The Dynamic Changes in Roles of Learning through the Baldwin Effect

Reiji Suzuki*
Graduate School of Information Science, Nagoya University
Furo-cho, Chikusa-ku, Nagoya 464-8601, Japan
reiji@nagoya-u.jp
Phone/Fax +81-52-789-4258
http://www.alife.cs.is.nagoya-u.ac.jp/~reiji/
and
Takaya Arita
Graduate School of Information Science, Nagoya University
Furo-cho, Chikusa-ku, Nagoya 464-8601, Japan
arita@nagoya-u.jp
Phone/Fax +81-52-789-3503
http://www.alife.cs.is.nagoya-u.ac.jp/~ari/

Abstract

An interaction between evolution and learning called the Baldwin effect is known as the two-step evolutionary scenario caused by the balances between benefit and cost of learning in general. However, little is still known about dynamic evolutions on these balances in complex environments. Our purpose is to give a new insight into the benefit and cost of learning by focusing on the quantitative evolution of phenotypic plasticity under the assumption of epistatic interactions. For this purpose, we have constructed an evolutionary model of quantitative traits by using an extended version of Kauffman's NK fitness landscape. Phenotypic plasticity is introduced into our model, in which whether each phenotype is plastic or not is genetically defined and plastic phenotypes can be adjusted by learning. The simulation results have clearly shown that the drastic changes in roles of learning cause the three-step evolution through the Baldwin effect and also cause the evolution of the genetic robustness against mutations. We also conceptualize four different roles of learning by using a hill-climbing image of a population on a fitness landscape.
keywords Baldwin effect, genetic robustness, phenotypic plasticity, evolution and learning, NK fitness landscape, artificial life.

*corresponding author

1 Introduction

There are different levels of interactions between genetic factors and the other environmental factors. The artificial life studies have clarified the complex dynamics of them by showing the possible scenarios of the evolution of artificial organisms. Especially, there have been various discussions on the Baldwin effect [1] as one of the interactions between evolution and learning, which suggests that individual lifetime learning can influence the course of evolution without the Lamarckian mechanism [19]. This effect explains these interactions by paying attention to balances between benefit and cost of learning through the following two steps [16]. In the first step, lifetime learning gives individual agents chances to change their phenotypes. If the learned traits are useful for agents and make their fitness increase, they will spread in the next population. In the second step, if the environment is sufficiently stable, the evolutionary path finds innate traits that can replace learned traits, because of the cost of learning. This step is known as genetic assimilation [18]. Through these steps, learning can guide the genetic acquisition of learned traits without the Lamarckian mechanism in general.

Hinton and Nowlan conducted the first computational experiment of the Baldwin Effect [7]. They assumed an extremely simplified version of a network connection model. There were 1000 individuals and each individual has 10 locus in which there were three alternative alleles 0, 1 and ?. 1 (or 0) represented the condition that the corresponding connection was present (or not present), and ? also represented that whether the corresponding connection was present or not was plastic and not genetically determined. Each individual conducted the learning processes for up to 1000 trials, in each of which a randomly generated condition (present or not present) was assigned to each plastic connection. The learning process stopped when all connections were present, and the fitness became $1 + 19n/1000$, where n was the number of learning trials that remain after the learning had stopped. The population was evolved by using standard roulette selection and one point crossover but mutational operation was not

used.

The essential point of this study is that they introduced the quantitative evolution of phenotypic plasticity (the evolution of the number of '?' in the individual) into their model, in other words, they allowed a population to adjust how much it depends on these two adaptive mechanisms through evolution. They revealed the existence of the Baldwin effect by showing the increase and subsequent decrease in the phenotypic plasticity. However, the learning mechanism in their model was too simple on the ground that its benefit was approximately proportional to the number of plastic phenotypes and the cost of learning was explicitly introduced. Thus, further investigations were necessary so as to understand this effect in more realistic situations, and then, this effect has been discussed in various contexts.

Suzuki and Arita focused on the iterated Prisoner's Dilemma game so as to discuss the emergence of the Baldwin effect in dynamic environments [15]. They introduced phenotypic plasticity into the deterministic strategies, and conducted the computational experiments, in which phenotypic plasticity is allowed to evolve. The Baldwin effect was observed in the experiments as follows: First, strategies with enough plasticity spread, which caused a shift from defect-oriented population to cooperative population. Second, these strategies were replaced by a strategy with a modest amount of plasticity generated by interactions between learning and evolution.

Downing discussed the mutual effects of a developmental process and a learning process on the evolution of developmental mechanisms [4]. He constructed the trilaterally adaptive system (TRIDAP) into which the evolutionary, developmental and learning mechanisms were incorporated. Its developmental system was composed of a Turing-machine-like system of which its rule and initial tape were genetically defined and it generated the initial string of the phenotype as a phenotype in Hinton and Nowlan's model. They successfully illustrated several roles of development in Baldwinian evolution, both inhibiting and enhancing.

The effects of epistasis are of interest in evolutionary studies because epistatic interactions among loci are ubiquitous in modern genetics and evolutionary biol-

ogy [21]. For instance, Mayley conducted an evolutionary experiment based on a genetic algorithm using the Kauffman's NK fitness landscape [11]. He adopted a learning process that searches for an adaptive phenotype in neighboring phenotypes. He pointed out that there should be a neighborhood correlation between genotype and phenotype space to guarantee a genetic assimilation to occur. Bull also discussed the evolution on the NK fitness landscape using a different type of probabilistic learning process and an evolutionary process implemented by a hill-climbing of a species on the landscape. He concluded that whether the learning can increase the fitness or not depends on the ruggedness of the landscape, the probability of learning, and the number of learning iterations [2]. However, all phenotypes were plastic and the quantitative evolution of phenotypic plasticity was not introduced into their models. In this sense, the two steps of the Baldwin effect were not clearly discussed in these models when compared with Hinton and Nowlan's model. In addition, Wiles et al. recently discussed the genetic redistribution effect that complex of genes can be integrated into functional groups as a result of environmental changes that mask and unmask selection pressures by using an extended version of Hinton and Nowlan's model [20]. They have shown that the decreased adaptivity of the trait of a specific gene caused by the genetic drift due to the masking of the fitness contribution of the trait can be compensated by a set of other genes and, as a result, these genes get be more tightly linked.

Our purpose is to give a new insight into the dynamic evolution of the benefit and cost of learning in complex environments. Especially, we focus on the effects of epistatic interactions on the quantitative evolution of phenotypic plasticity. As a first approach, we have investigated the quantitative evolution of phenotypic plasticity by using a less abstract model based on a neural network than conventional models [13]. The transitions of the phenotypic plasticity and the phenotypic variation revealed that the evolutionary scenario consists of three steps unlike the standard interpretation of the Baldwin effect.

The next approach, discussed in this paper, is to clarify the dynamic changes in roles of learning through the course of evolution by paying attention to the

effects of epistasis and to genetic robustness against mutations [15]. For this purpose, we have constructed an evolutionary model based on Kauffman's NK fitness landscape [9] in which we can explicitly adjust the degree of epistasis. We discuss the evolution of quantitative traits by extending the fitness evaluation of the NK model. We introduced the phenotypic plasticity into our model, in which whether each phenotype is plastic or not is genetically defined and the plastic phenotype can be adjusted by a simple learning process. By conducting experiments with various degree of epistasis, we show that the drastic changes in roles of learning cause a three-step evolution through the Baldwin effect and a subsequent evolution of the genetic robustness against mutations.

2 Three-step evolution through the Baldwin effect

We investigated the evolution of connection weights in a neural network as a situation where there are epistatic interactions among loci [13]. It was observed that the evolutionary scenario consists of three steps by focusing on the transitions of four indices as shown in Table 1. The *lifetime fitness* represents the actual fitness after learning in the population and the *innate fitness* is the potential fitness before learning based on initial phenotypes. The *phenotypic plasticity* represents the proportion of plastic phenotypes in the population. The *phenotypic variation* is the absolute difference in phenotypic values between before and after learning among plastic phenotypes.

The first step, that is the increase in both lifetime fitness and phenotypic plasticity, was simply caused by the benefit of learning. It is noteworthy that the second step has both properties of the first and second step in the standard interpretation of the Baldwin effect. The decrease in the phenotypic plasticity corresponds to the second step in the standard interpretation of the Baldwin effect in the sense the increased fitness by learning becomes dependent on fewer plastic phenotypes. At the same time, the increase in phenotypic variation means that the population becomes strongly dependent on the remaining plastic

phenotypes. Thus, we can also say that the population was still in the first step in this point of view. This phenomenon is supposed to be due to the implicit cost of learning caused by the epistatic interactions among plastic phenotypes through the learning processes. The third step corresponds to the second step in the standard interpretation because the genetic assimilation occurred in the remaining plastic phenotypes.

3 Model

3.1 NK Landscape with Real Valued Traits

We have constructed an evolutionary model based on Kauffman's NK fitness landscape [9], so as to discuss the evolution of phenotypic plasticity in quantitative traits with / without epistatic interactions among loci. There are P individuals in a population and each individual has N traits of which initial phenotypes are determined by genes in a N-length chromosome GI. Each gene represents the quantitative trait t_i (i=0, $\cdots$, N-1) which consists of a real value within the range $[0.0, 1.0]$. We adopt NK fitness landscapes for evaluation of fitness because we can explicitly adjust the degree of epistasis by using the parameter K. It represents the number of other traits that affect the fitness contribution of each trait. However, the standard NK fitness landscape only assumes the binary traits ("0" or "1"). Then we extended the definition of the fitness evaluation so as to deal with the fitness contributions of quantitative traits.

Each trait t_i has epistatic interactions among other K traits $t_{i+jmodN}$ (j=1, $\cdots$, K). For each t_i, we prepare a lookup table which defines its fitness corresponding to all possible (2^{K+1}) combinations of interacting traits in case that these phenotypes consist of only binary values ("0" or "1"). The value of each fitness in the lookup table is randomly set within the range $[0.0, 1.0]$. These tables are similar to those of the standard NK landscape.

The fitness for quantitative trait is defined as the linearly interpolated value among the fitness for binary combinations of interacting phenotypes using the

following equation:

$$f(t_i) = \sum_{c \in C_i} [f_{i,c} \cdot \prod_{j=i}^{i+K} \{(1.0 - b_{jmodN}) \cdot (1.0 - t_{jmodN}) + b_{jmodN} \cdot t_{jmodN}\}], \quad (1)$$

where $f(t_i)$ is the fitness of the trait t_i, C_i denotes the all possible 2^{K+1} combinations of binary traits, $f_{i,c}$ is the fitness of t_i when the combination of traits is c. b_j represents the j th binary phenotype in c. Figure 1 shows an example of the interpolation of the fitness of quantitative traits for $N = 2$ and $K = 1$. The table on the right side represents the lookup table which determines the fitness of t_0 corresponding to four binary combinations of t_0 and t_1. The left figure shows the interpolated fitness of t_0 generated by the right table and the equation (1). The individual fitness is regarded as the average fitness over all traits. Note that if we assume only binary phenotypic values, this model is equivalent to the standard NK fitness landscape.

3.2 Learning

Each agent has another N-length chromosome GP which decides whether the corresponding phenotype of GI is plastic ("1") or not ("0"). Each trait whose corresponding bit in GP equals to "1" is adjusted by repeating the following procedure L times. First, for each plastic trait t_i, we calculate the difference in t_i between time t and t+1 (Δt_i) using the following equation:

$$\Delta t_i = \begin{cases} -\beta(F_0 - F_c) & \text{if } max(F_0, Fc, F_1) = F_0, \\ \beta(F_1 - F_c) & \text{if } max(F_0, Fc, F_1) = F_1, \\ 0 & \text{otherwise,} \end{cases} \quad (2)$$

where F_c represents the individual fitness of the current combinations of traits and F_0 is the individual fitness when t_i is set to 0, F_1 is the individual fitness when t_i is set to 1. Next, we actually adjust all values of the plastic traits by adding Δt_i at the same time. This process means that the individual gradually adjust its own plastic phenotypes toward fitter extreme phenotypic value ("0.0" or "1.0") in proportion to the increase in the fitness. The gray arrows in Figure

1 show examples of Δt_0 and Δt_1. The black arrow corresponds to the resultant direction and distance of learning process.

In several computational models that include Hinton and Nowlan's one, each individual performed the learning process until the fitness reached an (local) optimal value. The cost of learning was explicitly introduced into these models as fitness tax, which was proportional to the learning period. Figure 2 shows examples of learning curves. The horizontal axis is time and the vertical axis is the fitness of the individual. The explicit cost of each learning process is proportional to (a0) and (a1) respectively. On the other hand, we adopt a short learning period (L), and measure the final lifetime fitness. The inverse of the fitness increase during this period ((b0) and (b1)) corresponds to the cost of learning, and we call it the implicit cost of learning in this study. The effect of the implicit cost is equivalent to the explicit cost if the learning curves of individuals are analogous as shown in Figure 2.

3.3 Evolution

After all individuals have finished their learning processes, the population in the next generation is generated by a simple genetic operation as follows: First, the worst individual's chromosomes (GI and GP) are replaced by copies of the best individual's. Then, every gene for all individuals is mutated with a probability p_m. A mutation in GW adds a randomly generated value within the range $[-d, d]$ to the current value and a mutation in GP flips the current binary value. If a mutated phenotypic value in GI exceeds the domain of the phenotypic space, another mutation is operated on the original value again. We adopted these procedures so as to observe the gradual transitions of four indices explained previously.

4 Experiments

4.1 Experiments without Epistasis (K=0)

We have conducted evolutionary experiments using the following parameters: P=20, N=15, K=0 or 4, L=5, β=10.0, p_m=0.003 and d=0.03. The initial population was generated on condition that initial values in GI were taken at random within the range $[0, 1]$ and the proportion of "1" in GP for each individual was uniformly distributed also within the range $[0, 1]$.

First, we have conducted the experiments without epistatic interactions among loci. Figure 3 shows the course of evolution over 20000 generations with K=0. The results shown are averages over 50 trials. The horizontal axis represents the generation in logarithmic scale. We adopted the use of a logarithmic scale because the speed change is a characteristic of three step evolution through the Baldwin effect, which is clearly shown in one figure by using a logarithmic scale. The lines represent the four indices as defined previously. Specifically, the *lifetime fitness* denotes the average actual fitness among all individuals calculated after the learning process, and the *innate fitness* is the average potential fitness calculated before the learning process using initial phenotypic values. The *phenotypic plasticity* represents the average proportion of "1" in all GP s and the *phenotypic variation* is the average absolute difference between the initial value and the resultant value adjusted by the learning process among all plastic phenotypes. Also, so as to clarify the existence of the implicit cost of learning caused by epistasis, we measured the lifetime fitness of the population if all phenotypes of the all individuals were set to plastic in each generation. We term it *totally-plastic fitness*.

As shown in the transitions of these indices, the evolutionary process basically consists of the standard two-step evolution through the Baldwin effect. From the initial population, we observe an increase in both lifetime fitness and phenotypic plasticity while the innate fitness remained steady. The phenotypic plasticity rapidly rose and exceeded 0.95 at around the 31th generation. This means that more plastic individuals could obtain higher fitness and could occupy

the population due to the benefit of learning.

Next, the innate fitness slowly increased and the phenotypic variation gradually decreased until around the 7000th generation. We can regard that the genetic assimilation occurred on the learned traits because the initial phenotypic values were getting closer to resultant phenotypic values after learning. The main reason for this phenomenon is due to the limitation in the number of iterated learning processes (L).

The totally-plastic fitness was always larger than (or equal to) the lifetime fitness throughout the experiment. It means that the increase in the phenotypic plasticity could always improve the fitness in this condition. The slight decrease in the phenotypic plasticity from the 1500 to 4000th generation were supposed to be caused by the genetic drift of GP. It is because that the totally-plastic fitness is equal to the lifetime fitness, and the evolution of the population was mainly driven by the changes in the initial phenotypic values GI.

However, in contrast with the evolutionary scenario in Table 1, the phenotypic plasticity increased again and kept high even after the genetic assimilation had completely finished, despite the fact that the learning did not increased the fitness of the population at all. Thus, another role of learning must occur after the Baldwin effect. This will be discussed later in detail.

4.2 Experiments with Epistasis (K=4)

Figure 4 shows the course of evolution for K=4. We have also conducted 50 trials in this case and observed that there were several variations in the timing of the increase and subsequent decrease in the phenotypic plasticity among these trials. Also, there were some exceptional cases in which the increase in the phenotypic plasticity in the several initial generations was not so significant. This is supposed to be due to the variations in the initial population or the NK landscape. So as to focus on the three-step evolution, we have picked up 40 trials that satisfied the condition in which the phenotypic plasticity exceeded 0.9 until 100th generation, and then, became smaller than 0.85 until 1000th generation. The Figure 4 shows the averaged result over these 40 trials.

From the initial population, we observe approximately the same transitions as those for K=0 during the first step, but the peak value of the phenotypic plasticity, 0.892 (at around the 28th generation), was relatively smaller than that for K=0.

However, a clearly different scenario caused by epistatic interactions among loci was observed further on. While the lifetime fitness still slowly increased, the phenotypic plasticity slightly but gradually decreased to about 0.867 and then the phenotypic variation increased until around the 500th generation. This phenomenon corresponds to the second step in the three-step evolution through the Baldwin effect, in which the benefit and cost of learning worked together as previously described. The cost of learning is considered to bring about the decrease in the phenotypic plasticity. A contribution of each phenotypic value to the individual's fitness strongly depends on the other phenotypic values when there are epistatic interactions. Similarly, the learning in a plastic phenotype also affects the learning processes of the other plastic phenotypes. However, when we calculate Δt_i for each plastic trait t_i respectively, we do not consider any changes in the other plastic traits. Thus, the learning in too many plastic phenotypes does not always yield an effective increase in the whole fitness. Actually, we see that the totally-plastic fitness became smaller than the lifetime fitness through the second step in this case. It means that the increase in the phenotypic plasticity yielded maladaptive effects during this step. This result clearly shows that the implicit cost of learning caused by the epistasis with limitation of learning iterations actually brought about the selection pressure that decreased the phenotypic plasticity even if its decrease was slight. It should be also noticed that this implicit cost was clearly observed when there was no explicit cost of learning (such as fitness tax) because the explicit cost always tends to make the phenotypic plasticity decrease strongly.

At the same time, the benefit of learning is reflected in the steady transition of the innate fitness and increase in the phenotypic variation, because these transitions mean that the lifetime fitness increased by learning was getting more strongly dependent on the remaining plastic phenotypes.

Finally, the innate fitness eventually began to increase, however in contrast, the phenotypic variation decreased. Thus, the genetic assimilation occurred in the remaining plastic phenotypes because these initial phenotypic values were getting closer to resultant phenotypic values after learning. In comparison with K=0, the innate fitness converged to around 0.67 and the genetic assimilation did not occur completely. This step approximately corresponds to the third step in our three-step evolution through the Baldwin effect, except that the phenotypic plasticity gradually increased again to high values as observed for K=0.

4.3 Evolution of Genetic Robustness

As discussed in the previous section, we found that the three-step evolution through the Baldwin effect emerges when there are epistatic interactions among loci. However, it is still open to question why the phenotypic plasticity increased again through the last step when K=4. Here, we focus on another different role of learning, that is, the genetic robustness against mutations [3, 5, 8].

Instead of measuring the genetic robustness, we measured the genetic vulnerability in view of its adaptive property based on the following procedures: First, for every individual in each generation, we generated a copy of the individual. Then, we conducted the mutational operations on its randomly selected genes in GI (or GP) for 5 times. We defined the genetic vulnerability of GI (or GP) as the average difference between the average lifetime fitness in the original population and in the mutated individuals. Thus, the genetic vulnerability becomes smaller when the genetic robustness gets larger. Here, we focus on the increase and decrease in these indices through the course of evolution respectively.

Figure 5 shows the evolution of the genetic vulnerability of GI and GP in the same experiments as Figure 4. We see that the genetic vulnerability of GP increased and subsequently decreased. Its peak exists between the second and third step when K=4. This implies that, in the second step, the population became strongly dependent on learning despite the decrease in the phenotypic

plasticity, because the mutations tend to make the number of plastic phenotypes small during this step.

Also, we observe a peak of the genetic vulnerability of GI between the second and third step of the Baldwin effect. The increase in the second step implies that the initial values of phenotypes became more important factors for the learning processes in the other plastic phenotypes due to the epistatic effects than the previous step.

Its gradual decrease through the third step was accompanied by the increase in the phenotypic plasticity which was restrained by the implicit cost of learning in the previous step. The increase in phenotypic plasticity was caused by the selective pressure for the evolution of the genetic robustness against mutations on GI. As the genetic assimilation of the initial phenotypic values proceeds, the effects of mutations tend to become deleterious because they often make the initial phenotypic values slightly deviate from the optimal values. Thus, the existence of phenotypic plasticity becomes beneficial even after the Baldwin effect finished. Such a phenomenon is expected to occur in a situation that the initial value of each phenotype value and its plasticity are genetically determined independently, and the effects of mutations are basically deleterious. In addition, the reason why the implicit cost of learning vanished through the third step is due to the fact that epistatic effects among plastic phenotypes got smaller as the initial phenotypes approached to the learned phenotypes through the genetic assimilation in this step.

Figure 6 shows the evolution of genetic vulnerability of GI and GP in the same experiments as Figure 3. We can observe that the population maintained the phenotypic plasticity high after the population had stabilized when K=0. It more clearly shows the existence of the selection pressure on the genetic robustness, because the genetic assimilation completely finished in this case. We conducted additional experiments and confirmed that the phenotypic plasticity rapidly converged to 0.5 when the evolution of GP was driven only by the genetic drift in this case (not shown). In addition, the genetic vulnerability of GI increased and converged to around 0.00035 along with the increase in the

innate fitness.

Canalization, that is a decrease in variance of phenotypes in a population [17], has strong relationships with the evolution of phenotypic plasticity, although Mills and Watson recently pointed out that the canalization is not required for the Baldwin effect to occur [12]. There are two different types of canalization depending on the source of variation: the genetic canalization that is the evolution of the insensitivity of a phenotype to mutations, and the environmental canalization that is the evolution of the insensitivity to the environmental perturbations [6]. The evolution of the genetic robustness against mutation accompanied by the increase in the phenotypic plasticity as discussed above corresponds to the former type of the canalization in the sense that the plasticity in our model can absorb the perturbational effects on the phenotypes caused by deleterious mutations. In addition, the decrease in the phenotypic plasticity observed in the second step when K=4 in our model seems to correspond to the latter type of canalization as observed in Hinton and Nowlan's model. But it is a by-product of the adaptive evolution of phenotypic plasticity caused by the balances between benefit and cost of learning as discussed before.

5 Effects of the Number of Learning Iterations

Finally, we discuss the effects of the number of learning iterations (L) on the course of evolution. This is another parameter which is supposed to affect balances between the benefit and cost of learning, because individuals have more chances to modify its phenotypic values in order to increase their own lifetime fitness as L becomes larger. We adopted the following parameters: P=20, N=15, L=1, 3, 5, 7, 9, β=10.0, p_m=0.003, d=0.03 and K=4.

Figure 7 shows the transitions of the phenotypic plasticity when L=1, 3, 5 (the default value in previous experiments), 7 and 9. These results are averages over 10 trials for initial 3000 generations which is sufficient to discuss the effect of L on the three-step of the Baldwin effect. This figure shows that only when L=5, the clear increase and subsequent decrease in phenotypic plasticity, which

corresponds to the transition in the first and second step, occurred. When L=1 and 3, the phenotypic plasticity slowly and monotonously increased and we could not find its peak until the end. In these cases, the small number of learning iterations did not bring about so significant an increase in the lifetime fitness as to the rapid increase in the first step.

On the other hand, the phenotypic plasticity rapidly increased from the initial generations when L= 7 and 9, which is approximately similar to the case of L=5. However, we could not observe its apparent decrease in later generations in these cases. It is mainly due to the fact that the sufficient amount of the benefit of learning compensated for the effects of implicit cost of learning caused by the epistatic interactions among loci. Thus, the evolutionary scenario became similar to the two-step evolution through the Baldwin effect which was observed in the experiments without epistasis.

These results imply that a modest limitation of learning ability is required for the three-step evolution to occur. If the limitation of learning ability is too weak, the implicit cost of learning is cancelled. On the contrary, if the limitation of learning ability is too strong, the phenotypic plasticity shows no significant increase the first step. As a result, the second step does not occur in these extreme cases.

6 Conclusion

In the literature of Darwinian evolution, effects of nongenetic factors on genetic evolution (such as Waddington's genetic assimilation or the Baldwin effect) had not been treated as important mechanisms of possible evolutionary change for a long time [19], while these effects have been investigated by using theoretical or constructive approaches in the field of artificial life or complex systems for more than a decade. However, recent progresses in the molecular and developmental biology have experimentally demonstrated that these mechanisms actually exist and play important roles for genetic evolution in many aspects. Also, it is reported that the expressions of quantitative traits are based on the complex

regulations controlled by the quantitative trait loci and many environmental factors [10]. Thus, it is the time to investigate into evolutionary models based on theoretical or constructive approaches with epistatic effects in conjunction with experimental biology in order to understand these mechanisms in real environments.

As a first approach, we have discussed the quantitative evolution of phenotypic plasticity based on an extended version of the NK fitness landscape. By conducting the evolutionary experiments with various degree of epistasis, we found that a three-step evolution through the Baldwin effect emerged when the degree of epistasis was relatively large. It also turned out that the phenotypic plasticity brought about the genetic robustness against mutations after the third step of the Baldwin effect.

In conclusion, what needs to be emphasized is that the drastic changes in roles of learning emerged through the course of evolution in order, and further that each role was the main selective pressure that guided the complex evolution of phenotypic plasticity. Here, we conceptualize this phenomenon by using a hill-climbing image of a population on a fitness landscape as shown in Figure 8. Also, Table 2 shows how the benefit and cost of learning drastically changed through this phenomenon. Figure 8 shows an example of a fitness landscape which consists of all possible phenotypic combinations. Let us assume that the initial population existed on the black filled circle on the right hand. The gray region around it represents the potential area where the current population can reach through learning.

Our experiments suggest that the role of learning changes as follows: 1) The learning in many phenotypes allows the population to search adaptive phenotypes in every direction on the phenotypic space owing to the benefit of learning. 2) However, the implicit cost of learning, which is caused by epistatic interactions among plastic phenotypes with the limitation of learning ability, limits the size of the searchable area on the phenotypic space. Thus, the learning in less phenotypes enables the population to get to more adaptive phenotypic combinations by transforming the shape of the potential area as shown in Figure 8.

This corresponds to the decrease in the phenotypic plasticity and increase in the phenotypic variation. 3) If the potential area reaches a maximum phenotypic combination, the learning guides the genetic combination to approach the maximum because of the cost of learning resulted from the limit of the learning ability. This phenomenon corresponds to genetic assimilation. 4) When the genetic combination completely reaches the maximum, the learning in every direction prevents mutations from dropping down the population from the optimum. This state continues until the population looses its stability due to some kind of internal or external factors.

In this scenario, the Baldwin effect corresponds to 1)-3). It is important for such a complex evolutionary scenario to occur that the initial value of each phenotype and its plasticity are genetically determined independently, and there exist the epistatic interactions among loci and the modest limitation of learning ability. They bring about different implicit costs of learning in the second and third step as shown in Table 2. In addition, when the effects of mutations become basically deleterious after the Baldwin effect, the evolution of the genetic robustness of the initial phenotypic values against mutations can also occur.

We believe that our synthetic and conceptual investigations into the dynamic evolution of the benefit and cost of learning can help further understanding of the phenotypic plasticity in real biological systems.

References

[1] Baldwin, J. M. (1896). A new factor in evolution. *American Naturalist, 30*, 441–451.

[2] Bull, L. (1999). On the Baldwin effect. *Artificial Life, 5(3)*, 465–480.

[3] DeVisser, J. A. G. M., Hermisson, J., Wagner, G. P., Meyers, L. A., Bagheri-Chaichian, H., Blanchard, J. L., Chao, L., Cheverud, J. M., Elena, S. F., Fontana, W., Gibson, G., Hansen, T. F., Krakauer, D., Lewontin, R. C., Ofria, C., Rice, S. H., VonDassow, G., Wagner, A. and Whitlock, M. C

(2003). Perspective: Evolution and detection of genetic robustness. *Evolution, 57(9)*, 1959–1972.

[4] Downing, K. L. (2004). Development and the Baldwin effect. *Artificial Life, 10(1)*, 39–63.

[5] Edlund J. A. and Adami, C. (2004). Evolution of robustness in digital organisms. *Artificial Life, 10(2)*, 167–179.

[6] Gibson, G. (2000). Canalization in evolutionary genetics: A stabilizing theory? *BioEssays, 22*, 372–380.

[7] Hinton, G. E. and Nowlan, S. J. (1987). How learning can guide evolution. *Complex Systems, 1*, 495–502.

[8] Jen, E.(Ed.) (2005). *Robust design: A repertoire of biological, ecological, and engineering case studies*. New York: Oxford University Press.

[9] Kauffman, S. (1993). *The Origins of order: Self-organization and selection in evolution*. New York: Oxford University Press.

[10] Mackay, T. F. C. (2001). Quantitative trait loci in drosophila. *Nature Reviews Genetics, 2(1)*, 11–20.

[11] Mayley, G. (1996). Landscapes, learning costs and genetic assimilation. *Evolutionary Computation, 4(3)*, 213–234.

[12] Mills, R. and Watson, R. A. (2005). Genetic assimilation and canalization in the Baldwin effect. In Capcarrere, M. S., Freitas, A. A., Bentley, P. J., Johnson, C. G., Timmis, J. (Eds.), *Proceedings of the Eighth European Conference on Artificial Life* (pp. 353–362). Berlin: Springer.

[13] Suzuki, R. and Arita, T. (2003). The Baldwin effect revisited: Three steps characterized by the quantitative evolution of phenotypic plasticity. In Banzhaf, W., Christaller, T., Dittrich, P., Kim, J. T. and Ziegler, M. (Eds.), *Proceedings of the Seventh European Conference on Artificial Life* (pp. 395–404). Berlin: Springer.

[14] Suzuki, R. and Arita, T. (2004). Drastic changes in roles of learning in the course of evolution. In Pollack, J., Bedau, M. A., Husbands, P., Ikegami, T. and Watson, R. A. (Eds.), *Proceedings of the Ninth International Conference on the Simulation and Synthesis of Living Systems* (pp. 369–374). Cambridge, MA: MIT Press.

[15] Suzuki, R. and Arita, T. (2004). Interactions between learning and evolution: Outstanding strategy generated by the Baldwin effect. *Biosystems, 77(1–3)*, 57–71.

[16] Turney, P., Whitley, D., and Anderson, R. W. (1996). Evolution, learning, and instinct: 100 years of the Baldwin effect. *Evolutionary Computation, 4(3)*, 4–8.

[17] Waddington, C. H. (1942). Canalization of development and the inheritance of acquired characters. *Nature, 150*, 563–565.

[18] Waddington, C. H. (1953). Genetic assimilation of an acquired character. *Evolution*, 7:118–126.

[19] Weber, B. H. and Depew, D. J. (Eds.) (2003). *Evolution and learning - The Baldwin effect reconsidered* -. Cambridge, MA: MIT Press.

[20] Wiles, J., Watson, J., Tokes, B. and Deacon, T. (2005). Transient phenomena in learning and evolution: Genetic assimilation and genetic redistribution. *Artificial Life, 11(1–2)*, 177–188.

[21] Wolf, J. B., Brodie III, E. D. and Wade, M. J. (Eds.) (2000). *Epistasis and evolutionary process*. New York: Oxford University Press.

Table 1: Three-step evolution through the Baldwin effect characterized by the four indices.

step	lifetime fitness	innate fitness	phenotypic plasticity	phenotypic variation	standard interpretation
1st	increasing	steady	increasing	steady	1st
2nd	increasing	steady	decreasing	increasing	1st and 2nd
3rd	slightly increasing	increasing	steady	decreasing	2nd

Table 2: The benefit and cost of learning which caused the three-step evolution through the Baldwin effect.

step	benefit of learning	cost of learning
1st	search for an adaptive phenotypic combination on a phenotypic space in every direction by adjusting many phenotypic values	———
2nd	directional and long-distance search by adjusting the small number of phenotypic values	epistatic interactions among plastic phenotypes with limitation of learning ability
3rd	genetic robustness against mutations	limitation of learning ability
equilibrium state	genetic robustness against mutations	———

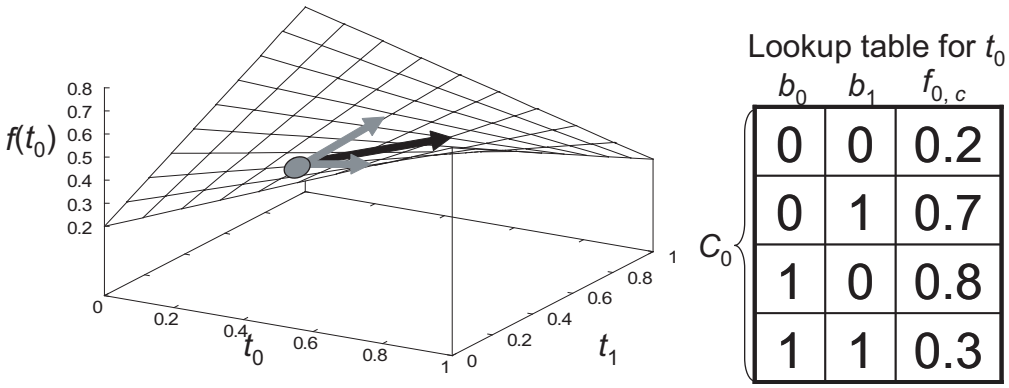

Figure 1: The example of the interpolated fitness of the trait t_0 for N=2 and K=1.

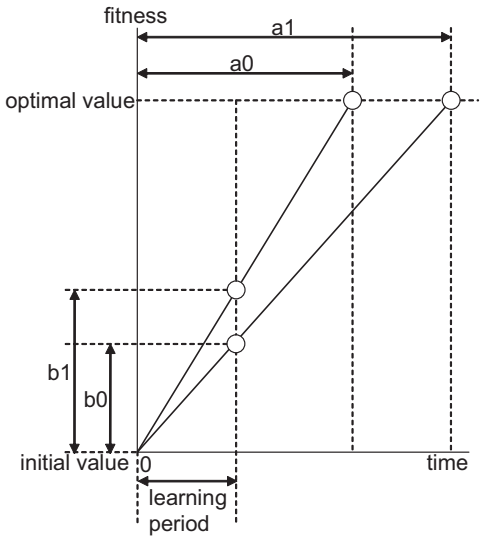

Figure 2: The learning curves.

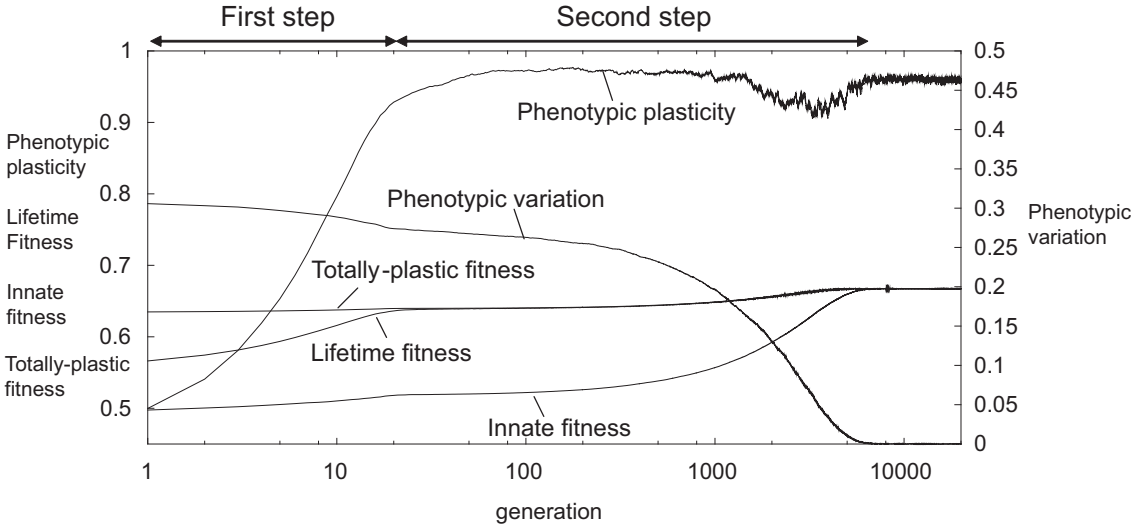

Figure 3: Evolutionary dynamics of fitness and phenotypic plasticity for K=0.

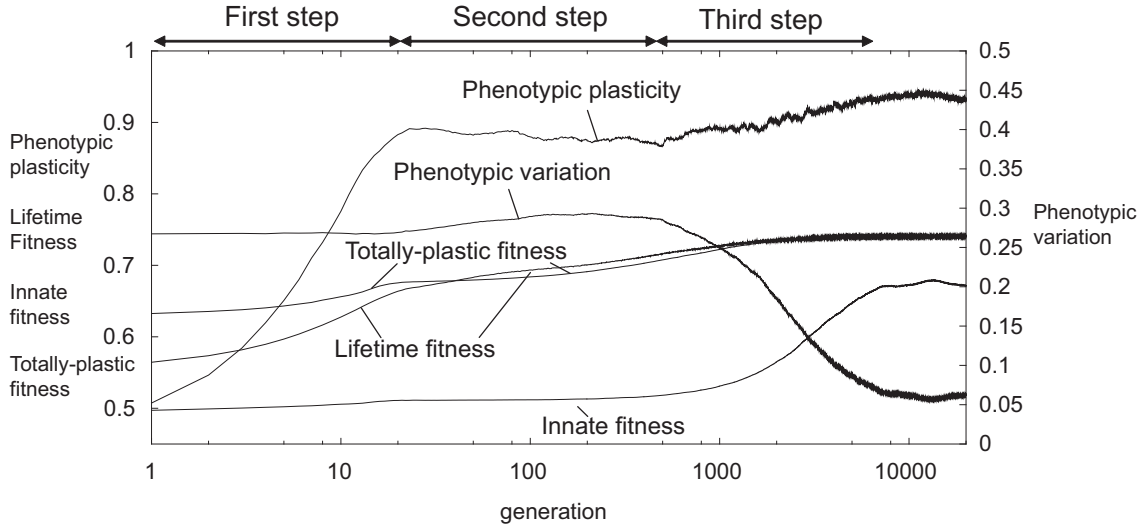

Figure 4: Evolutionary dynamics of fitness and phenotypic plasticity for K=4.

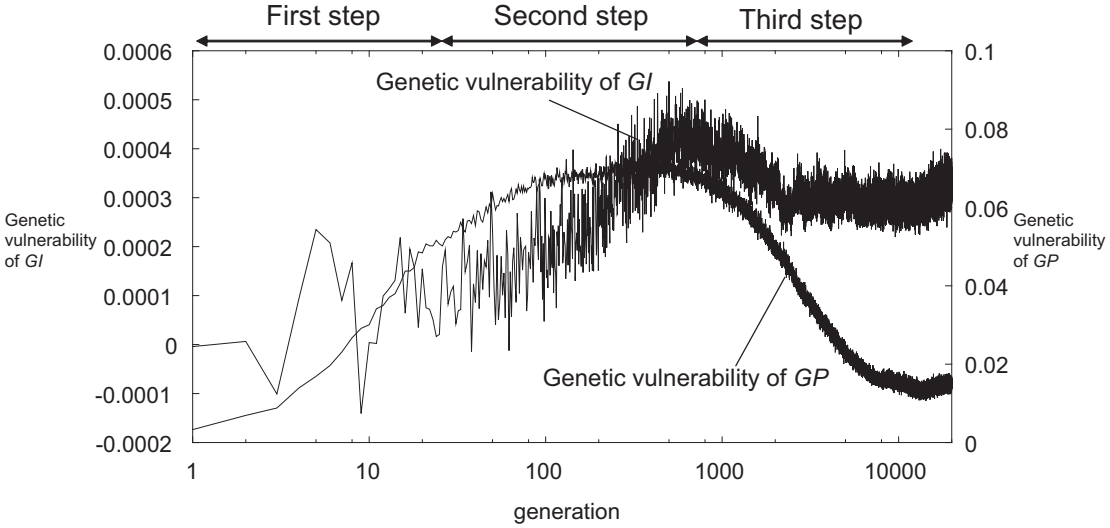

Figure 5: The evolution of the genetic vulnerability for K=4.

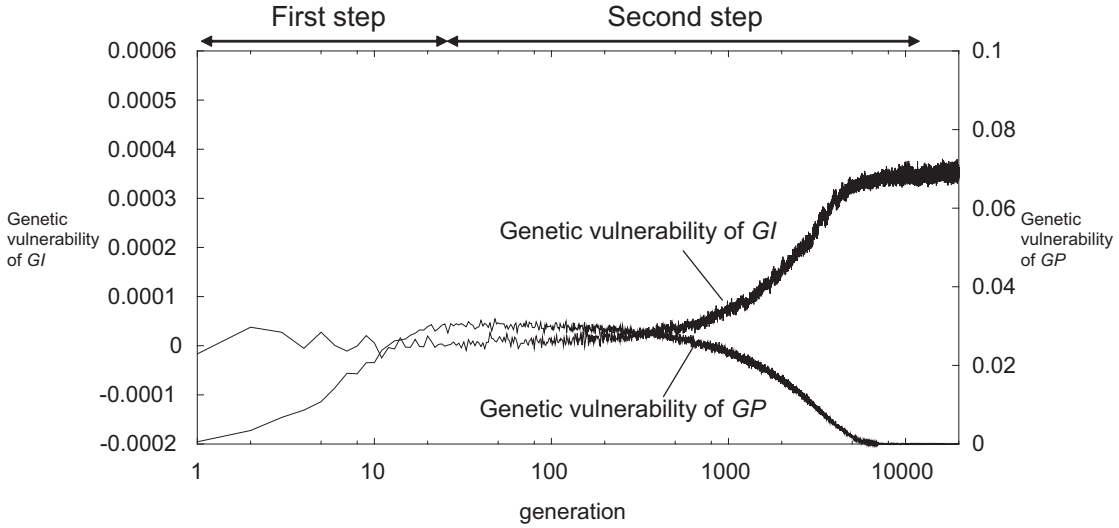

Figure 6: The evolution of the genetic vulnerability for K=0.

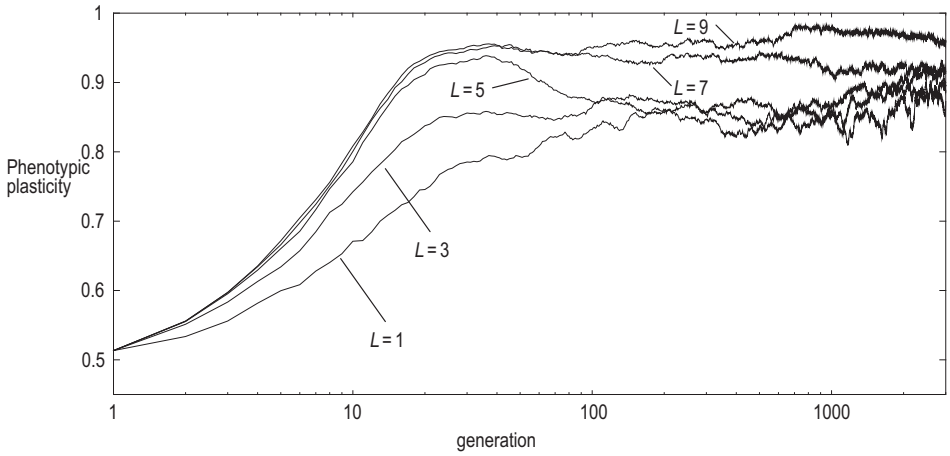

Figure 7: The evolution of phenotypic plasticity in various cases of L (K=4).

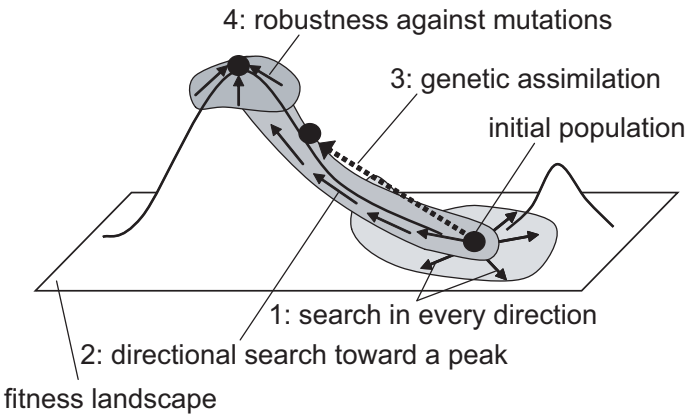

Figure 8: The roles of learning on fitness landscape.