A simple computational model of the evolution of a communicative trait and its phenotypic plasticity

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Abstract

We consider a simple computational model of the evolution of a quantitative trait and its phenotypic plasticity based on directional and positive frequency-dependent selection in order to explore whether and how leaning might facilitate evolution under the dynamics that arise from communicative interactions among individuals. In the model, each individual expresses, at many different times in its lifetime, its real-valued trait depending on the probability distribution determined by its own genotypes. In communicative interactions between two individuals, the contribution of an interaction to the fitness is high when their trait values are close to each other as well as large, which represents the positive frequency-dependent and directional components of selection, respectively. The iterative interactions allow individuals to acquire a more adaptive trait pair through trial and error. Under the stochastic evolution process with the limited number of individuals, we show that learning allows the population to avoid getting stuck in the global but low optimum of the innate and individual-level fitness landscape via both aspects of the components of selection, and brings about the successful evolution by increasing the genetic variation of the population. We also analyze how such an effect of learning can be realized by measuring the degree of the two different contributions for increasing the adaptivity and similarity of communicative traits, respectively. We show that this effect of learning arises from these different types of contributions depending on the biological and environmental conditions such as the mutation rate and the duration of

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communicative interactions. We further show the condition for the complete genetic assimilation to occur.

Keywords: Baldwin effect, frequency-dependent selection, learning, fitness landscape, genetic algorithm.

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1. Introduction

Phenotypic plasticity refers to the variation in the phenotype of a given genotype when individuals undergo development in different environments (West-Eberhard, 2003). A wide range of organisms exhibit traits with adaptive plasticity from a plasticity in morphological traits such as a predator-induced polyphenism in insects to human behavioral adaptation based on intelligent learning mechanisms (Gilbert and Epel, 2009). The ontogenetic adaptation based on the phenotypic plasticity is recognized as one of the key factors that brings about the adaptive evolution of novel traits in recent evolutionary biology (West-Eberhard, 2005; Gilbert and Epel, 2009). Wund provided a summary of eight hypotheses on how plasticity might influence evolution with several empirical supports, mainly focusing on the adaptation to a new environment (Wund, 2012). For example, the hypotheses include that phenotypic plasticity promotes persistence in a new environment and also include the hypothesis that a change in the environment can release cryptic genetic variation via phenotypic plasticity, in turn impacting the rate of evolutionary responses.

In particular, the Baldwin effect (Baldwin, 1896) is known as a hypothesis on the indirect effects of learning and ontogenetic adaptations on evolution, especially on a process in which an acquired trait, through the initial interactions between individuals and their environment, can become a genetic trait without direct inheritance of the acquired trait (so-called Lamarckian mechanism), although the concept of the Baldwin effect differs by authors (see (Weber and Depew, 2003; Turney et al., 1996; Pigliucci et al., 2006; Crispo, 2007)). Since this effect has been demonstrated by the seminal studies based on computational (Hinton and Nowlan, 1987; Maynard-Smith, 1987) and mathematical (Ancel, 1999) approaches, it has been one of the important topics in the interdisciplinary fields of mathematical biology (Sznajder et al., 2012), and artificial life or artificial intelligence (Belew and Mitchell, 1996; Turney et al., 1996; Suzuki and Arita, 2007a). An important finding is that benefit and cost of learning can accelerate or decelerate adaptive evolution by

increasing or decreasing the slope of the surface of adaptive landscapes (Price et al., 2003; Mills and Watson, 2006; Suzuki and Arita, 2007a; Paenke et al., 2009). Recently, this effect has also been observed empirically in the evolution of learning behavior for oviposition choice by *Drosophila melanogaster* (Mery and Kawecki, 2004) and in the colonization process of North America by the house finch (Badyaev, 2009) as well as in the landmark experiments of the evolution of morphological development of flies shown by Waddington (Waddington, 1953).

Effects of learning on evolution have been discussed in various environmental situations including complex environments that create rugged fitness landscapes (Borenstein et al., 2006; Suzuki and Arita, 2007b), an extraordinary environment after a sudden change (Lande, 2009), regularly or irregularly changing environments (Sasaki and Tokoro, 1997; Ancel, 1999; Curran et al., 2007; Arnold et al., 2010), socially learning populations in structured environments (Jones and Blackwell, 2011; Kobayashi and Wakano, 2012) and so on. In this paper, we particularly focus on the effects in the context of communicative or game-theoretical interactions such as the iterated prisoner's dilemma (Suzuki and Arita, 2004; Tanabe and Masuda, 2012), signaling games (Zollman and Smead, 2010), coordination games (Suzuki and Arita, 2008), producer-scrounger games (Katsnelson et al., 2012), and linguistic interactions (Turkel, 2002; Munroe and Cangelosi, 2002; Watanabe et al., 2008; Chater et al., 2009; Yamauchi and Hashimoto, 2010; Azumagakito et al., 2012; Suzuki and Arita, 2012). It has been reported that the evolution of learning abilities can facilitate the shift of the population from less adaptive to more adaptive in this type of dynamic environments (Suzuki and Arita, 2004; Zollman and Smead, 2010; Suzuki and Arita, 2008, 2012; Tanabe and Masuda, 2012). Suzuki and Arita focused on the evolution of strategies for iterated Prisoner's Dilemma (Suzuki and Arita, 2004). They introduced a learning rule termed as Meta-Pavlov learning into strategies as phenotypic plasticity that 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 defective populations to cooperative populations. Second, these strategies were replaced by a strategy with a modest amount of plasticity. Tanabe and Masuda recently showed the similar scenario using a simpler reinforcement learning with adaptive aspiration levels (Tanabe and Masuda, 2012). Zollman *et al.* analyzed a simple model of evolution of language based on Lewis's signaling game as well as the model of prisoner's dilemma (Zollman and Smead, 2010). They observed that the

presence of plastic individuals alters the trajectory of evolution by directing the population away from non-adaptive signaling population and toward the optimal population, which was termed the *Baldwin optimizing effect* by them. Suzuki and Arita also showed that such an adaptive shift can occur repeatedly using a computational model of the coevolution of sending and receiving behaviors of signals and their plasticity (Suzuki and Arita, 2008, 2012). These studies imply that learning could be an important driving force for the adaptive evolution in the broader context of communicative interactions. However, as far as we know, there still have been no clear attempts to understand it explicitly from this point of view.

In the study reported here, we explore whether and how leaning can facilitate evolution under the dynamics that arise from communicative interactions among individuals. We focus on the evolution of the fundamental traits underlying communicative interaction such as the rules or conventions for successful communication that can bring about collective behaviors. We assume that such traits can evolve under a directional selection because the traits can be modified incrementally so that they can bring about more benefit from communicative interactions. At the same time, we also assume such traits must be shared by those who wish to communicate. Accordingly, at least some of the selection will be positive frequency-dependent. This might prevent the evolution based on directional selection. We believe that this abstracts an fundamental problem of the evolution of communicative traits in general context. For example, in language evolution it has been pointed out that mutations in grammar cannot be beneficial because a mutant's peers might not understand him (Pinker and Bloom, 1990; Glackin, 2010). Our question is whether and how such a negative effect of frequency-dependent selection can be overcome by the evolution of phenotypic plasticity, and thus the population can evolve by the directional selection.

For this purpose, we construct a minimal model of the evolution of a quantitative trait for communicative interactions and its phenotypic plasticity based on a genetic algorithm. In the model, each individual expresses, at many different times in its lifetime, its real-valued trait depending on the probability distribution determined by its own genotypes. In communicative interactions between two individuals, the contribution of an interaction to the fitness is higher when their trait values are closer as well as larger, which represents both the positive frequency-dependent and directional components of selection. The iterative interactions allow individuals to acquire a more adaptive trait pair through trial and error.

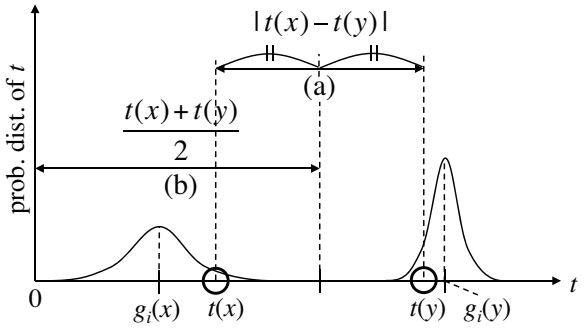

Figure 1: A communicative interaction between individuals and the corresponding fitness evaluation. The x-axis represents the trait value and the y-axis corresponds to the probability distribution of the trait values $t(x)$ and $t(y)$ when $g_p(x)$ is larger than $g_p(y)$. In this case, $t(x)$ tends to be more distant from $g_i(x)$ than $t(y)$ from $g_i(y)$ due to the higher degree of phenotypic plasticity of the individual x. The fitness contribution of these set of traits is calculated by using (a) and (b). The similarity and adaptivity correspond (a) and (b), respectively.

In the experiments, we first show that such a positive frequency-dependent selection creates a global but lower optimum of the innate and individual-level fitness landscape, depending on the fixation of that fitness landscape. This retards adaptive evolution. Then, we show that learning based on the evolving phenotypic plasticity allows the population to avoid getting stuck in that optimum, and brings about the successful evolution of communicative traits by increasing the genetic variation of the population in general. We also analyze how such an effect of learning can be realized by measuring the degree of the two different contributions for increasing the adaptivity and similarity of communicative traits, respectively. We show that this effect of learning arises from these different types of contributions depending on the biological and environmental conditions such as the mutation rate and the duration of communicative interactions. We further discuss the condition in which the typical evolution process of the complete genetic assimilation (Waddington, 1953) occurred.

2. Model

There are N individuals in the population. Each individual, numbered x, has a real-valued trait whose value $t(x)$ is determined by a pair of real-valued

genotypes: the innate trait value $g_i(x)$ and its phenotypic plasticity $g_p(x)$. Both are $\in [0, \infty)$. Note that this trait will be expressed many different times in the organism's lifetime.

A pseudocode of the model is shown in Algorithm 1. In each generation, $N/2$ pairs of two individuals are created randomly without duplication of individuals. For each pair, C steps of communicative interaction are conducted. The parameter C could be regarded as the duration of communicative interactions. We assume individuals have the ability to improve their communicative traits through simple learning iterations. Each step is composed of two phases: a trial phase for a new trait set and a choice phase of the adaptive trait set. In the trial phase, each communicating individual x has a new trait value $t(x)$, generated using the following equation:

$$t(x) = N(g_i(x), g_p(x)^2), \tag{1}$$

where $N(\mu, \sigma^2)$ is a function that returns a random value from a Gaussian distribution with mean μ and standard deviation σ. One can think of $g_i(x)$ determining the innate trait value of $t(x)$ and $g_p(x)$ determines the degree of deviation of $t(x)$ from $g_i(x)$ due to the phenotypic plasticity as illustrated in Fig. 1.

The traits of individuals x and y, $t(x)$ and $t(y)$, jointly makes a fitness contribution $f_c(x, y)$ to each individual:

$$f_c(x, y) = e^{-|t(x)-t(y)|} \times \frac{t(x) + t(y)}{2} \tag{2}$$

The smaller the difference between the two trait values ((a) in Fig. 1) is and the higher the average of the two traits is ((b) in Fig. 1), the higher the fitness contribution becomes. This situation can be interpreted in terms of communicative interaction as follows: The trait value represents a fundamental communicative feature that needs to be shared between individuals for successful communication (e.g., a communication channel or linguistic convention). Both individuals can obtain a communicative benefit only when their trait values are close enough. The trait value represents the maximum benefit from a communication when both individuals adopt exactly the same trait value (e.g., the same linguistic convention). Thus, higher trait values have the potential to produce larger communicative benefits, but whether that potential can be realized depends on the proximity of the communicating individuals' trait values. Consequently, there is a possibility that the

Algorithm 1 A pseudocode of the model. $fitness_x$ represents the lifetime fitness of the individual x. *similarityCont* and *adaptivityCont* represent the similarity contribution and the adaptivity contribution, respectively.

generate a population of N individuals.
for g=0 to $G-1$ **do**
  create $N/2$ pairs of individuals randomly without duplication.
  for each pair of the individual x and y **do**
    $fitness \Leftarrow 0$
    $bestFitness \Leftarrow 0$
    for c=0 to $C-1$ **do**
      (trial phase)
      $t(x) = N(g_i(x), g_p(x)^2)$
      $t(y) = N(g_i(y), g_p(y)^2)$
      $f_c(x,y) = e^{-|t(x)-t(y)|} \times \frac{t(x)+t(y)}{2}$
      (choice phase)
      if $f_c(x,y) \geq bestFitness$ **then**
        $bestFitness \Leftarrow f_c(x,y)$
        $similarityCont \Leftarrow |g_i(x) - g_i(y)| - |t(x) - t(y)|$
        $adaptivityCont \Leftarrow (t(x) + t(y))/2 - (g_i(x) + g_i(y))/2$
      end if
      $fitness \Leftarrow fitness + bestFitness$
    end for
    $fitness \Leftarrow fitness/C$
    $fitness_x \Leftarrow fitness$
    $fitness_y \Leftarrow fitness$
  end for
  generate the next generation using a fitness-proportional selection with mutations.
end for

evolution process of the population becomes very slow if the number of individuals is limited and the evolution process is stochastic, while there always exists a possibility that the population can become more adaptive by collectively adopting a higher trait value. The pair of individuals estimates the fitness contribution of the set of these new traits.

Then, in the choice phase, the agent pair adopts the best trait combination among the ones estimated in the all previous (trial) phases. In other words, they update the trait combination only when it brings the better fitness contribution than those estimated ones in preceding communicative interactions. The final fitness contribution in this step is calculated from the trait set that has been adopted in the choice phase rather than the one estimated in the trial phase. Thus, the fitness contribution increases or does not change with each step.

The *lifetime fitness* of each individual, which determines reproductive success, is defined as the average of the fitness contributions received over its lifetime (C steps). The offspring in the next generation are selected from the current population using "roulette wheel selection" based on the Monte Carlo method (in which the probability that an individual will be chosen as a parent is proportional to its lifetime fitness). Then, $g_i(x)$ and $g_p(x)$ of the all offspring is mutated with a probability p_{mi} and p_{mp}, respectively. A mutation adds a randomly selected value from $N(0, \sigma_m^2)$ to the current value. If the resulting value is < 0, another mutation is applied to the original value again. This evolutionary process is repeated for G generations.

3. Results

We analyzed whether and how learning can facilitate the evolution of more adaptive communicative traits with different settings of the number of learning iteration C and the mutation rate $p_m = p_{mi} = p_{mp}$. We used $N = 300$ and $\sigma_m = 1.0$. The initial population was generated with initial values of $g_i(x)$ sampled from $|N(0, 1)|$, and initial values of $g_p(x)$ were all 0.

Fig. 2 shows example evolution processes with the several conditions of C and p_m, showing that the lifetime fitness tended to increase more successfully as C and p_m increased. Fig. 3 is the summarized results of the all experiments for detailed quantitative analyses. There are 10 subfigures (a)-(j). (a)-(g) represent several average values in the last ($G = 20,000$th) generation, respectively. (h)-(j) also represent different indices explained later. The horizontal and vertical axes correspond to the experimental settings of the

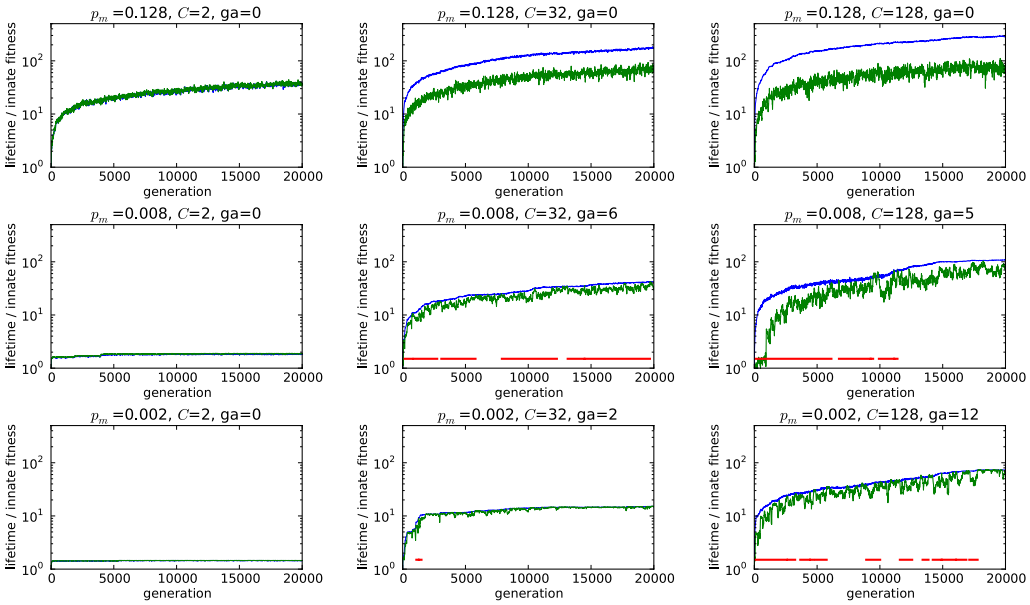

Figure 2: The example evolution processes with the several conditions of C and p_m. The blue (upper) line represents the average lifetime fitness and the green (lower) line represents the average innate fitness. Each red (bottom) straight-line segment also corresponds to the period counted as a single occurrence of the Baldwin effect (genetic assimilation) in Fig. 3 (h).

number of learning iteration C and the mutation rate p_m respectively. The additional labels "N" and "I" on the horizontal axis correspond to the two different control experiments based on neutral evolution and the evolution of the innate traits without learning respectively, which will be defined later. These values are averages over 100 trials for each experimental setting. Note that we regarded the values less than 0.001 as 0.001 in Fig. 3, in order to omit to show extremely small values in the logarithmic scale. We did not observe negative values smaller than -0.001. We focused on the various indices at the last (G=20,000th) generation because they reflect the tendency of evolution in each condition although the evolution process still continued in some cases.

3.1. Control experiments based on neutral evolution and evolution with no plasticity

First, we focus on the results of the two control experiments in order to show the effects of a positive frequency-dependent selection that can prevent the adaptive evolution of communicative traits in this model.

One is evolution based on neutral evolution of traits. In these experiments, we fixed the lifetime fitness of all individuals to 1 so that all individuals are selected completely at random through the evolution process. In Fig. 3, the data with the label "N" correspond to the results in this case with different mutation rates. Fig. 3 (a) shows that the lifetime fitness was fixed as 1 (by definition). The average and the standard deviation (SD) of the innate trait value $g_i(x)$ increased as the mutation rate p_m increased as seen in Fig. 3 (c) and (d). This is merely due to the genetic drift with higher mutation rates in the positive domain of the trait value. Under neutral evolution, the higher mutation rate can bring about the faster deviation from the initial trait values and the higher genetic variation of the population. The $g_p(x)$ also showed the similar tendency, which is also due to the same reason as the one for $g_i(x)$.

The data with the label "I" correspond to the results in the other control experiment with evolution but without phenotypic plasticity or learning. We defined the *innate fitness* as the evaluation value obtained if each individual's plasticity $g_p(x) = 0$, meaning that it uses the innate trait value $g_i(x)$ as the trait $t(x)$ for evaluation ($t(x) = g_i(x)$). We fixed $g_p(x)$ as 0 in this experiment. Fig. 3 (c) shows that the average innate trait $g_i(x)$ evolved to be the smaller values than those in the corresponding cases of the neutral evolution, although the innate fitness slightly increased as the mutation rate increased. The standard deviation of $g_i(x)$ became also much smaller but slightly increased as the mutation rate increased. This is expected to be due to the strong positive frequency-dependent selection for the dominant trait value in the population. When the mutation rate was small, we often observed that the increase in the average $g_i(x)$ temporally became very slow over many generations, meaning that the population got stuck on a single peak of the innate and individual-level fitness landscape (explained in detail later). This is expected to be (at least partly) due to the limited number of individuals and the stochastic processes of mutation and selection, because the adaptive evolution toward higher trait values is always theoretically possible in any situations of the population. In such a situation, mutant individuals who had higher trait values obtain lower fitness than the one obtained by the

communication between the individuals with dominant trait values. This prevented the population from evolving toward higher trait values. However, when the mutation rate was high enough, the population tended to move slowly toward the higher trait values because some mutant individuals with higher trait values often succeed in adaptive communication with each other using higher trait values. Note that the evolution process showed the similar tendency when we conducted the experiments with higher mutation rates. This is expected to be due to fact that genetic values do not deviate from the original values so significantly even when a mutation occurs almost every generation, because of the small value of σ_m we adopted here[1].

In sum, there is the strong positive selection pressure on the innate traits for communicative interaction if the individuals cannot acquire the adaptive trait through learning processes. However, the high mutation rate enables the population to evolve to acquire the more adaptive traits even in this situation.

3.2. Effects of learning on evolution

In order to grasp the general dynamics of the evolution, we show example evolution processes with the several conditions of C and p_m as illustrated in Fig. 2. Each subfigure shows the change in the average lifetime and innate fitness of the population in a trial with the corresponding condition. In all cases, both the lifetime and the innate fitness increased from the small values around 1. They often tended to stay around small values when C and p_m were small, while they gradually increased when C or p_m were large. We also see that the lifetime fitness tended to be much higher than the innate fitness as these parameters increased.

We analyzed quantitatively whether and how learning facilitated the adaptive evolution of communicative traits as shown in Fig. 3. When we focus on the evolution of the fitness, it turned out from Fig. 3 (a) and (b) that both the lifetime and innate fitness in all cases with learning were higher than those in the corresponding cases with the same p_m without learning (or no plasticity) in the control experiments ("I"). We also see that they increased gradually as p_m increased, which is clearly due to the increased genetic variation as explained before. However, it should be noticed that such

[1]We also conducted experiments with σ_m=10.0. We found that there was the peak of the lifetime fitness when p_m was intermediate ($p_m = 2.0^6 \times 10^{-3}$) in the case of "I".

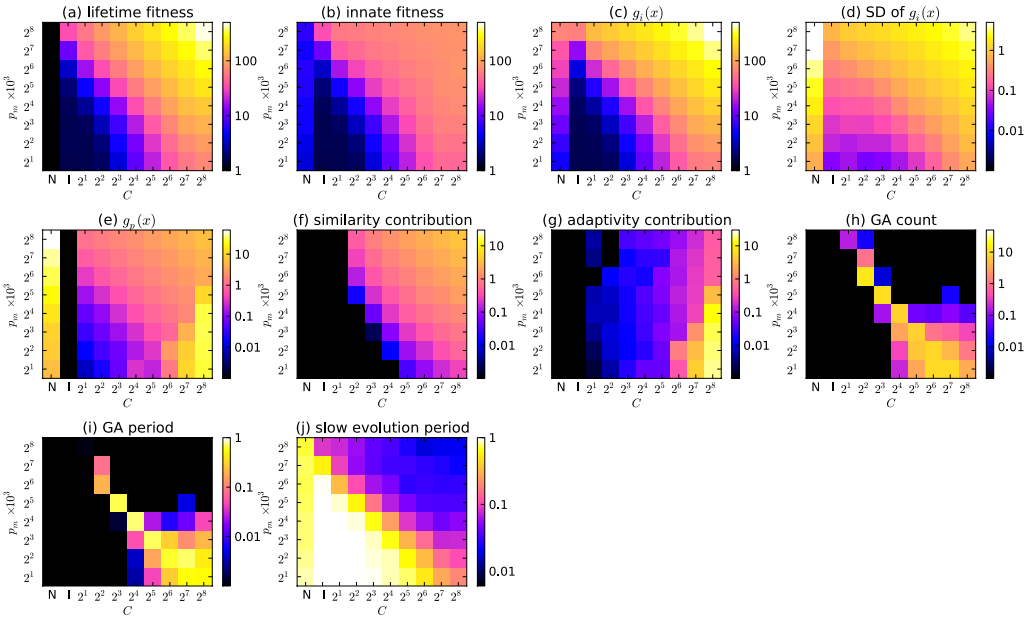

Figure 3: The statistics of the evolution process with different settings of the mutation rate (p_m) and the number of learning iterations (C). "N" and "I" represent the control experiments of the neutral evolution and the evolution with no plasticity respectively.

contribution of the high mutation rate to the fitness increase was increased significantly as the duration of communication C increased. In this sense, learning can facilitate the adaptive evolution of both acquired and innate traits in this model.

Next, we focus on the evolution of genotypes. Fig. 3 (c) and (d) shows that both the average and the standard deviation of the innate trait $g_i(x)$ became larger as these parameters increased. This high genetic variation is expected to contribute to the adaptive evolution of the population because the individuals with the large innate trait values can quickly establish more adaptive communication with learning individuals. However, instead, this variation made the innate fitness much smaller than the lifetime fitness as C or p_m increased, meaning that the population tended to become dependent on learning more strongly. Fig. 3 (e) shows that this genetic variation of innate traits was brought about by the successful evolution of necessary and sufficient degree of the phenotypic plasticity. Interestingly, we see that there are the two different peaks of the phenotypic plasticity on this two-

dimensional space of the experimental conditions of C and p_m. A lower peak exists on the condition in which both C and p_m were the highest, and the significantly higher peak existed on the condition in which C was the highest but p_m was small ($p_m = 2.0^3 \times 10^{-3}$). Specifically, the plasticity increased monotonously as p_m increased when C was small ($\leq 2^5$) and it was the highest when p_m was the largest. Although such a tendency still exists when C was high ($\geq 2^6$), another peak with the significantly high value also appears when p_m was small. This implies that there are two different way of contribution of learning to evolution when the duration of communication is sufficiently long.

In order to quantify how the evolved plasticity contributed to the fitness increase through learning process, we measured the two indices. One is the similarity contribution $|g_i(x) - g_i(y)| - |t_l(x) - t_l(y)|$, where $t_l(x)$ represents the trait value of the individual x at the last step. This is the decrease in the difference between the last (best) trait values in pairs from that between their innate values $g_i(x)$. The other is the adaptivity contribution $(t_l(x) + t_l(y))/2 - (g_i(x) + g_i(y))/2$. This is the increase in the average trait values in pairs from their average innate values $g_i(x)$. The former corresponds to the degree to which the learning process decreased the gap between the two trait values of communicating individuals, and the latter corresponds to the degree to which the learning process increased the potential adaptivity of the trait values. The higher each index is the higher contribution to the lifetime fitness that individuals receive from the communicative interaction. We see from Fig. 3 (f) and (g) that both indices gradually increased as C increased due to the increased number of chances to obtain the better trait set. We also see that the similarity contribution increased gradually as p_m increased while the adaptivity contribution decreased in general. The gradient of the similarity contribution well coincides with the gradient of the plasticity toward the lower peak that exists on the condition in which both C and p_m was the highest. This means that the evolved plasticity in the corresponding conditions mainly contributed to keep less innately adaptive individuals from being eliminated from the population by increasing their lifetime fitness through learning processes. This is also expected to have increased the intrinsic genetic variation significantly as shown in Fig. 3 (d).

On the other hand, the gradient of the adaptivity contribution well coincides with the gradient of the plasticity toward its higher peak that exists on the condition in which C was the highest but p_m was small ($p_m = 2.0 \times 10^{-3}$). This means that the evolved high plasticity in the corresponding condi-

tions mainly contributed to enable the potentially adaptive individuals with slightly higher innate trait values to acquire much higher lifetime fitness by making use of the large number of learning iterations. This is expected to have brought the sufficient amount of genetic variation for the population to evolve successfully even when there was the small genetic variation due to the low mutation rate.

Finally, we focus on how often the Baldwin effect or genetic assimilation occurred in each experimental condition. We regard the complete genetic assimilation as a process in which an acquired trait, through the initial interactions between individuals, can become a genetic trait in that the individuals do not require any learning abilities to express that trait. In our model, such an assimilation process occurs only when the innate phenotypes in the population become a unique value and the plasticity becomes sufficiently small, meaning that the lifetime fitness and the innate fitness become the same. Thus, in each trial, we checked the occurrence of the evolution process in which the difference in the average lifetime fitness and the average innate fitness in the population becomes higher than 1 at least for 100 generations, and then subsequently becomes less than 1, and the lifetime fitness increased by more than 1 during the process itself. Fig. 3 (h) shows the average count in each condition and Fig. 3 (i) represents the average proportion of generations during which the population was in this situation over all generations. Each red (lower) bar in Fig 2 also show the duration of a single occurrence of this process. We see that such an assimilation process occurs often in the limited cases when the effects of mutation and learning are well balanced in that the speed of evolution of innate and lifetime fitness are quite similar. Especially, it occurred more often when the mutation rate was small but the duration of interactions was long and the population tended to be in this situation for longer generations as shown in Fig. 3 (i), implying that the genetic assimilation played an important role throughout the adaptive evolution process in these cases. On the other hand, it rarely occurred in the other conditions.

We further investigated the effects of learning on the evolution of the innate traits by focusing on the occurrences of its very slow evolution process quantitatively. We defined the situation in which the evolution of the innate fitness was slow temporally as a period in which it took more than 1000

generations for the average $g_i(x)$ to increase by 0.5 [2]. Fig. 3 (j) shows the average proportion of generations during which the population was in this period over all generations. It turned out that when p_m and C were small, the evolution of the population tended to be slow for long (or almost all) generations. As both parameters increased, it tended to become slow less often, showing that learning can facilitate adaptive evolution. It should be noticed that the amount of reproductive advantage the fittest organisms have will decrease as the average fitness increase in this model, because we adopted the reproductive success depends on the relative difference in fitness. Fig. 3 (j) might reflect this effect of this fitness definition on evolution to some degree especially in the cases where the average lifetime fitness reached higher values.

Besides, we have conducted additional experiments under the condition that each individual communicates with 10 other individuals in each generation. We found that the general tendency of evolution process was qualitatively the same as the one explained above, although the lifetime and innate fitness tended to become small because they were necessary to communicate with different individuals with different trait values.

In sum, we found that the necessary and sufficient amount of phenotypic plasticity evolved and it facilitated the adaptive evolution of the communicating individuals by increasing the genetic variation of the innate traits for the directional selection to work constantly.

4. Discussion

In order to understand the roles of learning in evolution observed in this model, we visualize the observed scenario using a concept of a dynamically changing fitness landscape. Fig. 4 depicts the movement of the population on a dynamic fitness landscape. The horizontal axis represents the innate trait value $g_i(x)$. We focus on an individual x of which $g_i(x)$ is represented as an open circle. The distribution of $g_i(x)$ of the other individuals in the

[2]Specifically, we conducted the following procedures: 0) We regard a start generation (g_s) as the initial generation. 1) From g_s, we incrementally search for the end generation (g_e) in which the average $g_i(x)$ becomes higher than the one in g_s by 0.5. 2) The period from g_s to g_e is regarded as a slow evolution period if it is longer than 1000 generations. 3) g_e is regarded as a new g_s, and the process goes back to 1) until the search reaches the last generation.

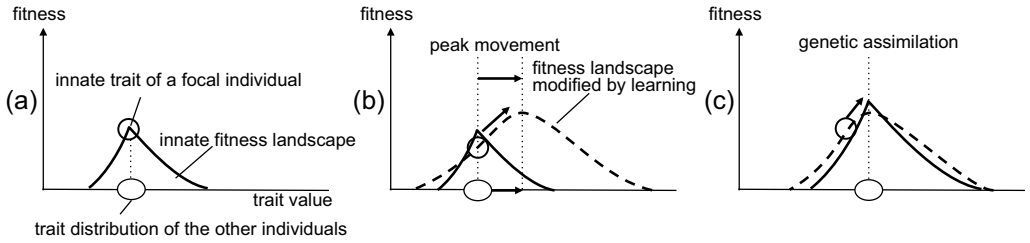

Figure 4: A visual conceptualization of adaptive evolution under both directional and positive frequency-dependent selection facilitated by the phenotypic plasticity, based on an individual-level fitness landscape.

population is represented as an open oval. The solid line shows the innate fitness of the focal individual, and the dashed line shows the lifetime fitness of the focal individual.

Let us start from the state in which the traits of the population have become a specific value i.e. the population exists on a peak of the innate fitness landscape as shown in Fig. 4 (a). In this situation, all individuals obtain the benefits from communicative interactions due to the highest coherence of their communicative traits. However, the fitness reward associated with communicative success can be improved by collectively moving to a higher trait value. But, the population cannot move if the individuals have no learning ability: mutant individuals with trait values higher than the dominant value will be communicatively unsuccessful if they cannot adapt to accommodate communication with the dominant trait value.

If individuals have an ability to learn, they can dominate the population. This increases the height of the peak of the learned fitness landscape and broaden its base as shown in Fig. 4 (b). Furthermore, it moves the peak of the learned fitness landscape toward the larger trait values. This enables the individual to move to the dynamically appearing higher peak of the innate fitness landscape because the trait distribution of the other individuals also moves toward the larger trait values. This directional evolution can occur continuously creating higher peaks, but the population sometimes completely reaches a peak due to the genetic assimilation of the acquired trait as shown in Fig. 4 (c).

Fig. 5 shows an example of the (a) innate and (b) lifetime fitness landscapes in the population of two individuals x and y, which supports our scenario. The x and y axes represent innate trait values of two individuals

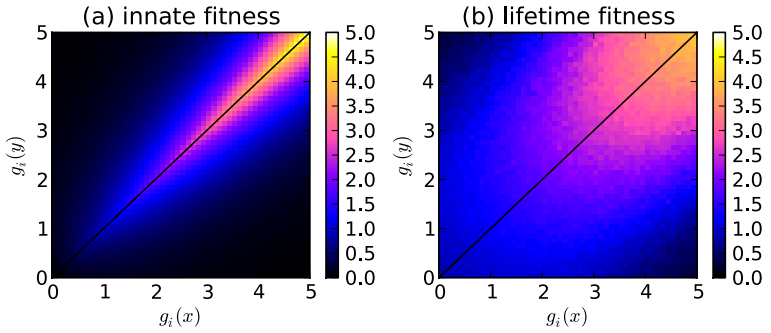

Figure 5: An example of the landscapes of (a) innate and (b) lifetime fitness in the population of two individuals. We used $g_p(x) = g_p(y) = 1.0$ and $C = 16$. The black line corresponds to the condition $g_i(x) = g_i(y)$.

$g_i(x)$ and $g_i(y)$. We used $g_p(x) = g_p(y) = 1.0$ and $C = 16$ for calculation of their lifetime fitness. The values are the average over 100 trials. We see from Fig. 5 (a) that there is a narrow and steep ridge on the condition that the innate traits are identical. The population gets stuck in the ridge if the population is basically allowed to move only in a horizontal or vertical direction due to a small mutation rate, which corresponds to the state in Fig. 4 (a). On the other hand, we see a broad and gentle ridge in Fig. 5 (b). This makes the genetic variation of the innate traits large. Furthermore, if we assume the innate trait value of an individual is fixed, there is a peak on the condition that the innate trait of the other individual is larger than the fixed one. This movement of the peak allows the population to evolve gradually, which corresponds to the state in Fig. 4 (b). In addition, we also see that the height of the ridge of the lifetime fitness is slightly lower than the one of the innate fitness due to the implicit cost of learning caused by the increased phenotypic variations, which makes the genetic assimilation to occur and corresponds to the state in Fig. 4 (c). The reason why the population can evolve slowly toward more adaptive communications even without learning under the condition of high mutation rates is expected to be due to the fact that the population is sometimes allowed to move in a diagonal direction in the innate fitness landscape because many mutant individuals with higher trait values can appear in the population.

Hinton and Nowlan's pioneering work (Hinton and Nowlan, 1987) clarified that learning can facilitate evolution on a "needle in a haystack" fitness landscape. Also, it is sometimes pointed out that learning enables the population

to cross valleys on rugged fitness landscape (Price et al., 2003; Borenstein et al., 2006; Suzuki and Arita, 2007b). Among these discussions, the role of learning in evolution is conceived of merely smoothing the shape of a pre-existing rugged fitness landscape. In the scenario presented here, under the stable condition in which the population got stuck in the peak of the innate fitness landscape due to the strong positive frequency dependent selection, the communicative interactions among individuals with learning ability can bring about movement of peaks of the learned fitness landscape, which could be regarded as an important role of learning in evolution on dynamically changing fitness landscapes.

The evolutionary significance of the Baldwin effect or genetic assimilation has been discussed in the context of language evolution (Pinker and Bloom, 1990; Szathmáry, 2010; Glackin, 2010), and several studies have discussed how and what kind of linguistic abilities could be genetically acquired by the Baldwin effect (Turkel, 2002; Munroe and Cangelosi, 2002; Watanabe et al., 2008; Chater et al., 2009; Yamauchi and Hashimoto, 2010; Suzuki and Arita, 2008, 2012). Jablonka and her colleagues proposed the *assimilate-stretch principle*, which states that the genetic assimilation of learned behaviors makes the individuals' learning ability residual and enables them to learn other additional adaptive behavior, which results in stretching of the repertoire of adaptive behaviors (Dor and Jablonka, 2000; Jablonka and Lamb, 2005). They pointed out that the evolution process of complex linguistic abilities of humans (e.g., the capacity of recognizing discrete conceptual categories, rapid processing of the speech channel, and lexical memory) could be explained by this principle. The repeated occurrences of the genetic assimilation observed in our experiments could be regarded as an example of such an evolution process, and may imply that this kind of scenario is likely to occur in evolution of communicative traits, although our scenario is not limited to human language evolution.

5. Conclusion

We have discussed whether and how learning can facilitate the adaptive evolution of communicative traits using a simple computational model of the evolution of a quantitative trait and its phenotypic plasticity based on directional and positive frequency-dependent selection. Under the stochastic evolution process with the limited number of individuals, the experimental results showed that there are the two different ways for learning to facilitate

adaptive evolution by preventing the population from getting stuck on the single peak of the innate and individual-level fitness landscape caused by the positive frequency-dependent selection. When the mutation rate was high, the evolved lower plasticity mainly contributed to keep less innately adaptive individuals from being eliminated from the population, increasing the intrinsic genetic variation. Furthermore, if the mutation rate was small, we found another role of the evolved higher phenotypic plasticity that enabled the potentially adaptive individuals to acquire much more adaptive traits. We also found the conditions in which the complete genetic assimilation occurred repeatedly.

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