Maturation and the evolution of imitative learning in artificial organisms

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Abstract. The traditional explanation of delayed maturation age, as part of an evolved life history, focuses on the increased costs of juvenile mortality due to early maturation. Prior quantitative models of these trade-offs, however, have addressed only morphological phenotypic traits, such as body size. We argue that the development of behavioral skills prior to reproductive maturity also constitutes an advantage of delayed maturation and thus should be included among the factors determining the trade-off for optimal age at maturity. Empirical support for this hypothesis from animal field studies is abundant. This paper provides further evidence drawn from simulation experiments. "Latent Energy Environments" (LEE) are a class of tightly controlled environments in which learning organisms are modeled by neural networks and evolved according to a type of genetic algorithm. An advantage of this artificial world is that it becomes possible to discount all non-behavioral costs of early maturity in order to focus on exclusively behavioral consequences. In spite of large selective costs imposed on parental fitness due to prolonged immaturity, the optimal age at maturity is shown to be significantly delayed when offspring are allowed to learn from their parents' behavior via imitation.

1 Introduction

To many computational modelers using neural networks (NNets) as models of learning, it is second-nature to provide as much training experience for their NNets as possible, since almost always this leads to increased performance. But when these NNets are used in conjunction with genetic algorithms (GAs) as biologically realistic models of the *evolution* of learning individuals, it is necessary to consider the problem as it occurs in "first Nature": Is the increased time required by extended training worth the evolutionary costs of providing it? The fact that many organisms spend their prolonged immaturity as part of family units that can -- potentially -- shape the experience of the learning juvenile in predictable, heritable ways makes the role of the learning period especially important as we begin to contemplate proto-cultural effects on cognitive development. In this paper we will address restricted versions of these questions, focusing exclusively on imitative types of learning between parent and child.

In the study of the evolution and adaptation of life history traits in animals, the commonly accepted theory states that any particular trait accomplishes a trade-off between the different selective pressures acting simultaneously upon the phenotypic variants of that trait. One such trait that is central in behavioral and developmental psychology as well as in theoretical biology is the age at which an individual reaches the adult stage. To use a more precise terminology, it is preferable to consider the maturation age, or age at maturity, defined as the onset of the stage when the individual is capable to reproduce.

The selective pressures concerning the evolution of age at maturity considered in theoretical biology are the adaptive costs and benefits associated with anticipating or delaying maturation age. Typically (see, e.g., Stearns 1992), the costs of delayed maturation include: (i) lower population reproductive fitness due to longer generation time; (ii) lower individual reproductive fitness due to decreased probability to reach the mature stage; and (iii) parenthood cost due to longer immature period requiring parental care. Conversely, the benefits of delayed maturation typically include: (i) higher fecundity of the parent who can grow for a longer time and better endure the reproductive effort; and (ii) lower instantaneous juvenile death rates due to better quality of offspring or parental care.

Ecological field studies attempting to quantify the magnitude of selective pressures toward delayed maturation necessarily focus on easily measurable life traits, such as body size and weight. On the other hand, animal and human psychology studies concerned with social cognition emphasize the improvement of offspring phenotypes taking place through cultural learning. For example, Tomasello, Kruger and Ratner (1993) identify three types of cultural learning: imitation, instruction, and collaboration. It would be desirable to be able to quantitatively correlate such behavioral advantages with life history models of delayed maturation. However, the difficulty in measuring phenotypic traits associated with behavioral development, necessary to apply analytical trade-off models, causes any behavioral benefit of delayed maturity to be neglected in studies of the evolution of maturation.

We propose a simulation model in which the advantage of parental care is in fact the only benefit of delayed maturation. Lower death rates may result from an improvement of phenotypic behavior before the adult stage is reached. In the model, this improvement is acquired by the offspring through learning by imitation of its parent. Notice that it makes sense to model learning only before maturation, because parents must

act as teachers to their immature offspring. Johnson (1982) has associated learning with immaturity in the study of costs and benefits of phenotypic plasticity as a life history trait. Cavalli-Sforza and Feldman (1981) have characterized cultural transmission as a diffusion process based on a model of imitation. Another seminal evolutionary account of cultural transmission by imitation has been given by Boyd and Richerson (1985). While it is not our intention to propose imitation as a universal mechanism for cultural transmission, Tomasello *et al.* (1993) identify imitative learning as the first of the three forms in which cultural learning manifests itself during ontogeny. Therefore, we use imitation to model one possible mechanism by which parents may confer a cultural advantage to their immature offspring. A similar approach has been taken by Denaro and Parisi (1994); more abstract models of cultural advantage have also been explored (Belew 1990).

In this paper we illustrate quantitative evidence in support of the hypothesis that the fitness improvement of phenotypes by means of their learned behavior plays an important role in the evolution of maturation age. We simulate the evolution of age at maturity as a genotypic trait regulating the duration of phenotypic learning by imitation. The model exhibits all of the costs of delayed maturation enumerated above. We hypothesize that the ability to learn imitated behaviors is adaptive, and hence expect that there be selective pressure toward delayed maturation to allow such learning to occur.

The next section contains an overview of the simulation tool used as the framework for our numerical experiments. Section 3 describes how the model and simulations have been implemented. Sections 4 and 5 illustrate and analyze the experimental results of simulated experiments. Finally, section 6 draws some conclusions.

2 LEE model

Simulation experiments described in this paper make use of a recent artificial life model and simulator, called Latent Energy Environments (henceforth LEE).[1] A detailed description of the model and other experiments using it can be found elsewhere (Menczer and Belew 1994, 1995), but its features most relevant to this work are summarized below.

In LEE, environments of measured complexity can be constructed due to a careful definition of the energy consumed by organisms, and of the work they must do to realize it. Energy is always conserved, and behavioral strategies are evolved by populations so as to allow an efficient exploitation of the available energy. Environmental complexity is therefore measured with respect to the difficulty of the survival task.

Organisms in a LEE population live in a shared environment consisting of a rectangular grid with toroidal edge conditions. Each organism behaves according to a neural network mapping sensory information to motor actions (see below). Populations of such organisms are born, reproduce, and die according to the algorithm presented in Figure 1. Organisms in an initial population are randomly constructed and given an initial, random reservoir of energy distributed uniformly in the interval $[0,\alpha]$. A constant, "metabolic" energy cost is paid anytime an individual is active. If an organism's energy level ever reaches zero, it dies. On the other hand, if an organism is consistently capable of consuming more energy than its metabolism demands, it reproduces and gives half of its energy to the

[1] The most recent release of the LEE software package and documentation is available by URL http://www-cse.ucsd.edu/users/fil or anonymous ftp://cs.ucsd.edu/pub/LEE.

```
for each time cycle {
      for each alive organism {
            sense world;
            compute actions;
            move;
            Energy ← Energy + Σ_{actions} Benefits − Σ_{actions} Costs;
            if ( Energy > α ) {
                  reproduce;      /* copy genotype         */
                  mutate;         /* change new genotype   */
                  develop;        /* get new phenotype     */
            }
            else if ( Energy < 0 ) die;
      }
      replenish world;
}
```

Figure 1. Basic LEE algorithm. The constant α is the reproductive threshold.

offspring anytime its energy passes a fixed reproductive threshold.

A LEE population therefore evolves according to a "steady-state" GA. Reproduction is asexual, and the only genetic operator is mutation on the network weights. Rather than assuming a constant population size, and scaling all fitness values proportionally (as in a conventional GA), the size of the population varies with time, and extinction is possible. "Fitness" is most appropriately measured in terms of energy intake per unit time, which is directly correlated to number of offspring per lifetime. The progression of the adaptive process is measured in terms of time (life cycles) rather than generations.

An organism is modeled by its energy reservoir, a sensory-motor system, and a NNet. The sensory system consists of a set of sensors, each mapping a state of the world onto one of the inputs of the network. Two types of sensors were used in these experiments, "contact" and "ambient" sensors. A contact sensor can only sense elements in the cell directly in front of the organism. An ambient sensor is capable of sensing elements anywhere in an oriented, local range of the organism. These senses are continuous values with the strength of the signal decreasing with distance. In the experiments reported here there are three ambient sensors, two with a range of three moves oriented to the sides of the organism, and one with a range of five moves oriented straight ahead. There is also one contact sensor, oriented ahead as well. The sensory system is illustrated in Figure 2.

The motor system is made of a pair of "binary" motors, which are controlled by two binary NNet output units. These allow organisms to make one of four possible moves: stay still, turn left or right 90 degrees, or move ahead by one cell. The sensors and motors are connected by a single "hidden" layer of seven units.

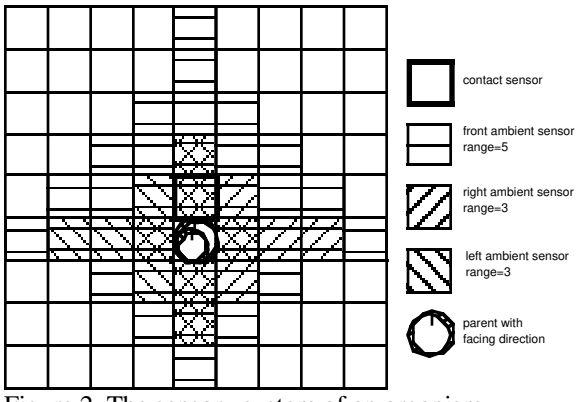

Figure 2. The sensory system of an organism. The ranges of the different sensors are shown. An immature offspring is also represented "on the shoulders" of its parent.

Due to the constant rate of replenishment of environmental resources to be shared and the constant reproductive threshold, fitness is strongly density dependent (Menczer and Belew, 1995). The measured age distribution is stationary for most of the duration of the simulation -- except for the very initial phase in which there is an exponential demographic explosion, quickly taken over by damped oscillations. This model is similar to others' (e.g., Kozlowski and Wiegert 1987), where zero population growth is assumed at any given instant of time. A good measure of fitness is then given by the R_0 statistics, defined as the expected number of offspring per parent. Indeed, over a wide range of LEE environmental conditions, the number of offspring is found to be well correlated with population size, p. Following these considerations, we will use p as the single fitness measure for the evolving population in the remainder of this paper.

3 Experiment setting

The particular task implemented in LEE for these experiments consists of approaching atoms scattered in the environment with uniform distribution in space and time. This is much like *positive chemotaxis* (Gruau and Whitley 1993). When an organism reaches a cell containing an atom, it acquires energy E. Atoms appear in the environment at a rate of r per unit time. We can easily calculate the carrying capacity of such environments for a population of organisms moving according to a type of "random walk"[2]:

$$p = \frac{rE}{c} \qquad (1)$$

where c is the cost of making a move (Menczer and Belew, 1995). The organisms rapidly evolve to implement this easy and relatively efficient random behavior, therefore the population size initially oscillates around the value predicted by Equation (1). This linear dependence between environmental resources and population size is a very good prediction over a wide range of LEE experiments. Adaptive behaviors implementing energy-seeking strategies better than random walks result in population sizes beyond the limit of Equation (1).

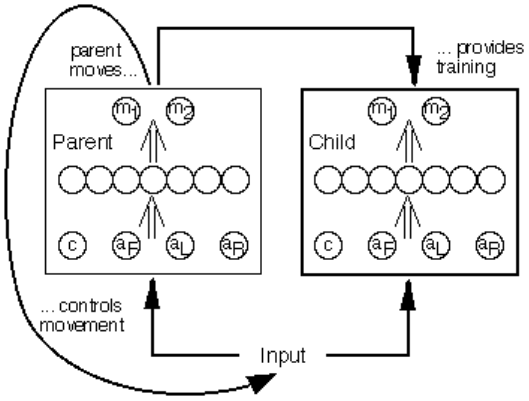

Figure 3. Imitative learning architecture.

To study age at maturity, the life of organisms is divided into two distinct stages separated by an "age of maturity." One new gene, called a_m (age at maturity), represents the age of the transition to maturity in number of life cycles. Values of a_m are between zero (no immaturity) and the age at death (no maturity). During its immature phase, an organism differs in exactly two respects from its mature form. First, the immature phenotype is not allowed to reproduce. Its energy at birth is conserved until maturity, and its moving costs are entirely transferred to its parent. Second, juveniles undergo a process of learning by imitation. The details of this construction are suggested

[2] We will use the term "random walk" in the remainder of the paper somewhat unconventionally. Due to the toroidal shape of the LEE world and the uniform distribution of atoms in our experiments, a behavior effectively equivalent to a random walk can be achieved by always moving forward. The equivalence is in terms of encounters with atoms, while the behavior is obviously different from truly random, Brownian movement.

by Figure 3. Parent and child experience identical input stimuli from the world, but the output computed by the parent's NNet is used to determine both organisms' movement. The motor units of the parent's NNet are also used as a training signal for the child's NNet. Our metaphor for this stage is to think of the offspring as being carried over the shoulders of its parent. When an organism reaches age a_m it becomes an adult, normal member of the population.

Several objections to this construction can be anticipated. First, some may find the notion of "imitation" captured here inappropriately direct and imposed. Certainly the real, biological organism is faced with a much more difficult learning task, for example requiring an appropriate correspondence between the sensory stimuli of *watching* its parent perform a behavior and the proprioceptive and kinesthetic experience of *executing* this behavior itself. We view the biological basis of such identification an extremely important research issue. Our present model elides the issue entirely by assuming complete, perfect identification. Second, why is the immature stage of an organism made to coincide exactly with its learning phase? One motivation behind this modeling choice is the simple observation that in many species learning is in fact contemporaneous with the initial, pre-reproductive phase of life. But our central motivation is that this equivalence is a methodological necessity in order to argue that learning plays a role in the evolution of life history strategies. By limiting learning to the immature stage, we intrinsically transfer the costs of immaturity to the learning process.

With these caveats, let us review the way in which the three costs of delayed maturation (cf. Section 1) are modeled. First, longer generation time is implicit in the LEE model: longer immature stages correspond to shorter times in which offspring can be generated, and thus to lower R_0 fitness. Second, increasing the immature stage decreases the probability to reach maturity, due to the constant expected probability of death of the parent per life cycle. When a parent dies, its "orphan" immature offspring are dropped into the environment before becoming adults. An orphan cannot move until it reaches its mature age, but may run out of energy and die before becoming adult. The probability of survival to mature age is higher the closer to maturation the offspring is when the parent dies. The third cost of the immature stage is associated with parental care: a parent pays an additional energy toll equal to the cost of a move for each offspring it carries on its shoulders. Energy is still conserved, since the immature offspring incur no living costs as long as they are carried on their parents' shoulders.

Learning is the only benefit of delayed maturity in our model. Thus if a delay of maturation age is observed in simulations, it can only be attributed to the advantage provided by learned behaviors. Note that this benefit is conferred upon the offspring and not the parent. In evolutionary ecology, similar benefits for the offspring are mainly attributed to morphologic development; for example, offspring size has been considered by Kozlowski and Wiegert (1987). The benefit modeled here is less explicit because mediated through the behavior of the offspring: juvenile mortality can be decreased thanks to the experience accumulated by offspring during their immature life stage, by way of parental imitation.

The a_m gene is allowed to evolve, being selected together with the rest of the organisms' genotypes as described above. It may be mutated (with probability 0.1) at reproduction by a random additive deviate uniformly distributed in the interval $[-a_m, +a_m]$. Negative values are clamped to $a_m = 0$. The rest of the genotype contains a description of the NNet's connections between its four inputs, seven hidden

units, and two outputs. Connection weights, represented as real numbers, are also mutated (with probability 0.15) by random additive deviates uniformly distributed over the range [-2.5, +2.5].

Learning is modeled via forced motor imitation of the parent, given that both parent and its offspring receive the same sensory input. The actions encoded in the parent's outputs are used as teaching signals for the offspring on its shoulders. Standard back-propagation of error is then used to correct the connection weights of the offspring (Rumelhart, Hinton and Williams 1986). However, the learning rate parameter is set to a relatively high value (0.8). The reason is that the learning NNets of immature organisms in this model are give a relatively short training experience (the duration of the immaturity stage), and previous research has shown that the more volatile learning associated with high learning rates can work in the context of evolving populations (Belew, McInerney and Schraudolph 1991). It is also important to note that in this simulation weight changes only affect phenotypes, so that learning is strictly non-Lamarckian.

4 Results

In this section we outline the main results of a series of experiments aimed at evaluating the potential role of imitation learning in the evolution of age at maturity. Simulations were run for 150,000 cycles (as defined above). The first simulations are controls designed to facilitate the interpretation of subsequent experiments.

In Figure 4 population size as a function of time (measured in cycles) is plotted for two single runs in which a_m is held at the constant values 0 (no immaturity) and 100 cycles. The first case (a_m = 0) provides a baseline with neither the costs nor advantages of an immature period. The observable increase in population corresponds to an improvement in the approaching behaviors of the organisms, due to the evolution of their network weights. The second case (a_m = 100) gives us a measure of the magnitude of the cost of delayed maturation: this is large enough to drive the population to extinction in less than 25,000 cycles. Thus we expect strong selective pressure against delayed maturation. For comparison, the (analytically derived) expected size of a population of random walkers is also shown in the Figure; cf. Equation (1).

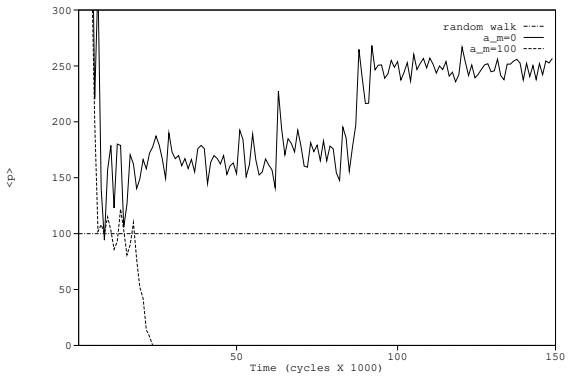

Figure 4. Population size for different fixed ages at maturity. Data are collected form two single simulation runs. Extinction occurs in the case of non-zero maturation age. Random walk in this and the following Figure is from Equation (1). At the beginning of the simulations, a large amount of environmental resources results in an exponential peak in population size (not shown in the plot for clarity). Then density dependence sets in because the rate of replenishment of resources is held constant, and the population is limited by the carrying capacity of the environment.

In order to study the evolution of life history strategies, however, the age of maturity must itself be assumed to be an evolved trait, the ability to learn via imitation being an independent variable. Our next experiments therefore add the a_m gene to the genome of the evolving organisms. The population is initialized with a_m uniformly distributed over the interval [0, 100]. Figure 5 shows the population sizes for two populations, one in which imitative learning is enabled during the immature phase and one in which it is not. In both cases, after the

initial stochastic fluctuations, the populations are able to evolve individuals with behaviors significantly more adaptive than random. However, with imitative learning, the evolution of good approaching behaviors is significantly accelerated.

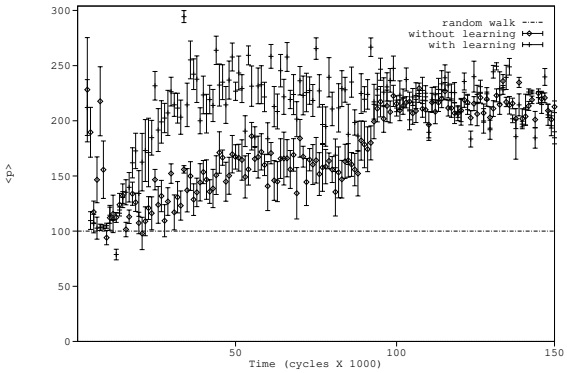

Figure 5. Population size with evolving maturation age gene. Errors correspond to one standard deviation over repeated simulation runs. The case with learning from imitation results in faster fitness improvement.

Figure 6 plots the population's average value for the evolved a_m gene in the two experiments, again with and without imitative learning. In the absence of learning, other costs of immaturity dominate and a_m rapidly evolves to zero. When the young are also allowed to learn via imitation, however, a significant delay in the extinction of the immature phase is observed. It is important to note that this is an evolutionary delay experienced by the species, not to be confused with the developmental delay controlled by the a_m and experienced by individuals. It is also important to recognize that the inclusion of imitative learning does not keep a_m from becoming zero; it only prolongs this process. The fact is, however, that real, biological environments rarely enjoy the equilibrium conditions of our artificial LEE world. Factors that maintain plasticity -- by definition making an organism more responsive to changes in its environment -- are therefore of real consequence.

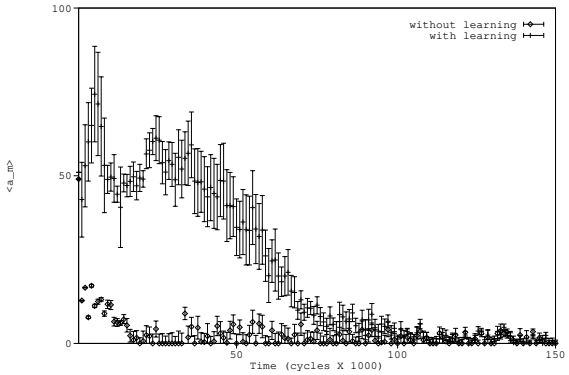

Figure 6. Evolution of maturation age gene. Errors correspond to one standard deviation over repeated simulation runs. Maturation age is delayed significantly when imitation occurs.

In summary, the delay observed represents evidence that there is a trade-off between costs and advantages of delayed maturation. This trade-off is quantitatively estimated by the value -- slightly above 50 cycles -- around which the population's average maturation age oscillates initially (cf. Figure 6). Since in our model the only advantage can be that of offspring learning via parental imitation, we must conclude that cultural transmission of adapted behaviors is one of the evolutionary factors creating selective pressure in favor of longer immature stages.

5 Analysis

The role learning and other forms of phenotypic plasticity can play in accelerating evolutionary change is increasingly well explored. For example, the "Baldwin effect" (Baldwin 1896, Waddington 1942) has come to describe a mechanism by which a learner, prohibited from encoding the *consequences* of learning directly onto the genome (as Lamarck proposed), can nevertheless enjoy a selective advantage as a direct consequence of its learning. In brief, the ability of an organism to adapt within its lifetime towards beneficial characteristics of its environment (i.e., learn) increases the probability that other genetic traits

serendipitously correlated with this same benefit will increase in frequency. A range of evidence for this important, albeit subtle connection between learning and evolution has been found by a number of investigators using computer simulations (Hinton and Nowlan 1987, Nolfi, Elman and Parisi 1990, Belew 1990, Ackley and Littman 1992, Gruau and Whitley 1993, Belew and Mitchell 95). Our experiments are consistent with these accounts. Once again, our learning organisms' ability to explore a range of behavioral strategies within their lifetimes means that the evolutionary process is capable of exploiting much more information about adaptively favorable characteristics of the environment.

But if learning by imitation during immaturity provides selective pressure toward delayed maturation, why does a_m eventually converge to zero, as shown in the last phase of its evolutionary course in Figure 6? The Baldwin effect mentioned above gives us a simple interpretation of this fact as well. During the juvenile period, modifications that learning causes upon the phenotype are eventually re-discovered by the evolutionary process and thereby affect the genotype. Once this happens, learning is no longer useful because offspring at birth are already capable of the behaviors that in earlier generations they could only acquire by imitating their parents. Therefore learning no longer confers an advantage to immature offspring. The costs of delayed maturation remain the same, and the missing benefit causes the immature period to disappear. The next two sub-sections analyze just how the Baldwin effect operates in our experiments, interpreting the phenomenon in both the phenotypic spaces of behaviors and connection weights.

5.1 Correlation of learned and evolved tasks

It may seem trivial that the imitated approach behavior is evolved, given its strong correlation with fitness: the behavior being learned is identical to the one that is eventually evolved. Recall that in the original construction an offspring's NNet learns a mapping from the shared input to the parent's output. We can imagine that the Baldwin effect might be weaker if the learned behavior were less well correlated with the ability to approach atoms.

To explore these questions, we consider a variant of our model in which, in addition to imitating their parents, immature offspring are trained to predict their parents' sensory input following the current move. This task, while different from the approaching task, may still be helpful to survival because of offspring taking advantage of their parents' experience (Nolfi *et al.* 1990). Genetically, the single a_m is replaced by two new genes. The duration of the prediction learning phase is regulated by a 'prediction gene' a_p while the imitation learning phase is regulated by an 'imitation gene' a_i During the prediction phase the offspring performs prediction learning using the parent's input, while imitation is as described in Section 3. Thus imitation and prediction are effectively decoupled, and prediction learning becomes an independent mechanism to model the cultural transmission of a parent's experience to its immature offspring. Between birth and age $min(a_i, a_p)$, both imitation and prediction learning occur. Between $min(a_i, a_p)$ and $max(a_i, a_p)$, only one of the two tasks is learned, namely, the one corresponding to the greater gene value. Both genes evolve analogously to a_m. The model stipulates that an organism is able to reproduce only after age $max(a_i, a_p)$. This way both the prediction and imitation processes have associated costs as necessary to model the trade-off. Furthermore, assuming that the selective advantage of prediction learning may be smaller than the one of imitation learning, the parental cost of carrying offspring on one's shoulders is reduced to zero.

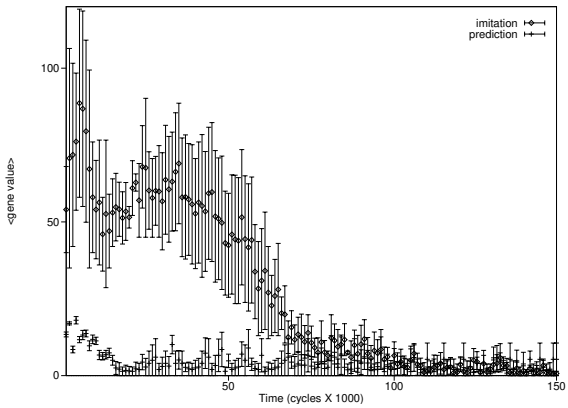

Figure 7. Evolution of prediction and imitation genes. Errors correspond to one standard deviation over repeated simulation runs.

The result of the experiment is shown in Figure 7. The a_p gene goes to zero very quickly, much like a_m without imitation learning (cf. Figure 6). On the other hand, the a_i gene evolves like a_m with imitation learning enabled (cf. Figure 6). This result suggests that prediction learning is not advantageous during immaturity. Note that the prediction task using ambient sensors is very difficult, due to their non-invertible mapping from world states to NNet inputs; for a more detailed discussion of this problem see (Menczer and Belew 1994). Therefore we do not have evidence that prediction learning is not useful, however we are unable to demonstrate its advantage in this context.

5.2 Baldwin effect in weight and behavior spaces

We now return to consider our standard model, in which juveniles are trained by their parent's actions and this imitation is controlled by a single a_m gene. The most straightforward analysis of the Baldwin effect consists of verifying that phenotypic modifications made by learning have become unnecessary due to behaviorally equivalent changes in the genome. In our simulations, learning is being modeled by changes made to the NNets weights as the result of error back-propagation. One simple measure of the magnitude of this change is given by the Euclidean distance in the space of the network connection weights:[3]

$$\Delta \underline{w} = \sqrt{\sum_{i,j} \left(w_{ij}^{after} - w_{ij}^{before} \right)^2} . \qquad (2)$$

Here the superscripts indicate the NNet's initial, genetically determined weights "before" learning and the final weights "after" learning. The Baldwin effect would predict that this distance decreases to zero as learned changes are transferred to the genome. Since our experiments typically allow the amount of learning to vary with the a_m gene, care must be taken to evaluate the magnitude of these changes after a constant amount of learning.

We therefore consider all the organisms in a typical simulation run. The initial genotypic weights of their NNets are recorded when the organisms are immature (at birth), and then subjected off-line to a fixed period of imitation learning (100 cycles of back-propagation) based on the parents' behaviors. This procedure guarantees that if the Euclidean distance decreases, this is due to the Baldwin effect rather than an artifact of variable immaturity periods. At the same time, the learning process takes place under identical environmental conditions as during the actual simulation.

Figure 8 shows a scatter plot of the average values of NNet weight change (2), vs. the corresponding a_m gene value. The two are very well correlated ($\mu \sim 0.936$): earlier maturation corresponds consistently with smaller phenotypic change in weight space. This confirms our interpretation that the Baldwin effect makes imitation learning

[3] In fact, this measure depending only on initial and final positions is too simple. The distance travelled by a learning NNet also depends critically on features of the learning surface, type of local, gradient search performed by the learning system, etc. (Belew *et al.* 1991)

unnecessary and thus the immature stage disappears.

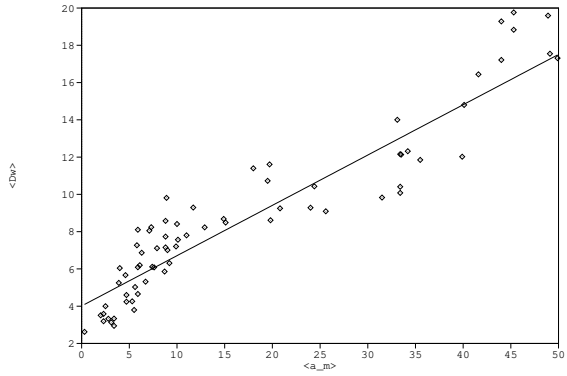

Figure 8. Scatter plot showing correlation between a_m and $\Delta\underline{w}$ (measured over a fixed number of back-propagation cycles at different evolutionary times between 50,000 and 120,000 life cycles). Both statistics are averaged over the population and over 10 different simulation runs. Linear regression fit of the data is also shown.

Another way to analyze the Baldwin effect is by directly inspecting phenotypic behaviors during evolution. Movement is designed to be the canonical behavior in LEE organisms. A necessary component of optimal foraging behavior in the environments used in the experiments outlined in this paper is to move forward when the contact sensor signals the presence of an atom in the cell immediately facing the organism. An indicative measure of *poor* behavior is therefore *P(~F)*, the fraction of times a mature organism presented with an atom in the next cell does *not* move forward to consume it. When the population converges to *low* values of *P(~F)* at birth, we have an indication that the *good* behavior has been encoded into the genotypic weights, making it unnecessary to learn during life.

Figure 9 shows a scatter plot of the average values of *P(~F),* measured off-line and without learning, vs. the corresponding a_m gene value. The two are again well correlated (μ~0.765). As the Baldwin effect works, organisms are born with progressively better behavior. The fraction of times organisms move forward in the presence of an atom in the facing cell increases in the course of evolution, and the average maturation age consequently decreases due to the costs of immaturity.

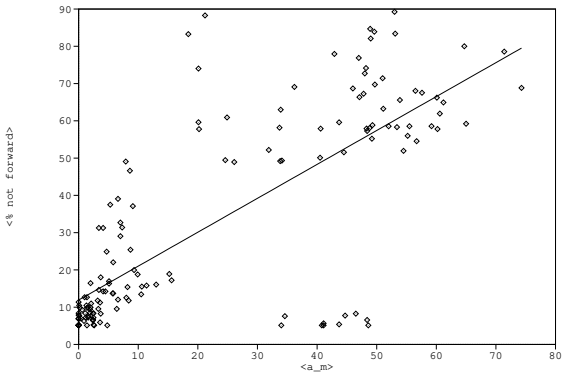

Figure 9. Scatter plot showing correlation between a_m and *P(~F)* (measured at different evolutionary times between 0 and 150,000 life cycles). Both statistics are averaged over the population and over 10 different simulation runs. Linear regression fit of the data is also shown.

6 Conclusion

The simulation experiments reported in this paper have shown that behavioral improvements of the immature phenotype may be among the factors determining the trade-off between costs and benefits of delayed maturation. We have shown in the previous section that learning during immaturity provides selective pressure toward delayed maturation. We have also demonstrated that the Baldwin effect can use this extended window of juvenile plasticity to good evolutionary advantage.

We believe that our experiments bring an important new dimension to the discussion of maturation as an evolved life history, i.e., the important role *cultural* influences can play. Following Boyd and Richerson (1985), we mean "culture" to refer to: "the transmission from one generation to the next, via teaching and imitation, of knowledge, values, and other factors that influence behavior" [p. 2]. In the present case, the "knowledge" being transmitted is

exclusively the *shaping of early experience* of impressionable young learners, via imitation of their parents' behaviors. Our model also provides an interesting contrast to more tangible forms of transmission, for example those mediated by external representational media (Hutchins and Hazelhurst 1991). Evidence for cultural transmission is most clear when it leaves a trace of such external artifacts, but our experiments demonstrate that culture can also take the form of behavioral "thatching" across generations. The more plastic the juvenile, the more possible it is that environmental exigencies can perturb its "normal" developmental trajectory. At the same time, increasingly adaptive young provide even more opportunities for culturally mediated knowledge to improve a population fitness.

Prior quantitative approaches to the study of this important life history trait are often subject to the limitations of field experiments, and therefore these have mainly focused on structural benefits of delayed maturation, such as body size, weight, etc. Animal psychology studies of cultural transmission, on the other hand, have difficulty in casting the phenotypic nature of learned behaviors into the more general evolutionary framework. The simulation approach employed here provides a bridge between the two sides.

Modeling the cultural transmission of learned behaviors from parents to offspring by forced imitation of motor responses to sensory stimuli is not intended to point to imitation as the only or the best mechanism by which an immature organism can improve its reproductive fitness as well as that of its offspring. One direction for further research is to consider other forms, such as instructed learning and collaborative learning (Tomasello *et al.* 1993), to gain a more general understanding of the mechanisms by which cultural transmission can influence the evolution of life history traits.

A related approach is to study the adaptive value of maturation using a task that must be learned by an organism during its life and which cannot evolve directly. In such a situation the Baldwin effect must be limited. This approach is the object of current research, with preliminary results that confirm our expectation from the results illustrated here: maturation age evolves to a value greater than zero, and remains at such value indefinitely (Cecconi and Parisi 1994). Another adaptive advantage of phenotypic plasticity may be found when the population evolves in a non-stationary environment (Menczer 1994). If environmental changes occur at a rate faster than the time necessary for evolution to encode adaptive changes in genotypes, then we expect the Baldwin effect to create selective pressure for maintaining a period of phenotypic adaptation, thus allowing evolution to track the environment.

Learning by imitation is a relatively simple paradigm within which we have been able to model the costs of delayed maturity on both mature organisms (parents) and immature ones (offspring). The adaptive advantage of transferring a parent's experience onto its offspring by "carrying them on its shoulders" has been shown to be large enough to permit a significant delay in maturation age, at least until the adaptive behaviors are transferred to the genotypes of the population via the Baldwin effect. Our data, therefore, supports behavioral and cultural models of maturation age as a life history trait. Since imitation seems to be a very basic form of cultural transmission in social animals, this result not surprisingly points to one account for the long immaturity stages in these species. Clearly, many other factors determine the trade-off for optimal age at maturity, but critical data about cultural learning are available for only a very few species, e.g., apes and humans (see Tomasello *et al.* 1993). We hope our research stimulates field studies aimed at providing experimental

evidence for (or against!) the behavioral benefits of delayed maturation illustrated here.

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