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Symbiotic sympatric speciation: consequence of interaction-driven phenotype differentiation through developmental plasticity

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Abstract A mechanism of sympatric speciation is presented based on the interaction-induced developmental plasticity of phenotypes. First, phenotypes of individuals with identical genotypes split into a few groups, according to instability in the developmental dynamics that are triggered with the competitive interaction among individuals. Then, through mutational changes in the genes, the phenotypic differences are fixed to genes, until the groups are completely separated in genotype as well as phenotype. It is also demonstrated that the proposed theory leads to hybrid sterility under sexual recombination, and thus speciation is completed in the sense of reproductive isolation. As a result of this postmating isolation, the mating preference evolves later. When there are two alleles, the correlation between alleles is formed to consolidate speciation. When individuals are located apart in space, different species are later segregated spatially, implying that the speciation so far regarded to be allopatric may be a result of sympatric speciation. Relationships to previous theories, frequency-dependent selection, reinforcement, Baldwin's effect, phenotypic plasticity, and resource competition are briefly discussed. Relevance of the results to natural evolution is discussed, including punctuated equilibrium, incomplete penetrance in mutants, and the change in flexibility in genotype–phenotype correspondence. Finally, we discuss how our theory is confirmed both in the field and in the laboratory, in an experiment using *Escherichia coli.*

Key words Dynamical systems · Development · Phenotypic plasticity · Postmating isolation · Mating preference · Genotype–phenotype mapping

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Introduction

In spite of progress in the understanding of evolution since Darwin (1859), speciation is not yet fully understood. In their recent book, Maynard Smith and Szathmary (1995) wrote that "we are not aware of any explicit model demonstrating the instability of a sexual continuum."

To discuss the problem of speciation, let us start by reviewing basic points in evolution theory, although this might appear elementary here.

i. Existence of genotype and phenotype.
ii. Fitness for reproduction is given as a function of the phenotype and the environment. The "environment" can include interaction with other individuals. In other words, the reproduction rate of an individual is a function of its phenotype and the environment, i.e., F(phenotype, environment).
iii. Only the genotype is transferred to the next generation (Weissman doctrine).
iv. There is flow only from genotype to phenotype (the central dogma of molecular biology). For example, through the developmental process the phenotype is determined depending on the genotype. Now, the process is summarized as *genotype* → *development* → *phenotype*.

Here we adopt these standard assumptions. (Although assumption iii may not be valid for some cases known as epigenetic inheritance, we accept the assumption here, because the relevance of epigenetic inheritance to evolution is still controversial, and the theory to be proposed is valid in the presence of epigenetic inheritance, although it does not require it.)

In standard evolutionary genetics, assumption iv is further replaced by a stronger one, i.e., iv′, "phenotype is a *single* valued function of genotype." If this were always true, we could replace F(phenotype, environment) in point ii by $F(f$(genotype), environment) and we could then discuss the evolutionary process in terms of the population dynamics of only genotype (and environment). This is the basic point in population genetics.

Indeed, this reduction to genes is valid for gradual evolution. It is also supported by the following mathematical argument. The change of genotype is slower in time scale than that of the phenotype. As is known, variables with a slower time scale act as a control parameter to faster ones, if the time scale separation is large enough (and if the dynamics in the fast time scale do not have such instability that leads to bifurcation).

Still, explanation of speciation, especially sympatric speciation, is not so easy following this standard evolutionary genetics. If slight genetic change leads to slight phenotype change, then individuals arising from mutation from the same genetic group differ only slightly, according to this picture. Then, these individuals compete with each other for the same niche. Unless the phenotype in concern is neutral, it is generally difficult for two (or more) groups to coexist; those with a higher fitness would survive. One possible way to avoid this difficulty is to assume that two groups are "effectively" isolated, so that they do not compete. Some candidates for such isolation are searched. The best-known example is spatial segregation, known as allopatric speciation. Because we are interested in sympatric speciation, this solution cannot be adopted here. (As is discussed later, sympatric speciation can bring about allopatric speciation, but not the other way around.) Furthermore, there is direct evidence that sympatric speciation really occurred in evolution, for example, in the speciation of cichlids in some lakes (Schiliewen et al. 1994).

As another candidate for separation, mating preference is discussed (Maynard Smith 1966; Felsenstein 1981; Doebeli 1996; Howard and Barlocher 1998). Recently, there have appeared some models showing the instability of a sexual continuum, without assuming the existence of discrete groups in the beginning. Probably the argument based on the runaway is most persuasive (Lande 1981; Turner and Burrows 1995; Howard and Barlocher 1998). Even though two groups coexist at the same spatial location, they can be genetically separated if two groups do not mate with each other. Hence, the mating preference is proposed as a mechanism for sympatric speciation. However, in this theory, why such mating preference itself occurs is not answered. Accordingly, it is not completely self-contained as a theory.

Another recent proposal is given as evolutionary branching in adaptive dynamics by Metz's group (Geritz et al. 1998). For example, by taking (almost) neutral fitness landscape and exclusion of individuals with similar phenotypes, branching in phenotype is demonstrated, although finite mutation step size has to be assumed. By extending this idea to include sexual reproduction, Dieckmann and Doebeli (1999) have succeeded in showing that two groups are formed and coexist, to avoid the competition among organisms with similar phenotypes, assuming a rather flat fitness landscape (see also Kondrashov and Kondrashov 1999; Kawata and Yoshimura 2000); this provides one explanation of sympatric speciation, although it is not so clear how the phenotype that is not as important in fitness works strongly as a factor for exclusion for a closer phenotype value. Furthermore, we are more interested here in the differentiation of phenotypes that are functionally different and not neutral.

So far, in these studies, the interaction between individuals leads to competition for their survival. Difficulty in stable sympatric speciation without mating preference 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. Of course, if the two groups were in a symbiotic state, coexistence could help the survival of each. However, the two groups have little difference in genotype at the beginning of the speciation process, according to assumption iv′. Then, it would be quite difficult to imagine such a "symbiotic" mechanism. Indeed, in all the studies just mentioned, iv′ i.e., a single valued phenotype from a single genotype for a given environment (including interaction), is assumed. (Under a *stochastic* environmental condition, this assumption may not be so clear if the environment has uncertainty [Yoshimura and Shields 1987]. Still, in this case also phenotype is uniquely determined, so long as the environment at each instant is specified).

Now, the problem we address here is as follows. If we do not assume iv′ (but by assuming i–iv), is there any mechanism by which two groups mutually require each other for survival in the beginning of the separation of the two groups? In this article, we propose such a mechanism, and provide a sympatric speciation scenario that is robust against fluctuations. Note that the foregoing difficulty comes from the assumption that the phenotype is a single-value function of the genotype. Is this single-value condition always true? To address this question, we reconsider the genotype–phenotype (G-P) relationship. Indeed, there are three reasons that we doubt this single-value state.

First, Yomo and his colleagues have reported that specific mutants of *Escherichia coli* show (at least) two distinct types of enzyme activity, although they have identical genes (Ko et al. 1994). These different types coexist in an unstructured environment of a chemostat, and this coexistence is not due to spatial localization. Coexistence of each type is supported by the other. Indeed, when one type of *E. coli* is removed externally, the remaining type starts differentiation again to recover the coexistence of the two types. The experiment demonstrates that the enzyme activity of these *E. coli* are differentiated into two (or more) groups by the interaction with each other even though they have identical genes. Here the spatial factor is not important because this experiment is carried out in a well-stirred chemostat.

Second, some organisms are known to show various phenotypes from a single genotype. This phenomenon is often related to malfunction of a mutant (Holmes 1979) and is called low or incomplete penetrance (Opitz 1981).

Third, a theoretical mechanism of phenotypic diversification has already been proposed as the isologous diversification for cell differentiation (Kaneko and Yomo 1994, 1997, 1999; Furusawa and Kaneko 1998). The theory states that phenotypic diversity will arise from a single genotype and develop dynamically through intracellular complexity and intercellular connection. When organisms with plastic developmental dynamics interact with each other, the dynam-

ics of each unit can be stabilized by forming distinct groups with differentiated states in the pheno-space. Here the two differentiated groups are necessary to stabilize each of the dynamics. Otherwise, the developmental process is unstable, and through the interaction the two types are formed again when there is a sufficient number of units. This theoretical mechanism is demonstrated by several models and is shown to be a general consequence of coupled dynamical systems (Kaneko 1990, 1994).

The isologous diversification theory shows that there can be developmental "flexibility" in which different phenotypes arise from identical gene sets, as in the incomplete penetrance aforementioned. Now we have to consider how this theory is relevant to evolution. Indeed, the question of the relationship between the developmental process and evolution has been addressed over decades (Maynard Smith et al. 1985; Gilbert et al. 1996). We consider correspondence between genotype and phenotype seriously by introducing a developmental process with which a given initial condition leads to some phenotype according to a given genotype. "Development" here means a dynamic process from an initial state to a mature state through rules associated with genes (In this sense, it is not necessarily restricted to multicellular organisms).

Model

To consider the evolution with developmental dynamics, it is appropriate to represent phenotype by a set of state variables. For example, each individual i has variables $(X_t^1(i), X_t^2(i), \ldots, X_t^k(i))$, which define 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 characterizing the behavior of the organism. The state is not fixed in time, but develops from the initial state at birth to a matured state when the organism is ready to produce its offspring. The dynamics of the state variables $(X_t^1(i), X_t^2(i), \ldots, X_t^k(i))$ is given by a set of equations with some parameters.

Genes, as 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), 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 set corresponding to genes can be represented by parameters $\{g^1(i), g^2(i), \ldots g^m(i)\}$ that govern the dynamics of phenotypes, because the parameters in an equation are not changed through the developmental process whereas 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 does this set of parameters change slightly by mutation. For example, when $\{X_t^\ell(j)\}$ represents the concentrations of metabolic chemicals, $\{g^1(i), g^2(i), \ldots g^m(i)\}$ is the catalytic activity of enzymes that controls the corresponding chemical reaction.

Now, our model is set up as follows.

1. Dynamic change of states giving a phenotype: The temporal evolution of the state variables $(X_t^1(i), X_t^2(i), \ldots, X_t^k(i))$ is given by a set of deterministic equations, which are described by the state of the individual, and parameters $\{g^1(i), g^2(i), \ldots g^m(i)\}$ (genotype), and the interaction with other individuals. This temporal evolution of the state consists of internal dynamics and interaction.

   1-1. The internal dynamics (say, a metabolic process in an organism) are represented by the equation governed only by $(X_t^1(i), X_t^2(i), \ldots, X_t^k(i))$ (without dependence on $\{(X_t^\ell(j)\}$ $(j \neq i)$), and are controlled by the parameter sets $\{g^1(i), g^2(i), \ldots g^m(i)\}$.

   1-2. Interaction between the individuals: The interaction is given through the set of variables $(X_t^1(i), X_t^2(i), \ldots, X_t^k(i))$. For example, we consider such an interaction form that the individuals interact with all others through competition for some "resources." The resources are taken by all the individuals, producing competition among all individuals. Because we are interested in sympatric speciation, we take this extreme all-to-all interaction by taking a well-stirred soup of resources, without including any spatially localized interaction.
2. Reproduction and death: Each individual produces offspring (or splits into two) when a given "maturity condition" for growth is satisfied. This condition is given by a set of variables $(X_t^1(i), X_t^2(i), \ldots, X_t^k(i))$. For example, if $(X_t^1(i), X_t^2(i), \ldots, X_t^k(i))$ represents a cyclic process corresponds to a metabolic, genetic, or other process that is required for reproduction, we assume that the unit replicates when the accumulated number of cyclic processes goes beyond some threshold.
3. Mutation: When each organism reproduces, the set of parameters $\{g^j(i)\}$ changes slightly by mutation, by adding a random number with a small amplitude δ, corresponding to the mutation rate. The values of variables $(X_t^1(i), X_t^2(i), \ldots, X_t^k(i))$ are not transferred but are reset to initial conditions. (If one wants to include some factor of epigenetic inheritance, one could assume that some of the values of state variables are transferred. Indeed, we have carried out this simulation also, but the results to be discussed are not altered or confirmed more strongly.)
4. Competition: To introduce competition for survival, death is included both by random removal of organisms at some rate as well as by a given death condition based on their state. For a specific example, see the Appendix.

Sympatric speciation observed

From several simulations satisfying the condition of the model just described, we have obtained a scenario for a sympatric speciation process. Besides the results in a simple model given in the Appendix, the same scenario is also

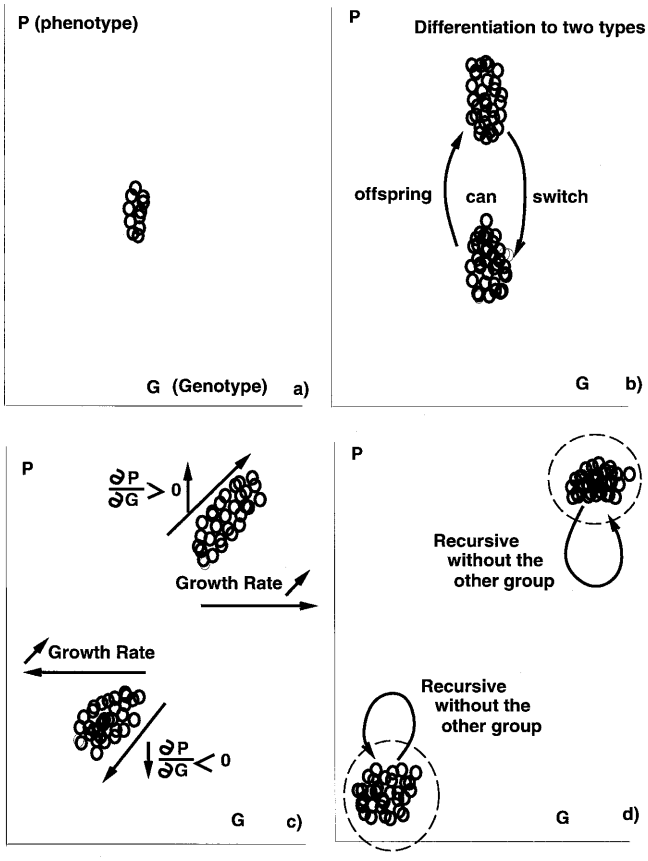


Fig. 1. Schematic representation of the speciation scenario obtained from our simulation and theory. A pair (phenotype, genotype) is plotted successively with time: **a** the stage of interaction-induced phenotypic separation; **b** the stage of genotype–phenotype feedback amplification; **c** the stage of genetic fixation; **d** speciation completed. (From Kaneko and Yomo 2002)

confirmed in a model with a catalytic reaction network mentioned in the last part of the Appendix (Takagi et al. 2000). The speciation process we observed is shown schematically in Fig. 1, where the change of correspondence between a phenotypic variable ("P") and a genotypic parameter ("G") is plotted at every reproduction event. This scenario is summarized as follows.

In the beginning, there is a single species, with one-to-one correspondence between phenotype and genotype. Here, there is little genetic and phenotypic diversity that are continuously distributed (see Fig. 1a). We assume that the isologous diversification starts because of developmental plasticity with interaction, when the number of these organisms increase. Indeed, the existence of such phenotypic differentiation is supported by isologous diversification theory and also by several numerical experiments, which gives the following stage I.

Stage I: Interaction-induced phenotypic differentiation

When there are many individuals interacting for finite resources, their phenotypes start to differentiate even though the genotypes are identical or differ only slightly. Phenotypic variables split into two (or more) types (see Fig. 1b). This interaction-induced differentiation is an outcome of the aforementioned mechanism. Slight phenotypic difference between individuals is amplified by the internal dynamics, while the phenotypes tend to be clustered into two (or more) types through the interaction between organisms. Here the two distinct phenotype groups (brought about by interaction) are called "upper" and "lower" groups, tentatively.

This differentiation is brought about because the population consisting of individuals taking identical phenotypes is destabilized by the interaction. Such instability is, for example, caused by the increase of population or decrease of resources, leading to strong competition. Of course, if the phenotype $X_t^j(i)$ at a matured state is rigidly determined by developmental dynamics, such differentiation does not occur. The only assumption we make in the present theory is that there exists such developmental plasticity in the internal dynamics when the interaction is strong. Recall again that this assumption is theoretically supported.

Note that at this stage the difference is fixed at neither the genetic nor the phenotypic level. After reproduction, an individual's phenotype can switch to another type.

Stage II: Amplification of the difference through the genotype–phenotype relationship

At the second stage, the difference between the two groups is amplified at both the genotypic and the phenotypic level; this is realized by a kind of positive feedback process between the change of genotype and phenotype.

First, the genetic parameter(s) separate as a result of the phenotypic change, which occurs if the parameter dependence of the growth rate is different between the two phenotypes. Generally, there are one or several parameter(s) g^ℓ, such that the growth rate increases with g^ℓ for the upper group and decreases for the lower group (or the other way around) (Figs. 1c, 2).

Certainly, such a parameter dependence is not exceptional. As a simple illustration, assume that the use of metabolic processes is different between the two phenotypic groups. If the upper group uses one metabolic cycle more, then the mutational change of the parameter g^ℓ to enhance the cycle is in favor for the upper group, whereas the change to reduce it may be in favor for the lower group. Indeed, all numerical results support the existence of such parameters. This dependence of growth rate on the genotypes leads to the genetic separation of the two groups, so long as there is competition for survival, to keep the population numbers limited.

The genetic separation is often accompanied by a second process, the amplification of the phenotypic difference by the genotypic difference. In the situation of Fig. 1c, as a parameter G increases, a phenotype P (i.e., a characteristic quantity for the phenotype) tends to increase for the upper group and to decrease (or to remain the same) for the lower group.

It should be noted that this second stage is *always* observed in our model simulation when the phenotypic differ-

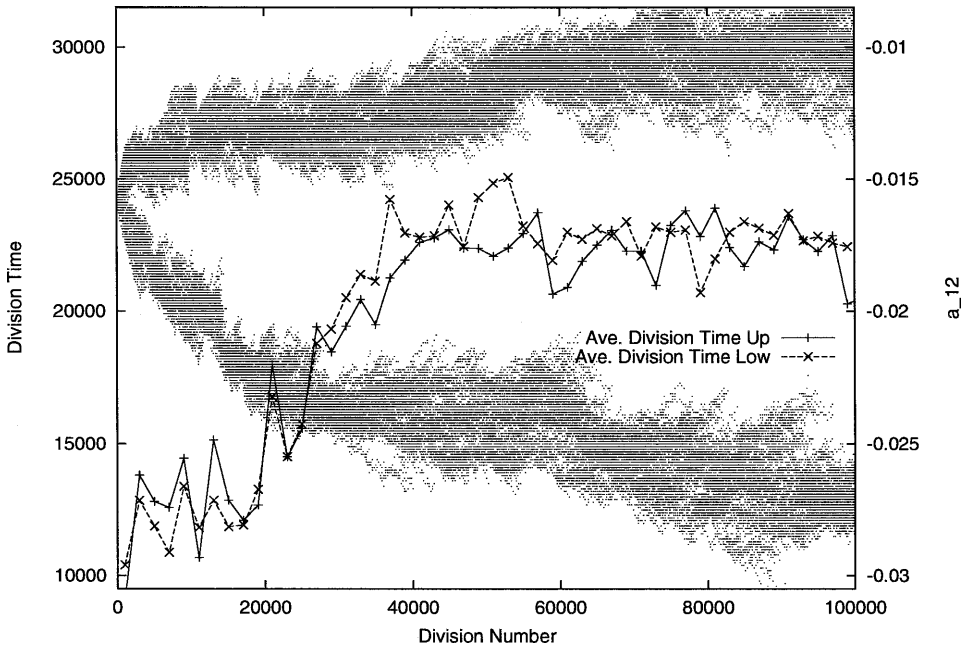


Fig. 2. Evolution of the genotypic parameter. The parameter $g = a^{12}(i)$ is plotted as a dot at every division (reproduction) event, with the abscissa as the division number. The average time necessary for division (reproduction) is plotted for the upper and lower groups, where the average is taken over 2000 division events (6th–8th generation). As the two groups are formed around the 2000th division event, the population size becomes twice the initial, and each division time is also approximately doubled. Note that the two average division speeds of the two groups remain of the same order, even when the genetic parameter evolves in time. (From Kaneko and Yomo 2000)

entiation at the first stage occurred. 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 g^m (e.g., enzyme catalytic activity) to enhance the cycle is in favor for the upper group, while the change to reduce it may be in favor for the lower group. Indeed, all the numerical results carried out so far support that such parameters always exist. This dependence of growth on genotypes leads to genetic separation of the two groups.

Stage III: Genetic fixation

After the separation of two groups has progressed, each phenotype (and genotype) starts to be preserved by the offspring, in contrast to the situation at the first stage. However, up to the second stage, each of the two groups with different phenotypes cannot exist in isolation by itself. When isolated, offspring with the phenotype of the other group start to appear. The two groups coexist depending on each other (see Fig. 1d).

Only at this third stage does each group start to exist on its own. Even if one group of units is isolated, no offspring with the phenotype of the other group appears. Now the two groups exist on their own. Such a fixation of phenotypes is possible through the evolution of genotypes (parameters). In other words, the differentiation is fixed into the genes (parameters). Now each group exists as an independent "species," separated both genetically and phenotypically. The initial phenotypic change introduced by interaction is fixed to the genes, and the "speciation process" is completed.

In contrast, at the second stage, the separation is not fixed rigidly. Units selected from one group at this earlier stage again start to show phenotypic differentiation, followed by genotypic separation, as demonstrated by several simulations. After some generations, one of the differentiated groups recovers the genotype and phenotype that had existed before the transplant experiment; this means that there remains some plasticity at this stage, which is in sharp contrast with the third stage.

Remarks on speciation by interaction-induced phenotypic differentiation

Dynamic consolidation to genotypes

At the third stage, two groups with distinct genotypes and phenotypes are formed, each of which has one-to-one mapping from genotype to phenotype. This stage now is regarded as speciation. (In the next section, we show that this separation satisfies hybrid sterility in sexual reproduction and is appropriately called speciation.) When we look at the present process only by observing initial population distribution (in Fig. 1a) and the final population distribution (in Fig. 1d), without information on the intermediate stages (given by Fig. 1b,c), one might think that the genes split into two groups by mutations and that as a result two phenotype groups are formed, because there is flow only from genotype to phenotype. As we know the intermediate stages, however, we can conclude that this simple picture does not hold here. Here, phenotype differentiation drives the genetic separation, in spite of the flow only from genotype to phenotype. Phenotype differentiation is consolidated to genotype, and then the offspring take the same phenotype as their ancestor.

Robust speciation

Note that the speciation process of ours occurs under strong interaction. At the second stage, these two groups form a symbiotic relationship. As a result, the speciation is robust in the following sense. If one group is eliminated externally, or extinct accidentally at the first or second stage, the remaining group again forms the other phenotype group and then genetic differentiation is started again. The speciation process here is robust against perturbations.

Coevolution of differentiated groups

Note that each of the two groups forms a niche for the other group's survival mutually, and each of the groups is specialized in this created niche. For example, some chemicals secreted by one group are used as resources for the other, and vice versa.

Hence, the evolution of two groups is mutually related. At the first and second stages of evolution, speed of reproduction is not so different between the two groups. Indeed, at these stages, the reproduction of each group is strongly dependent on the other group, and the "fitness" as a reproduction speed of each group by itself alone cannot be defined. At stage II, the reproduction of each group is balanced through the interaction, so that one group cannot dominate in the population (see Fig. 2).

Phenotype differentiation is necessary and sufficient for the sympatric speciation in our theory

Sufficient. If phenotypic differentiation at stage 1 occurs in our model, then the genetic differentiation of the later stages *always* follows, in spite of the random mutation process included. How long it takes to reach the third stage can depend on the mutation rate, but the speciation process itself does not depend on the mutation rate. However small the mutation rate may be, speciation (genetic fixation) always occurs.

Once the initial parameters of the model are chosen, it is already determined whether interaction-induced phenotype differentiation will occur. If it occurs, then genetic differentiation always follows.

Necessary. On the other hand, in our setting, if interaction-induced differentiation does not exist initially, there is no later genetic diversification process. If the initial parameters characterizing nonlinear internal dynamics or the coupling parameters characterizing interaction are small, no phenotypic differentiation occurs. Also, the larger the resource per individual, the smaller the effective interaction. Then, phenotypic differentiation does not occur. In these cases, even if we take a large mutation rate, differentiation into distinct genetic groups does not appear, although the distribution of genes (parameters) is broader. Also, we have also made several simulations starting from a population of units with widely distributed parameters (i.e., genotypes). However, unless the phenotypic separation into distinct groups is formed, the genetic differentiation does not follow (Fig. 3a,b). Only if phenotype differentiation occurs does genetic differentiation follow (Fig. 3c,d).

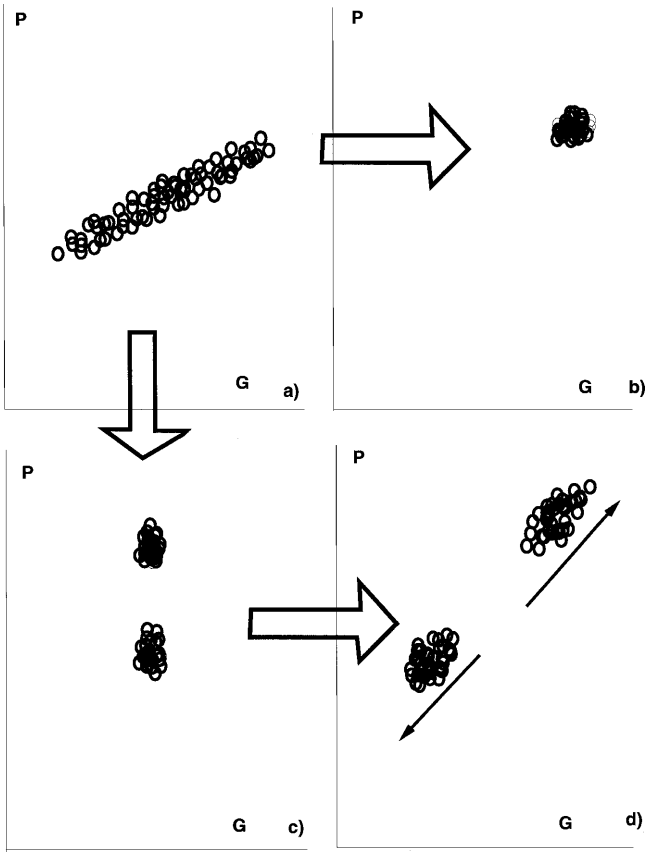


Fig. 3. Schematic representation of evolution starting from large genetic variance. **a** Initial genotypic and phenotypic distribution. **b** Without the phenotypic differentiation, no speciation follows. **c** If phenotypic differentiation occurs, then speciation follows later **d**

For some other models with many variables and parameters, the phenotypes are often distributed broadly, but continuously without making distinct groups. In this case again, distinct genetic groups do not appear through the mutations, although the genotypes are broadly distributed (Fig. 4).

Postmating isolation

The speciation process is defined both by genetic differentiation and by reproductive isolation (Dobzhansky 1937). Although evolution through stages I to III leads to genetically isolated reproductive units, one might still say that it should not be called speciation unless the process shows isolated reproductive groups under sexual recombination. In fact, it is not trivial if the present process works with sexual recombination because the genotypes from parents are mixed by each recombination. To check this problem, we have considered some models in which sexual recombination occurs to mix genes. To be specific, reproduction occurs when two individuals i_1 and i_2 satisfy the

maturity condition, and the two genotypes are then mixed. As an example we have produced two offspring $j = j_1$ and j_2, from the individuals i_1 and i_2 as

$$g^{\ell}(j) = g^{\ell}(i_1) r_j^{\ell} + g^{\ell}(i_2)\left(1 - r_j^{\ell}\right) + \delta \qquad (1)$$

with a random number $0 < r_j^{\ell} < 1$ to mix the parent genotypes (see also Appendix).

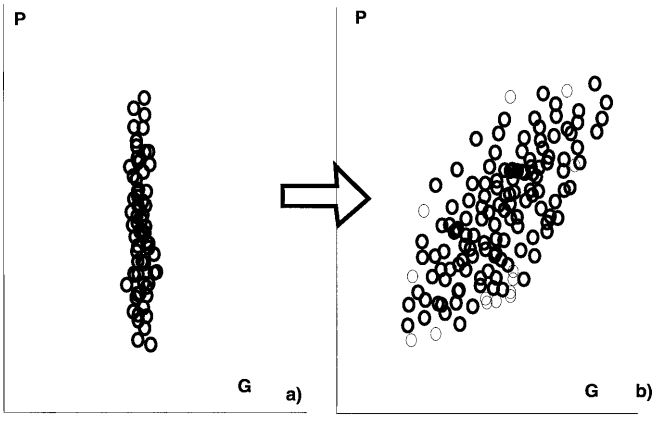


Fig. 4. Schematic representation of evolution when phenotype is distributed without clear differentiation to discrete state. **a** Initial distribution of phenotypes and genotypes. **b** After evolution, genes are also distributed broadly, but no speciation follows

In spite of this strong mixing of genotype parameters, the two distinct groups are again formed. Mating between the two groups can, of course, produce an individual with parameters in the middle of the two groups. When the parameters of an individual acquire values intermediate between those of the two groups, at whatever phenotype it can assume, its reproduction takes a much longer time than that of the two groups. Before the reproduction condition is satisfied, the individual has a higher probability to be removed by death. As the separation process of the two groups progresses further, an individual with intermediate values never reaches the condition for reproduction before it dies.

This postmating isolation process is demonstrated clearly by measuring the average offspring number of individuals over given parameter (genotype) ranges and over some time span. An example of this average number of offspring is plotted in Fig. 5 with progress of the speciation process. As the two groups with distinct parameter values are formed, the average offspring number of an individual having parameters between those of the two groups starts to decrease. This number soon 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, without any assumption about mating preference. Now genetic differentiation and reproductive

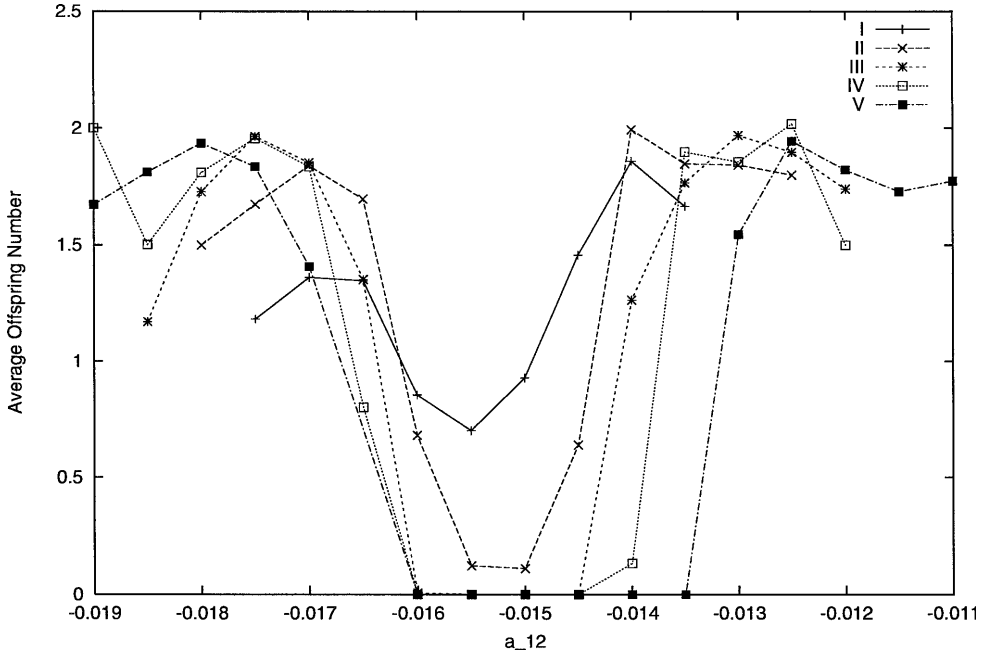


Fig. 5. The average offspring number before death is plotted as a function of the parameter (genotype), for simulations with sexual recombination. As an extension to include sexual recombination, we have also studied a model in which two organisms satisfying this threshold condition mate to reproduce two offspring. When they mate, the offspring have parameter values that are a randomly weighted average of those of the parents (as given in the text). We have measured the number of offspring for each individual during its life span. By taking a bin width 0.005 for the genotype parameter $g = a^{12}$, 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* ($3.75–4.5 \times 10^4$). As shown, a hybrid offspring will be sterile after some generations. Here we have used the model of the Appendix and the initial condition as in Fig. 1 and imposed recombination, with the parameters $p = 1.5/(2\pi)$ and $s^1 = s^2 = s^3 = 2$. In the run, the population fluctuates around 340. (From Kaneko and Yomo 2000)

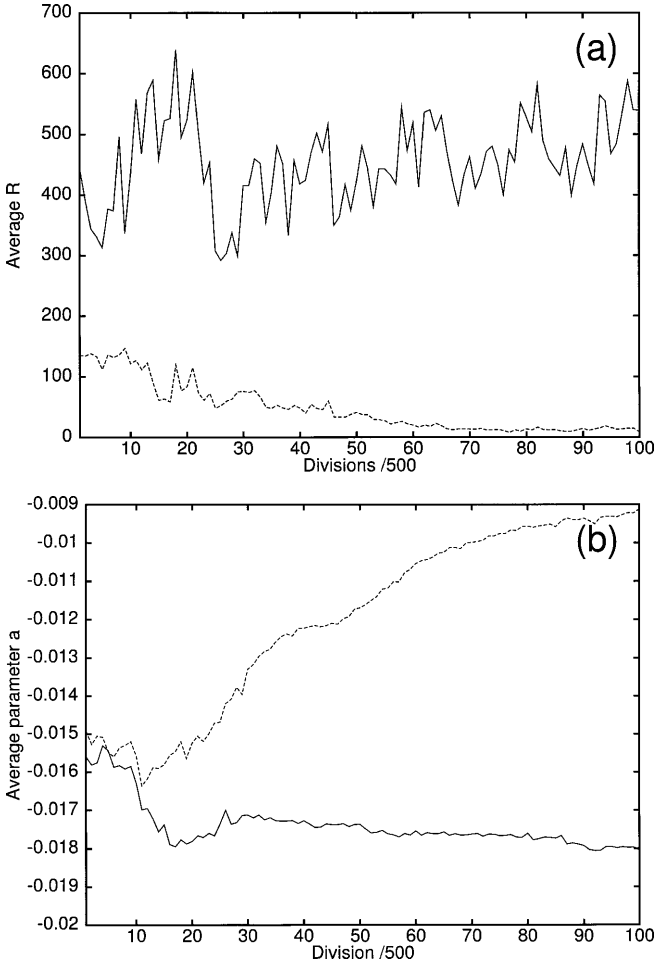


Fig. 6. An example of the speciation process with sexual recombination and the evolution of mating preference, with the model described in the Appendix. Here two groups of distinct phenotype (large X^1, small X^2) and (small X^1, large X^2) are formed at the first few generation, which we call "up" and "down" groups. We have measured the average $\overline{X^j}$ at reproduction events, $\overline{a^{\ell m}}$, $\overline{\rho^j}$ for each group per 500 divisions. (The population here is roughly 500, and thus the average is roughly over one generation.) Change of the average $\overline{R^j}$, $\overline{a^{\ell m}}$, and $\overline{X^1}$ are plotted with divisions (generations). $\delta\rho = 20$. **a** $\overline{X^1}$ (up group; *solid line*), $\overline{X^1}$ (down group; *broken line*); **b** $\overline{a^{12}}$ (up group; *solid line*), $\overline{a^{12}}$ (down group; *broken line*); **c** $\overline{\rho^1}$ (up group; *solid line*), $\overline{\rho^1}$ (down group; *broken line*), $\overline{\rho^2}$ (up group; *broken line*), $\overline{\rho^2}$ (down group; *thin broken line*). (Adapted from Kaneko and Yomo 2002)

isolation are satisfied. Hence, it is proper to call the process through stages I to III speciation.

Evolution of mating preference

So far we have not assumed any preference in mating choice. Hence, a sterile hybrid continues to be born. It is then natural to expect that some kind of mating preference evolves to reduce the probability of producing a sterile hybrid. Here we consider how mating preference evolves as a result of postmating isolation.

As a simple example, it is straightforward to include loci for mating preference parameters. We assume another set of genetic parameters that controls the mating behavior. For example, each individual i has a set of mating threshold parameters $(\rho^1(i), \rho^2(i), \ldots \rho^k(i))$, corresponding to the phenotype $(X^1(i), X^2(i), \ldots, X^k(i))$. If $\rho^\ell(i_1) > X^\ell(i_2)$ for some ℓ, individual i_1 denies mating with i_2 even if i_1 and i_2 satisfy the maturity condition. In simulation with a model with $\{\rho^m(i)\}$, we choose a pair of individuals that satisfy the maturity condition and check if one does not deny the other. Only if neither denies mating with the other does mating occur to produce offspring, when the genes from parents are mixed in the same way as in the previous section. If these conditions are not satisfied, the individuals i_1 and i_2 wait for the next step to find a partner again (see also Appendix).

Here the set of $\{\rho^m\}$ is regarded as a set of (genetic) parameters, and changes occur by mutation and recombination. Mutation is given by addition of a random value to $\{\rho^m\}$. Initially, all $\{\rho^m\}$ (for $m = 1, \ldots, k$) are smaller than the minimal value of $(X^1(i), X^2(i), \ldots, X^k(i))$, so that no mating preference exists. If some $\rho^\ell(i)$ becomes larger than some of $X^\ell(i')$, mating preference appears.

An example of numerical results is given in Fig. 6, where the change of phenotype X^m and some of the parameters g^j are plotted. Here, by phenotype differentiation, one group ("up" group) has a large X^m value for some $m = \ell$ and almost null values for some other $m = \ell'$. Hence, sufficiently large positive $\rho^{\ell'}$ gives a candidate for mating preference.

Right after the formation of two genetically distinct groups that follows phenotype separation, one of the mating threshold parameters $(\rho^1(i_1))$ starts to increase for one group. In the example in Fig. 6, the "up" group has a phenotype with (large X^1, small X^2) and the other ("down") group with (small X^1, large X^2). There, the up group starts to increase $\rho^1(i_{up})$, and $\rho^1(i_{up}) > X^1(i_{down})$ is satisfied for an individual i_{down} of the down group. Now the mating between

the two groups is no longer allowed, and mating occurs only within each group. The mating preference thus evolved prohibits intergroup mating, producing sterile hybrids.

Note that the two groups do not simultaneously establish the mating preference. In some case, only one group has positive ρ^{ℓ}, which is enough for the establishment of mating preference, while in some other cases one group has positive ρ^1 and the other has positive ρ^2, wherein the mating preference is more rigidly established.

Although the evolution of mating preference here is a direct consequence of postmating isolation, it is interesting to note that the coexistence of the two species is further stabilized with the establishment of mating preference. Without this establishment, there are some cases in which one of the species disappears due to fluctuation after a very long time in the simulation. With establishment, the two species coexist much longer (at least within our time frame of numerical simulation).

Formation of allele–allele correlation

In a diploid there are two alleles, and the two alleles do not contribute equally to the phenotype. For example, often only one allele contributes to control of the phenotype. If by recombination the loci from two alleles are randomly mixed, then the correlation between genotype and phenotype achieved by the mechanism so far discussed might be destroyed. Indeed, this problem was pointed out by Felsenstein (1981) as one difficulty for sympatric speciation.

Of course, this problem is resolved if genotypes from two alleles establish high correlation. To check if this correlation is generated, we have extended our model to have two alleles and examined if the two alleles become correlated. Here, we adopted the model studied so far and further added two alleles (see also Appendix). In mating, the alleles from the parents are randomly shuffled for each locus. In other words, each organism i has two sets of parameters, $\{g^{(+)\ell}(i)\}$ and $\{g^{(-)\ell}(i)\}$. Each $g^{(+)m}(i)$ is inherited from either $g^{(+)m}$ or $g^{(-)m}$ of one of the parents, and the other $g^{(-)m}(i)$ is inherited from either $g^{(+)m}$ or $g^{(-)m}$ of the other parent. Here parameters at only one of the alleles work as a control parameter for the developmental dynamics of the phenotype.

We have carried out some simulations of this version of our model (Kaneko, unpublished). Here again speciation proceeds in the same way, through stages I, II, and III. Hence our speciation scenario works well in the presence of alleles.

In this model, the genotype–phenotype correspondence achieved at stage III could be destroyed if there was no correlation between two alleles. Hence we have plotted the correlation between two alleles by showing the two-dimensional pattern $(g^{(+)1}(i), g^{(-)1}(i))$ in Fig. 7. Initially there was no correlation, but through temporal evolution the correlation is established. In other words, speciation in the phenotype is consolidated to the genes and later is consolidated to correlation between two alleles.

Allopatric speciation as a result of sympatric speciation

As already discussed, speciation, according to our theory, starts from phenotypic differentiation and proceeds to genetic differentiation and then to postmating isolation, and finally to premating isolation (mating preference). This order might sound strange in contrast to the commonly adopted point of view, but we have shown that this order is a natural and general consequence of a system with developmental dynamics with potential plasticity by interaction.

In a biological system, we often tend to assume a causal relationship between two factors from observation of merely correlation of the two factors. For example, when the residence area of two species that share a common ancestor species is spatially separated, we often guess that this spatial separation is a cause for speciation. Indeed, allopatric speciation is often adopted for explanation of speciation in nature.

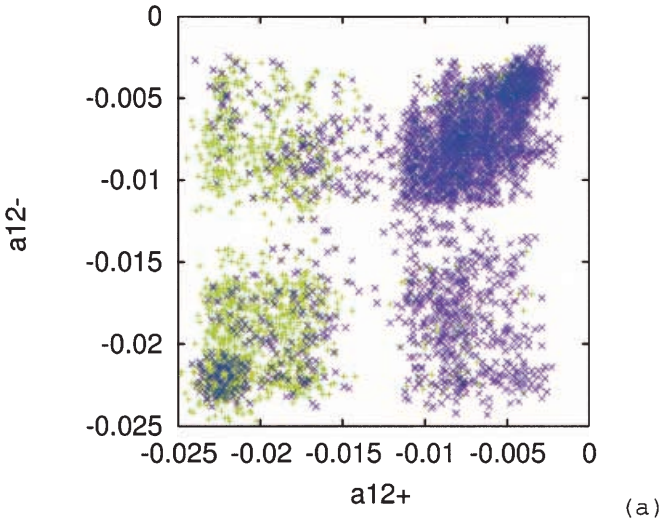
However, in many cases, what we have observed in the field is just correlation between spatial separation and speciation; which of these is the cause is not necessarily proved. Rather, spatial segregation can be a result of (sympatric) speciation. Consider, for example, the segregation of residential areas in a city between rich and poor people. Most of us do not assume that people in the "rich" area are rich because they live there. Rather, most think that the spatial separation is a *result* of differentiation in wealth, but not a *cause*. In the same way, it is sometimes dangerous to assume allopatric speciation even if the residence areas of two species are separated.

By extending our theory so far, we can show that spatial separation of two species has resulted from the sympatric speciation discussed here. To study this problem, we have extended our model by allocating to each organism a resident position in a two-dimensional space. Each organism can move around the space randomly but slowly, while resources leading to the competitive interaction diffuse throughout space much faster. If the two organisms that satisfy the maturation condition meet in the space (i.e, they are located within a given distance), then they mate with each other to produce offspring.

In this model, we have confirmed that sympatric speciation first occurs through stages I–III. Later, these two differentiated groups become spatially segregated (Fig. 8). Now, sympatric speciation is shown to be consolidated to spatial segregation (Kaneko, in preparation).

The spatial segregation here is observed when the range of interaction is larger than the typical range of mating. For example, if mobility of resources causing competitive interaction is larger than the mobility of organisms, spatial segregation of sympatrically formed species is resulted.

Instead of a spatially local mating process, one can assume a slight gradient of an environmental condition, for example, a gradient in resources. In this case, again, it is expected that sympatric speciation takes place first and spatial separation occurs later accordingly.



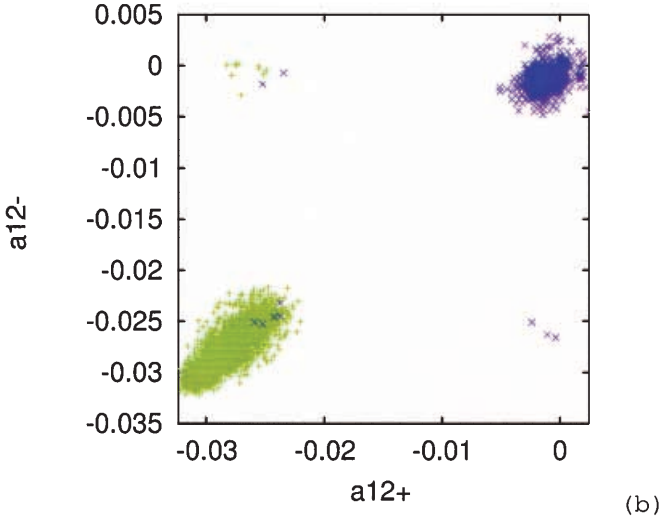


Fig. 7. Correlation in alleles. Using the model in the Appendix, the plot of $(a^{(+)12}(i), a^{(-)12}(i))$ is plotted at every reproduction event, with $p = 1.6$, $s_1 = s_2 = s_3 = 4$, $\delta\rho = 20$. Initially, genotype parameter a^{ij} is set at $-0.01/(2\pi)$. Two alternate groups with distinct phenotypes are plotted with alternate colors. **a** Plots at the division from 20000th to 36000th. Here phenotypic differentiation already occurred but the genetic separation is not completed. **b** Plots at the division at a much later stage (from 50000th to 63000th) where genetic separation has already occurred

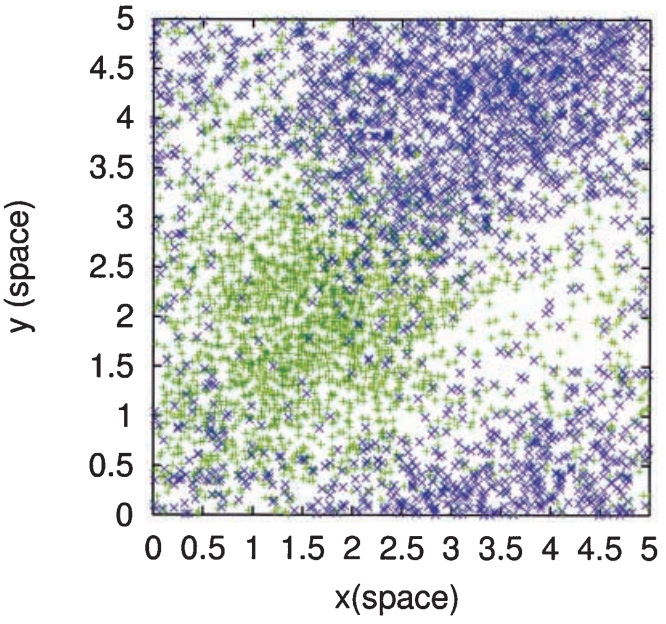


Fig. 8. Spatial separation of two species, observed in the model of the Appendix. Model in spatially local mating. Each unit moves in position with Brownian motion, given as the random number over $[-\delta_f, \delta_f] = [-0.0025, 0.0025]$, while units are located within a two-dimensional square of the size 5×5, with periodic boundary condition. Mating is possible if two units satisfying mature condition are located within the distance 0.25. The parameters are set as $p = 1.6$, $s_1 = s_2 = s_3 = 2$; initial genotype parameter aij is set at $-0.01/(2\pi)$. Position of the units at every division is plotted, at each division event from 45000 to 50000. Two alternate groups with distinct phenotypes and genotypes are plotted with alternate colors

To sum up, we have pointed out here the possibility that some speciation that is considered to be allopatric can be a result of sympatric speciation by our mechanism. Sympatric speciation is later consolidated to cause spatial segregation of organisms.

Comparison with previous theories

Our theory reviewed here is related to several earlier theories. Here we briefly discuss the relationships and differences.

Frequency-dependent selection

Because our mechanism crucially depends on interaction, one might think that it is a variant of frequency-dependent selection. The important difference here is that phenotype may not be uniquely determined by the genotype, even though the environment (including the population of organisms) is given. In frequency-dependent selection, genetically (and accordingly phenotypically) different groups interact with each other, and the fitness depends on the population of each group (Futsuyma 1986). At the third stage of our theory, the condition for this frequency-dependent selection is satisfied, and evolution progresses with frequency-dependent selection. However, the important point in our theory lies in the earlier stages where a single genotype leads to different phenotypes. Indeed, this intrinsic nature of differentiation is the reason why the speciation process here works at any (small) mutation rate and also under sexual recombination, without any other ad hoc assumptions.

Baldwin's effect

In our theory, phenotype change is later consolidated to the genotype. Indeed, genetic "takeover" of phenotype change

was also discussed as Baldwin's effect (Baldwin 1896; Bonner 1980), where the displacement of phenotypic character is fixed to the genes. The process of genes becoming assimilated to reinforce that which is already occurring is called Baldwin's effect in general. The fixation to genotype at the second stage in our theory is understood as an example of this Baldwin's effect, whereas phenotypic differentiation in the first stage is also essential to the speciation mechanism proposed here. Here, we have demonstrated the relevance of Baldwin's effect to speciation explicitly, by considering the developmental process using a model with (metabolic) reaction dynamics.

Application of Baldwin's effect to the developmental process itself was discussed by Waddington (1957). In his discussion, the phenotype character is given by an epigenetic landscape. In our case, phenotypic differentiation is formed through the developmental process to generate different characters as a result of the interaction. Distinct characters are stabilized with each other through the interaction. With this interaction dependence, the two groups are necessary to each other, and a robust speciation process is the result. Although the application of Baldwin's effect to speciation is novel here, the stabilization of development by genetic assimilation was discussed by Newman (1994).

Baldwin's effect is often discussed in the context of transmission of a novel type of behavior. Such behavior is sometimes related to the morphology of an organism. For example, consider the sympatric speciation of cichlids in African lakes. It is known that two species coexist that eat the gills of other fish from either the left or the right side, respectively. This difference in behavior is also reflected in the morphology of the mouth of the fish. The mouth of each species is curved to the right or to the left, respectively, so that it is easier for them to attack the gills from one side. In other words, distinct types of behavior and developmental process are tightly coupled. Our theory suggests that developmental plasticity induced by interaction with other fish first led to differentiation of phenotypes into "right-side" and "left-side" types, with regards to both behavior and development. Later, this difference was assimilated to the genes, as shown by the second stage of our theory that is given by Baldwin's effect. We suggest that speciation into these two types thus progressed rapidly in these lakes.

Reinforcement

Because the separation of two groups with distinct phenotypes is supported by the interaction, the present speciation mechanism is possible without supposing any mating preference. In fact, the hybrid becomes inferior in reproduction rate, and mating preference based on discrimination in the phenotype is shown to evolve. Indeed, a mechanism to amplify the differentiation by mating preference has been searched for as reinforcement since Dobzhansky (1937). Our theory also gives a plausible basis for the evolution of mating preference without assuming ad hoc reinforcement, or without any presumption as to the inferiority of the hybrid.

Phenotypic plasticity

Note that our phenotypic differentiation through development is different from the so-called phenotypic plasticity in which a single genotype produces alternative phenotypes in alternative environments (Callahan et al. 1997; Spitze and Sadler 1996; Weinig 2000). In contrast, in our case, distinct phenotypes from a single genotype are formed under the same environment. In fact, in our model, this phenotypic differentiation is necessary to show later genetic differentiation. Without this differentiation, even if distinct phenotypes appear for different environments as in phenotypic plasticity, genetic differentiation does not follow. In spite of this difference, it is true that both are concerned with flexibility in phenotypes. Some phenotypic plasticity so far studied may bring about the same developmental flexibility as ours under a different environmental condition.

Resource competition

In our case, competitive interaction is relevant to speciation. Indeed, coexistence of two (or more) species after the completion of the speciation is discussed as resource competition by Tilman (1976, 1981). Although his theory gives an explanation for coexistence, the speciation process is not discussed, because two individuals with a slight genotypic difference can have only a slight difference there. In our theory, even if the genotypes of two individuals are the same or only slightly different, their phenotypes can be of quite different types. Accordingly, our theory provides a basis for resource competition also.

Relevance of our theory to biological evolution

The general conclusion of our theory is that sympatric speciation can generally occur under strong interaction, if the condition for interaction-induced phenotype differentiation is satisfied. We briefly discuss the relevance of our theory to biological evolution.

Tempo in the evolution

Because speciation as discussed here is triggered by interaction, the process is not so much random as deterministic. Once the interaction among individuals brings about phenotypic diversification, speciation always proceeds directionally without waiting for a rare, specific mutation. The evolution in our scenario has a deterministic nature and a fast tempo for speciation, which is different from the typical stochastic view of mutation-driven evolution.

Some of the phenotypic explosions in the history of evolution have been recorded as having occurred within short geologic periods. Following these observations, punctuated equilibrium was proposed (Gould and Eldredge

1977). Our speciation scenario possibly gives an interpretation of this punctuated equilibrium; it may have followed the deterministic, faster way of interaction-induced speciation.

Why low penetrance is frequent in mutants

In the process of speciation, the potentiality of a single genotype to produce several phenotypes is consumed and may decline. After the phenotypic diversification of a single genotype, each genotype newly appears by mutation and takes one of the diversified phenotypes in the population. Thus, the one-to-many correspondence between the original genotype and phenotypes is utilized. Through the present process of speciation, the potentiality of single genotypes to produce various phenotypes decreases unless the new genotypes introduce another positive feedback process to amplify the small difference.

As a result, one may see single genotypes expressing only one (or a small number of) phenotypes in nature. Because most organisms at the present time have gone through several speciation processes, they may have reduced their potential to produce various phenotypes. According to our theory, if the organisms have high potential, they will soon undergo a speciation process and their potential will decrease. In other words, natural organisms tend to lose the potential to produce various phenotypes in the course of evolution. As a reflection on the evolutionary decline of the potential, one can expect that mutant genotypes tend to have a higher potential than the wild-type genotype. As already mentioned, low or incomplete penetrance (Opitz 1981) is known to occur often in mutants, compared with higher penetrance in the wild type. Our result is consistent with these observation, because wild types are, in most cases, a consequence of evolution, where the one-to-one correspondence is recovered, whereas mutants can have higher potential to have a loose correspondence.

Relevance of developmental plasticity to speciation

Relationship between development and evolution has been discussed extensively. Our theory states the relevance of developmental plasticity to speciation. Taking our results and experimental facts into account, one can predict that organisms emerging as a new species have a high potential to produce a variety of phenotypes. It is interesting to discuss why insects, for example, have a higher potential for speciation from this point of view. Also, examining if living fossils such as *Latimeria chalumnae* and *Limulus* have a stable expression of a small number of phenotypes is rewarding.

In our speciation theory, plasticity declines through evolution. Of course, there should be some occasions when potentiality is regained so that evolution continues. For example, changes of environment may influence the developmental dynamics to regain loose correspondence, or introduction of novel degrees of freedom or genes may provide such looseness. Endosymbiosis can be one of such causes. Also, changes in interaction through spatial factors may introduce novel instability in dynamics, resulting in loose correspondence.

It should also be noted that developmental processes are often sensitive to environmental conditions, as recent studies on environmental endocrine disruptors have suggested. The developmental plasticity, once lost by the completion of speciation, may be regained by some changes of environmental conditions, which are given by interactions with other organisms.

Unified theory for speciation in sexual and asexual (and unicellular) organisms

One important point in our theory is that speciation in asexual and sexual organisms is explained within the same theory. Of course, the standard definition of species using hybrid sterility is applied only to sexual organisms. However, it is true that the asexual organisms, or even bacteria, exhibit discrete genotypes and phenotypes. It is suggested that "species," i.e., discrete types with reproductive isolation, may exist in asexual organisms (Roberts and Cohan 1995; Holman 1987). There is also discussions that the potential for speciation in asexual organisms is not lower than that in sexual organisms. In this sense, the present theory sheds new light on the problem of speciation in asexual organisms as well.

Reversing the order

According to our theory, sympatric speciation under sexual reproduction starts first from phenotypic differentiation, and then genetic diversification takes place, leading to hybrid sterility, and finally the speciation is fixed by mating preference. This order may be different from studies most commonly adopted (while the order from phenotype to genotype was discussed as Baldwin's effect). Hence, our theory will be verified by confirming this chronic order in the field. One difficulty here, however, lies in that the process from phenotypic differentiation to the last stage is rather fast according to our theory. Still, it may be possible to find this order in the field, by first searching for phenotypic differentiation of organisms with identical genotype and under the identical environment. In this respect, the data of cichlids of Nicaraguan lake may be promising (Wilson et al. 2000), because phenotypic difference corresponding to a different ecological niche is observed even though a clear genetic difference has not yet been observed.

Experimental verification

Discussion of the mechanism of evolution using past data, however, often remains anyone's guess. Most important in our scenario, in contrast, is its experimental verifiability, as the process of speciation is rather rapid. For example, the evolution of *E. coli* can be observed in the laboratory, as has

been demonstrated by Kashiwagi et al. (1998, 2001) and Xu et al. (1996). Phenotypic differentiation of *E. coli* is experimentally observed when their introduction is strong. Because the strength of interaction can be controlled by resources and population density, one can check whether evolution at the genetic level is accelerated through interaction-induced phenotypic diversification (Kashiwagi et al. 2001). Examination of the validity of our speciation scenario will provide a first step to such study.

Summary: dynamic consolidation

We have shown that developmental plasticity induced by interaction leads to phenotypic differentiation, which is consolidated to the genes. Thus, distinct species with distinct genotypes and phenotypes are formed, leading to hybrid sterility; later, mating preference evolves. Further, this differentiation can be fixed to correlation in alleles or to spatial segregation. The original differentiation in the phenotype can be understood as symmetry arising from a homogeneous state, in terms of physics, whereas successive consolidation of the changed symmetry to different properties observed at later stages is more important for biological evolution. This dynamic process of consolidation is a key factor in development and evolution (see also Newman 2002).

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Appendix: an example of our model

A coupled map model

To be specific, we consider the following model.

We studied a simple abstract model of evolution with an internal dynamical process for development. In the model, each individual i has several (metabolic or other) cyclic processes, and the state of the jth process at time n is given by $X_n^j(i)$. With k such processes, the state of an individual is given by the set $(X_n^1(i), X_n^2(i), \ldots, X_n^k(i))$, 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 changes temporally according to a set of deterministic equations with some parameters. To be specific, our toy model consists of the following dynamics (Kaneko 1998).

1. Dynamics of the state. Here, we split $X_n^j(i)$ into its integer part $R_n^\ell(i)$ and the fractional part $x_n^\ell(i) = \mathrm{mod}[X_n^\ell(i)]$. The integer part $R_n^j(i)$ is assumed to give the number of times the cyclic process has occurred since the individual's birth, while the fractional part $x_n^\ell(i)$ gives the phase of oscillation in the process. The dynamics of the variables $x_n^j(i)$ consist of a mutual influence of cyclic processes and interaction with other organisms. As a simple example, the former is assumed to be given by $\sum_m \frac{a^{\ell,m}}{2}\sin\left(2\pi x_n^m(i)\right)$, while the latter is given by the competition for resources among the N_n organisms existing at the moment, given by $I^\ell(i) = p\sin\left(2\pi x_n^\ell(i)\right) + \frac{s^\ell - \sum_j p\sin 2\pi\left(x_n^\ell(i)\right)}{N_n}$. (The second term comes from the constraint $\Sigma_i I^\ell(i) = s^\ell$, i.e., the condition that N individuals compete for a given resource s^ℓ at each time step. The first term represents the ability to secure the resource, depending on the state.) Our toy model is given by

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

2. Growth and death. Each individual splits into two when a given condition for growth is satisfied. Taking into account that the cyclic process corresponds to a metabolic, genetic, or other process that is required for reproduction, we assume that the unit replicates when the accumulated number of cyclic processes goes beyond some threshold. Thus, the condition is given by $\Sigma_\ell X_n^\ell(i) \geq$ Thr (the maturity condition). (Thr is set at 1000 for most simulations.) The state $X_n^\ell(i)$ is reset to a random value between 0 and 1, when the corresponding individual splits. To introduce competition, individuals are eliminated by a given death condition, as well as by random removal with a given rate. As for the former condition, an individual with $X_n^\ell(i) < -10$ (i.e., with a reverse process) is removed.
3. Genetic parameter and mutation. Following the discussion in the text, genes are represented as parameters in the model, because the control parameters affect the dynamics of phenotypic variables, but no direct reverse process exists, as dictated by the central dogma of molecular biology. Here, genotypes are given by a set of parameters $a^{m\ell}(i)$, representing the relationship between the two cyclic processes ℓ and m ($1 \leq \ell, m \leq k$). This set of parameters changes slightly through mutation when offspring is reproduced. With each division, the parameters $a^{m\ell}$ are changed to $a^{m\ell} + \delta$ with δ, a random number over $[-\varepsilon, \varepsilon]$, with small ε, corresponding to the mutation rate.

In the present model, due to the nonlinear nature of the dynamics, x_n^ℓ often oscillates in time chaotically or periodically. Hence, it is natural to use $X^\ell(j)$, including its integer part, as a representation of the phenotype because its integer part represents the number of cyclic processes used for reproduction.

An alternative model using catalytic reaction network

We have also carried out some simulations of a model with a reaction network where $X^m_t(i)$ represents the mth chemical concentration of an individual i. Each individual obtains resources depending on its internal state. Through the foregoing catalytic reaction process, some products are synthesized from the resources. When they are beyond a given threshold, they split into two, as given in the model for isologous diversification (Kaneko and Yomo 1994, 1997, 1999; Furusawa and Kaneko 1998). With the increase of the number of individuals, they compete for resources, while they are removed randomly to include competition. Because genes code the catalytic activity of enzymes, the rate of each reaction in the catalytic network is controlled by a gene. Hence, as a genetic parameter g^ℓ, the parameter for each reaction rate is adopted. Through the mutation of this reaction rate, the speciation process discussed throughout this article is also observed (Takagi et al. 2000).

Sexual reproduction

To include sexual recombination, we have extended our model so that organisms satisfying the threshold condition mate to reproduce two offspring. When they mate, the offspring have parameter values that are intermediate to those of the parents. Here, the offspring $j = j_1$ and j_2 are produced from the individuals i_1 and i_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 $0 < r^\ell_j < 1$ to mix the parental genotypes.

Mating preference

Here the set of $\{\rho^m\}$ is introduced as a set of (genetic) parameters, and changes are by mutation and recombination. The mutation is given by addition of a random value over $[-\delta_\rho, \delta_\rho]$. Initially $\rho^m \leq 0$ (for $m = 1, \ldots, k$) is set smaller than the minimal value of $(X^1(i), X^2(i), \ldots, X^k(i))$, so that no mating preference exists. If $\rho^m(i)$ becomes larger than some of $X^m(i')$, mating preference appears.

Model with two alleles and random shuffling by mating

Here we assume that each individual has two sets of parameters $\{a^{\ell m}(j)\}$, given by $a^{(+)\ell m}(j)$ and $a^{(-)\ell m}(j)$. In mating, the alleles from the parents are randomly shuffled for each locus ($\ell \cdot m$). Each $a^{(+)\ell m}(i)$ is inherited from either $a^{(+)\ell m}(p_1)$ or $a^{(-)\ell m}(p_1)$ of one of the parents, p_1, and the other $a^{(-)\ell m}(i)(p_2)$ is inherited from either $a^{(+)\ell m}(p_2)$ or $a^{(-)\ell m}(p_2)$ of the other parents, p_2. For the dynamics for X, only $\{a^{(+)\ell m}(i)\}$ is used. The other parameter $a^{(-)\ell}m$ is not used but can be used as $\{a^{(+)\ell m}(i_{\text{offspring}})\}$ of the offspring after the shuffling.

Spatial model

To an individual i in the model of with sexual reproduction, spatial position $(x_t(i), y_t(i))$ is assigned. The individual shows Brownian motion in the two-dimensional space, by adding a random number over $[-\delta_f, \delta_f]$ to $(x_t(i), y_t(i))$ to each step. They move within a square of a given size with a periodic boundary condition. If two individuals i and j that satisfy the maturity condition ($\Sigma_m X^m_t(i) > \text{Thr}$) are within a given distance d, they can reproduce two offspring, which are located between $(x_t(i), y_t(i))$ and $(x_t(j), y_t(j))$.

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