Mathematical study of cooperative behavior in social amoeba and plant-mycorrhizal system

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Mathematical study of cooperative behavior in social amoeba and plant-mycorrhizal system

by

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Contents

Preface

In nature, all organisms interact with other individuals of the same species and different species. In the gut of human, for example, there are a tremendously many bacteria. Gut flora are known to contribute to human health (Lupp et al. 2012). In this thesis, I report theoretical studies on the altruism of social amoeba and the mutualism between plant and fungus.

Altruism is cooperative behavior that an individual helps other individuals of the same species by paying cost. Eusociality of social insects, such as ants, bees and termites, is one of the most spectacular examples of altruism. In the insect eusocialty, only a small fraction of individuals reproduce and others help the reproductive individuals as workers or soldiers. Their altruism is very well explained by the theory of kin selection (Hamilton, 1964). In chapter 1, I focus on the altruism of social amoeba as represented by *Dictyostelium discoideum*. Social amoeba lives like a single cell amoeba when there are many foods such as bacteria. However, when bacteria are depleted, the number of cells aggregate and make a fruiting body. Fruiting body consists of spores and a stalk. A stalk supports spores for their dispersal. In the fluiting body formation of *D. discoideum,* some cells of an aggregate are differentiated to form stalk and die after dispersal of spores. Therefore, making a fruiting body is an example of altruism. However, this altruistic behavior is threatened by a mutant strain called cheater. A cheater makes less stalk when it is mixed with the wild type strain. Although some cheaters do not have the ability to form a stalk, other cheaters called "facultative

cheater" can make a stalk normally when they form a fruiting body by themselves (Strassmann & Queller, 2011). Cell differentiation is controlled by signaling chemicals, such as DIF-1(Kay et al., 1993) and c-di-GMP(Chen & Schap, 2012). In chapter 1, I construct a mathematical model of cell differentiation mediated by DIF-1 and discuss the evolution of cells with respect to their ability to secrete DIF-1 and to their sensitivity to DIF-1 produced by other cells.

Mutualism is cooperative behavior between different species. A familiar example is a relationship of a flowering plant and their insect pollinators. Many flowering plants make nectar, and give it to the insect that visits them. Insect can move a broad area and carry pollens of the plant. Mutualism also occurs on a smaller scale. For example, a unicellular organism *Paramecium bursaria* has hundreds of chlorella, *Chlorella variabilis*, within the cell. *P. bursaria* supplies CO2 and NH3 to *C.variabilis.* (Brown & Nielsen, 1974). On the other hand, *P. bursaria* can endure starvation because of sugar supplied by *C.variabilis.* An advantage of mutualism is that an organism can use the ability of the partner. In the case of *P. bursaria* and *C. variabilis*, *P. bursaria* acquires ability to photosynthesize by symbiosis with *C. variabilis*. Mutualism between plant and myccorhizal fungus is another example. Most terrestrial plants have fungi in its roots. Such fungi are called mycorrhizal fungi. Mycorrhizal fungi can increase tolerance to abiotic stress such as drought and heavy metals (Sikes et al. 2010). In chapter 2 of this thesis, I focus on the phosphorus uptake of fungus, or mycorrhizal fungi (Sikes et al. 2010). Plants can obtain carbon in the atmosphere by photosynthesis. However, they have to get nutrients from soil. Root of plant may be insufficient to

sequester nutrients in the soil. Since hypha of mycorrhizal fungi is much thinner than roots, fungi can collect soil nutrients more efficiently than the plant. In the mutualism between a plant and mycorrhizal fungus, the plant gives carbohydrate to the fungus and the fungus gives soil nutrition to the plant. In chapter 2, I consider the situation that both plant and fungus grow simultaneously, and discuss the optimal resource allocation of both parties to their partners.

These two chapters address the optimal behavior of organisms. Especially, in chapter 1, I showed that complex behavior of cheater in social amoeba can be explained by simple mathematical models for the evolution of cells' ability of producing and responding to the signaling chemical. Knowledge of molecular and cell biologies have been accumulated rapidly in recent years. Now it is a time for mathematical models to play an important role to bridges molecular and cell biologies and the behavior of the whole organisms and the evolution.

The followings are more detailed explanations for the contents of two chapters:

Chapter 1:

The social amoeba (or cellular slime mold) is a model system for cell cooperation. When food is depleted in the environment, cells aggregate together. Some of these cells become stalks, raising spores to aid in their dispersal. Differentiation-inducing factor-1 (DIF-1) is a signaling chemical produced by prespore cells and decomposed by prestalk cells. It affects the rate of switching between prestalk and prespore cells, thereby

achieving a stable stalk/spore ratio. In this study I analyzed the evolution of the stalk/spore ratio. Strains may differ in the production and decomposition rates of the signaling chemical, and in the sensitivity of cells to switch in response to the signaling chemical exposure. When two strains with the same stalk/spore ratio within their own fruiting body are combined into a single fruiting body, one strain may develop into prespores to a greater degree than the other. Direct evolutionary simulations and quantitative genetic dynamics demonstrate that if a fruiting body is always formed by a single strain, the cells evolve to produce less signaling chemical and become more sensitive to the signaling chemical due to the cost of producing the chemical. In contrast, if a fruiting body is formed by multiple strains, the cells evolve to become less sensitive to the signaling chemical and produce more signaling chemical in order to reduce the risk of being exploited. In contrast, the stalk-spore ratio is less likely to be affected by small cheating risk.

Chapter 2:

Plant interacts with mycorrhizal fungus in the roots. Plant gives carbohydrate produced by photosynthesis to the fungus. On the other hand, fungus allocates soil nutrition such as phosphorus to the plant. Therefore, relationship between plant and fungus is mutualism. In this study, I analyzed optimal allocation of carbon by a plant to its mycorrhizal fungus, and the optimal allocation of phosphorus by the mycorrhizal fungus to the plant. I considered the resource allocation by both players that achieve the fastest growth of the whole system when it grows exponentially. I assume the resource

acquisition rates of Cobb-Douglas type. I also analyze the dynamic optimization condition (singular subarc) for each player to maximize its own fitness given the partner's growth schedule. As a result, each player should allocate more to the partner when the resource provided by the partner is more important to its own resource acquisition. The ESS resource allocation by each player is equivalent to the one achieving the cooperative optimum.

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Chapter 1

Evolution of stalk/spore ratio in a social amoeba: cell-to-cell interaction via a signaling chemical shaped by cheating risk.

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1. Introduction

The cellular slime mold, or social amoeba, exists as a unicellular form that divides and multiplies rapidly when the microhabitat includes abundant bacteria. However, when food (i.e., bacteria) is depleted, cells aggregate to form a fruiting body (Bonner 1967) within which cells differentiate into spores and stalks. Some of these spores are able to find a new microhabitat with plenty of food and resume a unicellular phase with fast population growth. In contrast, stalk cells lift spores to aid in their dispersal and then die. Therefore, becoming a stalk cell is an altruistic behavior (Strassmann et al. 2000). In the field, social amoebae undergo a repeating cycle of proliferation and dispersal phases.

This system is an example of altruism and cell cooperation. Although a fruiting body is usually made of clones containing a single strain, in the laboratory social amoebae can make a fruiting body out of a mixture of two strains (Ostrowski et al. 2008; Flowers et al. 2010). Some strains are called cheaters because they contribute less to stalk development and more to spore development than the wild type when they are mixed (Strassmann et al. 2000). These cheaters have a reproductive advantage over the wild type due to the higher proportion of spores, and will spread within the population (Gilbert et al. 2007). Some cheaters can make a fruiting body in a single-strain aggregation that looks similar to that of the wild type (Santorelli et al. 2008; Strassman and Queller 2011). Several mechanisms to prevent cheaters from spreading in a population have been identified. Cells may make a fruiting body preferentially with the same strain of *Dictyostelium discoideum* (Queller et al. 2003; Ostrowski et al. 2008;

Flowers et al. 2010), as well as with a related species, *D. purpureum* (Mehdiabadi et al. 2006), and the bacteria *Myxococcus xanthus* (Fiegna and Velicer 2005). Altruistic behavior, such as making a fruiting body, evolves easily if an organism repeatedly interacts with its identical clone or its close relatives (Hamilton, 1964). In addition, Foster et al. (2004) showed that the gene *dimA* is not only necessary for making a fruiting body, but is also needed to become spore in mixed fruiting body; therefore, this pleiotropy reduces the risk of cheating in *D. discoideum*.

This system has also been studied theoretically as a case study for the maintenance of altruism. Many theoretical models that have evaluated the evolution of cooperation simply assumed cells to have different degrees of cooperation, or tendencies to become a stalk cell, without specifying the underlying mechanism (Matsuda and Harada 1990; Brannstrom and Dieckmann 2005; Dionisio and Gordo 2007). However, it has been known for many years that the developmental fate of a cell in a social amoeba is not fixed, but is decided based on interactions with other cells in the aggregation. A cell aggregation including both prespore cells and prestalk cells may be divided experimentally into two: one consisting mostly of prespore cells and the second consisting mostly of prestalk cells. In both of these groups, the relative abundance of the two cell types changes, resulting in an intermediate ratio of the two cell types (Kay et al. 1993). Thus, a proper ratio of stalk cells and spore cells is recovered spontaneously. Such complex behaviors in developmental stage can be explained by simple molecular mechanism.

In the social amoeba *Dictyostelium discoideum,* the differentiation of cells in

an aggregation into stalk versus spore cells is controlled by the chemical signals. Recently Chen and Schaap (2012) found cyclic di-(3': 5')-guanosine (c-di-GMP) monophosphate triggers stalk cell differentiation. Although c-di-GMP will become major factor of cell differentiation, the study is not sufficient to make model. Therefore, in this chapter, I focus on differentiation inducing factor-1 (DIF-1) which has been studied very well in *D. discoideum* (Town et al. 1976). It is produced by prespore cells (Kay and Thompson 2001) and decomposed by prestalk cells (Kay et al. 1993). In addition, it induces the differentiation of prespore cells into prestalk cells and suppresses differentiation of prestalk cells into prespore cells (Town et al. 1976) . DIF-1 is an important factor that influences the altruistic behavior of *D. discoideum*. Negative feedback from cell determination mediated by DIF-1 results in a stable ratio of prestalk/prespore cells that will later differentiate into stalks and spores.

Parkinson et al. (2011) proposed a mathematical model in which the fraction of cells constituting the stalk of the fruiting body is a product of two factors, both being normalized between 0 and 1. The first factor is cell sensitivity (called responsiveness) to a stalk-inducing factor (StIF) such as DIF-1. The second factor is the relative magnitude of the production of StIF, the level of which is assumed as an arithmetic average production rate of cells. This model predicted that if a mutant with a slower production rate of StIF and a sensitive response to StIF is mixed with wild type, the mutant cells become stalks more frequently than the wild type cells. Subsequently they produced a mutant with this property (i.e., a mutant of the *lirA* gene that produced less StIF and had a higher response to StIF than the wild type). This result showed that one gene could

control both StIF production and responsiveness. However, their model does not consider the evolution of sensitivity nor production.

In this chapter, I model the mechanism of cell differentiation in *D. discoideum* controlled by a signaling chemical within cell aggregation, and discuss how the number of different cell types may be adjusted. When two strains are mixed in a fruiting body, one strain may predominantly become prespore cells, while the other contributes more to prestalk cells, despite the fruiting body being formed by cells of single strains with the same stalk/spore ratio. I then consider the evolution of parameters such as the rates of production and decomposition of the signaling chemical, the rates of cell determination and switching, and cell sensitivity to the signaling chemical concentration. I show that if two strains are mixed in a fruiting body, the social amoeba evolves to produce more signaling chemical than if the strains are never mixed. When each fruiting body consists of cells from the same strain, the ratio of prestalk cells to prespore cells quickly reaches the optimal value. Evolution slows down due to the cost of producing the chemical signal, the rate of chemical production decreases and the cell sensitivity to the chemical increases. In contrast, if multiple strains are mixed in a fruiting body, a higher production rate of chemicals and an intermediate level of sensitivity evolve in order to prevent the risk of being exploited by a cheater.

2. Model

I begin with the control of the stalk/spore cell ratio within an aggregation consisting of a single strain by the signaling chemical. Within the aggregation, cells change state

between prespore cells and prestalk cells. After the proportion of cells in these states reaches equilibrium, prestalk cells and prespore cells develop into spores and stalks, respectively.

Here, I consider the dynamics of the cell state change illustrated in Figure 1. I denote the number of prespore cells by P and that of prestalk cells by T. Cells change their states at the rates controlled by the amount of the signaling chemical, which is denoted by C.

$$\frac{dT}{dt} = f(C)P - g(C)T , \tag{1a}$$

$$\frac{dP}{dt} = -\frac{dT}{dt} . \tag{1b}$$

Here the total number of cells $N = P + T$ is a constant. In Eq. (1a), $f(C)$ is the rate of switching from prespore cells to prestalk cells. $f(C)$ is an increasing function of C, implying that signaling chemical promotes conversion from prespore cells to prestalk cells. $g(C)$ is the rate of switching from prestalk cells to prespore cells. Because the signaling chemical suppresses this conversion, $g(C)$ is a decreasing function of C. I represent the rates of switching between prespore cells and prestalk cells as follows:

$$f(C) = f_0 C , \tag{1c}$$

$$g(C) = \frac{g_0}{C} , \tag{1d}$$

where f_0 and g_0 are constants indicating the rate of conversion.

The concentration of the signaling chemical follows:

$$\frac{dC}{dt} = aP - bCT . \tag{2}$$

The first term on the right hand side indicates that the signaling chemical is secreted by

prespore cells at a rate of a per cell. In contrast, prestalk cells have an enzyme called DIF dechlorinase, which inactivates the signaling chemical. Because DIF dechlorinase is known to be localized within prestalk cells, I assume that the rate of inactivation of the signaling chemical by prestalk cells follows the law of mass action: i.e., the rate increases in proportion to the substrate concentration. In Eq. (2), the rate of decomposition per cell is bC, proportional to the amount of signaling chemical, where b is a proportionality coefficient of the inactivation of signaling chemical by a prestalk cell.

2.1. *Ratio of prestalk cells to prespore cells*

Figure 2a illustrates the time course of the cell fractions in each state, which is given by Eq. (1). An intermediate ratio of T to P that is globally stable exists. Irrespective of the initial value, the ratio of prestalk cells to prespore cells converges to that value. I can calculate that ratio at equilibrium as follows:

$$\frac{\hat{T}}{\hat{P}} = \left(\frac{f_0}{g_0}\right)^{1/3} \left(\frac{a}{b}\right)^{2/3}. \qquad (3)$$

Symbols with a hat indicate the values at the equilibrium of Eq. (1). The proportion of prespore cells to prestalk cells depends on f_0/g_0, which is the relative sensitivity to the signaling chemical between two reactions. It also depends on a/b, the ratio of the secretory capacity of signaling chemical and the inactivating capacity of signaling chemical.

Note that the equilibrium ratio of prespore cells and prestalk cells given by Eq.

(3) is independent of the total number of cells *N*. This explains an empirical observation that, after the cell mass divides, the cells in each new cell mass can again differentiate and achieve a stable proportion of prespore and prestalk cell types (Kay and Thompson 1999), and that the final proportion is independent of the total number of cells N (Kay et al. 1993; Loomis 1996). Eq. (3) also shows that two strains may have exactly the same stalk/spore ratio if the combination of parameters given by Eq. (3) is the same between strains, even if their individual parameters differ (see Fig. 2a).

2.2. *Mixture of strains*

Next, I consider the case in which multiple strains are mixed in the same fruiting body. I assume that there are two strains indicated by suffix i (=1, 2), and that both strains secrete and inactivate the same chemical, signaling chemical. Further, cells of the two strains switch between the two states according to the concentration of signaling chemical. T_i and P_i are the numbers of prestalk cells and prespore cells of strain i, respectively. The dynamics are as follows:

$$\frac{dT_i}{dt} = f_i(C)P_i - g_i(C)T_i, \qquad i=1, 2. \tag{4a}$$

$$\frac{dP_i}{dt} = -\frac{dT_i}{dt}, \qquad i=1, 2. \tag{4b}$$

I assume that growth rate is the same between strains. Following this assumption, each strain shares 50% of the total cell number *N*, and I have $T_i + P_i = N/2$ for *i*=1, and 2. $f_i(C) = f_{0i}C$ is the rate of switching from a prespore cell to a prestalk cell of strain i, and $g_i(C) = g_{0i}/C$ is the rate of switching from a prestalk cell to a prespore cell of

strain i. Cells of different strains may switch their states, but their sensitivity to the signaling chemical may differ. a_i and b_i are the production rate and inactivation rate of strain i, respectively. The dynamics of the chemical signal is given by:

$$\frac{dC}{dt} = \sum_{i=1}^{2} \left(a_i P_i - b_i C T_i \right). \tag{4c}$$

Figure 2a illustrates a case in which each fruiting body consists of a single strain. Here, two strains form a fruiting body of the same ratio of T to P when each fruiting body consists of cells from the same strain. However, when the strains are mixed in a fruiting body with 1:1 ratio, one strain develops more prespore cells and the other strain develops more prestalk cells (Fig. 2b). These values are calculated by Eqs. (4). The strain that contributes less to stalk development may be called a cheater (Strassmann et al., 2000).

For the equilibrium condition of Eq. (4a), I have the following equation for the stalk/spore ratio:

$$\frac{\hat{T}_i}{\hat{P}_i} = \frac{f_{0i}}{g_{0i}} \hat{C}^2, \qquad i=1 \text{ and } 2, \tag{5a}$$

$$\hat{C} = \frac{\sum_j a_j \hat{P}_j}{\sum_j b_j \hat{T}_j}. \tag{5b}$$

Eq. (5a) indicates that the stalk/spore ratio at equilibrium is proportional to the ratio f_{0i}/g_{0i}. Hence, if there are two aggregated strains that differ in this ratio, the one with the smaller ratio can be regarded as a cheater because it contributes less to stalk formation. On the other hand, the cheater stain may not have a smaller stalk/spore ratio when it forms a fruiting body consisting of cells from the same strain. This ratio is

given by Eq. (3), which depends not only on f_0/g_0, but also on a/b. Thus, this simple model explains opponent-dependent cheating (Strassmann et al., 2000).

3. Evolution

When food is abundant, cells of *D. discoideum* multiply by cell division; however, when they face a shortage of food, they start forming fruiting bodies. Only spore cells can disperse to new microhabitats with food, but the dispersal success of spore cells depends on the number of stalk cells that lift up the spores. This cycle of unicellular life with asexual proliferation and fruiting body formation with dispersal occurs repeatedly. If the whole population is composed of multiple strains that differ in the number of surviving spore cells, the strain with the highest expected number of surviving spore cells increases in proportion, and eventually dominates the population after many cycles of proliferation and dispersal phases. In this section, I model this process of natural selection and discuss the evolutionary outcome.

(a) *No mixing of strains in a fruiting body*

I begin with the case in which each fruiting body consists of a single strain. I consider a population composed of two strains: ξ and $1-\xi$ are the fractions of strain 1 and strain 2 in the beginning of a cycle, respectively. Suppose that each fruiting body includes N cells. These cells differentiate into stalk cells and spore cells, following the procedure described in the last section. Stalk cells and spore cells are determined by the prestalk cells and prespore cells at the equilibrium of the dynamics Eqs. (1) and (2).

These values are calculated by Eq. (3) with $\hat{P} = N - \hat{T}$.

The success of dispersal and settlement to a new microhabitat with a sufficient amount of food is an increasing function of the number of stalk cells and is denoted by $S\left(\hat{T}\right)$. Specifically, I assume the following dispersal success function:

$$S\left(\hat{T}\right) = \frac{S_0 \hat{T}^l}{\alpha^l + \hat{T}^l}. \qquad (6)$$

$\hat{T}$ is the total number of prestalk cells in the equilibrium of Eqs. (1) and (2). Prestalk cells at equilibrium will later differentiate into stalk cells. If the number of prestalk cells is too small, spores may slip from the top of a fruiting body (Saito et al. 2008). Therefore, $S\left(\hat{T}\right)$ is very small if the number of stalk cells is small. Dispersal success increases with the number of stalk cells, but the rate of increase becomes lower for a large stalk cell number. Eq. (6) saturates for a very large *T*. The parameter α expresses the number of prestalk cells when $S\left(\hat{T}\right) = 0.5$. Parameter *l* specifies the way the success rate increases with *T*. If *l* is 1, the success rate increases linearly for small *T*. If *l* is 2, the success rate increases with *T* as a quadratic function for small *T*, and the whole curve is of an S-shape. If *l* is large, the success rate is close to a step function -- it is very small for *T* less than α, and close to the maximum if *T* is larger than α. The same function is used for describing biochemical reaction rate, where *l* is called Hill coefficient. Although I show the results only for the case *l*=2 in figures, the results did not change much if *l*=1.

In addition, I consider the cost of signaling chemical secretion, which is

expressed as a factor e^{-ka_i}. k is the magnitude of the cost to produce each unit amount of signaling chemical. Although signaling chemical itself is a small chemical, many proteins are involved in synthesizing it (Kay et al. 1999; Saito et al. 2008). Here, I simply assume that there exists some cost for producing the signaling chemical. The number of surviving spores of a fruiting body is a product of the number of prespore cells at equilibrium ($\hat{P}$), the dispersal success function ($S\left(\hat{T}\right)$), and a factor indicating the signaling chemical secretion cost (e^{-ka_i}). I assume that the cost of changing sensitivity is negligible, because there are many ways to change sensitivity such as changing conformation of receptor that is not necessarily accompanied by a significant cost. For a fruiting body composed only of strain i cells, the number of surviving spores of strain i is:

$$W_i = \hat{P}_i S\left(\hat{T}_i\right) e^{-ka_i}, \qquad \text{for } i=1, 2. \tag{7}$$

W_i is proportional to the fitness of strain i. The fraction of strain 1 at the beginning of the next cycle can be expressed in terms of the fraction of strain 1 in the current cycle.

$$\xi^{next} = \frac{W_1 \xi}{W_1 \xi + W_2 (1-\xi)}. \tag{8}$$

If this process of natural selection repeats over many generations, one strain may outcompete the other. If mutation introduces a new genotype into the population, it may go extinct or replace the old type. Mutation and replacement occur many times over the course of evolution, and the traits of the organism change slowly.

Figure 3a illustrates the evolutionary changes in the production rate (a) and the sensitivity to the chemical (f_0) caused by recurrent invasion of mutants and

subsequent replacement. Because the mutants are assumed to be close to the parent in phenotype, evolution appears as a continuous change in the traits. The horizontal axis shows the production rate of signaling chemical a, and the vertical axis shows the sensitivity of cells to the signaling chemical. The traits quickly converge onto a curve in which the optimal ratio of stalk cells to spore cells is realized (see below). After the convergence to this curve, changes along the curve occur slowly, where the production rate a decreases and the sensitivity f_0 increases.

If mutants are close to the phenotype of the parent, then the population should show continual and smooth changes in phenotype, which can be described in terms of multivariate quantitative genetics (Lande 1976; Barton and Turelli 1991; Iwasa et al. 1991) or of adaptive dynamics (Mets et al. 1992; Dieckmann and Law 1996; Geritz et al. 1998).

In Appendix A, I derive the evolutionary dynamics of parameters, such as the rate of signaling chemical production a and the sensitivity of the switching rate to the chemical f_0, with other parameters (b and g_0) fixed. I first calculate the fitness of a mutant in the population occupied by the resident and then derive the selection gradient from the differential fitness. The following equations describe quantitative traits (Iwasa et al. 1991):

$$\begin{pmatrix} \Delta\bar{a} \\ \Delta\bar{f}_0 \end{pmatrix} = \begin{pmatrix} G_a & B \\ B & G_{f_0} \end{pmatrix} \begin{pmatrix} \beta_a \\ \beta_{f_0} \end{pmatrix}, \tag{9a}$$

where the left hand side is a vector of a single generation change in the two traits. The matrix on the right hand side is the additive genetic variance-covariance matrix, where

G_a and G_{f_0} are additive genetic variances of a and f_0, respectively, and B is the additive genetic covariance between these two traits. The right-most vector is for the selection gradients with the following elements:

$$\beta_a = \left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)\frac{\partial T}{\partial a} - k, \tag{9b}$$

$$\beta_{f_0} = \left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)\frac{\partial T}{\partial f_0}. \tag{9c}$$

The factor common to both quantities is $\left(\frac{dS}{dT}\right)/S - 1/(N-T) = \frac{d}{dT}\ln\left(S(T)\bullet(N-T)\right)$. See appendix A for the derivation of Eq. (9).

If I neglect k, the dynamics Eq. (9) indicates a monotonic increase in $(N-T)\bullet S(T)$ by the evolutionary changes in the traits. The fast dynamics would then show quick convergence to the combination of parameters that achieves the optimal stalk size maximizing the number of surviving cells, $\max_{T+P=N}\{P\bullet S(T)\}$. On a (a,f)-plane, the points that achieve the optimal T appear as a curve $f_0 a^2 = \text{constant}$. There are numerous ways to realize the optimum fraction of prestalk cells, and all the points on this curve are equilibria of the fast dynamics with k neglected.

After convergence to this curve, the system moves slowly along it, due to the cost of producing signaling chemical, $k>0$. The system moves in the direction of reducing the cost of producing signaling chemical; namely a decreases. The analysis in Appendix A shows that, if strains do not mix, cells evolve to produce less signaling chemical and to be more sensitive to the chemical (Fig. 3a). In this way, cells can reduce the signaling chemical secretion cost and maintain the optimal proportion of prestalk cells and prespore cells.

(b) *When a fruiting body consists of multiple strains*

I next consider the case in which some fruiting bodies are a mixture of two strains. Specifically, I consider the following scenario. In the beginning of the feeding-proliferation phase, a single cell may start to proliferate rapidly. When the cell number reaches N, a shortage of food in the microhabitat is experienced, triggering the formation of an aggregation. The cells then form a fruiting body by the previously described mechanism. In this case, each fruiting body is composed of cells of a single strain.

In contrast, the feeding-proliferation phase may start from two initial cells that divide and increase in number at equal rate. When the total number of cells reaches N, the food in the local microhabitat is depleted. Half of N cells originated from a single cell and the other half originated from the other cell. In this case, a single fruiting body might be a mixture of two strains forming a single fruiting body if the initial two cells are of different strains.

Let m be the fraction of fruiting bodies consisting of cells originating from the two initial cells, and $1-m$ be the fraction of fruiting bodies consisting of cells originating from a single cell. Note that all of the fruiting bodies include the same number of cells N. Let ξ be the fraction of cells of strain 1 at the beginning of the feeding-proliferation phase of a cycle.

In fruiting bodies consisting of cells from a single initial spore, the fruiting body consists of strain 1 only, or of strain 2 only, with fractions of ξ and $1-\xi$,

respectively. The number of successfully dispersed spores from a fruiting body of strain *i* cells is given by Eq. (7).

In contrast, among fruiting bodies each starting from two initial spores the body consists of cells from strain 1 only, or strain 2 only, or an equal mixture of strain 1 and strain 2, the frequencies of which are ξ^2, $(1-\xi)^2$ and $2\xi(1-\xi)$, respectively. The number of successfully dispersed spores is also given by Eq. (7) if the original two cells are of the same strain. However, if a fruiting body is made of two different strains, the number of surviving spores of strain 1 and strain 2 are:

$$W_1^{mix} = \hat{P}_1^* \bullet S\left(\hat{T}_1^* + \hat{T}_2^*\right)e^{-k a_1}, \tag{10a}$$

$$W_2^{mix} = \hat{P}_2^* \bullet S\left(\hat{T}_1^* + \hat{T}_2^*\right)e^{-k a_2}, \tag{10b}$$

respectively. Symbols with an asterisk are for the quantities in a fruiting body with two different strains, which are calculated from Eq. (5) and $T_1^* + P_1^* = T_2^* + P_2^* = N/2$. The survivorship of spores depends on the sum of the number of prestalk cells, $\hat{T}_1^* + \hat{T}_2^*$. Note that if cells of two strains behave exactly the same way, $W_1^{mix} = W_2^{mix}$ being equal to half of $W_1 = W_2$ holds, because the total number of prespore and prestalk cells combined from each strain is equal to $N/2$.

Noting that 1-*m* and *m* are the fractions of fruiting bodies starting from one cell and two cells, respectively, the fraction of fruiting bodies consisting only of strain 1 is $(1-m)\xi + m\xi^2$. In a similar manner, the fraction of fruiting body consisting only of strain 2 is $(1-m)(1-\xi) + m(1-\xi)^2$. The fraction of fruiting body with a 50% mixture of the two strains is $m \bullet 2\xi(1-\xi)$. The number of cells of strain 1 and of strain 2 are:

$$R_1 = \left\{(1-m)\xi + m\xi^2\right\}W_1 + \left\{2m\xi(1-\xi)\right\}W_1^{mix}, \tag{11a}$$

$$R_2 = \left\{(1-m)(1-\xi) + m(1-\xi)^2\right\}W_2 + \left\{2m\xi(1-\xi)\right\}W_2^{mix}. \tag{11b}$$

Their relative fraction is equal to the fraction of strain 1 at the beginning of the next cycle.

$$\xi^{next} = \frac{R_1}{R_1 + R_2}. \tag{11c}$$

Repeating these dynamics, I have an equation describing whether a mutant strain can invade and replace the resident strain.

If mutations occur recurrently and if the mutants are close to the parent in phenotype, the evolutionary trajectory of traits can be modeled; Figure 3 illustrates examples. Unlike in the case without mixing shown in Figure 3a, now the evolutionary equilibrium has a positive rate of chemical production $\bar{a}$ and an intermediate level of sensitivity to the chemical $\bar{f}_0$ (Fig. 3a). Figure 3b shows the evolutionary trajectories when the degree of mixing *m* is larger than in Figure 3b. Evolution would produce the equilibrium state with a faster production rate and lower sensitivity.

Figure 4a illustrates that the equilibrium values of the production rate of signaling chemical a decreases and the sensitivity f_0 increases as the magnitude of mixing of different strains in a fruiting body *m* increases. However, the fraction of prestalk cells decreases very little even if *m* increases (Fig. 4b).

In Appendix B, I analyze the selection gradient of this system by calculating the differential fitness between a mutant and a resident strain when the two strains have similar parameters: f_0, g_0, a, and b. Quick convergence of the system to the optimal

fraction of stalk cells occurs first, resulting in a point on the curve $f_0 a^2 = const$. This is followed by two additional terms: the cost of producing signaling chemical (k) appears in the dynamics of $\bar{a}$, which tends to move the system toward a lower production rate a and a higher sensitivity f_0. In contrast, a term in the dynamics of sensitivity f_0 has a negative sign, which results in a system with lower sensitivity to signaling chemical (small f_0) and a higher production rate (larger a). An opportunity to be exploited by other strains drives the evolution of lower sensitivity. As a consequence of balance between these two forces, the final equilibrium is determined where both the sensitivity to the chemical and the production rate of the chemical are of an intermediate magnitude. The term to reduce the sensitivity f_0 is proportional to m, the fraction of fruiting bodies that are a mixture of the offspring of two cells. As m increases, f_0 becomes smaller and a becomes larger. This is clearly shown in Figures 3 and 4.

4. Discussion

In this chapter I studied the evolution of the stalk/spore ratio in the social amoeba, or cellular slime mold, which is a problem of altruism or cooperation because a cell contributing to the stalk is a self-sacrificing action to help other cells to disperse. Many previous theoretical studies of this system assumed that each cell had a fixed probability to commit an altruistic act (Matsuda and Harada 1990; Brannstrom and Dieckmann 2005; Dionisio and Gordo 2007), neglecting the mechanism for cell-cell interactions via the diffusing chemical.

I first modeled the production and decomposition of signaling chemical by

prespore cells and by prestalk cells, respectively, and the switching rate of cell states depending on signaling chemical concentration. This can be used to explain a cheating behavior observed in *Dictyostelium discoideum* (Strassmann and Queller 2011) wherein a mutant cell with a higher f_0/g_0 ratio but a lower a/b ratio than the wild type can have exactly the same stalk/spore ratio in a single-strain fruiting body. However, when the mutant cells are mixed with the wild type cells in a single fruiting body, the mutant cells can predominantly become prespore cells, contributing little to stalk formation. This cheating behavior can be understood in terms of the cell's lower sensitivity to the signaling chemical and faster production of the signaling chemical.

I then discussed the evolutionary change of each rate parameter, including the production rate and decomposition rate of signaling chemical, and cell sensitivity to signaling chemical for the switching rate in two different directions (i.e., from a prespore cell to a prestalk cell, and from a prestalk cell to a prespore cell). By direct computer simulations of the recurrent invasion of mutants that are close to the parent type, subsequent replacement would cause an evolutionary trajectory as illustrated in Figure 3. The traits first converge quickly to a curve, followed by a slow change along the curve. To understand this evolutionary trajectory, I developed a quantitative genetic formalism, which explained that if each fruiting body is formed from cells of a single strain, then the evolution of parameters should result in approximately the maximum number of surviving spores $\max_T S(T) \bullet (N - T)$. After realizing the optimal stalk size by this quick evolution, the cells then evolve slowly to produce less chemical and be more sensitive to the chemical, due to the cost of producing signaling chemical.

However, in the field, different strains of social amoeba exist in close proximity (Strassmann et al. 2000), suggesting some opportunity for different strains to mix in a fruiting body (Fortunato et al. 2003). In the laboratory I can make a fruiting body from a mix of two strains. If two strains mix, our model predicts that the social amoeba will evolve to produce more signaling chemical and cells will become less sensitive to signaling chemical, compared to a case without mixing. The degree to which different strains mix varies depending on the microhabitats, as well as the dispersal and proliferation ability of the strains (Fortunato et al. 2003). It is likely that the environment is spatially heterogeneous and that signaling chemical productivity and cell sensitivity to the chemical might evolve to different degrees within specific microhabitats. Rodrigues and Gardner (2012) emphasize the importance of spatiotemporal heterogeneity in cooperation. This may explain the heterogeneity observed among strains sampled from the field (Strassmann et al. 2000). Because signaling chemical causes cell differentiation through a complex molecular network (Fortunato et al. 2003; Parkinson et al. 2011), additional examinations of this network are needed for assessing signaling chemical cell sensitivity.

If each fruiting body consists of the same strain (m=0), then the relatedness among cells to be stalk and those to be spore is one, and I expect perfect cooperation achieving the maximum number of surviving spores. In addition, the method of communication among cells that is the least costly should evolve, which explains the evolution of low production rates of signaling chemical and a high sensitivity of cells to the chemical. However, as opportunities to form a multi-strain fruiting body increase,

perfect cooperation becomes more difficult. As a consequence, the cells become less sensitive to the signaling chemical to prevent the risk of being exploited by cheater strains. However, just reducing cell's sensitivity leads to a small stalk size when the strain makes fruiting body by itself. This is disadvantageous because the dispersal success of spores becomes low. As a result, in order to keep the stalk size close to optimal, the production rate of signaling chemical should increase. As a consequence, the size of the stalk may not change much between the mixing and nonmixing situations, but cell sensitivity to signaling chemical and the chemical production rate should change greatly (Fig. 4b). If this scenario holds, even if the issue is stalk size, the outcome may appear in cell-to-cell communication rather than the size of the stalk itself.

The model I adopted in this chapter includes many simplifications. Some of the limitations of the model can be removed in future theoretical studies. First, in this model I focused only on the production of the signaling chemical and cell sensitivity to it in a phenomenological way. I may be able to incorporate the details of the molecular mechanisms of within-cell kinetics. Second, when two strains are mixed, I focused on cheater production. However, other effects of mixing strains are also known. For example, the migration distance travelled by a mixed aggregation is shorter than that by a single-strain aggregation (Strassmann and Queller 2011). Third, I assumed that the total number of cells included in a fruiting body is a fixed constant, but there can be a systematic difference with respect to cell division ability among different strains (Strassmann and Queller 2011).

Despite the limitations of this model, the study clearly demonstrates that when I study the evolution of the stalk/spore ratio, I must incorporate the mechanisms for cell-type determination and how different cells interact with each other via chemical signals. For example, I discovered that the main outcome of conflict appears in the chemical production rate and cell sensitivity, much more clearly than in the stalk size itself. Without considering the cell-to-cell interaction, I would fail to identify this sign of the conflict. In forming the model, I adopted the interaction between chemicals and cells known for DIF-1. However the framework of the model in which a signaling chemical controls the cell fate is probably also applicable to other signaling chemicals. For example, Chen and Schaap (2012) found that c-di-GMP induces stalk cell differentiation. It will be an interesting question how much modification of the model is requred when function of c-di-GMP becomes known in more details in considering the evolution of cooperation through c-di-GMP communication.

I expect that many evolutionary phenomena make more sense if I consider the mechanism for cell interaction, which is more difficult to interpret if I simply regard the fraction of stalk size as an indicator of the cooperation of the strain. I must consider the molecular mechanism and gene network at least in the simplest manner. On the other hand, cell and developmental biology have focused on the mechanisms by which the cell responds to different inputs and how the whole aggregation of cells or the whole individual should behave. Without considering the evolutionary process, I would miss an important process that determines chemical reactions and sensitivity. I are certain that theoretical modeling that incorporates both evolutionary biology and cellular and

molecular mechanisms will become an important tool for modeling evolution in the near future.

5. Appendices

Appendix A: *Evolution when fruiting bodies are made of clones containing a single strain*

The simplest way of introducing a continual change in parameters is as follows. Let $W = S(T)(N-T)e^{-ka}$ be the fitness function (I remove the hat in the following equations). From the equilibrium of the dynamics of prespore cells and prestalk cells, given by Eq. (3) and $T+P=N$, I can express T as the function of parameters: a, b, f_0, and g_0. Hence $T = T(a, b, f_0, g_0)$. In Figure 3, a and f_0 change over evolution, whereas b and g_0 are fixed (i.e., common to all the phenotypes).

To derive the trait dynamics, I first calculate the selection differential by considering two strains with very similar parameters. Suppose strains 1 and 2 are the invader and the resident.

$$W_1 = S(T_1)P_1e^{-ka_1}, \text{ where } T_1+P_1=N, \ \frac{T_1}{P_1}=\frac{f_{01}}{g_{01}}C_1, \text{ and } C_1=\frac{a_1P_1}{b_1T_1}. \quad \text{(A.1a)}$$

$$W_2 = S(T_2)P_2e^{-ka_2}, \text{ where } T_2+P_2=N, \ \frac{T_2}{P_2}=\frac{f_{02}}{g_{02}}C_2, \text{ and } C_2=\frac{a_2P_2}{b_2T_2}. \quad \text{(A.1b)}$$

By setting $T = T_2$ and $\Delta T = T_1 - T_2$, and assuming that the latter is small in magnitude, I can rewrite $P_2 = N - T$, $T_1 = T + \Delta T$, and $P_1 = N - T - \Delta T$. I assume that $\Delta f_0 = f_{01} - f_{02}$ and $\Delta a = a_1 - a_2$ are small, and $g_{01} = g_{02}$ and $b_1 = b_2$. Then the differential fitness is

$$\begin{aligned}\Delta F &= W_1 - W_2 = S(T_1)P_1e^{-ka_1} - S(T_2)P_2e^{-ka_2} \\ &\approx \left[\frac{dS}{dT}\bullet(N-T) - S(T)\right]e^{-ka}\bullet\Delta T_1 - S(T)(N-T)e^{-ka}k\Delta a\end{aligned}. \quad \text{(A.2)}$$

The quantitative genetics formalism (Lande 1976; Barton and Turelli 1991; Iwasa et al.

1991) then gives the dynamics in Eq. (9) in the text.

$\partial T/\partial a$ and $\partial T/\partial f_0$ are the dependence of the mutant's stalk size T on its signaling chemical production rate a_1 and the sensitivity f_{01} of the mutant, respectively. From the formula of Eq. (3), I have $\Delta T = \frac{T(N-T)}{3N}\left(\frac{\Delta f_0}{f_0} + 2\frac{\Delta a}{a}\right)$, which leads to

$$\frac{\partial T}{\partial a} = \frac{T(N-T)}{3N}\frac{2}{a} \text{ and } \frac{\partial T}{\partial f_0} = \frac{T(N-T)}{3N}\frac{1}{f_0}. \qquad \text{(A.3)}$$

Both of these are positive. Hence, if I neglect the cost of chemical k, the dynamics given by Eq. (A.3) indicate fast convergence to the line of equilibria $\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T} = 0$, which appears as a curve on a (a, f)-plane. As this quantity is equal to the derivative of $S(T)\bullet(N-T)$, the dynamics (with k neglected) describe the monotonic increase in $S(T)\bullet(N-T)$, the number of surviving spores.

Appendix B: ***Evolution when some fruiting bodies are a mixture of two strains***

I define the following functions as the fitness of strains 1 and 2, respectively:

$$F_1 = (1-m)E[1|1] + m\left[\xi E[1|1] + (1-\xi)E[1|2]\right], \tag{B.1a}$$

$$F_2 = (1-m)E[2|2] + m\left[\xi E[2|1] + (1-\xi)E[2|2]\right]. \tag{B.1b}$$

where $E[1|1] = W_1$, $E[2|2] = W_2$, $E[1|2] = 2W_1^{mix}$, and $E[2|1] = 2W_2^{mix}$.

The frequency of strain 1 in the next generation is $\xi^{next} = F_1\xi / \left(F_1\xi + F_2(1-\xi)\right)$. The change in one generation is $\Delta\xi = \xi^{next} - \xi = \frac{\xi(1-\xi)}{F_1\xi + F_2(1-\xi)}\left(F_1 - F_2\right)$. The differential fitness is

$$\begin{aligned} F_1 - F_2 &= (1-m)\left(E[1|1] - E[2|2]\right) \\ &\quad + m\left[\xi\left(E[1|1] - E[2|1]\right) + (1-\xi)\left(E[1|2] - E[2|2]\right)\right], \end{aligned} \tag{B.2}$$

I can calculate the $E[i|j]$ as follows:

$E[1|1] = S(T_1)P_1 e^{-ka_1}$, where $T_1 + P_1 = N$, $\frac{T_1}{P_1} = \frac{f_{01}}{g_{01}}C_1$, and $C_1 = \frac{a_1 P_1}{b_1 T_1}$. (B.3a)

$E[2|2] = S(T_2)P_2 e^{-ka_2}$, where $T_2 + P_2 = N$, $\frac{T_2}{P_2} = \frac{f_{02}}{g_{02}}C_2$, and $C_2 = \frac{a_2 P_2}{b_2 T_2}$ (B.3b)

$E[1|2] = S\left(T_1^* + T_2^*\right)P_1^* 2e^{-ka_1}$, (B.3c)

$E[2|1] = S\left(T_1^* + T_2^*\right)P_2^* 2e^{-ka_2}$, (B.3d)

where $T_1^* + P_1^* = \frac{N}{2}$, $T_2^* + P_2^* = \frac{N}{2}$, $\frac{T_1^*}{P_1^*} = \frac{f_{01}}{g_{01}}\left(C^*\right)^2$, $\frac{T_2^*}{P_2^*} = \frac{f_{02}}{g_{02}}\left(C^*\right)^2$, and

$C^* = \frac{a_1 P_1^* + a_2 P_2^*}{b_1 T_1^* + b_2 T_2^*}$.

I consider strain 2 as the resident and if the mutant, strain 1, can invade and replace strain 2. Here, I consider the case in which two strains are similar with respect

to parameters: $\Delta f_0 = f_{01} - f_{02}$, $\Delta g_0 = g_{01} - g_{02}$, $\Delta a = a_1 - a_2$ and $\Delta b = b_1 - b_2$ are all small in magnitude. Hence, I can assume that $T_1 - T_2 = \tau_1$, $T_1^* - T_2/2 = \tau_1^*$, and $T_2^* - T_2/2 = \tau_2^*$ are small quantities. Using these and the constraints of $T_1 + P_1 = T_2 + P_2 = N$ and $T_1^* + P_1^* = T_2^* + P_2^* = N/2$, I have $P_1 - P_2 = -\tau_1$, $P_1^* - P_2 = -\tau_1^*$, and $P_2^* - P_2 = -\tau_2^*$. Please note that when two strains are exactly the same, $\tau_1 = \tau_1^m = \tau_2^m = 0$ holds. Furthermore I set $T = T_2$. Using this I have the following relationships:

$$\begin{aligned} E[1|1] - E[2|2] &= S(T_1)P_1 e^{-ka_1} - S(T_2)P_2 e^{-ka_2} \\ &\approx \left[\frac{dS}{dT} \bullet (N-T) - S(T)\right] e^{-ka} \bullet \tau_1 - S(T)(N-T)e^{-ka} k\Delta a \end{aligned}, \tag{B.4a}$$

$$\begin{aligned} E[1|1] - E[2|1] &= S(T_1)P_1 e^{-ka_1} - S(T_1^* + T_2^*)P_2^* 2e^{-ka_2} \\ &\approx \left[\frac{dS}{dT} \bullet (N-T) - S(T)\right] e^{-ka}(\tau_1 - \tau_1^* - \tau_2^*) + S(T)e^{-ka}(-\tau_1^* + \tau_2^*), \\ &\quad -S(T)(N-T)e^{-ka} k\Delta a \end{aligned} \tag{B.4b}$$

and

$$\begin{aligned} E[1|2] - E[2|2] &= S(T_1^* + T_2^*)P_1^* 2e^{-ka_1} - S(T_2)P_2 e^{-ka_2} \\ &\approx \left[\frac{dS}{dT} \bullet (N-T) - S(T)\right] e^{-ka}(\tau_1^* + \tau_2^*) + S(T)e^{-ka}(-\tau_1^* + \tau_2^*). \\ &\quad -S(T)(N-T)e^{-ka} k\Delta a \end{aligned} \tag{B.4c}$$

Hence, the differential fitness divided by the mean fitness $(F_1 - F_2)/\overline{F}$ is

$$\begin{aligned} \frac{\Delta F}{F} &= \left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)\left\{(1 - m + m\xi)\tau_1 + m(1-2\xi)(\tau_1^* + \tau_2^*)\right\} \\ &\quad + m\frac{1}{N-T}\left\{-\tau_1^* + \tau_2^*\right\} - k\Delta a \end{aligned}. \tag{B.5}$$

The fitness is written as $F(T_1, T_1^*, T_2^*, a_1)$ where T_1, T_1^*, T_2^* are dependent on the mutant's parameters f_{01}, g_{01}, a_1, and b_1. The last argument indicates the direct dependence of the fitness to a_1. Eq. (B.5) then indicates

$$\frac{\partial \ln F}{\partial T_1} = \left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)(1-m+m\xi), \tag{B.6a}$$

$$\frac{\partial \ln F}{\partial T_1^*} = \left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)m(1-2\xi) - m\frac{1}{N-T}, \tag{B.6b}$$

$$\frac{\partial \ln F}{\partial T_2^*} = \left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)m(1-2\xi) + m\frac{1}{N-T}, \tag{B.6c}$$

$$\frac{\partial \ln F}{\partial a_1} = -k. \tag{B.6d}$$

The trait dynamics are

$$\Delta\bar{f}_0 = G_{f_0}\frac{\partial}{\partial f_0}\ln F = G_{f_0}\left[\frac{\partial \ln F}{\partial T_1}\frac{\partial T_1}{\partial f_0} + \frac{\partial \ln F}{\partial T_1^*}\frac{\partial T_1^*}{\partial f_0} + \frac{\partial \ln F}{\partial T_2^*}\frac{\partial T_2^*}{\partial f_0}\right]$$
$$= G_{f_0}\left[\left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)\left\{(1-m+m\xi)\frac{\partial T_1}{\partial f_0} + m(1-2\xi)\left(\frac{\partial T_1^*}{\partial f_0} + \frac{\partial T_2^*}{\partial f_0}\right)\right\} + \frac{m}{N-T}\left(-\frac{\partial T_1^*}{\partial f_0} + \frac{\partial T_2^*}{\partial f_0}\right)\right]$$

Similarly, I have

$$\Delta\bar{a} = G_a\left[\left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)\left\{(1-m+m\xi)\frac{\partial T_1}{\partial a} + m(1-2\xi)\left(\frac{\partial T_1^*}{\partial a} + \frac{\partial T_2^*}{\partial a}\right)\right\} + \frac{m}{N-T}\left(-\frac{\partial T_1^*}{\partial a_0} + \frac{\partial T_2^*}{\partial a}\right) - k\right]$$

Now I consider how T_1^* and T_2^* may depend on parameters. I note the following relationship:

$$\frac{T_1^*}{N/2 - T_1^*} = \frac{f_{01}}{g_{01}}\left(C^*\right)^2, \quad \text{and} \quad \frac{T_2^*}{N/2 - T_2^*} = \frac{f_{02}}{g_{02}}\left(C^*\right)^2$$

If the deviation of T_1^* and T_2^* from $T/2$ are small, I have

$$\frac{\partial T_1^*}{\partial f_{01}} = \frac{2N}{T(N-T)}\left(\frac{\Delta f_0}{f_0} + \frac{2\Delta C^*}{C^*}\right), \text{and } \frac{\partial T_2^*}{\partial f_{01}} = \frac{2N}{T(N-T)}\left(\frac{2\Delta C^*}{C^*}\right), \tag{B.8}$$
$$\frac{\partial T_1^*}{\partial a_1} = \frac{\partial T_2^*}{\partial a_1} = \frac{2N}{T(N-T)}\frac{2\Delta C^*}{C^*}.$$

Hence $-\frac{\partial T_1^*}{\partial f_{01}} + \frac{\partial T_2^*}{\partial f_{01}} = -\frac{2N}{T(N-T)}\frac{\Delta f_0}{f_0} < 0$, and $-\frac{\partial T_1^*}{\partial a_1} + \frac{\partial T_2^*}{\partial a_1} = 0$.

$$\Delta\bar{a} = G_a\left[\left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)\left\{(1-m+m\xi)\frac{\partial T_1}{\partial a} + m(1-2\xi)\left(\frac{\partial T_1^*}{\partial a} + \frac{\partial T_2^*}{\partial a}\right)\right\} - k\right], \quad \text{(B.9a)}$$

$$\Delta\bar{f}_0 = G_{f_0}\left[\left(\frac{1}{S}\frac{dS}{dT} - \frac{1}{N-T}\right)\left\{(1-m+m\xi)\frac{\partial T_1}{\partial f_0} + m(1-2\xi)\left(\frac{\partial T_1^*}{\partial f_0} + \frac{\partial T_2^*}{\partial f_0}\right)\right\} - \frac{2mN}{T(N-T)^2}\frac{1}{f_0}\right]. \quad \text{(B.9b)}$$

The first term in the brackets in each equation is for fast dynamics, which cause the quick approach to a curve for the optimal stalk size $\frac{1}{S}\frac{dS}{dT} = \frac{1}{N-T}$, at which $S(T) \bullet (N-T)$ is maximized. This appears as a curve $f_0 a^2 = const$. An additional term in the dynamics of production rate $\bar{a}$ is to reduce the rate (selection gradient of -*k*). This makes the movement of the state along curve $f_0 a^2 = const$ toward a smaller *a* and higher sensitivity f_0. In contrast, an additional term of the dynamics for sensitivity $\bar{f}_0$ is also a negative selection gradient $-\frac{2mN}{T(N-T)^2 f_0}$. This causes the movement of the system along the curve $f_0 a^2 = const$ toward a lower sensitivity. Combining these two, I have the dynamics for which the balance of these two forces results in equilibrium with an intermediate magnitude of sensitivity f_0 and chemical production rate *a*. Note that the term toward reducing sensitivity f_0 increases with the fraction of fruiting bodies that are mixture of strains *m*.

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7. Figure Captions

Fig. 1 Interaction between prestalk and prespore cells via a signaling chemical. The signaling chemical is produced by prespore cells and decomposed by prestalk cells. In turn, the signaling chemical enhances the rate of transition from a prespore cell to a prestalk cell while reducing the rate of transition in the opposite direction.

Fig. 2 Equilibrium of cell differentiation. The number of prespore cells of strain 1 (P_1) and prestalk cells of strain 1 (T_1) are indicated by a cross and asterisk, respectively. The number of prespore cells of strain 2 (P_2) and prestalk cells of strain 2 (T_2) are indicated by an open circle and open square, respectively. Horizontal axis represents time. (a) Each strain makes a fruiting body by itself. (b) Two strains make a fruiting body. Parameters are: N=100000, a_1=0.25, b_1=0.4, f_{01}=0.2, g_{01}=1, a_2=0.25, b_2=0.8, f_{02}=0.8, g_{02}=1.0.

Fig. 3 Evolutionary trajectories. Horizontal axis is for the production rate of the signaling chemical (a), and vertical axis is for the sensitivity to the signaling chemical when a prespore cell switches to to a prestalk cell (f_0). (a) Two strains do not mix (m=0), and (b) two strains can mix (m = 0.03). Other parameters are: N=100,000, b=2, g=2, k=0.01, S_0=1, α=10,000, l=2.

Fig. 4 Evolutionarily stable states depending on the possibility of mixing. (a) Squares are for the evolutionarily stable sensitivity of cells to signaling chemical cell

(f_0), for which the label is given on the left. Circles are for the evolutionarily stable rate of signaling chemical production (a), for which the label is given on the right. Horizontal axis is for m, the fraction of fruiting bodies consisting of cells originating from two initial spores. (b) Evolutionarily stable fraction of prestalk cells. Other parameters are the same as in Fig. 3.

8. Figures

Figure 1

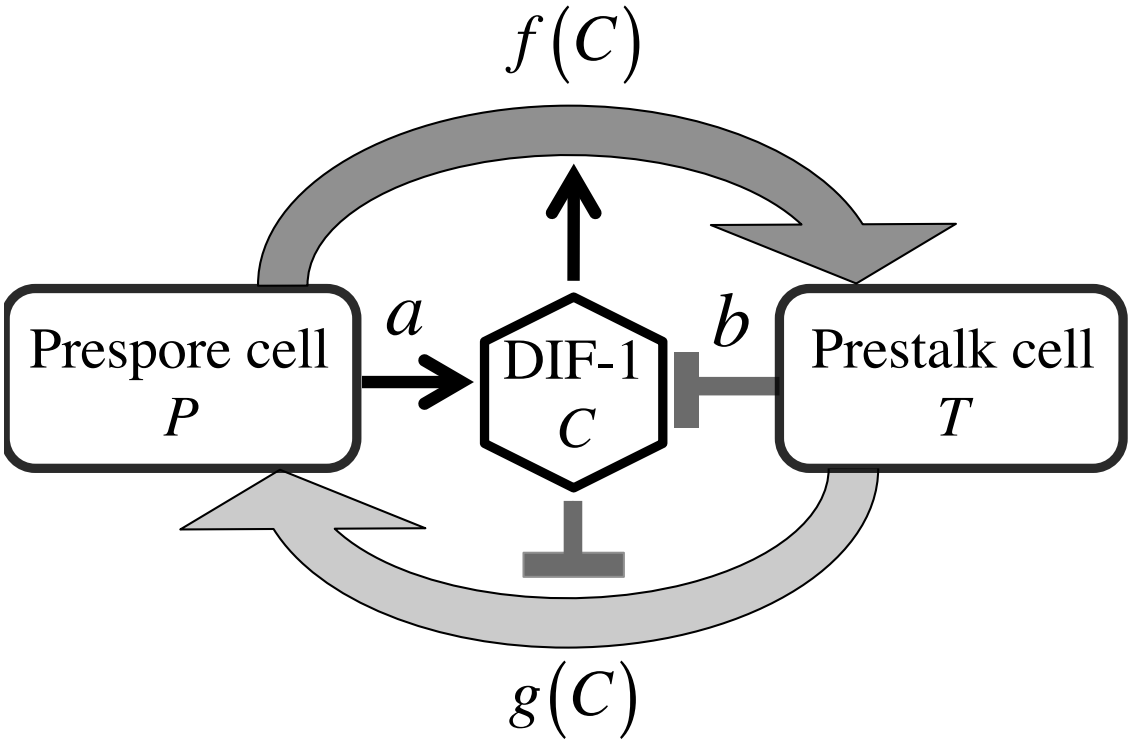


Figure 2

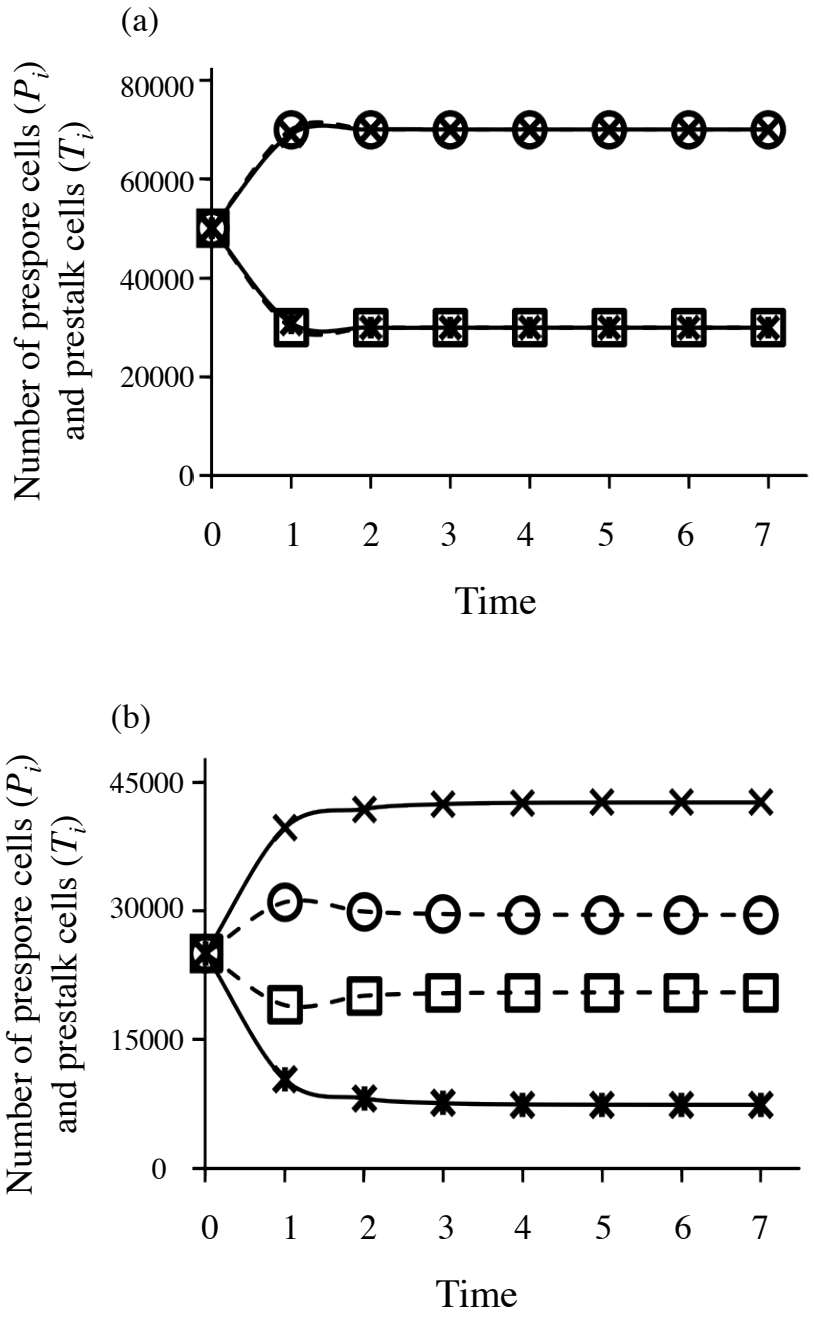

(a)
Number of prespore cells (P_i) and prestalk cells (T_i)
80000
60000
40000
20000
0
0 1 2 3 4 5 6 7
Time
(b)
Number of prespore cells (P_i) and prestalk cells (T_i)
45000
30000
15000
0
0 1 2 3 4 5 6 7
Time

Figure 3

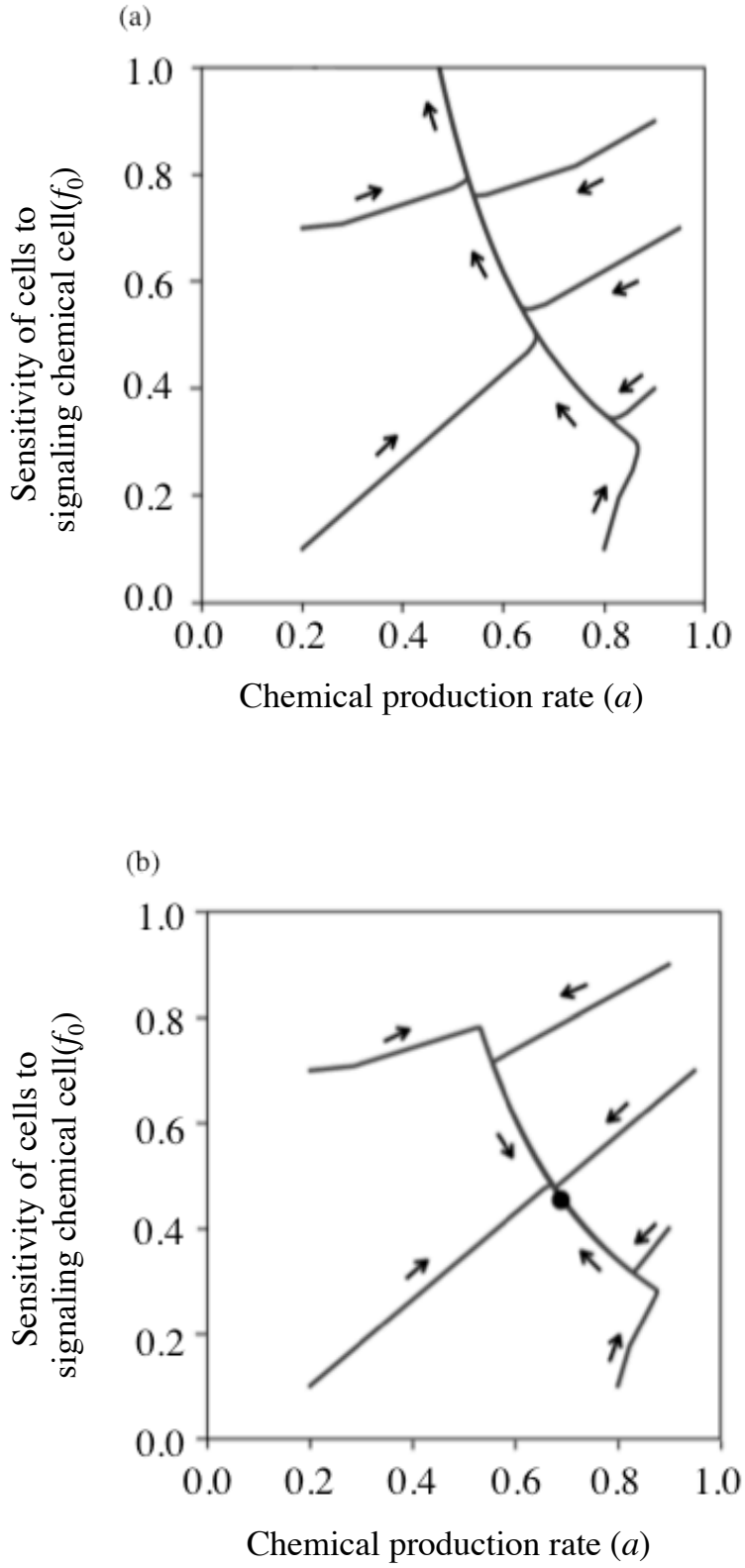
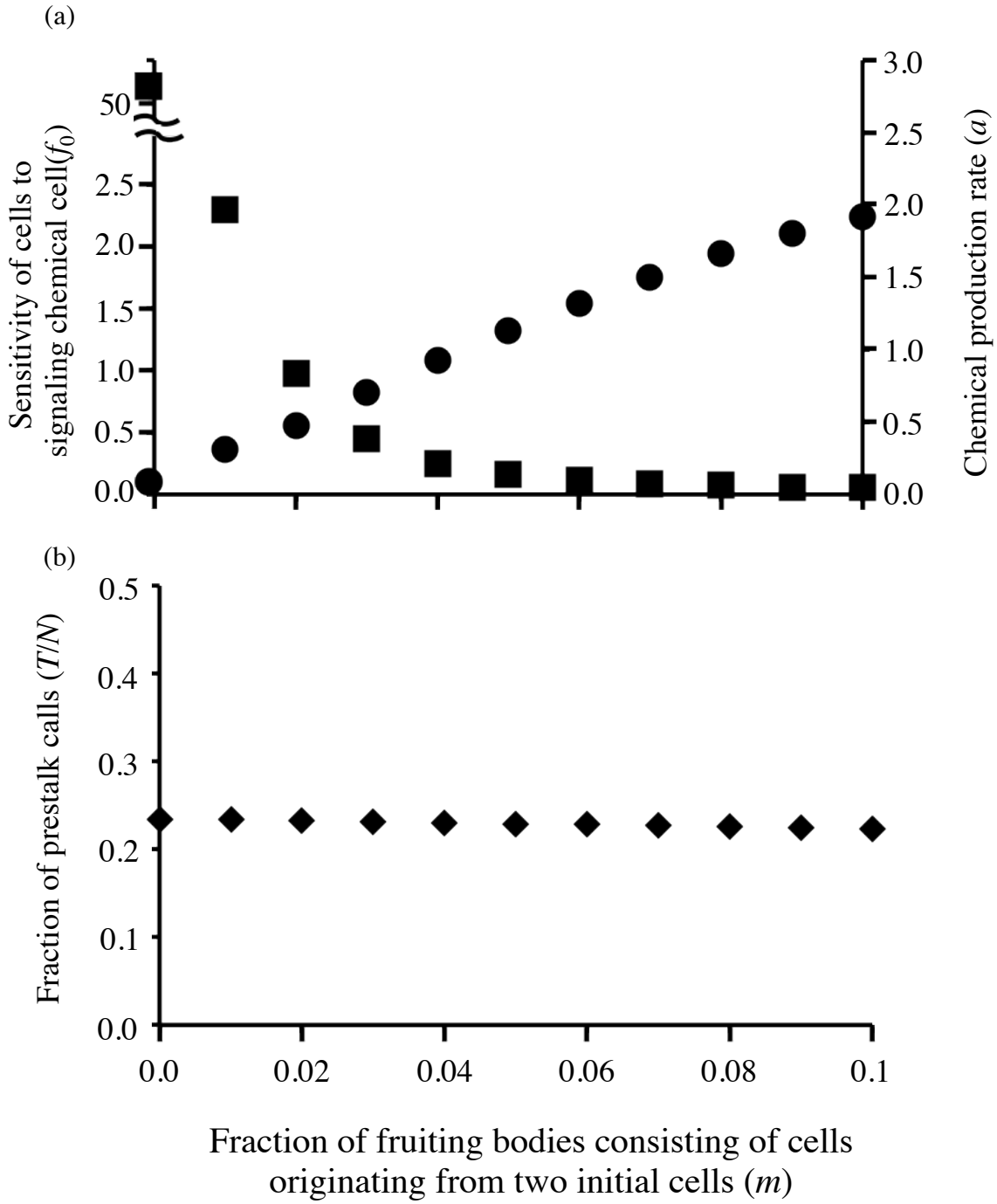

(a)
1.0
0.8
0.6
0.4
0.2
0.0
0.0
0.2
0.4
0.6
0.8
1.0
Sensitivity of cells to signaling chemical cell(f_0)
Chemical production rate (a)
(b)
1.0
0.8
0.6
0.4
0.2
0.0
0.0
0.2
0.4
0.6
0.8
1.0
Sensitivity of cells to signaling chemical cell(f_0)
Chemical production rate (a)

Figure 4


(a)
Sensitivity of cells to signaling chemical cell(f_0)
50
2.5
2.0
1.5
1.0
0.5
0.0
3.0
2.5
2.0
1.5
1.0
0.5
0.0
Chemical production rate (a)
(b)
Fraction of prestalk calls (T/N)
0.5
0.4
0.3
0.2
0.1
0.0
0.0
0.02
0.04
0.06
0.08
0.1
Fraction of fruiting bodies consisting of cells originating from two initial cells (m)

Chapter 2

Optimum resource allocation in the plant-fungus symbiosis for an exponentially growing system

The chapter was done in collaboration with Professor Yoh Iwasa.
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1. INTRODUCTION

Mutualism with soil fungi is prevalent throughout terrestrial plants. Most terrestrial plants develop root systems called mycorrhiza by interacting with soil fungi; i.e., mycorrhizal fungi. The plant produces carbohydrate from atmospheric CO_2 by photosynthesis and transfers some portion of carbohydrates to the fungal symbiont. On the other hand, the fungus takes up soil nutrients such as phosphate and provides a fraction thereof to the host plant.

Many theoretical studies have examined the resource exchange between host and symbiont. Schwartz and Hoeksema (Schwartz and Hoeksema, 1998; Hoeksema and Schwartz, 2003) applied biological market theory to the plant-mycorrhiza system. If the cost of acquiring ambient resources differs between two species, each species benefits by specializing toward acquisition of one resource and trading the other resource with the partner species.

De Mazancourt and Schwartz (2010) demonstrated that organisms could use resources more efficiently by trading resources. They considered two species that require two resources and whose resource acquisition abilities differ. If one species can use both resources more efficiently than the other species, the latter species goes extinct. However, if one species can use one resource more efficiently and the other species can use the other resource more efficiently, the two species can coexist. In addition, if growth is limited by the less abundant resource among the two, there exists a surplus of the second resource that the species cannot use efficiently. Then, by trading the resource

surpluses of both species, they can both use the resource more efficiently than in the case without trading.

In their theoretical model, Grman *et al*. (2012) combined the cooperative game model used for the plant-rhizobia system by Akçay and Roughgarden (2007) and the biological market theory developed by Schwartz and Hoeksema (1998). If the growth of organisms is dependent on two resources, two symbiotic conditions can exist. In the first set of conditions, both the plant and fungus specialize in taking up one resource. They obtain the other resource by trading: the plant makes carbohydrate by photosynthesis but does not take up phosphorus from the soil, while the fungus takes up only phosphorus from the soil. These conditions are optimal if light availability is low and phosphorus levels in the soil are moderate. In the second set of conditions, one organism takes up both resources and the other organism can only take up one. For example, when soil is rich in phosphorus, the plant specializes in taking up carbon, but the fungus takes up both carbon and phosphorus from the soil. However, neither Grman *et al*. (2012) nor De Mazancourt and Schwartz (2010) considered the dynamic aspects of the plant-fungus interaction.

Both the host plant and symbiotic fungus change their behavior in response to the environment. For example, the presence of fungi would enhance plant growth when soil nutrients are scarce, whereas fungi could deter plant growth when soil nutrients are abundant (Johnson *et al*., 1997). In response to such shifts in the relative benefit of the symbiosis, plants can decrease carbon allocation to symbiotic fungi, leading to reduced size of mycorrhiza (Bever *et al*., 2008). In addition, plants can engage in partner choice;

when a plant harbors many symbiotic fungi in its roots, it can selectively decrease allocation to the fungi that do not allocate very much phosphorus (Bever *et al*., 2008; Kiers *et al*., 2011). In turn, fungi can also control resource allocation to the host plant; when the fungus interacts with many roots, it can allocate more phosphorus to roots that provide more carbohydrate (Kiers *et al*., 2011). Changing resource allocation is sometimes considered "punishment" that stabilizes the mutualism (Kiers *et al*., 2011). However, this situation provides an opportunity for cheaters; for example, by mycoheterotrophic plants, which do not have chloroplasts and do not produce carbohydrate via photosynthesis (Leake, 1994). Nonetheless, fungi can allocate not only nutrients from the soil, but also carbohydrates that are allocated by other plants (Leake, 1994).

In this chapter, I examine the optimal resource allocation of a plant and fungus to their partner when the entire system grows exponentially. Rapid growth is critical for the survival of seedlings, and phosphorus provided by the fungus is key to seedling establishment (Smith and Read, 2010). Under these conditions, the exponential growth rate for the whole system is a natural measure for overall success. The rates of resource acquisition (photosynthesis by the plant and phosphorus absorption by the fungus) increase with increases in both carbon and phosphorus within the body of each player. Assuming Cobb-Douglas production functions, I show that given the optimal rate of resource allocation, each player should allocate more to the partner when the resource provided by the partner is more important to its own resource acquisition. Second, I show that the growth trajectory corresponding to the optimal allocation fractions

satisfies the conditions for the singular control subarc for dynamical optimization when each player maximizes its own fitness given the partner's growth schedule. Hence, the ESS resource allocation by two players in a non-cooperative game is equivalent to the cooperative optimum of resource allocation during the exponential growth phase.

2. MODEL

Consider two players: a plant and its symbiotic fungus in soil (Fig. 1). The plant performs photosynthesis and obtains carbon in its aboveground parts, while the fungus takes up nutrients, such as phosphorus, from the soil. The plant needs phosphorus supplied by the fungus to function properly. Hence, the plant allocates some fraction of its carbon to the soil fungus, which may in turn supply phosphorus to the plant in the future. In contrast, the fungus needs carbon supplied by the plant; thus, it allocates some phosphorus to the plant to secure future carbon. As a result, both players, plant and fungus, allocate some fraction of its acquired resource to the partner to secure future resources that are otherwise difficult to obtain directly. Here, I focus on a small, young plant individual (or seedling) and its soil fungus, in the state in which both grow exponentially through time.

I distinguish quantities of plant and fungi by $i = 1$ and $i = 2$, respectively. For simplicity, I assume that the soil fungus interacting with an individual plant exists as a large number of bundles or sheets of hyphae composed of genetically identical cells. Let C_i be the amount of carbon, and P_i be the amount of phosphorus contained in the plant ($i = 1$) and in the fungus ($i = 2$). Let $f\left(C_1, P_1\right)$ be the rate of carbon acquisition by

the plant, and $g(C_2, P_2)$ be the rate of phosphorus acquisition by the fungus. These resource acquisition rates are increasing functions of both carbon and phosphorus. I consider the following dynamics:

$$\frac{dC_1}{dt} = (1-u_1) f(C_1, P_1), \tag{1a}$$

$$\frac{dP_1}{dt} = u_2 \xi_2 g(C_2, P_2), \tag{1b}$$

$$\frac{dC_2}{dt} = u_1 \xi_1 f(C_1, P_1), \tag{1c}$$

$$\frac{dP_2}{dt} = (1-u_2) g(C_2, P_2), \tag{1d}$$

where u_1 is the fraction of carbon obtained by the plant that will be allocated to the fungus; it satisfies $0 \le u_1 \le 1$. This value can be chosen by the plant. In contrast, u_2 is the fraction of phosphorus sequestered by the fungus that will be allocated to the plant ($0 \le u_2 \le 1$). I consider that some loss occurs during this allocation process; hence, I consider the fractions lost, ξ_1 and ξ_2 to be factors smaller than 1 ($\xi_1 < 1$ and $\xi_2 < 1$).

The rate of photosynthesis increases with carbon C_1 and phosphorus P_1. In addition, the rate increases in proportion to plant size, if the ratio of carbon to phosphorus is kept constant. This last assumption is plausible for a young, small plant (or seedling) growing exponentially together with its symbiotic fungus. More specifically, I assume that the carbon acquisition rate of the plant and the phosphorus acquisition rate of the soil fungus satisfy the following relationships:

$$f(KC_1, KP_1) = Kf(C_1, P_1) \quad \text{for any positive } K, \tag{2a}$$

$$g(KC_2, KP_2) = Kg(C_2, P_2) \quad \text{for any positive } K. \tag{2b}$$

Eq. (2a) implies that if both C_1 and P_1 are multiplied by factor *K*, $f(C_1,P_1)$ is also multiplied by the same factor *K*. Hence, the rate of carbon acquisition increases in proportion to plant size, if the size by which the plant increases with the ratio of carbon and phosphorus is kept constant. In a similar manner, Eq. (2b) indicates that if both C_2 and P_2 are multiplied by factor *K*, $g(C_2,P_2)$ is also multiplied by the same factor *K*.

2.1. Optimal application to own partner

As a simple example of production functions satisfying Eq. (2), I assume that the photosynthetic rate is the power function of two resources: $f(C_1,P_1) \equiv f_0 C_1^{1-\alpha} P_1^{\alpha}$, where α indicates the relative importance of phosphorus ($0 < \alpha < 1$), and f_0 indicates the basic rate of carbon acquisition. In economics, $f(C_1,P_1)$ is called the "Cobb-Douglas production function" (see Fig. 2). In a similar manner, the rate of phosphorus sequestering by the fungus is $g(C_2,P_2) \equiv g_0 C_2^{\beta} P_2^{1-\beta}$, which increases with both carbon and phosphorus levels of the fungus. β indicates the relative importance of carbon in this function ($0 < \beta < 1$), and g_0 indicates the basic rate of phosphorus acquisition. Again, if both C_2 and P_2 are multiplied by factor *K*, $g(C_2,P_2)$ is also multiplied by the same factor *K*.

Now, I focus on the exponential growth solution for this system. Assume that $C_1 = c_{10}e^{rt}$, $P_1 = p_{10}e^{rt}$, $C_2 = c_{20}e^{rt}$, and $P_2 = p_{20}e^{rt}$, where *r* is the exponential rate of growth. Then, Eq. (1) becomes

$$rc_{10} = (1-u_1) f_0 c_{10}^{1-\alpha} p_{10}^{\alpha}, \tag{3a}$$

$$rp_{10} = u_2 \xi_2 g_0 c_{20}^{\beta} p_{20}^{1-\beta}, \tag{3b}$$

$$rc_{20} = u_1 \xi_1 f_0 c_{10}^{1-\alpha} p_{10}^{\alpha}, \tag{3c}$$

$$rp_{20} = (1-u_2) g_0 c_{20}^{\beta} p_{20}^{1-\beta}. \tag{3d}$$

After some arithmetic (explained in Appendix A), I have

$$r^{\frac{1}{\beta}+\frac{1}{\alpha}} = u_1 (1-u_1)^{\frac{1-\alpha}{\alpha}} u_2 (1-u_2)^{\frac{1-\beta}{\beta}} g_0^{\frac{1}{\beta}} f_0^{\frac{1}{\alpha}} \xi_1 \xi_2. \tag{4}$$

The optimum carbon allocation for the plant to the partner is the value of u_1 that maximizes the exponential rate of growth Eq. (4). By calculating the partial derivative of Eq. (4) by u_1 and then setting it to 0, I have

$$u_1^* = \alpha. \tag{5a}$$

In a similar manner, the optimal rate of phosphorus allocation for the fungus to the plant that achieves the maximum exponential growth rate *r* is:

$$u_2^* = \beta. \tag{5b}$$

See Appendix A for derivation.

Power α is a coefficient for the dependence of the rate of photosynthesis on the amount of phosphorus that is supplied by the fungus. If α is close to 0, the photosynthetic rate is almost independent of *P*, and if instead α is close to 1, it is proportional to *P* and is almost independent of *C*. The plant should allocate more to the fungus if the resource supplied by the fungus is more important for its own function (i.e., larger α). Eq. (5b) is a similar result for the optimal resource allocation for the fungus. If the phosphorous acquisition rate by the fungus is strongly dependent on its carbon rather than phosphorus level (large β), then the fungus should allocate a larger fraction of newly acquired phosphorus to the plant, rather than for its own use.

Fig. 3a illustrates a contour plot of the optimal allocation ratio by the plant to the fungus, where the horizontal axis is α, and the vertical axis is β. Fig. 1b depicts a similar contour plot of the optimal allocation ratio by the fungus to the plant.

3. DYNAMIC OPTIMIZATION OF EACH PLAYER

Eqs. (5a) and (5b) are the allocation values for two players given that they are constant over time. Here, I show that even if the allocation fraction is allowed to depend on time, these constant solutions are the optimal control for the respective players. This can be shown by introducing the analysis of dynamic optimization. By fixing phosphorus allocation by the fungus at a constant value $u_2(t) = \beta$, I search for the optimal control schedule of the plant $u_1(t)$, and show that the optimal solution is a constant $u_1(t) = \alpha$. The allocation ratio affects the dynamics linearly, and this solution is an intermediate value $0 < u_1(t) < 1$, which is called a "singular control subarc" (Intriligator, 1971). In a similar manner, I also show that the optimal control problem for the fungus is a constant solution $u_2(t) = \beta$, which maximizes the performance of the fungus under the condition of $u_1(t) = \alpha$.

I consider the optimal allocation schedule for the plant (player $i = 1$), denoted by $0 \le u_1(t) \le 1$ $(0 < t < T_f)$, which maximizes $f\left(C_1(T_f), P_1(T_f)\right)$, the rate of photosynthesis of the plant at time T_f. In calculating this dynamic optimization problem, I treat the behavior of the fungus as given. This is a typical problem of optimal control and can be solved using Pontryagin's maximum principle (Pontryagin *et al.*,

1962). The Hamiltonian of this dynamic optimization model for the plant (the first player) is defined as

$$H = \lambda_{C1}\left(1-u_1\right)f_0 C_1^{1-\alpha}P_1^{\alpha} + \lambda_{P1}u_2^{\ *}\xi_2 g_0 C_2^{\beta}P_2^{1-\beta} + \lambda_{C2}u_1\xi_1 f_0 C_1^{1-\alpha}P_1^{\alpha} + \lambda_{P2}\left(1-u_2^{\ *}\right)g_0 C_2^{\beta}P_2^{1-\beta}. \quad (6a)$$

λ_{C1}, λ_{P1}, λ_{C2}, and λ_{P2} are costate variables, corresponding to C_1, P_1, C_2, and P_2, respectively. These variables indicate the "marginal effect of a small unit increase in the corresponding variable toward enhancing fitness" (Intriligator, 1971; Iwasa and Roughgarden, 1984). They change with time following the differential equations below:

$$\frac{d\lambda_{C1}}{dt} = -\frac{\partial H_1}{\partial C_1} = -\left(\lambda_{C1}\left(1-u_1\right)+\lambda_{C2}u_1\xi_1\right)\left(1-\alpha\right)f_0 C_1^{-\alpha}P_1^{\alpha}, \quad (6b)$$

$$\frac{d\lambda_{P1}}{dt} = -\frac{\partial H_1}{\partial P_1} = -\left(\lambda_{C1}\left(1-u_1\right)+\lambda_{C2}u_1\xi_1\right)\alpha f_0 C_1^{1-\alpha}P_1^{-1+\alpha}, \quad (6c)$$

$$\frac{d\lambda_{C2}}{dt} = \frac{\partial H_1}{\partial C_2} = -\left(\lambda_{P1}u_2^{*}\xi_2+\lambda_{P2}\left(1-u_2^{*}\right)\right)\beta g_0 C_2^{-1+\beta}P_2^{1-\beta}, \quad (6d)$$

$$\frac{d\lambda_{P2}}{dt} = \frac{\partial H_1}{\partial P_2} = -\left(\lambda_{P1}u_2^{*}\xi_2+\lambda_{P2}\left(1-u_2^{*}\right)\right)\left(1-\beta\right)g_0 C_2^{\beta}P_2^{-\beta}, \quad (6e)$$

Using these functions, Pontryagin's maximum principle states that the optimal schedule is the one that maximizes the Hamiltonian H with respect to control variable u_1, given state variables and costate variables. This condition gives the following:

$$\begin{aligned} &u_1^{*} = 0 \ \ if \ \ \lambda_{C1} > \lambda_{C2}\xi_1 \\ &u_1^{*} = 1 \ \ if \ \ \lambda_{C1} < \lambda_{C2}\xi_1 \\ &0 < u_1^{*} < 1 \ \ if \ \ \lambda_{C1} = \lambda_{C2}\xi_1 \end{aligned} . \quad (7)$$

The costate variables satisfy the following differential equations:

$$\frac{d\lambda_{C1}}{dt} = -\frac{\partial H_1}{\partial C_1} = -\max\left[\lambda_{C1},\ \lambda_{C2}\xi_1\right]\left(1-\alpha\right)f_0 C_1^{-\alpha}P_1^{\alpha}, \quad (8a)$$

$$\frac{d\lambda_{P1}}{dt} = -\frac{\partial H_1}{\partial P_1} = -\max\left[\lambda_{C1},\lambda_{C2}\xi_2\right]\alpha f_0 C_1^{1-\alpha}P_1^{-1+\alpha}, \quad (8b)$$

$$\frac{d\lambda_{C2}}{dt} = \frac{\partial H_1}{\partial C_2} = -\left(\lambda_{P1} u_2^* \xi_2 + \lambda_{P2}\left(1 - u_2^*\right)\right)\beta g_0 C_2^{-1+\beta} P_2^{1-\beta}, \tag{8c}$$

$$\frac{d\lambda_{P2}}{dt} = \frac{\partial H_1}{\partial P_2} = -\left(\lambda_{P1} u_2^* \xi_2 + \lambda_{P2}\left(1 - u_2^*\right)\right)\left(1-\beta\right) g_0 C_2^{\beta} P_2^{-\beta}. \tag{8d}$$

The optimal control problem would typically be composed of the intervals in which the allocation ratio takes on terminal values (0 or 1) as well as an interval in which the allocation is an intermediate value (between 0 and 1). If the latter lasts for an interval of a positive length, it is called a "singular subarc" (Intriligator, 1971). In the singular subarc, $\lambda_{C1} = \lambda_{C2}\xi_1$ holds for some time interval with a positive length, which generates additional equations and allows us to derive the solution (see Appendix B for details).

Based on the other optimal growth schedule, the optimal solution is composed of three parts: (1) the initial phase in which allocation is either 0 or 1, which produces the most rapid convergence to the singular subarc, (2) a singular subarc in which both players receive a supply of carbon, and (3) the final period in which the plant stops supplying carbon to the fungus and uses all carbon income for itself. If the entire period of growth is sufficiently long (i.e. T_f is large), the singular subarc should comprise a large portion of the growth schedule.

I also consider the optimal allocation schedule for the fungus (the second player, $i = 2$), which is given by $0 \le u_2(t) \le 1$ $(0 < t < T_f)$, which maximizes $g\left(C_2\left(T_f\right), P_2\left(T_f\right)\right)$, the rate of photosynthesis of the plant at time T_f. In calculating this optimization, I treat the behavior of the plant as given. This can also be analyzed using Pontryagin's maximum principle.

If both players (plant and fungus) adopt intermediate allocation ratios, the growth path needs to be a singular subarc in the two optimal control problems. Along this exponential growth solution, the conditions required by Pontryagin's maximum principle are satisfied for two dynamic optimization problems (optimization of $u_1(\bullet)$ by the plant, and optimization of $u_2(\bullet)$ by the fungus). By combining these two conditions, the same solution results as in the previous section: $u_1 = \alpha$ and $u_2 = \beta$. The derivations are explained in Appendix B.

I note that there are two differences between the calculation in the present section and the arguments in the previous section. First, the calculation in the present section is the dynamic optimization, while that in the previous section was parametric optimization. The second, more important difference is a potential conflict of interest. In the present section, I discuss the solution in which each player maximizes its own objective function that differs between players, and the solution obtained is the evolutionarily stable strategy (Maynard Smith and Price, 1973; Maynard Smith, 1982) or the Nash equilibrium (Nash, 1951). The evolutionary stable dynamic allocation turned out to be the same as the solution for the cooperative optimum, which is the solution that achieves maximization of the common target; i.e., the most rapid growth rate *r* of the whole system.

4. DISCUSSION

In this chapter, I analyzed the optimal allocation between a terrestrial plant and a soil fungus. The plant acquires carbon by photosynthesis, and the soil fungus sequesters

phosphorus from the soil. Because both players require both resources, both parties might profit by allocating some fraction of its resource to the other player. I focused on the situation for which both the plant and fungus grow exponentially through time and determined the optimal fraction of resources allocated to the other player.

I assumed that the resource acquisition rates (photosynthesis by the plant and phosphorus uptake by the mycorrhizal fungus) were Cobb-Douglas type as a function of carbon and phosphorus content of the body. When I measure how a quantity Q depends on a parameter A, a widely used index for the sensitivity is "elasticity," which is defined as follows: $A(\Delta Q)/Q(\Delta A) = \partial \ln Q/\partial \ln A$, which is non-dimensionalized. For Cobb-Douglas production functions, the elasticity of the resource acquisition rate to the amount of resource supplied by the partner is constant at α and β for the plant and fungus, respectively. Cobb-Douglas production functions are commonly assumed in economic theory. The optimal allocation fraction is determined by the importance of the resource that is supplied by the opponent. More specifically, the plant's optimal allocation fraction to the soil fungus is equal to the elasticity of the photosynthetic rate on phosphorus (α), and the optimal allocation fraction of the fungus to the plant is equal to the elasticity of the nutrient acquisition rate on carbon (β). These optimal allocation fractions are independent of the functional form of the resource acquisition rate for the opponent.

4.1. Cooperative optimum and noncooperative equilibrium

Another interesting result is that the solution for the most rapid growth rate for both the

plant and fungus satisfies the ESS condition for each player to maximize its own performance. This implies that there happens to be no conflict of interest. Although the two players have different objective functions, to attain high fitness, they must realize the fastest rate of growth, which is achieved by the same allocation fraction as the cooperative optimum.

4.2. Relationship between the results and past theoretical models

Our model shows that if resource acquisition follows Cobb-Douglas production function, an organism will allocate more resource to the partner when the resource provided by the partner is more important to its survival. This is similar to previous studies that have assumed a stationary state of the plant and mycorrhizal fungus (e.g. De Mazancourt and Schwartz, 2010; Grman *et al*., 2012). Grman *et al*. (2012) demonstrated that the plant allocates more carbon if the soil contains more phosphorus. Although this situation corresponds to a high basic rate of resource acquisition of phosphorus (g_0) in our model, the result is different. According to our analysis, optimally growing plants should allocate the same fraction of photosynthates to the soil fungus regardless of resource acquisition of phosphorus. Whether this difference is caused by the difference in the functional forms of resource acquisition rates or by the contrast between a dynamically growing system versus a stationary system should be determined in future studies.

4.3 Generalization of functional forms

Under these conditions, I can show that the whole system grows exponentially through time. The exponential rate of growth r is the solution of the following equation:

$$\frac{u_1 u_2 \xi_1 \xi_2}{(1-u_1)(1-u_2)} = \varphi^{-1}\left(\frac{r}{1-u_1}\right)\psi^{-1}\left(\frac{r}{1-u_1}\right). \quad (9)$$

See Appendix C for derivation. The optimal allocation ratios of both players that achieve the fastest growth rate can be determined numerically.

4.4. Future extensions

Several extensions of the current model should be explored in future theoretical studies. A plant may obtain phosphorus directly from the soil without help by the fungus. On the other hand, some mycorrhizal fungi, such as ectomycorrhizal fungi, can obtain carbon directly. Then, I could discuss conditions under which the plant-fungus symbiosis is more beneficial to the plant or the fungus than the case without symbiosis.

In this chapter, I assume that a single plant individual interacts with a fungus composed of a single genotype. Given that the hyphae is produced by cell division, this is a plausible assumption. However, sometimes a single plant might interact with multiple strains of fungi, each of which might start from a separate cell that proliferates to produce an individual lineage. The plant may then undergo partner choice by preferentially supplying resources to a strain that provides a highly efficient supply of phosphorus. If this is possible, then the outcome of the plant-fungus interaction might be greatly modified. I can also consider partner choice by the fungus if a single hypha interacts with multiple plant individuals and preferentially supplies phosphorus to an

individual plant that provides a larger supply of carbon. These modifications to the model warrant additional theoretical treatment.

5. APPENDICES

Appendix A : ***Derivation of the optimal allocation for an exponentially growing system.***

From Eqs. (3), I can obtain the following equations:

$$\frac{r}{1-u_1} = f_0\left(\frac{p_{10}}{c_{10}}\right)^{\alpha}, \tag{A.1a}$$

$$\frac{r}{u_2\xi_2} = g_0\left(\frac{c_{20}}{p_{20}}\right)^{\beta}\frac{p_{20}}{p_{10}}, \tag{A.1b}$$

$$\frac{r}{u_1\xi_1} = f_0\left(\frac{p_{10}}{c_{10}}\right)^{\alpha}\frac{c_{10}}{c_{20}}, \tag{A.1c}$$

$$\frac{r}{1-u_2} = g_0\left(\frac{c_{20}}{p_{20}}\right)^{\beta}. \tag{A.1d}$$

From Eqs. (A.1a) and (A.1 c), I can derive

$$c_{20} = \frac{u_1\xi_1}{1-u_1}c_{10}. \tag{A.2a}$$

And from Eqs. (A.1b) and (A.1d), I have

$$p_{20} = \frac{1-u_2}{u_2\xi_2}p_{10} \tag{A.2b}$$

By substituting Eqs. (A.2) into Eq. (A.1d), I can get

$$\begin{aligned}\frac{r}{1-u_2} &= g_0\left(\frac{u_1\xi_1 u_2\xi_2}{(1-u_1)(1-u_2)}\right)^{\beta}\left(\frac{c_{10}}{p_{10}}\right)^{\beta} \\ &= g_0\left(\frac{u_1\xi_1 u_2\xi_2}{(1-u_1)(1-u_2)}\right)^{\beta}\left(\frac{(1-u_1)f_0}{r}\right)^{\frac{\beta}{\alpha}}.\end{aligned} \tag{A.3}$$

where I adopted Eq. (A.1a). Eq. (A.3) is rewritten as Eq. (4) in the main text. Taking the logarithm of both sides of Eq. (4), I have

$$\left(\frac{1}{\beta}+\frac{1}{\alpha}\right)\ln r = \ln u_1 + \frac{1-\alpha}{\alpha}\ln(1-u_1) + \ln u_2 + \frac{1-\beta}{\beta}\ln(1-u_2) + \ln g_0^{\frac{1}{\beta}} f_0^{\frac{1}{\alpha}}\xi_1\xi_2. \tag{A.4}$$

In the optimal carbon allocation, the plant should maximize the growth rate r. To find the optimal carbon allocation of the plant, I calculate the partial derivative of Eq. (A.4) by u_1 and set it equal to zero, giving

$$\left(\frac{1}{\beta}+\frac{1}{\alpha}\right)\frac{\partial \ln r}{\partial u_1}=\frac{1}{u_1}+\frac{1-\alpha}{\alpha}\frac{1}{1-u_1}=0 \quad . \tag{A.5}$$

As $0<u_1<1$, $u_1=\alpha$ is the optimal allocation ratio.

In the same way, I obtain $u_2=\beta$ as the optimal choice.

Appendix B: ***Derivation of optimal resource allocation ratios from singular control conditions in the dynamic optimization problems***

Here, I consider an exponential growth phase of the system in which both players (plant and fungus) allocate some resource to the partner. I show that the condition for an intermediate allocation to form a singular control subarc is satisfied for the optimal resource allocation ratios that maximize the exponential rate of growth of the whole system.

Here, I assume that all four variables are growing exponentially through time, and the ratio of the four variables is independent of time. let $\lambda(t) = \lambda_{C1}(t) = \lambda_{C2}(t)\xi_1$ and $\mu(t) = \lambda_{P1}(t)u_2^{*}\xi_2 + \lambda_{P2}(t)\left(1 - u_2^{*}\right)$. Then, Eqs. (8) become

$$\frac{d\lambda}{dt} = -\lambda(1-\alpha)f_0\left(P_1/C_1\right)^{\alpha}, \tag{B.1a}$$

$$\frac{d\lambda_{P1}}{dt} = -\lambda\alpha f_0\left(C_1/P_1\right)^{1-\alpha}, \tag{B.1b}$$

$$\frac{d\lambda}{dt} = -\xi_1\mu\beta g_0\left(P_2/C_2\right)^{1-\beta}, \tag{B.1c}$$

$$\frac{d\lambda_{P2}}{dt} = -\mu(1-\beta)g_0\left(C_2/P_2\right)^{\beta}, \tag{B.1d}$$

$$\frac{d\mu}{dt} = -u_2^{*}\xi_2\lambda\alpha f_0\left(C_1/P_1\right)^{1-\alpha} - \left(1-u_2^{*}\right)\mu(1-\beta)g_0\left(C_2/P_2\right)^{\beta}. \tag{B.1e}$$

Note that C_1/P_1 and C_2/P_2 are independent of time. By integrating Eq. (B.1a), I have

$$\lambda(t) = \lambda^0\exp[-Rt], \tag{B.2}$$

where $R = (1-\alpha)f_0\left(P_1/C_1\right)^{\alpha}$. λ^0 is the value of $\lambda(t)$ at $t = 0$. Substituting Eq. (B.2) into Eq. (B.1c),

$$\mu = \mu^0 \exp\left[-Rt\right], \tag{B.3}$$

where $\mu^0 = \lambda^0 \dfrac{(1-\alpha) f_0}{\xi_1 \beta g_0} \left(\dfrac{P_1}{C_1}\right)^{\alpha} \left(\dfrac{P_2}{C_2}\right)^{\beta-1}$. Using Eqs. (B.2) and (B.3), I can rewrite Eq. (B.1c) as,

$$\lambda^0 R = \xi_1 \mu^0 \beta g_0 \left(P_2/C_2\right)^{1-\beta}. \tag{B.4}$$

From Eq. (3d), I have $r = \left(1-u_2\right) g_0 \left(c_{20}/p_{20}\right)^{\beta}$, which is rewritten as,

$$\left(\frac{p_{20}}{c_{20}}\right)^{\beta} = \frac{\left(1-u_2\right) g_0}{r}. \tag{B.5}$$

Therefore, I can rewrite Eq. (B.4) as

$$\frac{\lambda^0}{\mu^0} R = \xi_1 \beta \frac{P_2}{C_2} \frac{r}{1-u_2}. \tag{B.6}$$

Here, I rewrite Eq. (B.1e) using Eqs. (B.3), (B.4), and (B.5) as follows,

$$R = u_2^* \xi_2 \alpha \frac{\lambda_0}{\mu_0} \frac{C_1}{P_1} \frac{r}{1-u_1} + r\left(1-\beta\right). \tag{B.7}$$

Using Eq. (B.6), I can rewrite Eq. (B.7) as

$$R = u_2^* \xi_1 \xi_2 \alpha \beta \frac{1}{R} \frac{C_1}{C_2} \frac{P_2}{P_1} \frac{r}{1-u_1} \frac{r}{1-u_2} + r\left(1-\beta\right). \tag{B.8}$$

Using Eqs. (A.2) and Eq. (B.8) becomes

$$R = r\left(\alpha\beta \frac{1}{R} \frac{r}{u_1} + 1 - \beta\right). \tag{B.9}$$

Using Eq. (A.1a) and $R = \left(1-\alpha\right) f_0 \left(P_1/C_1\right)^{\alpha}$, I have

$$\left(1-\alpha\right) f_0 \left(\frac{P_1}{C_1}\right)^{\alpha} = \left(1-u_1\right) f_0 \left(\frac{p_{10}}{c_{10}}\right)^{\alpha} \left(\alpha\beta \frac{1}{\left(1-\alpha\right) f_0 \left(P_1/C_1\right)^{\alpha}} \frac{\left(1-u_1\right) f_0 \left(p_{10}/c_{10}\right)^{\alpha}}{u_1} + 1 - \beta\right). \tag{B.10}$$

This equation leads to

$$1-\alpha=(1-u_1)\left(\alpha\beta\frac{1}{1-\alpha}\frac{1-u_1}{u_1}+1-\beta\right),$$

which leads to $u_1 = \alpha$, which is the same as the optimal rate that maximizes the exponential growth rate of the whole system.

In a similar manner, I can derive $u_2 = \beta$. Thus, the two singular control conditions combined would lead to the optimal allocation ratios maximizing the growth rate of the whole system.

Appendix C: ***Generalized functional forms for resource acquisition rates***

I can generalize the theory. By setting $K = 1/C$, Eq. (2) becomes $f(1, P/C) = (1/C) f(C, P)$, which is rewritten as $f(C, P) = C \bullet \varphi(P/C)$, where $\varphi(P/C) = f(1, P/C)$. Hence, I have

$$f(C_1, P_1) = C_1 \varphi\left(\frac{P_1}{C_1}\right), \tag{C.1a}$$

where $\varphi(x)$ is an increasing function. In a similar manner, I can derive the following form for the rate of phosphorus absorption:

$$g(C_2, P_2) = P_1 \psi\left(\frac{C_2}{P_2}\right). \tag{C.1b}$$

Substituting these functions, Eq. (1) becomes

$$\frac{dC_1}{dt} = (1 - u_1) C_1 \varphi\left(\frac{P_1}{C_1}\right), \tag{C.2a}$$

$$\frac{dP_1}{dt} = u_2 \xi_2 P_1 \psi\left(\frac{C_2}{P_2}\right), \tag{C.2b}$$

$$\frac{dC_2}{dt} = u_1 \xi_1 C_1 \varphi\left(\frac{P_1}{C_1}\right), \tag{C.2c}$$

$$\frac{dP_2}{dt} = (1 - u_2) P_1 \psi\left(\frac{C_2}{P_2}\right). \tag{C.2d}$$

Assuming exponential growth, $C_1 = c_{10} e^{rt}$, $P_1 = p_{10} e^{rt}$, $C_2 = c_{20} e^{rt}$, and $P_2 = p_{20} e^{rt}$, I can rewrite Eqs. (C.2) as

$$r = (1 - u_1) \varphi\left(\frac{p_{10}}{c_{10}}\right), \tag{C.3a}$$

$$r = u_2 \xi_2 \frac{p_{20}}{p_{10}} \psi\left(\frac{c_{20}}{p_{20}}\right), \tag{C.3b}$$

$$r = u_1 \xi_1 \frac{c_{10}}{c_{20}} \varphi\left(\frac{p_{10}}{c_{10}}\right), \tag{C.3c}$$

$$r = (1 - u_2) \psi\left(\frac{c_{20}}{p_{20}}\right). \tag{C.3d}$$

From Eqs. (C.3a) and (C.3d), I have

$$\frac{p_{10}}{c_{10}} = \varphi^{-1}\left(\frac{r}{1-u_1}\right) \quad \text{and} \quad \frac{c_{20}}{p_{20}} = \psi^{-1}\left(\frac{r}{1-u_2}\right), \tag{C.4}$$

which can be written in terms of inverse functions. By multiplying Eqs. (C.3b) and (C.3c), I have

$$r^2 = u_1 u_2 \xi_1 \xi_2 \frac{p_{20}}{p_{10}} \frac{c_{10}}{c_{20}} \psi\left(\frac{c_{20}}{p_{20}}\right) \varphi\left(\frac{p_{10}}{c_{10}}\right). \tag{C.5}$$

Substituting Eq. (C.4) into Eq. (C.5), I have Eq. (9), which specifies r implicitly.

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7. FIGURE CAPTIONS

Fig. 1 Scheme of the model

One plant and one mycorrhizal fungus interact with each other. The plant obtains carbon by photosynthesis and controls the allocation of carbon to the fungus. The fungus takes up phosphorus from the soil and controls the allocation of phosphorus to the plant. By allocating resource, they can help the growth of the partner, and the partner may allocate more resources in the future.

Fig. 2 Functional form of the Cobb-Douglas production function

Contour plots of the Cobb-Douglas production function for photosynthesis; (a) when both carbon and phosphorus are equally important (α = 0.5), (b) when phosphorus is more important than carbon (α = 0.8). The other parameter f_0 = 1.0.

Fig. 3 The optimal reaction of plant and fungus

(a) Contour plot of u_1^*, the optimal allocation ratio by the plant to the fungus. (b) Contour plot of u_2^*, the optimal allocation ratio of the fungus to the plant. The horizontal axis is α, the relative importance of phosphorus to photosynthesis by the plant. The vertical axis is β, the relative importance of carbon for phosphorus uptake by the fungus.

8. FIGURES

Figure 1

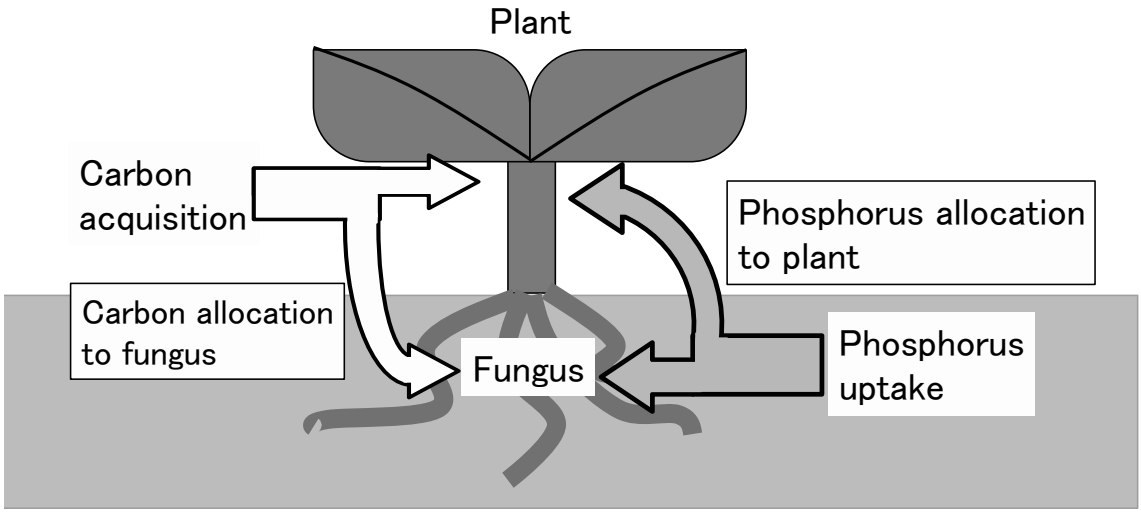
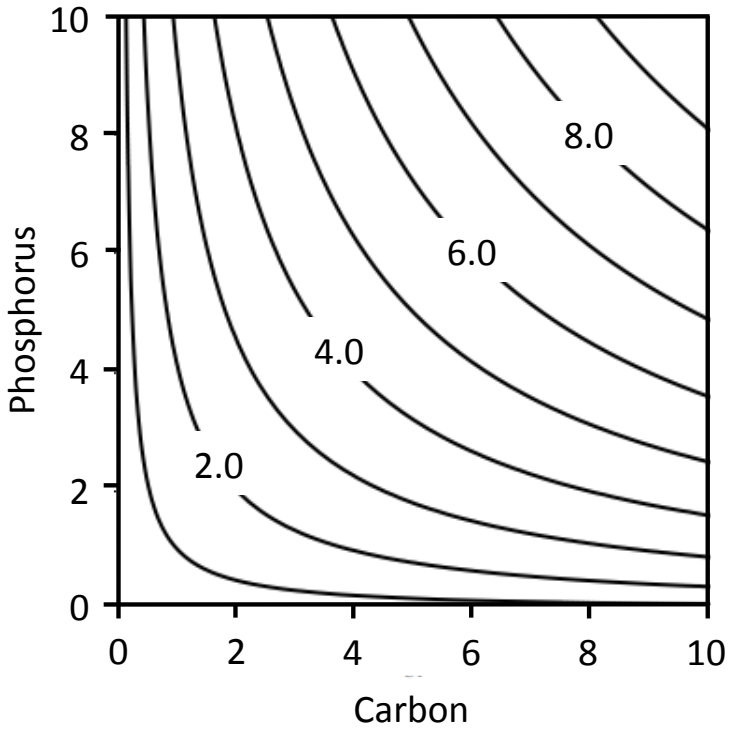


Figure 2

(a)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Carbon

(b)

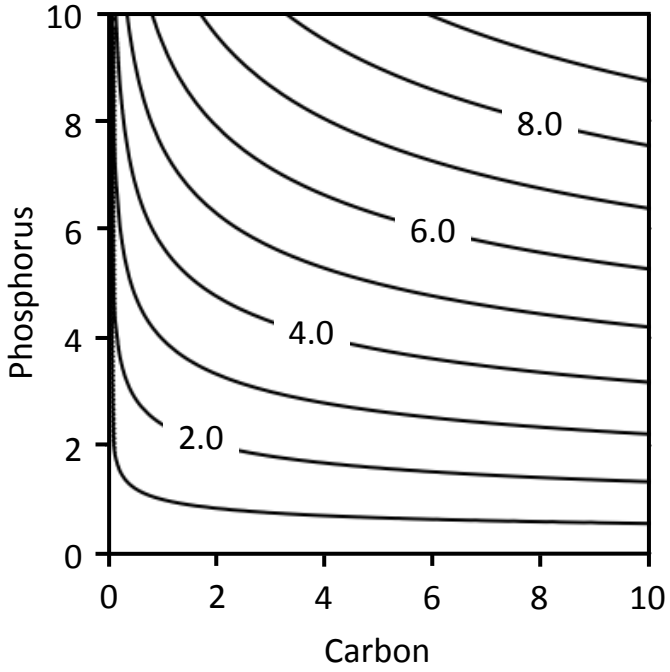
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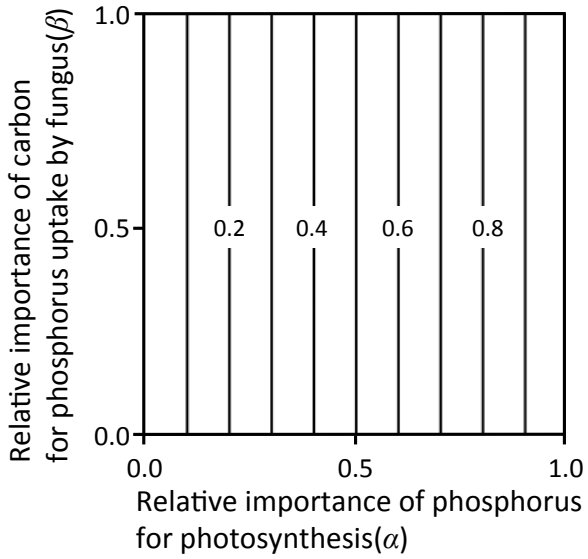
Figure 3

(a)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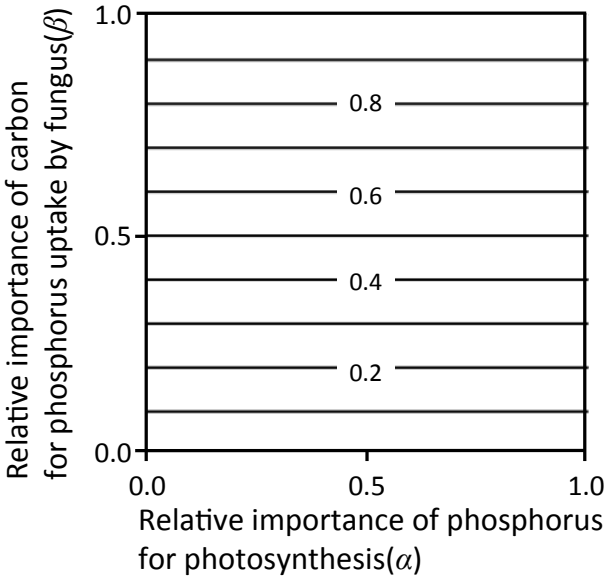
Relative importance of carbon for phosphorus uptake by fungus(β)
Relative importance of phosphorus for photosynthesis(α)

(b)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Relative importance of carbon for phosphorus uptake by fungus(β)
Relative importance of phosphorus for photosynthesis(α)

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