Sympatric Speciation from Interaction-induced Phenotype Differentiation

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Abstract

A novel viewpoint for evolution is presented, by taking seriously into account the relationship between genotype and phenotype. First, as a consequence of dynamical systems theory, phenotypes of organisms can be differentiated into distinct types through the interaction, even though they have identical genotypes. Then, with the mutation in genotype, it is shown that the genotype also differentiates into discrete types, while maintaining the 'symbiotic' relationship between the types. This process is robust against sexual recombination, because offspring with intermediate genotypes are less fit than their parents. Accordingly, a plausible scenario for sympatric speciation is presented. Relevance of our scenario to the historical evolution as well as to artificial evolution is discussed.

Introduction

The question why organisms are separated into distinct groups, rather than exhibiting a continuous range of characteristics(1), originally raised by Darwin(3), has not yet been fully answered, in spite of several attempts to explain sympatric speciation (see also Maynard-Smith and Szathmary(23; 2)). Here, we provide an answer to this question, by presenting a novel and plausible mechanism for the sympatric speciation, based on dynamical systems theory.

Difficulty in stable sympatric speciation, i.e., process to form distinct groups with reproductive isolation, lies in the lack of a known clear mechanism how two groups, which have just started to be separated, coexist in the presence of mutual interaction and mixing of genes by mating. So far people try to propose some mechanism so that the two groups do not mix and survive independently, as is seen in sexual isolation by mating preference (e.g., (24; 22; 27; 11; 21; 4)). However, this type of theory cannot answer how such mating preference that is 'convenient' for sympatric speciation, is selected. In addition to this drawback, there lies another serious problem. In the conventional theory of sympatric speciation, if one group may disappear by fluctuations due to finite-size population, the other group does not necessarily reappear. Coexistence of one group is not necessary for the survival of the other. Hence the speciation process is rather weak against possible fluctuations that should exist in a population of finite size.

Of course, if the two groups were in a symbiotic state, the coexistence would be necessary for the survival of each. However, the two groups have little difference in genotype in the beginning of speciation process, and it might be hard to imagine such a 'symbiotic' mechanism. Accordingly, it is generally believed that sympatric speciation, robust against fluctuations, is rather difficult. As long as we assume that the phenotype is a single-valued function of genotype for a given environment, this conclusion will be plausible and general.

Let us recall the standard standpoint for the evolution in the present biology(8; 1). (i) First, each organism has genotype and phenotype. (ii) Then, the fitness for survival is given for a phenotype, and Darwinian selection process acts for the survival of organisms, to have a higher fitness (iii) Only the genotype is transferred to the next generation (Weissman's doctrine) (iv) Finally, there is a direct flow only from a genotype to phenotype, i.e., a phenotype is determined through developmental process, given a genotype and environment (the central dogma of molecular biology). Although there may be some doubt in (iii) (and (iv)) for some cases, we follow this standard viewpoint here.

Note, however, that (iv) does not necessarily mean that the phenotype is 'uniquely determined'. In the standard population genetics, this uniqueness is assumed, but it is not necessarily postulated within the above standard framework. Indeed, there are three reasons to make us doubt this assumption of the uniqueness, one theoretical and two experimental.

First, we have previously proposed isologous diversification theory, where two groups with distinct phenotypes appear even from the same genotype(6; 7; 15; 16; 17). In this theory, due to the orbital instability in developmental process, any small difference (or fluctuation) is amplified to a macroscopic level, so that the dynamical state of two organisms (cells) can be different, even if they have a same set of genes. The organisms are dif-

ferentiated into discrete types through the interaction, where existence of each type is necessary to eliminate the dynamic instability in developmental process, which underlies when the ensemble of one of the types is isolated. Hence, existence of each type is required for the survival of each other, even though every individual has identical, or slightly different genotypes.

Second, it is well known experimentally that in some mutants, various phenotypes arise from a single genotype, with some probability(10). This phenomenon is known as low or incomplete penetrance(25). Although the existence of an organism with low penetrance is a 'headache' in genetics, it is observed even in *C elegans*. Although it might sound strange, it is an established fact that the uniqueness of phenotypes is not always true.

Last, the interaction-induced phenotypic diversification is clearly demonstrated in an experiment reported by one of the authors and his colleagues, for specific mutants of *E. coli*. In fact, they show (at least) two distinct types of enzyme activity, although they have identical genes. These different types coexist in a well stirred environment of a chemostat (20; 19), and this coexistence is not due to spatial localization. Here, coexistence of each type is supported by each other. In fact, when one type of *E. coli* is removed externally, the remained type starts to differentiate again to recover the coexistence of the original two types. It is now demonstrated that distinct phenotypes (as for enzyme activity) appear, according to the interaction among the organisms, even though they have identical genes. Indeed, the mechanism for this differentiation is understood theoretically by the above 'isologous diversification theory'.

Hence, we take this interaction-induced phenotypic differentiation from a single genotype seriously into account and discuss its relevance to evolution. We will show that this phenotypic differentiation is later fixed to genotypes through mutation to genotypes, in spite of the fact that we have assumed only the flow from genotype to phenotype, and will give a general mechanism for the sympatric speciation. Last, we will discuss relevance of our theory to biological evolution, as well as to artificial life studies.

Model

To study phenotypic and genotypic relationship, we have to consider a developmental process that maps a genotype to a phenotype. As an illustration, we consider an abstract model consisting of several biochemical processes. Each organism possesses such internal dynamic processes which transfer external resources into some products depending on the internal dynamics. Through this process, organisms mature and eventually become ready for reproduction.

Here, the phenotype is represented by a set of variables, corresponding to biochemical processes. Each individual i has several cyclic processes $j = 1, 2, \cdots, k$, whose state at time n is denoted by $X_n^j(i)$. With k such processes, the state of an individual is given by the set $(X_n^1(i), X_n^2(i), \cdots, X_n^k(i))$, which defines the phenotype. This set of variables can be regarded as concentrations of chemicals, rates of metabolic processes, or some quantity corresponding to a higher function. The state is not fixed in time, but changes temporally according to a set of deterministic equations with some parameters.

Genes, since they are nothing but information expressed on DNA, could in principle be included in the set of variables. However, according to the central dogma of molecular biology (requisite (iv) in *Introduction*), the gene has a special role among such variables. Genes can affect phenotypes, the set of variables, but the phenotypes cannot change the code of genes. During the life cycle, changes in genes are negligible compared with those of the phenotypic variables they control. In terms of dynamical systems, the genes can be represented by control parameters that govern the dynamics of phenotypes, since the parameters in an equation are not changed through the developmental process, while the parameters control the dynamics of phenotypic variables. Accordingly, we represent the genotype by a set of parameters. Only when an individual organism is reproduced, this set of parameters changes slightly by mutation.

To be specific we consider the following model consisting of the processes (i)-(iii) given below.

(i) Dynamics of the phenotypic state:

The dynamics of the variables $X_n^j(i)$ consist of a mutual influence of cyclic processes $\{ X_n^\ell(i) \}$ and interaction with other organisms $(X_n^\ell(i'))$. First, as a simple model we split the state variable $X_n^\ell(i)$ into its integer part $R_n^\ell(i)$ and the fractional part $x_n^\ell(i) \equiv mod[X_n^\ell(i)]$. The integer part $R_n^\ell(i)$ is assumed to give the number of times that the cyclic process has passed since the individual's birth, while the fractional part $x_n^\ell(i)$ gives the phase of oscillation in the process.

As a simple model, we assign a phase of oscillation to each cyclic process and assume that there are mutual influences depending on the phase state of processes. The ℓ-th process has a flow from other processes, while there is a flow from the process to the other processes. As a simple example, the internal dynamics of the cyclic process is assumed to be represented by $\sum_m \frac{a^{\ell,m}}{2} sin(2\pi x_n^m(i))$. Hence the internal dynamics are given by

$$X_{n+1}^\ell(i) = X_n^\ell(i) + \sum_m \frac{a^{\ell m}(i)}{2} sin(2\pi x_n^m(i)) - \sum_m \frac{a^{m\ell}(i)}{2} sin(2\pi x_n^\ell(i)).$$

Next, the interaction between individuals is introduced through competition for resources, with which each cyclic process progresses. The ability to obtain resources generally depends on the internal state of

the unit $x_n^j(i)$. Again, we choose our model so that only the phase is relevant to the interaction and take $psin2\pi(x_n^\ell(j))$ as the ability to obtain resources. Assuming that all elements (whose number is N_n) compete for resources s^j for each step, we take the following interaction term:

$$Interaction^\ell(i) = psin(2\pi x_n^\ell(i)) + \frac{s^\ell - \sum_j psin2\pi(x_n^\ell(j))}{N_n}.$$

Here, the second term comes from the constraint that $\sum_i Interaction^\ell(i) = s^\ell$, due to the condition that units compete for a given resource s^ℓ at each time step.

Now, by summing up the two processes, the developmental dynamics of phenotypes in our model is given by

$$X_{n+1}^\ell(i) = X_n^\ell(i) + \sum_m \frac{a^{\ell m}(i)}{2} sin(2\pi x_n^m(i)) - \sum_m \frac{a^{m\ell}(i)}{2} sin(2\pi x_n^\ell(i)) + psin(2\pi x_n^\ell(i)) + \frac{s^\ell - \sum_j psin2\pi(x_n^\ell(j))}{N_n}. \quad (1)$$

(ii) Growth and death: Each individual splits into two when a given condition for the growth is satisfied. Taking into account that the cyclic process is required for reproduction, we assume that a unit replicates when the accumulated number of cyclic processes goes beyond some threshold. As a specific example, the condition for the reproduction is given by $\sum_\ell R_n^\ell(i) \geq Thr$. The rotation number $R_n^\ell(i)$ is reset to zero when the corresponding individual splits.

To introduce the competition for survival, death of an individual has to be included. Here each individual is eliminated both by random removal of organisms at some rate as well as by a given death condition based on their state. The latter condition is given by the elimination of such individual that satisfies $R_n^\ell(i) < -10$. In other words, if the cyclic process of an individual progresses reversely too much, it dies.

(iii) Genetic parameter and mutation:

Next, genotypes are given by a set of parameters $a^{m\ell}(i)$, representing the relationship between the two cyclic processes ℓ and m ($1 \leq \ell \neq m \leq k$). Following the above argument, genes, represented by parameters in the model slowly mutate by reproduction. With each division, the parameters $a^{m\ell}$ are changed to $a^{m\ell} + \delta$, with δ as a random number over $[-\epsilon, \epsilon]$. Here the small parameter ϵ corresponds to the mutation rate.

Scenario for Symbiotic Sympatric Speciation

We have carried out several simulations of the model with $k = 3, 4$, and 5, and some other variants (18). Also, we have carried out the simulations of a model describing of a metabolic process of autocatalytic networks (26). Since a common speciation is obtained for all the models, we basically describe numerical results of the model in the last section, to demonstrate our general scenario for the sympatric speciation. An example of the speciation process in the model is shown in Fig.1 and Fig.2. The evolution of the genotype-phenotype relationship, plotted by (a^{23}, R^2) at every reproduction event, is given in the sequence of Fig.1, while the values of $a^{23}(i)$ are plotted at every reproduction in Fig.2. Note the change in the scale for the genotype axis by figures in Fig.1, which illsutrates the progress of separation in genotype parameters. As shown in Fig.1a, the phenotype differentiates initially into two groups and this phenotypic change is subsequently fixed to the genotypes. The scenario for the speciation here is described as follows (18). (see Fig.3 for schematic representation).

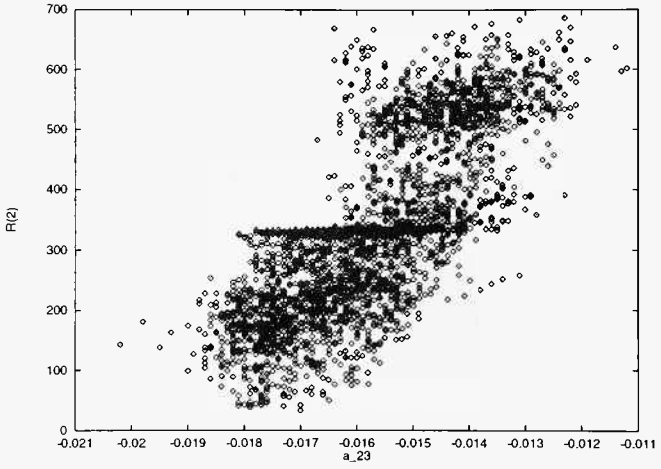

Fig. 1a)

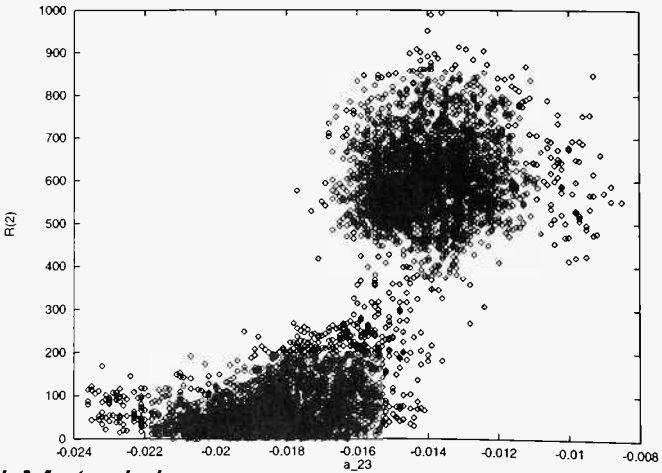

Fig.1 b)

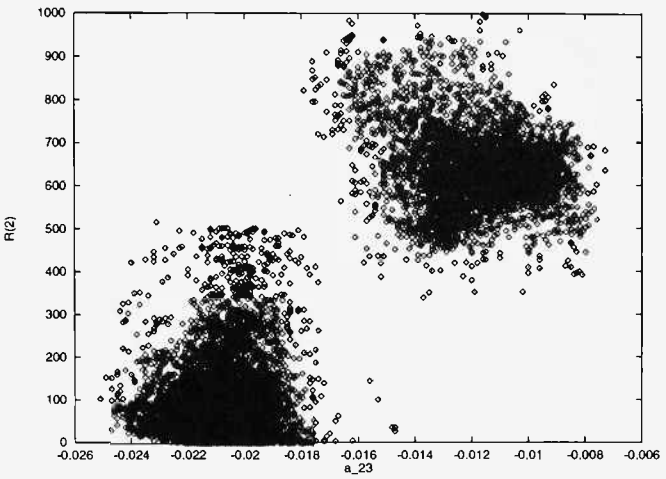

Fig.1 c)

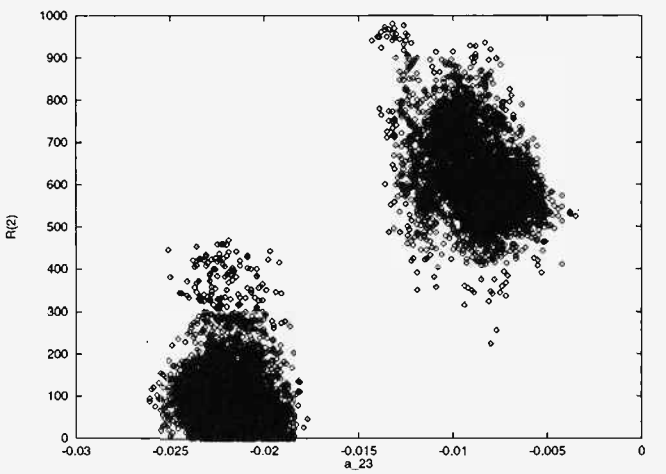

Fig.1 d)

Figure 1: Evolution of genotype-phenotype relationship. As a phenotype, the integer part $R^2(j)$, the number of cyclic process 2 used for reproduction, is adopted, while a^{23} is used as a genetic parameter. (a^{23}, R^2) is plotted for every division of individuals. The 501-3500th divisions are plotted in (a), 6000-10000 divisions in (b), 10000-20000 divisions in (c), and 20000-30000 divisions in (d). Initially, phenotypes are separated, even though the genotypes differ only slightly, as shown in (a). Later, the genotypes are also separated, according to the difference in phenotypes, as shown in (b) and (c). In the simulation shown in Fig.1. and Fig.2, the parameters are set at $p_k = 1.5/(2\pi)$, $s^1 = 10, s^2 = 8, s^3 = 5$, while threshold number Thr for the reproduction is set at 1000, and the mutation rate of the parameters is 0.001. The population size fluctuates around 500, after an initial transient. (Hence the generation number is given roughly by dividing this division number by 500.) Initially, the genotype parameters are set as $a^{ij} = \frac{-0.1}{2\pi}$.

Stage-1: Interaction-induced phenotypic differentiation

When many individuals interact competing for finite resources, the phenotypic dynamics start to be differentiated even though the genotypes are identical or differ only slightly. This differentiation generally appears if nonlinearity is involved in the internal dynamics of some phenotypic variables. Slight differences in variables between individuals are amplified by the internal dynamics (e.g., metabolic reaction dynamics). Through interaction between organisms, the difference in phenotypic dynamics are amplified and the phenotype states tend to be grouped into two (or more) types. The dynamical systems mechanism for such differentiation was first discussed as clustering (12), and then extended, to study the cell differentiation (6; 7; 13; 16; 17). In fact, the orbits of $(x_t^1(i), x_t^2(i), \cdots, x_t^k(i))$ lie in a distinct region in the phase space, depending on each of the two groups that the individual i belongs to. Note that the difference at this stage is not fixed in either the genotype or the phenotype. The progeny of a reproducing individual may belong to a distinct type from the parent. If a group of one type is removed, then some individuals of the other type change their type to compensate for the missing type. To discuss the present mechanism in biological terms, consider a given group of organisms faced with a new environment and not yet specialized for the processing of certain specific resources. Each organism has metabolic (or other) processes with a biochemical network. As the number of organisms increases, they compete for resources. As this competition becomes stronger, the phenotypes become diversified to allow for different uses in metabolic cycles, and they split into two (or several) groups. Each group is specialized in processing of some resources. Here, the two groups realize a differentiation of roles and form a symbiotic relationship. Each group is regarded as specialized in a different niche, which is provided by another group.

Stage-2: Co-evolution of the two groups to amplify the difference of genotypes

At the second stage of our speciation, difference in both genotypes and phenotypes is amplified. This is realized by a kind of positive feedback process between the changes in geno- and phenotypes.

This process consists of two parts. The first part, essential to the genetic fixation, is genetic separation due to the phenotypic change. This occurs if the parameter dependence of the growth rate is different between the two phenotypes. In other words, there are (one or) several parameters such that the growth rate increases with them for the upper group and decreases for the lower group (or the other way around).

Indeed, such parameter dependence is not exceptional.

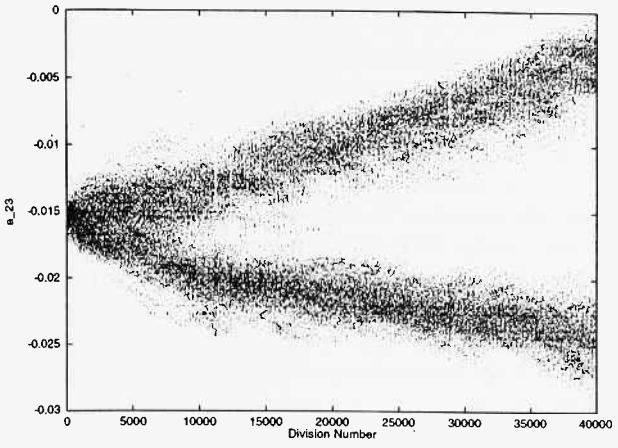

Figure 2: The evolution of the genotypic parameter, corresponding to Fig. 1. The parameter $a^{23}(i)$ is plotted as a dot at every division (reproduction) event, with the abscissa as the division number.

As a simple illustration, assume that the use of metabolic processes is different between the two groups. If the upper group uses one metabolic cycle more, then the mutational change of the parameter $a^{\ell m}$ to enhance the use of the cycle is in favor for the upper group, while the change to reduce it may be in favor for the lower group. Indeed, several numerical results (given e.g., in Fig. 1 and Fig. 2) support that there always exist such parameters. This dependence of growth on genotypes leads to genetic separation of the two groups.

Although the above process is most essential to speciation, the genetic separation is often accompanied by the second process, the amplification of phenotypic difference due to the genotypic difference. In the situation of Fig. 3, as the parameter $a^{\ell m}$ is increased, the phenotype variable R^j tends to increase and vice versa. This is possible if $\partial R^j / \partial a^{\ell m}$ is larger for the upper group. In a typical and clear example, as in Fig. 1 and Fig. 3c, $\partial R^j / \partial a^{\ell m}$ is positive for the upper group and negative for the lower group. With this process, the separation of the two groups is amplified both in genotypes and phenotypes. We again emphasize that the existence of such parameter(s) that satisfy the two conditions is not unusual.

With this separation of two groups, each phenotype (and genotype) tends to be preserved by the offspring, in contrast with the first stage. Now, distinct groups with recursive reproduction have been formed. However, up to this stage, the two groups with different phenotypes cannot exist by themselves in isolation. When isolated, offspring with the phenotype of the other group start to appear. The developmental dynamics in each group, when isolated, are unstable and some mutations start to be differentiated to recover the other group. The dynamics accordingly each phenotype is stabilized by

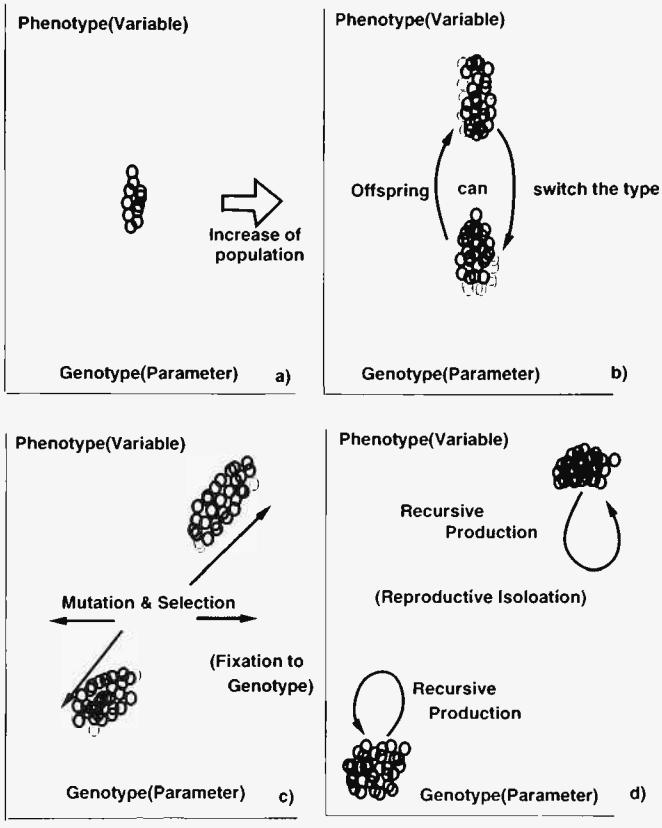

Figure 3: Schematic representation of speciation process, plotted as phenotype-genotype relationship. (a) Initially, there is a group of organisms with distribution centered around a given phenotype and genotype. (b) Then, with the increase of population, phenotype is differentiated into discrete types. (c) Then according to the difference of phenotype, genotype is also differentiated. (d) Finally, the two groups differentiate both in genotypes and phenotypes, and form distinct species. Indeed, these two groups are separated also by sexual recombination, since the hybrid offspring cannot produce its progeny.

each other through interaction. Hence, two groups are in a symbiotic state, and the evolution of one group is related with that of the other. To have such stabilization, the population of each group has to be balanced through the interaction. Even under random fluctuation by finite-size populations and mutation, the population balance of each group is not destroyed. Indeed, the growth speed of each group remains of the same order at this stage. With this co-evolutionary process, the phenotypic differentiation is fixed to the genotype.

Accordingly, our mechanism of genetic diversification is stable. This is why our mechanism works as a stable sympatric speciation, as will be shown in the next section.

Stage-3 Genetic Fixation and Isolation of Differentiated Groups

Complete fixation of the diversification to genes occurs at this stage. Here, even if one group of units is isolated, the offspring of the phenotype of the other group are no longer produced. Offspring of each group keep their phenotype (and genotype) on their own. This is confirmed by numerically eliminating one group of units.

Now, each group has one phenotype corresponding to each genotype, even without interaction with the other group. Hence, each group is a distinct independent reproductive unit at this stage. This stabilization of a phenotypic state is possible since the developmental flexibility at the first stage is lost, due to the shift of genotype parameters. The initial phenotypic change introduced by the interaction is now fixed to genes. Genetically distinct groups with independent reproduction are formed with this genetic fixation.

To check the third stage of our scenario, it is straightforward to study the further evolutionary process from only one isolated group. In order to do this, we pick out some population of units only of one type, after the genetic fixation is completed and both the geno- and phenotypes are separated into two groups, and start the simulation again. When the groups are picked at this third stage, the offspring keep the same phenotype and genotype. Now, only one of the two groups exists. Here, the other group is no longer necessary to maintain stability.

Speciation as Reproductive Isolation in the Presence of Sexual Recombination

The speciation process is defined by both genetic differentiation and by reproductive isolation (Dobzhansky 1937). Although the evolution through the stages I-III lead to genetically isolated reproductive units, one might say that the term 'speciation' should not be used unless the process shows isolated reproductive groups under the sexual recombination. In fact, one may wonder if the present differentiation scenario works under sexual recombination, since the two genotypes from parents are mixed in the offspring by recombination.

On the other hand, since the present scenario is robust against perturbations, such as the removal of one phenotype group, it may be expected to be stable against sexual recombination, which mixes the two genotypes and may bring about a hybrid between the two genotypes. To examine this stability, we have extended the previous model to include this mixing of genotypes by sexual recombination. To be specific, when the reproduction occurs for two individuals i_1 and i_2 that satisfy the threshold condition ($\sum_\ell R_n^\ell(i_k) > Thr$), the two genotypes are mixed, by producing two offspring $j = j_1$ and j_2, as

$$a^{\ell m}(j) = a^{\ell m}(i_1) r_j + a^{\ell m}(i_2)(1 - r_j) + \delta \qquad (2)$$

with a random number $0 < r_j < 1$ to mix the parents' genotypes, while the term by δ represents the random mutation. (Asexual reproduction is not included in the simulation). We have made several simulations of this type by choosing the same setting as the previous model.

In Fig.4, we have plotted the evolution of the parameter $a^{12}(i)$ by each reproduction event. As shown in Fig.4, the two distinct groups are again formed in spite of the above mixing of genotypes by sexual recombination.

Even if two separated groups may start to be formed according to our scenario, the above recombination can form 'hybrid' offspring with intermediate parameter values $a^{\ell m}$ between the two group. We have again plotted the evolution of the parameter $a^{12}(i)$ by each reproduction event. As shown in Fig.4, the two distinct groups are again formed in spite of the above mixing of genotypes by sexual recombination.

Of course, the mating between the two groups can produce an individual with the parameters in the middle of the two groups, according to eq.(2). However, an individual with intermediate parameters between the two groups starts to have a lower reproduction rate. Such individual requires much longer time to reach the threshold condition for reproduction whatever phenotype it takes. Before the reproduction condition is satisfied, the individual dies with a higher probability. Hence, an individual from the parents of two distinct groups becomes harder and harder to produce their offspring, with time.

To demonstrate this post-mating isolation, we have also measured the average offspring number of individuals over given parameter (genotype) ranges and over some time span, in Fig.5. As the two groups are formed with the split of the parameter values, the average offspring number of an individual having the control parameter between those of the two groups starts to decrease. Soon the number goes to zero, implying that the hybrid between the two groups is sterile. In this sense, sterility (or low reproduction) of the hybrid appears as a result. Hence it is proper to call the process I-III as speciation, since it satisfies genetic differentiation and

reproductive isolation (under the sexual recombination).

Note that we have not assumed any preference in mating choice. Rather, it is natural, according to the present scenario, that mating preference in favor of similar phenotypes evolves, since it is disadvantageous for individuals to produce a sterile hybrid. In other words, the present mechanism also provides a basis for the evolution of sexual isolation through mating preference(24; 22; 27; 11; 21; 4). For example, assume that each individual has a tendency to prefer a mating partner with a closer phenotype, or to avoid an individual with too much different phenotype. With this mating preference, the rate to form a sterile hybrid is reduced. Hence, the pre-mating isolation will evolve as a consequence of post-mating isolation. At any rate, the mating preference can strengthen the speciation process of our mechanism, but never hinders the mechanism from working.

It should be noted that our mechanism for the speciation can work in asexual and sexual reproduction in the same way. The phenotype (R^j) separates into two groups first in the present case with sexual recombination as in the previous asexual case. Later the change is mapped onto the parameters a^{lm}. The speciation process progresses following the three stages given in §3. Indeed the stability of the speciation against sexual recombination is naturally expected, since the coexistence of two distinct phenotype groups is supported by the isologous diversification, i.e., differentiation to distinct phenotypes under the same genotypes. Even though the genes are mixed, the phenotypes are tended to be separated into distinct groups. Hence the separation into distinct groups is not blurred by the recombination.

Relevance to Biology

According to our scenario, the speciation is a result of interaction-induced phenotypic differentiation. To check the condition for speciation, we have performed numerical experiments of our model, by choosing parameters so that differentiation into two distinct phenotype groups does not occur initially. In this case, separation into two (or more) groups with distinct pheno/geno-types is never observed, even if the initial variance of genotypes is large, or even if a large mutation rate is adopted.

Next, the genetic differentiation always occurs when the phenotype, (represented by the rate of each cyclic process R^k), differentiates into two (or more) distinct groups. After the initial separation into two groups, the fixation into parameters *always* follows, as long as mutation exists. Hence, phenotypic differentiation is a necessary and sufficient condition for the speciation process, in a standard biological situation, i.e., a process with reproduction, mutation, and a proper genotype-phenotype relationship.

Note that the interaction-induced phenotypic differ-

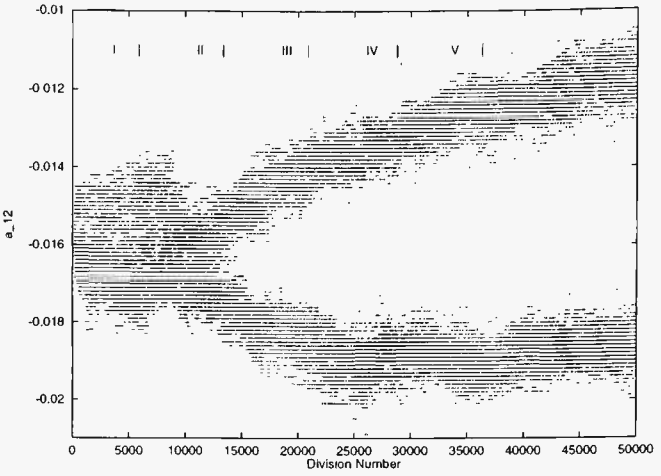

Figure 4: An examples of the speciation process with sexual recombination. The parameter a^{13} of divided units is plotted with the division event. The parameters are $p_k = 1.6/(2\pi)$, $s^1 = s^2 = s^3 = 2$, with initial parameters $a^{ij} = (-.1)/(2\pi)$. The total population fluctuates around 350 in this example.

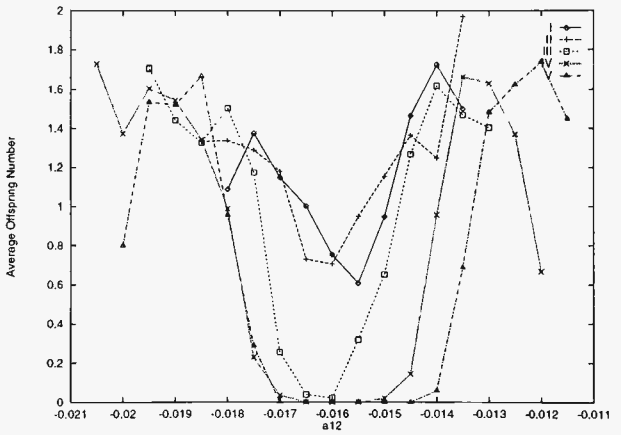

Figure 5: The average offspring number before death is plotted as a function of the parameter (genotype), for simulations of a model with sexual recombination. We have measured the number of offspring for each individual during its lifespan, for the data of Fig.4. By taking a bin width 0.005 for the genotype parameter a^{12}, the average offspring number over a given time span is measured to give a histogram. The histogram over the first 7500 divisions (about 20 generations) is plotted by the solid line (I), and the histogram for later divisions is overlaid with a different line, as given by II (over 7500-15000 divisions), III (1.5-2.25 $\times 10^4$), IV(2.25-3 $\times 10^4$), and V(3.75-4.5 $\times 10^4$). The stages I-V are also represented in Fig.4. As shown in the increase of the dip in the middles, a hybrid offspring will be sterile after some generations.

entiation is deterministic in nature. Once the initial parameters of the model are chosen, it is already determined whether such differentiation will occur or not. Hence, the speciation process is also deterministic in nature, in spite of stochastic mutation process therein. In fact, the speciation process (e.g., the time required for it or the population ratio between the two groups) changes only little between runs that adopt a different random number.

This speciation process is rather fast, once the condition for phenotypic differentiation is satisfied. In the simulations shown in the figures 1 and 2, the speciation is completed around the first 50 generations. Hence, our scenario may give a new and plausible explanation on the variation of time scales on which evolution proceeds, e.g., punctuated equilibrium(9).

In our speciation process, the potentiality for a single genotype to produce several phenotypes decreases. After the phenotypic diversification of a single genotype, each genotype again appears through mutation and assumes one of the diversified phenotypes in the population. Thus the one-to-many correspondence between the original genotype and phenotypes eventually ceases to exist. As a result, one may expect that a phenotype is uniquely determined for a single genotype in wild types, since most organisms at the present time have gone through several speciation processes. One can also expect that a mutant tends to have a higher potentiality to produce various phenotypes from a single genotype. Hence our theory explains why low or incomplete penetrance(10; 25) is more frequently observed in mutants than in a wild type.

Our theory is expected to shed a new light on a possible relationship between developmental process and speciation. For example, consider the questions why the insect has often a larger number of species, or why there are some species that stop evolution, such as *living fossils*. These questions are thought to be tightly related with the developmental flexibility. Our theory predicts that the evolution in phenotype and genotype is accelerated when the developmental process to map between the two types is flexible.

Finally, it should again be stressed that *neither any Lamarckian mechanism nor epigenetic inheritance is assumed* in our theory, in spite of the genetic fixation of the phenotypic differentiation. Only the standard process allowing from genotype to phenotype is included in our theory. Note also that genetic 'takeover' of phenotype change was also proposed by Waddington as genetic assimilation, in possible relationship with Baldwin's effect (28). Using the idea of epigenetic landscape, he showed that genetic fixation of the displacement of phenotypic character is fixed to genes. In our case the phenotype differentiation is not given by 'epigenetic landscape', but rather, the developmental process forms different characters through the interaction. Distinct characters are stabilized through the interaction. With this interaction dependence, the two groups are necessary with each other, and robust speciation process is possible.

Relevance to Artificial Life

Discussion of the mechanism involved in evolution often remains vague, since no one knows for sure what has occurred in history, within limited fossil data. Most important in our scenario, on the other hand, lies in experimental verifiability. As mentioned, isologous diversification has already been observed in the differentiation of enzyme activity of *E. coli* with identical genes(20; 19). We have already started an experiment of the evolution of *E. coli* in the laboratory(29), controlling the strength of the interaction through the population density. With this experiment we can check if the evolution on the genetic level is accelerated through interaction-induced phenotypic diversification, and can answer if our scenario really occurs in nature. In this sense, our evolution theory is testable in laboratory, in contrast with many other speculations. In the same sense, our study is relevant to the field of artificial life (AL), since AL attempts to understand some biological process such as evolution, by constructing an artificial system in laboratory or in a computer from our side.

A problem in most of the present AL studies lies in that it is too much symbol-based. They generally assume some rule for a biological system, represented as manipulation over symbols. Such process will eventually be written by a universal Turing machine. Hence it generally faces with the problem that the emergence may not be possible in principle in such system, since the emergence originally means a generation of a novel, higher level that is not originally written in a rule. The same drawback lies in the symbol-based study of evolution (i.e., a study starting from the evolution of symbols corresponding to genes), and indeed, the AL study on the evolution is often nothing but a kind of complicated optimization problem.

According to our theory, first the phenotype is differentiated, given by continuous (analogue) dynamical system, which is later fixed to genes that serve as a rule for dynamical systems. Now, rules written by symbols (genetic codes) are not necessarily the principal cause of the evolution(14).

In the present paper, we have mainly studied the speciation process from one species to two. However, our theory is straightforwardly extended to study further speciation processes. By including a larger number of processes, one can study successive speciation, as is relevant to the adaptive radiation. There, it is important to study the evolution process in an "open" phase space, i.e., with an increasing number of variables and parameters (phenotypes and genotypes). With this extension,

the origin and evolution of diversity will be understood, that is one of the focus issues in the artificial life studies.

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