Can optimality models and an ‘optimality research program’ help us understand some plant–fungal relationships?

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**Abstract**

In this paper we suggest that the field of fungal ecology may benefit from the use of optimality models in the context of an ‘optimality research program’ (ORP). An ORP is a research program in the sense of modern philosopher of science Lakatos’ [1978. The Methodology of Scientific Research Programmes: philosophical papers, vol. 1. Cambridge University Press, Cambridge] seminal work. An optimality research program has a lengthy history and record of success in the field of behavioural ecology, but has been seldom employed in fungal ecology. We discuss the ORP and provide some examples of how optimality models may be useful in fungal ecology. We suggest that such an approach may benefit experimental fungal ecologists by: providing a framework for organizing knowledge; generating hypotheses; helping in the planning of experiments; aiding in the interpretation of results; and directing the next steps of an experimental research program. We illustrate these benefits by sketching out how an ORP might be used to answer some fundamental questions about the interactions between host plants and arbuscular mycorrhizal fungi.

\section*{Introduction}

The role of mathematical modelling in ecological research is often misunderstood. Models, particularly very simple models, are commonly seen as armchair exercises by people unwilling or unable to do the heavy lifting required of an experimental research program. Judgments about the usefulness of a model (or lack thereof), however, beg the question: “useful for what?”

Ecologists use models for a variety of different reasons, which are not necessarily mutually exclusive. Appreciating these different reasons for constructing and using mathematical models, and judging the models accordingly, can help to illuminate the utility of particular modelling exercises. Five broad reasons for modelling are to: (1) make predictions; (2) enhance our understanding of a particular biological system; (3) improve experimental design, and hypothesis formation and testing; (4) organize knowledge; and (5) generalize beyond the results of a single experiment. Below we consider these reasons for modelling and provide some examples of their uses in fungal ecology, where possible, and in other fields of ecology if necessary to make our points.

For many non-modellers, making predictions is what modelling is all about. A good model is one that makes accurate predictions and a bad model is one that does not. Making predictions is indeed important, but it is neither the only reason to create models, nor are judgments about model quality, based on accuracy of predictions, straightforward. By way of demonstrating this point, consider the following example. Models that demonstrate ‘deterministic chaos’ are not the same as stochastic models (see Fig 1). In a stochastic model, prediction of future values is limited by the degree of...
that complexity begets accuracy. Bellman (1957) summed up understanding. Missing from a model and this can lead to a new conceptual predict satisfactorily can lead us to consider what might be initial conditions sufficiently? As Aris (1994) pointed out, this a mismatch between model predictions and experimental with which the initial conditions can be estimated. Does process, prediction of future values is limited by the accuracy variability in the random process. However, for a chaotic process, prediction of future values is limited by the accuracy with which the initial conditions can be estimated. Does a mismatch between model predictions and experimental measurements denote a poor model or failure to establish the initial conditions sufficiently? As Aris (1994) pointed out, this raises the question of whether we can ever trust the matching of results with experience?

Often, as much or more can be learned from examining when and where a model fails to predict accurately, as can be learned from a model that often predicts accurately. Statistical models usually predict better than mechanistic models, but the former usually offer little insight into what is happening biologically (Thornley & Johnson 2000). Failure to predict satisfactorily can lead us to consider what might be missing from a model and this can lead to a new conceptual understanding.

Another misconception about model accuracy is the idea that complexity begets accuracy. Bellman (1957) summed up the problem quite succinctly:

“It seems reasonable to suppose that the more realistic this mathematical model, the more accurate the prediction. There is however a point of diminishing returns... If we attempt to include too many features of reality in our mathematical model, we find ourselves engulfed by complicated equations containing unknown parameters and unknown functions. The determination of these functions leads to even more complicated equations with even more unknown parameters and functions, and so on. Truly a tale that knows no end. If, on the other hand, made timid by these prospects, we construct our model in too simple a fashion, we soon find that it does not predict to suit our tastes. It follows that the Scientist, like the Pilgrim, must wend a straight and narrow path between the Pitfalls of Oversimplification and the Morass of Overcomplication. Knowing that no mathematical model can yield a complete description of reality, we must resign ourselves to the task of using a succession of models of greater and greater complexity in our efforts to understand.”

Progress is not always achieved by increasing the complexity of a model. Sometimes progress comes from simplifying a model. As Bellman (1957) pointed out above, greater complexity often comes with more parameters (constants), and it has been said that one can fit an elephant with just five constants (Aris 1994). A model should be as simple as necessary, but not more so (Revett 2007). It can be impressive when a simple model is able to account for a complex phenomenon, as long as the ‘fit’ is not achieved by compromises elsewhere in the model. Certainly one hallmark of a useful model is that its constants refer to real, measurable, quantities.

“Similarly data must be of a certain degree of accuracy or it has no ability to [test] a theoretical viewpoint. Thus the attitude of never believing an experiment until it’s confirmed by theory has as much to be said for it as that which never believes a theory before it is confirmed by experiment” (Aris 1994). Experiments and observations are not infallible. Models can help us to understand how our data could have been generated and give us some confidence that they might actually be correct. The word ‘model’ implies a change of scale, a simplification. We attempt to strip away what we think are irrelevant details of the biology. We try to find some minimal set of mechanisms or circumstances that could produce the phenomenon of interest. Such a model does not tell us how the phenomenon occurs, but how it might occur. The model provides a testable hypothesis that can then be confronted with data. Without the model, all we have is the observed phenomenon. We might have the results of a set of randomized controlled experiments, but still, without a model that presents a plausible mechanism, a reasonable accounting, it is difficult to claim that we understand what is going on.

Mathematical models are not intended to be able to reproduce the world in mathematics (sensu Bellman 1963), but they can often suggest which hypotheses are worth pursuing, and they can help us to design experiments that may be more useful than if there were no theoretical insight. For example, Gange & Ayres (1999) used a very simple graphical model of arbuscular mycorrhizal fungi (AMF)–plant interactions, built on a few reasonable assumptions. They used this model to show clearly that changing AMF density in an experiment can easily lead to: increased, decreased, or no change in plant performance. It is a simple but effective demonstration that interpreting the results of an experiment
can be fraught with difficulty in the presence of non-linear treatment effects. It furthermore demonstrates the futility of conducting an AMP-reduction experiment with only two levels of the treatment.

The process, of formulating a mathematical model, will itself often point out gaps in our knowledge. As experimentalists, how do we identify what we do not yet understand – what should be the next experiment? Of course one way is just to work on something no one else has worked on. Ultimately that strategy may lead to a complete understanding of a system (to the extent that a ‘complete understanding’ is possible in science) but it can be an inefficient process. Attempting to capture mathematically what we do know about a system enforces a kind of rigor in our thinking, and in so doing exposes data that are unavailable but probably necessary for a fuller understanding (e.g. Newman et al. 2003). Many of these models may go unpublished as they serve their purpose in the lab. Nevertheless, some are published and they can help to set the future research agenda.

The optimization research program

The ‘optimization research program’ (ORP) was first formally expressed in the language of philosophy of science by Mitchell & Valone (1990) but has been widely used in ecology since the late 1970s. The optimization approach to the study of evolutionary adaptation is based on the principle that phenotypes, operating under a given set of constraints, are optimal, in that the fitness of the phenotype is locally maximized (e.g. Grafen 1984). The ORP is connected to the use of mathematical modelling techniques from a field called ‘operations research’. Operations research was conceived in World War II with an aim toward maximizing the provision of logistical support to the war effort (e.g. Churchman et al. 1957).

Optimization models generally contain a set of assumptions, either implicit or explicit, about constraints, optimization criteria and (in biology) heredity (Maynard 1978a, b, 1982). The ORP was developed to explicate these ‘essential assumptions’ (Mitchell & Valone 1990) and is based on the concept of the ‘scientific research program’ (SRP) pioneered by contemporary philosophers of science such as Lakatos (1978).

The framework of the ‘Lakatosian’ SRP is based on a series of hierarchical assumptions. These assumptions fall into three categories: (1) hard core assumptions; (2) protective belt assumptions; and (3) methodological assumptions. Hard core assumptions represent the core principles of the SRP, but do not alone generate predictions. Once empirically established, hard core assumptions are generally not subject to further testing, but instead provide the a priori assumptions used to build more specific models. Protective belt assumptions represent a series of ‘auxiliary hypotheses’ that provide a testable means of linking the hard core assumptions to observable data. Methodological assumptions pertain to the reliability of equipment involved in making empirical measurements. Within this framework, refuted predictions do not change the hard core assumptions, but instead call for the generation of new hypotheses or alteration of assumptions within the protective belt.

Methodological assumptions are straightforward and relevant to any research program, and will not be discussed further. Below we first elaborate on the hard core assumptions and then we develop the protective belt assumptions in more detail, since these are the crux of an ORP.

Hard core assumptions

To generate meaningful conclusions from a scientific research program, competing SRPs should be evaluated in light of how refuted predictions are treated (Lakatos 1978). In this way SRPs can be viewed as either ‘progressive’ or ‘degenerate’. The former uses refuted predictions to alter the protective belt hypotheses or assumptions resulting in new testable predictions that ultimately strengthen the hard core. In a degenerative research program, refuted predictions result in untested or untestable ad hoc explanations and these should be abandoned in favour of more progressive programs.

The optimization research program as presented by Mitchell & Valone (1990) addresses changes in the frequency of evolutionary ‘strategies’ over time (or what Grafen 1984 referred to as ‘the phenotypic gambit’; see Maynard 1978a for more discussion). Four central tenets comprise the hard core assumptions of the ORP and can be summarized as: (1) random mutation generates a small frequency of new strategies in each generation; (2) different strategies may have different fitnesses (where a strategy’s change in frequency is a function of its environment); (3) populations of strategies reside near some locally stable equilibrium point and this point represents an evolutionarily stable strategy (ESS); (4) the relative fitness of strategies near a locally stable equilibrium are not qualitatively influenced by the transmission properties of the strategies (i.e. linkage, recombination, epistasis, pleiotropy and drift).

Protective belt assumptions

As we mentioned above, there is a relationship, if often only implied, between the ORP and the use of the mathematics of ‘optimization theory’ (e.g. Chiang 1984; understood even more broadly than the branch of mathematics called ‘operations research’). Within an optimization framework, an individual ‘chooses’ the behaviour or strategy which maximizes an ‘objective function’ subject to a set of constraints, or formally written:

$$\max_x Y(x) \text{ subject to } z_1, z_2, \ldots, z_n$$

where $x$ is the behaviour or strategy, $Y(x)$ is the objective function and the $z_i$ are the constraints under which the individual must operate. The objective function is ultimately the local maximization of fitness (not the global maximization, sensu Wright 1967) and this forms part of the hard core assumptions. Thus, the most interesting insights that can be derived from this model are those that pertain to the ‘behavioural repertoire’ and the constraints on the system, rather than questions related to the maximum value of $Y(x)$. 
Specifically, the model may be used to address the relationship between the choice of behaviour ($x$) and a given set of constraints ($z_n$), and these may form all or a part of the protective belt assumptions.

A direct empirical measure of evolutionary fitness is rarely possible and is particularly intractable for organisms such as an asexually reproducing endophytic fungus, and so another protective belt assumption is needed. This assumption is made in the form of a ‘surrogate measure of fitness’. For example, in foraging theory it is commonly assumed that the animal is maximizing its energy intake per unit time spent foraging. This surrogate then forms part of the hypothesis being tested and may be rejected in favour of more appropriate surrogates, while retaining the hard core assumption that an individual chooses the behaviour which will maximize evolutionary fitness. Thus the program advances by posing and rejecting such models.

For arbuscular mycorrhizal fungi, it has been suggested that an appropriate surrogate for fitness might be the number of spores produced at the end of a season (Bever 2002). But notice that even here, reproductive fitness at the end of a season is not (necessarily) equivalent to lifetime evolutionary fitness. There are also other possibilities. Another surrogate might be to maximize the carbon gained per unit of phosphorous lost to the plant. Such a surrogate supposes that phosphorous is not (always) available in quantities in excess of the fungus’s needs. Still another surrogate might be simply the rate of biomass accumulation. This surrogate supposes that fungi with more biomass are better able to survive in difficult conditions and able to invest more energy in reproduction. Again, the program advances by posing such (protective belt) hypotheses, deriving predictions that follow from them for different environments and conditions, and then testing those predictions.

It is important to note that ‘choice’ may imply conscious choice, but need not do so. More broadly, choice refers only to the particular phenotype displayed by the organism. The hard core assumption is that natural selection is in some sense an optimizing process, subject to developmental, genetic, and environmental constraints. Natural selection does not (necessarily) optimize the global fitness of an organism, only the local fitness (cf. Wright’s (1967) adaptive landscape). The ‘subject to constraints’ caveat is crucial to the entire project. The ORP attempts to (simultaneously) answer two questions: (1) what is the ‘objective function’; and (2) what are the constraints on its maximization? The answer to the first question is only necessary because we must use a surrogate for fitness, otherwise the answer would simply be ‘lifetime evolutionary fitness’ and this would be a hard core assumption.

**Sketching the ORP approach**

So, the ORP is about determining the constraints that operate on how an organism’s phenotype maps onto its lifetime fitness. One might reasonably ask why the program is necessary at all and why is modelling necessary? By formally specifying the problem as a mathematical model we gain two benefits. First, we gain the transparency that models bring. All of our assumptions must be laid bare when we write down our thinking mathematically. Second, we gain precision of prediction. To distinguish among alternative competing hypotheses, we need a means of differentiating the predictions from each hypothesis. Mathematical models can give us precise predictions. Models can also show us where two (or more) alternative hypotheses cannot be distinguished. That is, there will be circumstances in which very different hypotheses yield very similar predictions but for entirely different reasons (e.g. Newman 1991). Models can quantitatively demonstrate this, and so tell us that a particular experiment, conducted under a particular set of conditions, will not be useful for distinguishing among these competing hypotheses (much as we discussed above, albeit in a more general context).

We will try to illustrate these ideas with a more or less explicit example. The relationship between arbuscular mycorrhizal fungi (AMF) and their host plants seems to vary from apparent mutualism to apparent parasitism, depending on the species involved and the environment in which they are found (e.g. Klironomos 2003). One question, the answer to which would help us understand this interaction, is ‘which organism controls the interaction?’ Obviously, there are three possibilities: the plant, the fungus, or the interaction is jointly controlled.

Developing this further, let us consider a stylized model (Fig 2). Let us assume that at the site of the arbuscule–root interface, exchange of carbon and phosphorus is passively controlled by the process of diffusion. In this stylized model, the plant might exercise control over the amount of substrate carbon that it translocates to the roots, and ultimately to the vicinity of the plant–fungus interface. By increasing the concentration of carbon at the site of the interface, the plant boosts the rate of diffusion across the plant–fungus interface. Similarly, the fungus might be able to control the amount of substrate phosphorous that it translocates to the site of the fungus–plant interface, again, boosting the concentration on the fungal side of the interface, and hence boosting the rate of diffusion across the interface.

Now, let us imagine the interaction from the plant’s perspective. Assuming that it has a mechanism to control the translocation of carbon to the site of the interface (which seems reasonable, see particularly Bever et al. 2009) then the ORP tells us that we expect the plant to ‘behave’ so that it maximizes its lifetime evolutionary fitness. Since fitness is often positively correlated with plant biomass, and easier to measure than seed production, we might choose as a surrogate for fitness: ‘maximization of plant biomass’. In cases where plant growth is limited (or co-limited) by phosphorus, and the fungus behaves as a simple ‘diffusion partner’, then the plant should translocate sufficient carbon to the interface to ‘manage’ AMF growth such that the plant obtains the maximum phosphorous benefit for the minimum carbon cost (see Fig 2) subject to the constraints. Constraints might include, but not be limited to: the plant’s rate of photosynthesis; nitrogen availability; biomass investments in reproductive structures; carbon requirements of the plant’s roots; and water availability.

On the other hand, suppose that the fungus can control the translocation of phosphorous to the plant–fungus interface...
and that the plant is a simple ‘diffusion partner’. The fungus should then ‘manage’ the plant’s growth so that the fungus obtains the maximum carbon benefit for the minimum phosphorous cost, subject to the constraints. Constraints might include, but not be limited to: the rate of phosphorous uptake; fungal demand for phosphorous; nitrogen availability; biomass investment in spores or hyphae; and water availability.

Either of these two models would produce testable predictions. They would be of the form: “given conditions \(x, y, z\), the plant should invest \(C\) units of carbon, but given conditions \(x', y', z'\) the plant should invest \(C' \neq C\) units of carbon.” It might be the case that measuring carbon investment is too difficult and instead we might measure only the plant and fungal growth. Some hypothetical examples of predictions are illustrated in Fig 3. Ideally, we would like to find circumstances in which these two models produce contrasting predictions so that we might critically evaluate them. We might, after some early tests, tentatively conclude that some of these constraints are incorrect or unnecessary, and that some constraints need to be added. We might also conclude that the surrogate for fitness is not quite correct. It might be that we have the wrong time scale over which the maximization ought to occur (e.g. not week to week, but year to year). Eventually, we might conclude that one model or the other does not adequately represent the biological situation and hence we might reject one, the other, or both of these models.

Mathematically these models are conceptually straightforward ‘optimal control’ problems. There is a single control variable (the P- or F-valve in Fig 2) that is controlled to maximize the growth of the organism exerting that control (e.g. Hocking 1991).

A third alternative is that neither the plant nor the fungus is a passive ‘diffusion partner’, but that both actively

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**Fig 2** – A stylized model of plant–arbuscular mycorrhizal fungus interaction. The grey boxes represent pools of carbon and phosphorous and the arrows represent directions of transport. Exchange of carbon and phosphorous happens across the interface between the plant and the fungus, and the rate of exchange is proportional to the difference in concentrations on either side of the interface. Plants might be able to influence this rate of exchange by translocating more or less substrate from the shoot to the root (and ultimately to the site of the interface) using what we labelled as the ‘P-valve’. Similarly, the fungus might use an ‘F-valve’ to regulate the rate of exchange of phosphorous substrate.

**Fig 3** – Hypothetical example of predictions that might arise from an analysis of the model implied by Fig 2. These examples assume that the plant controls the interaction; see text for details. (a) The plant biomasses that result from two different strategies, in a continuum of soil phosphorous environments. \(I = 0\) denotes the case where the plant invests nothing in the AMF. \(I = I'\) denotes the case where the plant follows the optimal investment strategy. (b) The fungal biomass for two cases, again in which the plant controls the interaction. \(I' > I\) denotes the P-uptake efficiency of the plant relative to the fungus. So in the case where \(I' > I\) the model might predict that the plant would invest less carbon in the fungus and so the total biomass would be lower. These examples are only illustrative of the kinds of predictions that might arise from a formal analysis of such a model.
control what they can about the interaction. In cases where the benefit to one 'player' depends in part on the behavioural strategy of the other, the mathematical problem becomes one of 'game theory' (e.g. Maynard 1982; Luce & Raiffa 1985).

For the sake of illustration, suppose that the 'game' is played in discrete rounds (e.g. every hour the plant and the fungus 'decide' how much carbon and phosphorus, respectively, to translocate to the plant-fungus interface). Also for illustration suppose that the plant and fungus can 'choose' from a discrete set of quantities. The game can then be represented in matrix form (see Fig 4). The analysis of such a game proceeds along two lines. First we determine the so-called 'Nash equilibrium'. There may be more than one Nash equilibrium, but for a game such as that depicted in Fig 4, there will be at least one Nash equilibrium (Luce & Raiffa 1985). At a Nash equilibrium, neither player can improve their payoff by playing any other strategy, so long as the other player continues to play its Nash equilibrium. In addition to Nash equilibria, we can also look for Pareto equilibria. At a Pareto equilibrium, no player can improve its payoff without reducing the payoff to the opposing player (see e.g. Luce & Raiffa 1985). Pareto equilibria are often referred to as 'cooperative solutions', while Nash equilibria are referred to as 'non-cooperative solutions'.

A natural question to ask in such a case is whether or not the players are 'cooperating'. What looks like cooperation may or may not be cooperation. That is, both players may be acting totally out of self-interest. Newman & Caraco (1989) modelled such a situation, using game theory, in the context of 'food-calling behaviour' in birds. In this behaviour, one individual, upon discovering food, emits a context specific call that attracts conspecifics to the site of the food discovery. This behaviour looks, prima facie, like cooperation or perhaps even altruism (e.g. Axelrod 1984). Newman & Caraco (1989) were able to show the environmental conditions that might lead to this behaviour developing as a form of cooperation, and when it might develop solely as a form of self-interest.

We suggested that, for illustration, we would consider a so-called 'discrete two-player game' (see Fig 4). The game is discrete because it takes place in discrete time and players have repertoires of discrete strategies to choose from. Such games are relatively easy to solve analytically, but may be significantly lacking in realism. We note here that continuous games do exist where players can choose strategies along a continuum, and that decisions are made in continuous time. We note too that there is no mathematical reason that the game must be a two-player game. Mathematical games of N-players are not uncommon, although they are more difficult mathematically (see e.g. Luce & Raiffa 1985). So, our fungus and plant might be viewed in game theoretic terms as a two-player (or more if we consider multiple species of fungi) continuous strategy, continuous time game, where the plant and fungus continuously controls its translocation rate for carbon and phosphorus, respectively.

**A game theoretic model and the ORP**

Let us now put some more realism into the 'toy example' discussed above. A recent study by McGill (2005) implicitly applied the framework of an optimization research program to address the evolution and regulation of the plant–mycorrhiza mutualism. McGill formalized this approach by using game theory to develop a mechanistic model of population dynamics and coevolution of mutualisms. The model is based on von Liebig's (1862) law of the minimum, which states that population growth will be constrained by whatever resource is most limiting. As with any model in an optimization research program, this model operates under a specified set of assumptions whereby the optimal strategy (behaviour or phenotype) chosen by the symbionts changes according to the constraints. The hard core assumptions of the model are implied – that is, that populations of strategies evolve through random mutations and natural selection. Specifically the model examines how the fitness of symbiotic partners (defined here as the fitness surrogate, per capita growth rate) varies depending on the resource availability to both partners. So the hard core assumption here is that plant and fungus each maximize individual evolutionary fitness, while Liebig's law informs the protective belt assumptions whereby fitness is defined in terms of resource availability. Again, the protective belt assumptions are subject to change under the ORP, if per capita growth rate is deemed an inappropriate surrogate of fitness when subject to empirical testing, then it may be changed for some other surrogate.

Fitness itself is difficult to measure empirically, particularly for taxa where it is not obvious how fitness might be defined. While McGill's (2005) model is not parameterized for a particular example, it does provide some good general insights into the possible mechanisms underlying the evolution of mutualism between trading partners. The model predicts that coevolving mutualisms behave much the same way as observed in the evolution of cooperation between conspecifics in that a Prisoner’s dilemma challenge (e.g. Axelrod 1984) arises whereby a ‘cheating’ strategy is always optimal. The model addresses this dilemma by predicting the existence of a mechanism that can overcome the Prisoner’s dilemma challenge and form an evolutionarily stable strategy (ESS; a strategy is an ESS if it cannot be invaded by any other strategy as long as all other members of the population continue to play their ESS; Maynard 1982). This is based on the
idea that some third party, not directly involved in the interaction, acts as an enforcer of fair-trade and prevents cheating. When a policing parameter was incorporated into this model, the result was that fair-trade was enforced and a mutualism evolved. This enforced fair-trade results in the formation of a Nash equilibrium point whereby the strategies of the symbionts are fixed and natural selection alone is sufficient to prevent alternative strategies from successfully invading. McGill (2005) suggests that this policing mechanism may take the form of simple diffusion, particularly in facultative bitrophic systems, where symbiotic partners are in close physical contact and nutrients are transported by diffusion across plant-mycorrhizal tissues. Thus, based on the observed outcome of the model, a hypothesis can be generated about the mechanisms underlying the evolution of a mutualistic interaction based on cooperative nutrient transfer.

Economic models and the ORP

Some of the same ideas explored by McGill (2005) were previously modelled as a ‘comparative advantage scenario’, a concept often used in economics, whereby an individual which produces a resource in excess exchanges this resource with a symbiotic partner for a limiting resource and vice versa (Schwartz & Hoeksema 1998). So-called ‘economic models’ are not uncommon in ecology (e.g. Newman & Elgar 1991). In the context of the present paper, it is worth pointing out that such approaches, although they often lack the explicit language of the ORP, are in fact examples of the ORP in action.

Economic models built on any of the ideas of neoclassical economics can be understood in ORP terms by making a connection between the economic concept of ‘utility’ and the biological concept of ‘fitness’. Neoclassical economics is the science of ‘allocating scarce resources’. So-called ‘efficient allocations’ are those that maximize aggregate utility. Caraco’s (1979a, b, 1981) early work in foraging behaviour offers some good examples of linking the economic idea of a utility function to evolutionary fitness.

Now, returning to the ‘comparative advantage scenario’, Schwartz & Hoeksema (1998) modelled mutualism as a biological analog to trade among nations (or households). Here we offer a brief excursion into the idea of the economics of trade to show that it fits comfortably within an ORP framework. Schwartz & Hoeksema’s model is more complex than our treatment here, but the basic ideas are similar.

First, consider a plant and its need for carbon and phosphorous. The idea in economics is that the plant ought to be willing to trade-off some amount of one element for some amount of the other. Fig 5 demonstrates this idea of a trade-off by plotting so-called ‘indifference curves’. Indifference curves in biology can be understood as curves of equal fitness. That is, the plant’s performance would be the same for any pair of points along the indifference curve. If plants could be said to have such a thing as preferences, they would generally prefer to be in areas of the C-P space that are farther from the origin than closer to the origin (i.e. they prefer more of both C and P to less of both). Starting from any point in the C-P space, there will be some indifference curve; the plant should be indifferent to any resource allocation along this curve. The specific shape of the individual curves will probably vary according to the distance of the curve from the origin (and in extreme cases this concept may well be thought of in terms of Liebig’s law of the minimum), but in general represent the idea that the plant
might be ‘willing’ to trade carbon for phosphorous at some specific rate of substitution.

If the fungus also has a set of indifference curves, these will probably be different from the plant’s indifference curves. To understand trade, suppose that the amount of C and P available in total is fixed by the maximum rate of photosynthesis on the one hand, and the soil P availability on the other. An economic analysis of this situation might employ an ‘Edgeworth Box Diagram’ (see e.g. Gowdy & O’Hara 1995). This box is constructed by taking the indifference curve diagram for the plant (Fig 5a) and a similar diagram for the fungus and rotating one of these diagrams to combine it with the other (Fig 5b). There will then be a curve called the ‘contract curve’ in this box defined by the set of points were individual indifference curves are just tangent to each other in the Edgeworth Box space. In Fig 5b we have drawn a hypothetical curve for the purposes of illustration.

Suppose we are at point ‘A’ in Fig 5b, the plant has much more carbon than the fungus and the fungus has much more phosphorous (arbitrary units and numbers chosen for illustrative purposes). In this case, the plant should be indifferent between any point along this particular indifference curve (i.e. its performance should be the same at point A as it is at point C). However, the fungus would do absolutely better at point C than for any point along its indifference curve. So although the fungus would do better at point C than point A, the plant would perform the same at either point. Similarly, the fungus should be indifferent between points A and B because they both fall along the fungus’ indifference curve. However, the plant would do absolutely better at point B than either points A or C.

We complete our analysis of this example by noting that the plant and the fungus could ‘trade’ carbon and phosphorous in such a way that they move from point A to some point along the contract curve between points B and C (inclusive). On the contract curve, the fungus and the plant achieve a Pareto optimal solution such that no further trading can make one ‘partner’ better off without making the other partner worse off. All points along the contract curve are ‘Pareto optimal’ (see e.g. Gowdy & O’Hara 1995). How the plant and fungus move from point A to the contract curve depends upon the rates of trade. The plant would prefer to trade carbon for phosphorous at a rate determined by the slope of the line R1, while the fungus would prefer to trade phosphorous for carbon at a rate determined by the slope of R3. Any rate of trade between R1 and R3 is also acceptable, with R2 representing a ‘fair’ solution (i.e. both partners split the gains to fitness from trading, equally between them). Which rate of trade ultimately emerges depends on the conditions under which trade takes place. Just like trade between nations, one side may have an advantage and be in a position to influence strongly the conditions under which trade may occur, and this comes back to our toy example in the previous section about which partner exerts control over the interaction.

Hopefully, this second toy example makes clear that whether one couches the plant–fungus interaction in terms of the economics of trade, or in the language of optimality theory, it is clear that both are simply examples of the application of an optimality research program.

Conclusions

The optimality research program has enjoyed broad success in the field of behavioural ecology over the past 30 years (Ydenberg et al. 2007). Such a research program is likely to be equally useful in fungal ecology. To apply such a program, researchers would need to think in evolutionary terms (something clearly already done) and to learn some of the formal mathematics of optimality theory. By using mathematical models, as shaped by the ORP, researchers may gain any or all of the advantages discussed in the Introduction. Such an approach takes some training but is well within the grasp of most modern ecologists due to the increasingly quantitative nature of our discipline.

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