captured. It selects pairs of parents whose offspring will form the next generation. We use FitPropSelection whereby an individual \(i\) is selected to be a parent with probability \(f_i / F_p\), where \(f_i\) is the fitness value of individual \(i\) and \(F_p\) is the sum of fitness values of all individuals in population \(p\). This selection process has been called fitness proportionate selection [14]. The process Recombine(parents), considers each pair of parents and creates a new diploid individual having two genotypes, one inherited from each parent as a result of recombining each parent’s two genotypes. We use UniformRecombination(parents) whereby one of the alleles from the two genotypes of a parent is selected with equal probability at each location, as is done when forming the phenotype by UniformDevelopment. This recombination method has been called uniform crossover [6]. Finally, the two genotypes of each individual in the new population may be altered by the process of mutation, implemented as Mutate(p). Each gene of each genotype of each individual in new population \(p\) is changed to the other allele value according to the given, per locus mutation probability \(m\).

1.2 Simulation Measures and Parameters

To observe the dynamics generated by simulations of our models of evolution, we consider three measures of a population at each generation. The first measure corresponds to a population’s level of adaptation to an environment and is its average fitness, being the average of the individual fitness values. The other two measures assess the genetic diversity of a population. One is the percent homozygotes of a population, evaluated across all genotype loci of all individuals in the population. A homozygote is the occurrence of equal-valued alleles at a given locus in the two genotypes of a diploid individual. We expect the percent homozygotes to be 0.50 in an initial population of random binary individuals. The other diversity measure is percent polymorphic, defined as the percentage of gene loci with less than 99 percent of their alleles being a single value across a given population [12]. We expect percent polymorphic to be 1.0 in a random, initial population.

Our experiments use a parameterized family of fitness functions, Step(stepSize). Each function compares a given phenotype to a random phenotype called the point. With a stepSize of \(k \geq 1\), \(k\) consecutive locations must equal the corresponding point locations to get credit for a match of
a step; there are $\text{genSize} / k$ non-overlapping steps. The fitness function returns the percentage of steps that match. The Step functions define fitness landscapes having a single peak (i.e., the point) with plateaus determined by the steps. Increasing the stepSize adds difficulty, as the evolutionary process must accumulate all of a stepSize group of alleles before an individual’s fitness improves above a current plateau. The stepSize corresponds to a degree of epistatic interaction between (successive) gene loci [9].

There are several factors that we do not change throughout the simulations. We employ a fixed-size population and set the population size to be 1000 for all experiments. This is a relatively small, finite population that makes the simulation experiments feasible, but that is large enough so as not to be dominated by small population effects, such as genetic drift [7]. Another factor not varied is genotype size, i.e., number of gene loci in a genotype. We have set the genotype size to be 100, again a compromise between computational efficiency and genetic diversity. Both population size and genotype size, while not at levels comparable to natural populations, are greater than what can be easily analyzed theoretically.

We consider two mutation probabilities in our study. The first is a probability of 0.0, which forces any improvement in fitness to come solely from the genetic diversity already resident in the population. For the second, we set the mutation probability to be such that an average of 10 alleles over the whole population are modified by mutation for each generation, yielding a per locus mutation probability of $5 \times 10^{-5}$ in our diploid setting with the population and genotype sizes we have chosen. This is near the mutation rates observed in nature [12]. Simulations were run for 2000 generations; each simulation experiment is repeated 20 times to generate average values and standard deviations. We gather values for the three measures defined above every 50 generations.

2. EXPERIMENTS AND RESULTS

2.1 Dominance Alignment

The extent to which a dominance pattern matches the genes expressed in an optimal phenotype for a given fitness function corresponds to the degree of dominance alignment with the environment. Our first experiment investigates the impacts that differing degrees of dominance alignment have upon our observed population measures during evolution from an initial, random population. This experiment is easy to perform given our Step fitness function with its point that corresponds to an optimal phenotype. We consider conditions where the dominance pattern is the same as the point of the Step function at every location, differs (i.e., is the opposite allele) at half of the locations, or differs at every location. We call these experimental conditions the EQDom100, EQDom50, and EQDom00 models, respectively. We compare the dynamics generated under these three models with the corresponding population measures generated by UniformDevelopment, i.e., without active dominance. Results are presented in Figures 1 and 2, for a stepSize of 4. Legends for figures throughout the paper are of the form model.stepSize, with 0 in between for the no mutation case, which is always plotted in bold.

The expected average fitness of a random population differs with degree of dominance alignment. In a population that meets the Hardy-Weinberg equilibrium condition [7], at each gene locus, 1/4 of the individuals have a homozygote matching that of the point, 1/4 have a homozygote that is not matching, and 1/2 have a heterozygote. Therefore, under UniformDevelopment, the probability that a locus of the point is matched by the phenotype of an individual is equal to 0.50, i.e., 1/4 matching homozygotes plus 1/2 of the 1/2 that are heterozygotes. Under the EQDom100 model, the probability is 0.75, i.e., 1/4 matching homozygotes plus the 1/2 that are heterozygotes. Under EQDom00, the probability is 0.25, being only the matching homozygotes. Under the EQDom50 model, the probability is the same as under the UniformDevelopment model. For a given stepSize of s, the probability that a step is matched by a random individual is equal to the probability of locus matching raised to the s power.

For a stepSize of 4, evolution with UniformDevelopment is able to locate and maintain a population with near optimal average fitness. Such an environment is said to be conquerable by a population of the given size. EQDom100 also is able to establish and maintain a near optimally fit population. After generation 1000, when average fitness values for both cases are clearly stable, average fitness is 0.9945 for EQDom100 and 0.9895 for the Uniform model. When the dominance pattern is partially aligned or completely unaligned, as for EQDom50 and EQDom00, respectively, average fitness values rise more slowly. The values ultimately reached remain relatively high, however, approaching 0.75 for EQDom00 and above 0.90 for the EQDom50 case. For this stepSize, the presence of mutations makes little or no difference in average fitness, the curves being essentially identical.

The results in Figure 2 indicate that the Uniform model achieves high average fitness at the cost of genetic diversity. Without mutations, almost all genetic diversity is lost in less than 500 generations. Percent homozygote values follow the rapid increase of average fitness and stabilize near 0.994. Under the EQDom100 model, percent homozygotes stabilizes at a lower level of 0.966, a small but significant difference. The positive impact of aligned dominance on genetic diversity is more apparent when considering percent polymorphic. Without mutations, percent polymorphic under the Uniform model approaches 0.0 before 500 generations, while under the EQDom100 model it just begins to fall. With mutations, the Uniform model soon reaches lev-
The environment with stepSize of 4 is already conquerable by the Uniform model. When the dominance pattern is well-aligned, the primary impact of dominance is to maintain a significant pool of recessive gene alleles that persists in the presence of mutations. It does this by hiding recessive alleles from the selection process through development of phenotypes that reflect advantageous gene alleles whenever possible. As a result, development by well-aligned dominance has a Diversifying Effect, yielding the same average fitness but greater genetic diversity when compared to the Uniform model. When the dominance pattern is not well-aligned, dominance has a Blurring Effect, reducing average fitness and yielding greater genetic diversity when compared to Uniform; the evolutionary process is not able to focus on potentially more fit individuals in this case.

What happens when the environment is more difficult to conquer due to greater epistatic interaction with higher stepSize? We next consider a stepSize of 8. Only 1 out of 256 randomly selected allele patterns match a step with stepSize of 8, compared to 1 out of 16 for a stepSize of 4. Figures 3 and 4 present these results.

Only the EQDom100 model is able to approach a near optimal population for this environment with average fitness approaching 0.994. The Uniform model realizes average fitness values that stabilize near 0.88; this environment is not conquerable for a population of the given size. The EQDom100 model is unable to overcome its misaligned dominance pattern, only realizing average fitness of 0.20. The EQDom100 model has the highest level of percent homozygotes, stabilizing near 97.5, below its average fitness. With mutations, percent homozygotes under the Uniform model approaches that of EQDom100 by generation 2000, even though average fitness is significantly lower. The other two dominance models arrive at levels of homozygotes well above their average fitness levels, indicating an inability to adapt to the environment. Percent polymorphic levels for EQDom100 are significantly higher than those for the Uniform model; even though average fitness nears 0.99, over 40% of the gene loci remain polymorphic in the presence of mutations. Increased genetic diversity paired with higher average fitness levels indicate the Adaptive Effect of well-aligned dominance in unconquerable environments. Only the EQDom100 model yields higher levels of genetic diversity, which more reflect an inability to adapt to the environment.

Our results indicate a consistent Diversifying Effect for aligned dominance. Aligned dominance allows the development process to ignore most recessive alleles that are introduced by mutations. This confirms the role of aligned dominance cited earlier as a definition as a "form of phenotypic robustness to mutation". While mutations have a consistent, positive impact on genetic diversity, they have little or no significant impact on average fitness for the rate considered. Improvements in average fitness that are seen with increased stepSize suggest that aligned dominance has a significant Adaptive Effect in more complex environments. Dominance has its direct impact on evolution during phenotype development. When dominance is well-aligned, it improves average fitness of a population by selecting adaptive alleles. UniformDevelopment only selects an adaptive allele one-half of the time at each heterozygote. The question arises as to the effect of dominance on the underlying fitness level of the population. In other words, what is the average fitness of a population without the immediate positive impact of aligned dominance? We can investigate this question by considering a modified model that turns off dominance for purposes of creating phenotypes for average fitness calculation, while leaving it active for creating the population.
from which parents are selected. Figure 5 shows average fitness results for stepSizes of 4 and 8, where EqDomMod is the modified model defined above.

For a stepSize of 4, during early generations the impact of aligned dominance on average fitness is primarily due to the impact of favorable allele selection during development. Average fitness of the modified model significantly trails that of both the Uniform and EQDom100 models, reaching maximum (negative) differences of about 0.40. Aligned dominance significantly slows the rate of increase in average fitness of the underlying genetic makeup of a population. For an easily conquered environment of this stepSize, well-aligned dominance has a Blurring Effect on the evolution of the genetic makeup of a population, being a slower average fitness increase combined with greater genetic diversity. There is a sufficient likelihood of individuals having the needed alleles, which aligned dominance can then select during development; the resultant lack of evolutionary focus is corrected by the development process through the lens of well-aligned dominance. For a stepSize of 4, average fitness of the modified model stabilizes at less than 0.95, while the Uniform model reaches near optimal average fitness after less than 500 generations. The persistent 0.60 difference in average fitness is eliminated by the action of aligned dominance during development.

For a stepSize of 8, we see a different pattern. In this more difficult environmental context, the Uniform model is not able to realize near optimal fitness, producing a population with average fitness about 0.88. The modified model realizes average fitness values that are significantly higher than those produced by the Uniform model. Well-aligned dominance now has a Baldwin Effect, combining a higher rate of increase in average fitness with a greater loss of genetic diversity when compared to the Uniform model. With increasing stepSize, individuals having adaptive alleles for all locations of a given step are less likely to occur. Aligned dominance guarantees these alleles are expressed when they do occur, increasing the fitness of the corresponding phenotypes that are then more likely to be selected as parents. Aligned dominance in this case helps focus the evolutionary process on these relatively rare individuals, leading to increased average fitness and fixing of adaptive alleles, as indicated by the associated loss of genetic diversity. We will discuss the Baldwin Effect [1] further when we add developmental learning to an adaptive dominance model.

### 2.2 An Adaptive Dominance Model

We have seen the advantages of well-aligned dominance in evolution. How could a well-aligned dominance pattern arise automatically during evolution? The answer may be related to how near optimal average fitness values arise, namely, through the impact of natural selection acting on phenotypes of a population. We assume that each gene allele brings along with it some information in the genotype as to how that allele can be expressed. Without this information, the gene would not be active in determining the phenotype. As a first model, let us assume that the amount or quality of that developmental information depends on the alleles found in the genotypes of recent ancestors. This information will be represented as a dominance pattern that
hitchhikes along on the genotype and is used during development to select and express gene alleles and impact the resultant phenotype.

We now specify our Domin model of dominance, which is a simple, adaptive model for the acquisition and inheritance of a dominance pattern. We associate with every individual a dominance pattern called a dominator with values \( \{0, 1, *\} \) for each gene location, where the asterisk indicates no dominance preference at a location. We determine the value at a location of an individual’s dominator in terms of the two genotypes and two dominators it has inherited. We explain determination of a location’s value in two steps. First, combine the values at the given location from the inherited dominators to create an initial value, as follows:

If the two dominators agree on a \( \{0, 1\} \) value, then make that the initial value of the new dominator;

Otherwise, make * the initial value of the new dominator.

When creating an initial, random population, all initial values of a dominator are *. Next, we consider the impact of the alleles from the two genotypes of the new individual, determining the final dominator value at each location, as follows:

If the two alleles differ (a heterozygote), then make the initial value the final dominator value;

If the two alleles are the same (a homozygote) and the initial value is *, then make the common allele the final dominator value;

If the two alleles are the same (a homozygote) but differ from the initial value, then make * the final dominator value;

If the two alleles are the same (a homozygote) and are the same as the initial value, then make the initial value the final dominator value;

This Domin model reflects a theory of dominance by which each allele brings along an encoding of information that assists the development process in expressing that gene value. An asterisk dominator value at a location indicates that both alleles have received equivalent support for being expressed during development, while a specific 0 or 1 value indicates one of the alleles has received greater support and therefore has a preference for being expressed. We have simplified our model for this experiment, making the dominance preference absolute during development; the preferred allele will be expressed, if it is present in the genotype. The Domin model propagates not only gene alleles but also a dominator that is indicative of recent, ancestral development choices. The evolutionary advantage of past development choices is implicit in the fact that the selection process chose the current individual and its ancestors to be parents.

The Domin model is inspired by research in biology that suggests a significant amount of an individual’s genotype encodes information for controlling phenotype development based upon available gene alleles, e.g., [16]. The effect of this developmental information is clearly seen in tissue and organ differentiation throughout a body. Changes in this developmental information could lead to changes in phenotype characteristics even without changes in genes. A related notion regarding the flexibility/fixedness of phenotype development is that of canalization [18]. Canalization is the notion that developmental reactions can be adjusted so that they bring about a nearly same outcome regardless of limited variation in environmental conditions. This concept is supported by the observed constancy of the so-called wild type of a species across similar environments. Dominance can be seen as a form of canalization, where despite the occurrence of a recessive gene at a heterozygote location, the dominant gene is expressed. From this perspective, dominance is a means of narrowing the reaction range of development [16] at a particular gene location. While an asterisk dominator value leaves possible a broad range of development reactions (i.e., either allele can be expressed), a 0 or 1 value narrows the reaction range to the dominant allele, if present.

The results for the Domin model with stepSize of 4 (not shown) indicate that its evolutionary behavior is nearly identical to that of EQDom100. In fact, results regarding the fixing of dominator locations indicate that, after just 100 generations, over 99 percent of dominator locations are fixed as \( \{0, 1\} \) values and that the values that are fixed are aligned with the environment. For this conquerable environment, average fitness approaches optimal values, significant genetic diversity is maintained, and the dominator becomes well-aligned under the Domin model.

When we consider a stepSize of 8, we get a different picture, however, as shown in Figures 6 and 7. Here, average fitness rises quickly at first under the Domin model, but then plateaus at about 0.81. Average fitness values under the Uniform model rise more slowly but stabilize at about 0.88, significantly higher than those realized by the Domin model. The Domin model maintains a higher level of genetic diversity, with about half of the alleles remaining polymorphic, though average fitness is significantly higher. As noted earlier, the Uniform model realizes higher average fitness through loss of diversity. Data as to the fixation of dominator location values under the Domin model indicate that, as average fitness stabilizes, about 0.90 of the dominator locations are fixed and, of those that are fixed, about 0.90 are aligned with the environment. A lack of dominator fixation and the existence of misaligned dominator locations work together to reduce average fitness. Overall, the Domin model has a Blurring Effect for this stepSize.

2.3 Dominance and Developmental Learning

Baldwin [1] suggested that the ability to adapt during development, i.e., to exhibit some degree of phenotypic plasticity, impacts the evolution of a population even in the
absence of direct inheritance of the developmental adaptations. G.G. Simpson [16] introduced the notion of reaction ranges within which phenotypic characteristics develop from genetic information. Reaction ranges can become narrowed by various forces during evolution, resulting in loss of phenotypic plasticity. Whereas development may be responsive to environmental interaction initially, it subsequently may become fixed or canalized [18]. Dominance can be seen as a way of canalizing the impact of developmental learning. The expression of a gene allele that is initially dependent upon interaction with the environment becomes canalized when fixated in a dominator. A correspondence between dominance and developmental learning is suggested by experimental results for the EQDom100 model, which are reminiscent of results regarding the impacts of developmental learning as stepSize increases [5]. At a stepSize of 4, developmental learning slightly slowed the rate of average fitness gain in the underlying population, relying upon developmental learning to select adaptive alleles. At a stepSize of 8, however, developmental learning produced the predicted Baldwin Effect, being an increased rate in improvement of average fitness and in fixation of favorable alleles as homozygotes.

Most of the existing research in artificial evolution regarding the Baldwin Effect has focused on impacts of developmental learning to genetic content [2]. Given our model of dominance, we can investigate its possible impacts on development, as well. We consider a model called LearnedDomin, which modifies the basic Domin model, as follows. During development, we first apply DominanceDevelopment, as defined before. Then, for each heterozygote of the genotype that has an asterisk value in the dominator, we try the alternate allele. If this change improves fitness of the phenotype, the alternate allele is expressed in the phenotype; otherwise, we leave the originally selected allele in the phenotype. Note that if an allele is fixated in the dominator, it is not considered for change by learning, as it has been canalized already [18]. The dominators and genotypes that are passed to the next generation are not directly affected by developmental learning. Dominators and genotypes are impacted only by the effects of selection on the resultant phenotypes.

With a stepSize of 4, the Domin model already approaches the same performance of the EQDom100 model, as almost all dominator locations quickly become aligned. Adding developmental learning has little or no additional effect upon average fitness and genetic diversity in that environment. Figures 8 and 9 show results for stepSize of 8, plotted as differences between the Domin and LearnedDomin models minus the Uniform model, with LearnedDomin results shown in bold. Both dominance models have an initial positive impact on average fitness, with the LearnedDomin model doubling the impact of the Domin model. As generations pass, however, average fitness under the Domin model falls behind the Uniform model, as was noted in earlier results. The LearnedDomin model, on the other hand, maintains a positive difference in average fitness throughout. The LearnedDomin model results in a persistent increase in percent homozygotes and a long-term reduction in per-
cent polymorphic for the context without mutations. This combination of impacts corresponds to a Baldwin Effect for the LearnedDomin model.

Under the LearnedDomin model, over 0.99 of the dominator locations become fixed and, of those, about 0.95 are aligned with the environment. This are significant increases in these measure values over results for the Domin model reported above. Final average fitness under the Learned-Domin model also increases to 0.92. Clearly, developmental learning has indirect impacts upon the effectiveness of domination. Maintaining an explicit dominance pattern represents another way that the evolutionary effects of developmental learning can be captured, which pattern in turn impacts the development of phenotypes and average fitness.

3. DISCUSSION

We have investigated the impacts of differently aligned dominance on evolution and proposed models for the acquisition of well-aligned dominance patterns by evolution. Current biological theories of dominance focus upon the role of cellular metabolic systems in controlling phenotype development. Genetic impacts on the concentration or activity of the system of enzymes that control gene expression are thought to account for dominance as well as for cellular differentiation during development [8], [15]. Our notion of a dominance pattern can be seen to be a representation of inherited genetic information that determines the cellular enzyme mix leading to dominance. Our models of dominance are clearly simplified abstractions of the complex interactions that occur biologically. Our simulation results indicate that relatively simple mechanisms could exist for the efficient acquisition of well-aligned dominance. More experience is needed with a wider range of fitness functions and with more complex models of dominance to better understand the creation and impact of dominance in evolution.

Our studies indicate a role for dominance in applications of evolutionary computation that adopt diploid-like representations. Diploid representations provide development with options for gene expression at heterozygotes when creating a phenotype. By remembering which alleles have been prevalent as homozygotes in parents selected from recent generations, a dominator makes it possible for development to guarantee that these alleles are expressed whenever they are present in a current genotype. These alleles are assumed to be adaptive, having been expressed in individuals that were selected for reproduction from earlier generations. We see that adding a form of developmental learning to dominance can speed the search for more fit individuals in more difficult, unconquerable environments. By being restricted to considering alternative options only for those heterozygotes that are not yet fixed by a dominance pattern, the learning process can be made more efficient.

4. REFERENCES