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COMPUTER SYMBIOSIS – EMERGENCE OF SYMBIOTIC BEHAVIOR THROUGH EVOLUTION

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Symbiosis is cooperation between distinct species. It is one of the most effective evolutionary processes, but its dynamics are not well understood as yet. A simple model of symbiosis is introduced, in which we consider interactions between hosts and parasites and also mutations of hosts and parasites. The interactions and mutations form a dynamical system on the populations of hosts and parasites. It is found that a symbiotic state emerges for a suitable range of mutation rates. The symbiotic state is not static, but dynamically oscillates. Harmful parasites violating symbiosis appear periodically, but are rapidly extinguished by hosts and other parasites, and the symbiotic state is recovered. The relation between these phenomena and "TIT for TAT" strategy to maintain symbiosis is discussed.

1. Introduction

Symbiosis is a sophisticated evolutionary tactic. Symbiosis is a close relationship between species by which both species in the relationship benefit. Such "cooperative" relationships may actually begin as prey–predator relationships. The Lotka–Volterra equation [1], for example, shows an out-phased oscillating behavior of hosts and parasites. A prey and a predator in such model are not mutally beneficial. When we call symbiosis, it requires cooperative behavior between hosts and parasites. We argue that some qualitative changes in both hosts and parasites are requisites to symbiosis. Such changes are brought about by mutation, which may make the coexistence of hosts and parasites possible.

Evolution does not occur in a *fixed* environment. In nature, species can interact with many other species, and the term "environment" should include all interactions from other species. The strength of the interaction is dependent on the population of other species, and generally is time-dependent as well.

Here, the "environment" of hosts consists of parasites and vice versa. Both mutants of parasites and hosts can have more offspring if they gain larger benefits from the interaction with each other in the population at that time. Parasites have a benefit by mutating in a direction that increases harm to the hosts. We are interested in the question of how the population dynamics of hosts and parasites can reach a state in which they help each other, instead of attaining a state with growing harmful parasites.

Some of the symbiotic relationships are so strong that the species involved can no longer dissolve into free independent species. A eukaryotic cell is known to have a strong symbiotic relationship with Mitochondorous [2]. Such strong symbiosis is often encoded at the genetic level.

Another class of symbiosis is called loose symbiosis [2]. Such symbiotic relationships are loose enough to dissolve. A well-known example is lichen, which are the symbiosis between fungi and algae. Depending on environmental conditions, lichen may dissolve into two independent living

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species, or establish symbiosis from independent species [2]. Jeon's study of amoebae shows us that such symbiosis can be generated within a few days [3]. Another related example is the interaction between a virus and a host. Through evolution, the virus and the host may find a relationship in which the former attacks the latter less harmfully. The class of loose symbiosis is the focus of our study.

In the present paper we introduce a simple model for symbiosis. Through the simulation of the model, we try to understand the condition of the emergence of symbiosis and the dynamical nature of such symbiotic states.

The model consists of the population dynamics of two distinct genetic systems, called "host" and "parasite". Both hosts and parasites can change their genotypes by mutation. The genotypes are coded by bit strings. The difference between a host and a parasite lies in the interaction between the two. A host is uni-directionally attacked by all types of parasites but one. Such an exceptional parasite can be a symbiotic partner to the host. The more strongly a parasite exploits a host, the more offspring it has. Through mutations, hosts and parasites have a chance to generate a symbiotic relationship. A symbiotic state, however, is often unstable. There is no explicit reason to prevent the parasite from mutating into more harmful types, instead of remaining symbiotic. As will be seen, the symbiotic state exists only if the mutation rate of hosts is slightly larger than that of parasites. Furthermore, it will be shown that the attractor of the symbiotic state is not a fixed point but a limit cycle.

2. Model

We represent each species of host and parasite by a simple binary sequence of length L. Thus hosts and parasites can have 2^L different genotypes. Each genotype is represented as $0 = 0000000$, $1 = 0000001$, $2 = 0000010$, $3 = 0000011, \ldots$. The proportion[#1] of each genotype j in the population is represented by continuous variables on the 2^L-dimensional space, and is denoted by h_j and p_j corresponding to a host j and a parasite type j, respectively. To construct our dynamical model, we take into account the following constraints:

(1) Mutation of hosts and parasites: Hosts as well as parasites can mutate to other genotypes by only 1 bit. Hosts cannot become parasites or vice versa, that is, they mutate only among themselves. Since we restrict the gene space in a bit string of length L, each genotype has L neighbors by a 1-bit mutation. Hosts may mutate to a type which suffers less damage from parasites. Parasites can also mutate to a more harmful type. By denoting the mutation rates of hosts and parasites as μ_H and μ_P, we get the terms $\mu_H \Sigma_{j'}(h_{j'} - h_j)$ and $\mu_P \Sigma_{j'}(p_{j'} - p_j)$ for the population dynamics of hosts and parasites, respectively. Here, the summation over j' runs over all 1-bit neighbors of the binary sequence j.

(2) Interaction of hosts and parasites: For most parasites, hosts are uni-directionally exploited by parasites. The more advantage a parasite receives through the interaction with a host, the less advantage (or the more damage) the host receives by the interaction. If both a parasite and a host happen to take advantage of each other, such a pair of parasites and hosts is said to be in a symbiotic relation. This constraint leads to positive growth rates for parasites and mostly negative for hosts. Since a symbiotic host receives a benefit from a symbiotic parasite, a growth rate should be positive for the symbiotic host and parasite. The interaction term is written as $\Sigma_{k=1}^{2^L} a_{kj}^P p_j h_k$.

(3) Interaction among parasites: Since different parasites compete at the same host, we assume a mutual suppressing term in the equation of parasite.

[#1] "Proportion" here is not normalized by unity. It is normalized by the population at some (arbitrary chosen) time. Since the total population size can change in time, neither Σh_j nor Σp_j is constant.

No mutual of self suppression is assumed for the host system. The host can only increase or decrease its number by interacting with parasites. This constraint leads to the term $-\sum_{i\neq j} p_i p_j$ on the growth of parasite j.

Combining the constraints (1)–(3), we can write down the following set of equations on the population of the jth species of hosts $h_j(t)$ and that of parasites $p_j(t)$:

$$\frac{dp_j}{dt} = \sum_{k=1}^{2^L} a^{\mathrm{P}}_{kj} p_j h_k - \sum_{i\neq j} p_i p_j + \mu_{\mathrm{P}} \sum_{j'} (p_{j'} - p_j), \tag{1}$$

$$\frac{dh_j}{dt} = \sum_{k=1}^{2^L} a^{\mathrm{H}}_{kj} h_j p_k + \mu_{\mathrm{H}} \sum_{j'} (h_{j'} - h_j). \tag{2}$$

If $a^{\mathrm{P}}_{kj} > 0$ and $a^{\mathrm{H}}_{kj} < 0$, the interaction term takes the same form as the Lotka–Volterra equation with prey and predator. If both a^{P}_{kj} and a^{H}_{kj} are positive, the host and parasite help each other to increase the population through the interaction.

The next choice of our model is the dependence of interaction terms a^{P}_{kj} and a^{H}_{kj} on types k and j. From the constraint (2), the terms must have the following properties:

(i) There must be a pair of host and parasite types such that both a^{P}_{kj} and a^{H}_{kj} are positive, that is, the interaction results in "cooperation". We call such pairs of indices k, j a *symbiotic pair*. The interactions are constructed to have the symbiotic pairs.

(ii) If a host and a parasite do not form a symbiotic pair, then the parasite gets a positive gain from the interaction whereas the host receives a negative gain. This difference of the sign in the additional interaction term sets apart a parasite from a host.

We adopt the simplest form with these conditions as follows:

$$a^{\mathrm{P}}_{kj} = f_{\mathrm{P}} + \mathrm{Ham}(k, j), \tag{3}$$

$$a^{\mathrm{H}}_{kj} = f_{\mathrm{H}} - \mathrm{Ham}(k, j), \tag{4}$$

where the coefficients f_{P} and f_{H} are positive, and the function $\mathrm{Ham}(k, j)$ denotes the Hamming distance between the binary sequences of k and j[#2]. In the present model, parasites have larger growth rates if their bit patterns *mismatch* those of hosts, while hosts have larger growth terms if their bit patterns *match* those of parasites.

We take $f_{\mathrm{H}} < 1$, so that only a pair with a perfect matching develops a symbiotic state. Since all pairs (m, m) are symbiotic, there are 2^L possible symbiotic pairs. It can be shown by linear stability analysis that a state with only one symbiotic pair ($p_j = h_j = 0$ for $\forall j \neq m$) is unstable. As we see in section 3, an observed symbiotic state indeed contains small population ratio of non-symbiotic pairs.

In what follows, we set the bit length L equal to 7, and represent each genotype as $0 = 0000000$, $1 = 0000001, \ldots, 127 = 1111111$.

It may be natural to include a term γh_i which describes the growth of hosts in the r.h.s. of eq. (2). Indeed, we have studied such a system. The results obtained are qualitatively the same as those reported below, provided that γ is not too large.

3. Emergence of periodic symbiosis

To judge whether or not our system is in a symbiotic state, we compute the following average interaction between hosts and parasites:

$$\overline{a^{\kappa}} = \frac{\sum_{j,k} a^{\kappa}_{kj} p_j h_k}{\sum_j p_j \sum_k h_k}, \tag{5}$$

where κ is either P or H. Each interaction among hosts and parasites is averaged over all species. $\overline{a^{\kappa}}$ measures the ratio of symbiotic pairs in the total pairs of hosts and parasites. A positive $\overline{a^{\mathrm{H}}}$ thus indicates that a system is in a symbiotic state.

[#2] The results in section 3 are not strongly dependent on the specific choice of a^{P}_{kj} and a^{H}_{kj}. Our conclusion is thought to be valid within the conditions of (i) and (ii) and for suitable parameter values.

Since the whole parasite receives positive interaction from hosts (see eq. (3)), $\overline{a^{P}}$ is always positive. On the other hand, $\overline{a^{H}}$ is positive only when symbiotic pairs are dominant. If the population is completely concentrated on a symbiotic pair, $\overline{a^{P}} = f_P > 0$ and $\overline{a^{H}} = f_H > 0$ would follow.

In wide parameter regimes, our system ends up with a state of extinction: Harmful parasites distribute widely in gene space, and hosts are exploited by the parasites[#3]. Escape from attack by the parasites is possible by mutation of hosts, but the escape is in vain if parasites are widely distributed. All hosts are thus extinguished by the parasites, which after all starve by loosing their hosts (preys).

If a parasite mutates to others whose bit sequence has a large Hamming distance from that of a host, the mutant can become more harmful to the host. On the other hand, hosts can escape from the attack of parasites by mutating to match the gene sequence of that parasite.

Parasites often spread further through gene space than hosts do. Hosts spread to follow the spread of parasites, but begin to shrink after a certain size is reached since parasites always outgrow hosts.

An example of successful symbiotic behavior is shown in figs. 1 and 2. Let us see how a symbiotic state is generated from the initial condition, by taking a case with the initial distribution of a parasite type 85 and a host type 86. Almost immediately, all hosts of type 86 change into 85 by mutation. Thus a symbiotic pair of type 85 is generated. The distribution in genotypes has a sharp peak around the symbiotic pair (see fig. 2). That symbiosis is accomplished is clearly seen in the effective interaction, as $\overline{a^{H}} > 0$ and $\overline{a^{P}} > 0$, as is shown in fig. 1. In this symbiotic state, both the populations of hosts and parasites increase, until the state becomes unstable and dissolves. Through the spread in gene space, parasites reach the most harmful species to the host type 85. Thus the

[#3] The extinction can be removed by introducing the growth term in our model. In order to focus on the emergence of symbiosis, we have not included this term. This stresses the harm of some parasites.

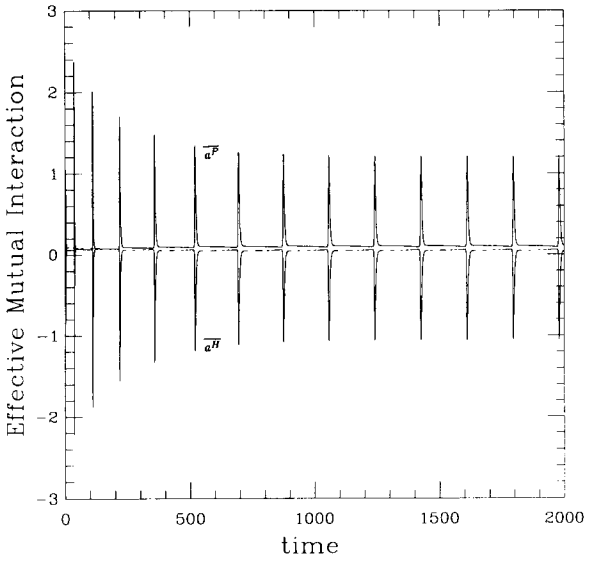

Fig. 1. Temporal evolution of order parameters $\overline{a^{H}}$ and $\overline{a^{P}}$. A symbiotic state occurs when both parameters have positive values. If the order parameter for hosts takes a negative value, it is out of the symbiotic state. A symbiotic state disintegrates periodically when the values of $\overline{a^{H}}$ and $\overline{a^{P}}$ are close together. The simulation is executed with parameter values $f_P = 0.05$, $f_H = 0.1$, $\mu_P = 0.001$ and $\mu_H = 0.002$.

parasite with the genotype 42 ($= 127 - 85$) appears. (Note that 85 = 1010101 and 42 = 0101010.) Since the parasite can exploit the host most strongly, its number increases. In order to recover the original symbiotic state, this harmful parasite must be eliminated. In the present case this elimination occurs through the decrease of host type 85 by mutation and the suppression of parasite type 42 by other parasites. A symbiotic pair of host and parasite drives down the parasite type 42, and a symbiotic state of type 85 has again been established.

In our present model, we have not found a fixed symbiotic state. After the system returns to a symbiotic state, the state lasts for a while but it again is destroyed. The present symbiotic state thus appears as a temporally periodic state. Two eras repeat periodically: a long symbiotic era (180 times steps in fig. 1) where the population increases slowly, and a shorter era (10 times steps in fig. 1) in which harmful parasites suddenly in-

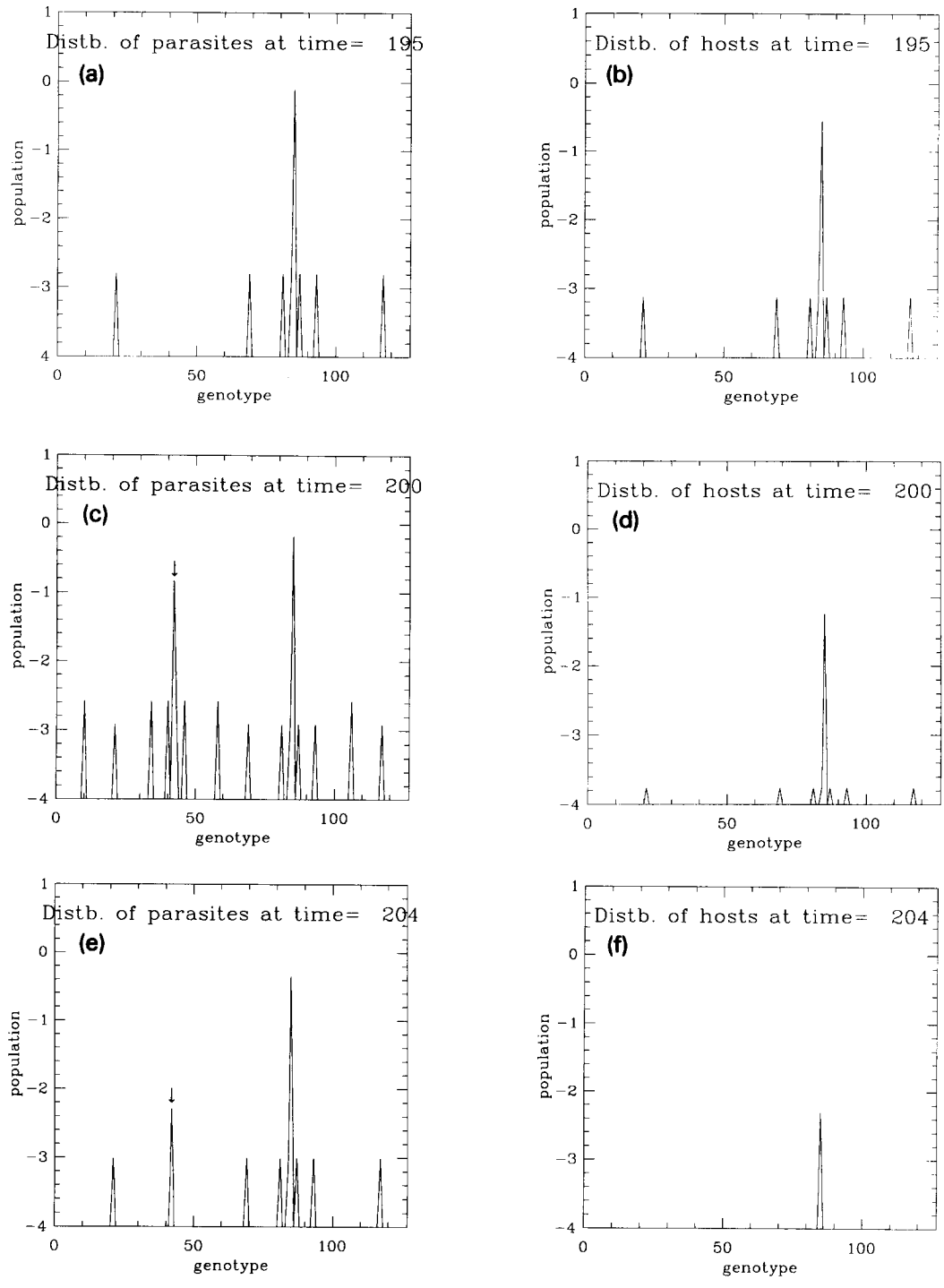

Fig. 2. Population distribution of hosts and parasites, which is simulated with the same parameter values as in fig. 1. Each horizontal line describes a genotype of species. Initially there exists one type of host with one type of parasite. A symbiotic pair of type 85 is accompanying 7 other types (a, b). At the disintegration of the symbiotic state, parasites spread in gene space but hosts begin to shrink (c, d). For symbiosis to be recovered, a host must fight with the most harmful parasite, which is indicated by a down arrow (e, f). If the host can beat the most harmful parasite, a symbiotic state is completely recovered.

crease, and then is eliminated accompanied by the decrease of populations of hosts. It should be noted that the duration of the latter era is much shorter (less than 1/10) than that of the symbiotic era. Neither a quasiperiodic nor a chaotic, but only a periodic temporal change has been observed.

Non-existence of fixed symbiotic states can be understood as follows: If our dynamics is in a symbiotic state, the population of hosts increases by the interaction term $a^{H}_{kj}p_j h_k$. As the population of hosts increases, the increase rate of harmful parasites gets larger by the interaction term $a^{P}_{kj}p_j h_k$. Since this increase rate is larger than that of other parasites and hosts, the population of harmful parasites eventually becomes large enough to destroy the symbiosis. Thus the symbiotic state cannot be temporally fixed.

The appearance of symbiosis is dependent on the initial distribution of hosts and parasites in gene space. We have studied some typical initial distributions as follows:

(a) One type of parasite versus a random or homogeneous distribution of host types.

(b) One type of host versus a random or homogeneous distribution of parasite types.

(c) Both types are randomly distributed.

(d) One type of parasite versus one type of host.

Of the above conditions, we have not found a stable symbiotic state for the conditions (b) and (c). Since various types of parasites already exist, hosts cannot escape from them, and are extinguished.

For suitable sets of parameters, a periodic symbiotic state is observed under the conditions (a) and (d). Hosts mutate so that they can form a symbiotic pair with the initially given type of parasite. Symbiosis lasts for a long time (say 200 time steps) and disintegrates, but it recovers through the punishment of harmful parasites, as has been discussed in the above. Even if the initial Hamming distance between hosts and parasites is large (say the types 85 and 42), the symbiotic pair is developed if the mutation rate of hosts is moderately large.

Next, let us discuss the condition of emergence of the symbiotic behavior in our model. By varying parameters in our model, we have found that the above dynamical symbiotic state exists only in a small parameter regime. The following conditions are found to be necessary for successful symbiosis:

(1) A mutation rate of hosts (μ_H) should be larger than that of parasites (μ_P). Too large mutation rate of hosts, however, brings the extinction again.

(2) The gain of hosts by the symbiotic pair (f_H) should be larger than that of parasites (f_P). Too large f_H again leads to the extinction.

The first restriction ($\mu_H > \mu_P$) is necessary for the escape of host from the attack of harmful parasites. The second condition ($f_H > f_P$) is understood as follows: Under the opposite condition ($f_H < f_P$), a larger growth rate of the symbiotic parasite generates more diverse parasites, which are harmful to the symbiotic host. As a result, the host is exploited by such parasites. If $f_H > f_P$, this tendency is suppressed.

The meaning of the restriction that neither f_H nor μ_H should be too large is more subtle. If these parameters are too large, the host population increases faster than that of parasites. The large population of hosts, however, leads to the growth of non-symbiotic parasites.

If the parameters do not satisfy the condition of symbiosis, a symbiotic state exists only for the initial stages and disintegrates by the spread of parasites (see figs. 3 and 4) for the extinction process. The spread of parasites in their gene space continues until it covers the whole gene space. Such a situation leads to the condition (b), and the hosts are completely exterminated by those parasites. Suppression of the spread of parasites is essential to the recovery of symbiotic behavior.

Emergence of TIT for TAT

The above mechanism of the suppression of harmful parasites is suggestive of *TIT for TAT* strategy [4] in the iterated prisoner's dilemma

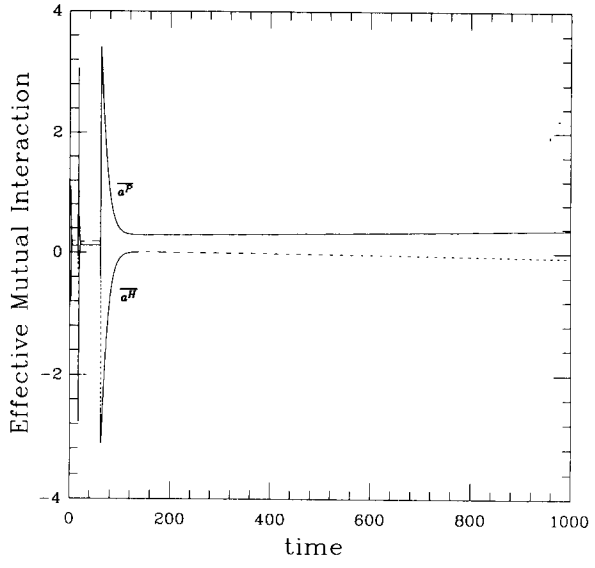

Fig. 3. A disintegration of a symbiotic state at time = 300 can be seen in the temporal evolution of order parameters. The simulation is executed with parameter values $f_P = 0.1$, $f_H = 0.2$, $\mu_P = 0.001$ and $\mu_H = 0.002$.

game. In the game, a community of programs has played a prisoner's dilemma with each other. Each player can defect or cooperate by some program. For just one iteration of game, the strategy "detect" may work, but for a long run of iterated games, it may not be a good strategy. Indeed, it is known that the robust program takes a strategy of TIT for TAT [4]. A player following the strategy of TIT for TAT trusts its component unless being defected. Once the opponent defects, it also defects.

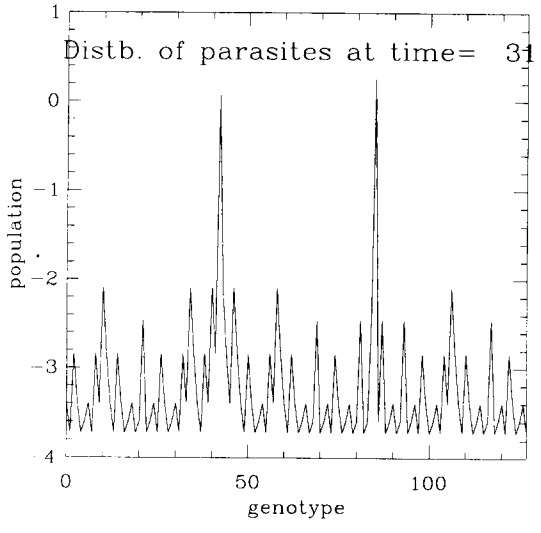

Fig. 4. If the host cannot beat the most harmful parasite, parasites spread out in gene space. After this event, all hosts are extinguished by parasites. This unsuccessful case is simulated with the same parameter values as in fig. 3.

A strategy similar to TIT for TAT is attained through a mutual supervision in a game of the evolution of norms [5].

In our model, harmful parasites (with large Hamming distance from hosts) are playing the role of "defect". If the proportion of such parasites increases, the number of parasites is eventually decreased by the decrease of the host population. Thus the existence of harmful parasites is also harmful to sustain the population of parasites, in the long run. To have sustained population size, we need a mechanism to suppress such harmful parasites. In our model, the interaction among parasites corresponds to mutual supervision. This mutual supervision inhibits the increase of the most harmful ("selfish") parasite like a type 42 in our example. Thus symbiosis is maintained by the mutual supervision, as in the case of the TIT for TAT strategy in the iterated prisoner's dilemma game.

We also note that the instant decrease of the harmful parasite is essential to the recovery of symbiosis in our model. Otherwise, the genotypes of parasites distribute widely, leading to the extinction of hosts. This instant decrease of harmful parasites is analogous to the instant punishment in TIT for TAT strategy in the iterated prisoner's dilemma game [4]. The instant "TIT" against a bad strategy (e.g. defect against any strategy) is necessary to the robustness of cooperative strategy in the game.

Of course, neither host nor parasite plays a game with a strategy in our model. Both of them are just under random mutation without any purpose. As an emergent dynamical behavior, however, harmful parasites are extinguished instantly through supervision and mutation. If we observe our results from a macroscopic level, we can say that harmful parasites are "punished" by an "invisible hand". Thus we can conclude that the

"TIT for TAT" in our model appears as the *emergent* behavior to attain the symbiotic behavior.

4. Discussion

Let us consider the significance of our results from more general points of view, and discuss possible extensions.

First, our model adaptively changes the landscape through the interaction of hosts and parasites. Walks in a fixed landscape have recently been discussed especially in a rugged landscape [6]. In the evolution of species, the environment itself is generated by all species, and there is no a priori landscape. A model with a given landscape misses an essential quality of evolution. To study the evolutionary process, we need a model with a landscape which changes temporarily, depending upon the population of all species. The present model gives a simple example of this class of dynamics.

Second, our symbiosis appears only as a dynamical state. This class of symbiosis is predicted by Margulis [2] as "loose symbiosis". Symbiosis can be formed but it may dissolve again. It is a function of time. There are several reasons for the dissolution of a symbiotic relationship, as Margulis has pointed out. She also argues that the growth rate of partners should be approximately equal in order to keep a symbiotic relationship [2].

In the present model, the approximately same positive values for $\overline{a^{\mathrm{P}}}$ and $\overline{a^{\mathrm{H}}}$ lead to approximately equal growth rates for hosts and parasites. A dissolution of the symbiotic state of the present model is caused by the inbalance of these order parameters.

The importance of host–parasite interaction for the development of polymorphism has often been stressed (see e.g. ref. [7]). Polymorphism means a coexistence of different phenotypes of the same species. Let us consider a collection of ensembles of hosts and put parasites on each ensemble. Hosts in each ensemble may evolve to different genotypes (e.g. a different symbiotic pair). Thus polymorphism of hosts is induced by the interaction with parasites. If there exists a competition among different host types, this picture may not be justifiable.

Since our model is one of the simplest within the constraints in section 2, we expect that our results are rather general. Of course, other choices of interactions may be of importance to study specific models for symbiosis. Modification of parasite–parasite interaction or the inclusion of host–host interaction may be important. We have also studied a model in which parasites suppress themselves. It seems that the inclusion of this self-suppression term destroys the symbiotic state. Other choices of host–parasite interaction terms may be worth considering.

In our model we have assumed that all parasites and all hosts interact with each other. This assumption may be artificial. It may be better to use a model with the interaction only among restricted sets of hosts and parasites. A simple example is a model on a lattice (see also ref. [8]). It is expected that this kind of restricted interaction will enhance the stability of the symbiotic state, since the host which promotes a symbiotic relation with some parasite has a small chance to be attacked by other harmful parasites. In our long-ranged coupling model, however, such symbiotic hosts may be exploited by other parasites.

Another important future problem is the use of sex [9]. As stressed by Hamilton and others, recombination (i.e. cross-over) can be more effective than mere mutation. One of the reasons for this is that mere mutation cannot memorize the effective genetic sequences against the past parasites, but recombination does. Recombination is thought to be especially useful to protect from the attack of parasites, since it makes a large uncorrelated jump.

In our problem, recombination is useful for hosts for the same reason, to escape from the attack of parasites. Recombination, however, may in fact hinder the symbiotic relationship, since recombination may instantly create non-symbiotic hosts and parasites.

A creation of new species by recombination strongly depends on the population distribution.

Since recombination merely crosses over the already existing types, a symbiotic pair generates the symbiotic genotypes through recombination.

Thus the above drawback may be removed. It is an open question, however, if our system attains such concentrated distribution under the existence of recombination.

If we regard a parasite in our model as a virus, we may ask why the mutation rate of hosts must be higher than that of viruses, to attain symbiosis. This would seem counter-intuitive unless one thinks of our "hosts" as antibodies in the host's immune system. The mutation rate of the immune system is known to be very fast to generate various types of antibodies, and our condition of "symbiosis" is satisfied. Through the mechanism we have discussed in the present paper, coexistence of less harmful viruses with the immune system is attained in the course of evolution. The loss of harm from viruses through evolution is frequently seen in nature.

Lastly, we have to point out that there is another form of symbiosis which we have not discussed in the present paper. This is the process which involves the sharing of information (gene sequence) and the creation of new species through it. For example, Margulis [2] has put forward a theory on the origin of eukaryotic cell through such joining of different species. For such a class of symbiosis, we need a model which includes the process of merging gene sequences and creating new species. This class of models will be discussed in the future[#4] [11].

In the present paper, we have discussed the emergence of symbiotic behavior. Without any supervising, our system attains a symbiotic relationship by suppressing harmful parasites. Emergence of such relationship seems relevant to cooperativity in computer community [12]. Since our model gives a simple example of symbiosis, it may be useful to extend our results to computer community, and study the emergent symbiotic computation.

[#4] A joint process of this type is also discussed in the "Urobors model" for the immune system [10].

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References

[1] M. Peschel and W. Mende, The Predator-Prey Model (Springer, Berlin, 1986).
[2] L. Margulis, Symbiosis in Cell Evolution (Freeman, San Francisco, 1981).
[3] K.W. Jeon, Science 202 (1978) 635.
[4] R. Axelrod, The Evolution of Cooperation (Basic Books, New York, 1984).
[5] R. Axelrod, Am. Political Sci. Rev. (1987) 44.
[6] S. Kauffman and S. Levin, J. Theor. Biol. 128 (1987) 11.
[7] R.M. Anderson and R.M. May, Parasitology 85 (1983) 411–426;
H. Ishikawa, Symbiosis and Evolution (Baifukan, Tokyo, 1988) (in Japanese).
[8] D. Hillis, Physica D 42 (1990) 228–234, these Proceedings.
[9] W.D. Hamilton, R. Axelrod and R. Tanase, Sexual reproduction as an adaptation to resist parasites, preprint; W.D. Hamilton, presented at the Ninth Annual CNLS Conference on Emergent Computation, Los Alamos, 22–26 May 1989.
[10] T. Ikegami, Ph.D. Thesis (1989);
T. Ikegami and K. Kaneko, to be published.
[11] T. Ikegami and K. Kaneko, to be published.
[12] B.A. Huberman, Physica D 42 (1990) 38–47, these Proceedings.