

Optimality explanations: a plea for an alternative approach

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Abstract Recently philosophers of science have begun to pay more attention to the use of highly idealized mathematical models in scientific theorizing. An important example of this kind of highly idealized modeling is the widespread use of optimality models within evolutionary biology. One way to understand the explanations provided by these models is as a censored causal explanation: an explanation that omits certain causal factors in order to focus on a modular subset of the causal processes that led to the explanandum. In this paper, I first argue that the censored causal model approach fails to establish a permanent explanatory role for optimality models in biology and mischaracterizes the explanatory virtues of biological optimality modeling. In addition, I argue that many biological optimality explanations cannot be characterized as censored causal explanations. In response, I propose an alternative approach that analyzes optimality models' reliance on synchronically representing a system's constraints and tradeoffs as well as their employment of various kinds of idealization in order to provide equilibrium explanations.

Keywords Explanation · Optimality · Idealization · Tradeoffs · Models

Introduction

Optimality models are widely used within biology to investigate adaptations by representing the evolution of a particular phenotype as a function of the relative fitnesses of a set of possible trait values. For example, optimality models have been used to study organisms' foraging behaviors (Pyke 1984; Stephens and Krebs 1986). A simple optimality model explains why, for instance, when two kinds of food are

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readily available and require the same amount of time to process, organisms choose the one that maximizes energy intake (since energy intake is assumed to be directly correlated with fitness). This explanation makes no direct reference to the step-by-step evolution that leads to this preference—analysis of the optimality model only identifies a particular trait value as the fittest (i.e. locally optimal) of the available phenotypes given the constraints of the foraging problem. The optimality explanation then assumes that this locally optimal strategy is the equilibrium point of the evolving population.¹

Optimality models have long been a source of controversy within evolutionary biology. Some have argued that although they are useful given that we currently know little about genetic dynamics, they ought to be phased out as we obtain information about genetic systems (Lewontin 1979). This, of course, means that optimality models' place within evolutionary biology is only provisional—they are pragmatically motivated short cuts. Others have argued that optimality models are important for determining whether or not particular kinds of adaptationism are true (Orzack and Sober 1994, 1996).² This role, however, is also temporary; once adaptationism is tested this use of optimality models will no longer be necessary. In addition, few (if any) evolutionary biologists actually use optimality models in order to test adaptationism in this way.

Recently the philosophical literature on optimality models has shifted its focus away from their roles in the confirmation of adaptationist hypotheses, towards their ability to provide a unique kind of explanation of evolutionary phenomena. One approach to understanding the explanations of biological optimality models is as a kind of “censored causal explanation” (Elgin and Sober 2002; Orzack and Sober 1994, 1996; Potochnik 2007, 2010). A censored causal explanation is an explanation that purposely omits certain causal factors in order to focus on a modular part of the causal process that led to the explanandum. For example, Steven Orzack and Elliott Sober employ this terminology when they claim that optimality models are “censored” models, “in which the only evolutionary force is natural selection” (Orzack and Sober 1994, p. 363). Following this approach, Angela Potochnik has recently argued that their ability to provide a kind of censored causal explanation secures optimality models a permanent explanatory role within population biology (Potochnik 2007, 2010). I will call this general approach the *censored causal model approach* to understanding the explanations of biological optimality models.

In this paper, I first argue that the censored causal model approach is unable to establish a permanent explanatory role for optimality models within biology and mischaracterizes the nature of the explanations provided by biological optimality models. In addition, I argue that many biological optimality explanations are not censored causal explanations. In response, I propose an alternative approach to

¹ I will refer to any explanation that makes essential use of an optimality model as an optimality explanation. The nature of the relationship between models and the explanations they can provide, or can aid in providing, will have to be discussed elsewhere.

² Optimality modeling is also central to long-standing debates concerning the adaptationist approach (Gould and Lewontin 1979), Sociobiology, (Kitcher 1985), and evolutionary psychology. Their importance to these debates highlights the need for a better account of how optimality models provide scientific explanations.

understanding the explanations provided by biological optimality models—one that analyzes optimality models' reliance on synchronically representing a system's constraints and tradeoffs as well as their essential use of various kinds of idealization in order to provide equilibrium explanations.

In the following section I present a widely cited example of a biological optimality explanation. Next, I present the censored causal model approach. Then, I argue that the censored causal model approach is unable to establish a permanent explanatory role for optimality models in biology and mischaracterizes the explanatory virtues provided by biological optimality models. The final section argues that many biological optimality explanations cannot be characterized as censored causal explanations and proposes an alternative approach to understanding how optimality models explain biological phenomena.

An example of a biological optimality explanation

Biological optimality models aim to represent the available phenotypic strategies along with the constraints and tradeoffs involved in the selection of a trait. Once these components of the model are specified, the optimality modeler can deduce which of the available strategies will optimize the criterion of the model. Ideally, in biological contexts, this criterion will be fitness (or inclusive fitness), but in most cases a more easily measured proxy is used—e.g. average energy intake. Biological optimality models usually assume that natural selection will move the population towards (and maintain) the strategy, or mix of strategies, that optimizes the model's criterion. In addition, these models commonly assume that the selection pressures within the model will be able to overcome any other factors influencing the evolution of the trait; e.g. drift. These assumptions entail that the strategy that optimizes the criterion of the model is the equilibrium point of the evolving population.³

For example, G. A. Parker utilized an optimality model in his attempts to explain why dung flies (*Scatophaga stercoraria*) copulate for 36 min on average (Parker 1978). First, Parker observed that female dung flies mate with multiple males. He then discovered, by experimentation, that when this occurs the second male fertilizes far more eggs (80 %) than the first (20 %). Consequently, after copulating with a female, a male dung fly spends some time guarding her before flying off in search of other mates. The total behavioral cycle time is given by summing search time, copulation time and guard time. Parker then observed that the average time spent searching plus guarding was 156 min. Therefore the total cycle will last $156 + c$ min, where c is the amount of time spent copulating. In Parker's model, the x -axis represents the total time expenditure on all three tasks, and the y -axis represents the (average) number of eggs fertilized (Fig. 1 below).

By experiment, Parker found that increasing the copulation time increases the average number of eggs fertilized. However, there is an important tradeoff: time

³ Optimality modeling also includes game-theoretic models. Game-theoretic techniques are utilized when the trait depends on the strategies being played by other members in the population.

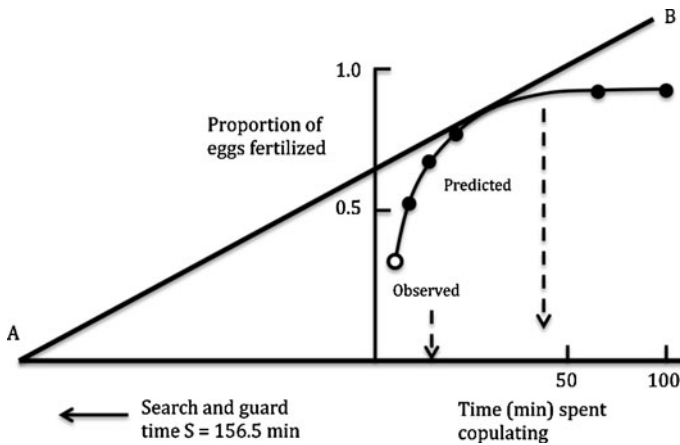


Fig. 1 (Parker's optimality model used to investigate the copulation time of dung flies (Parker and Stuart 1976). © 1976 by The University of Chicago. All rights reserved

spent copulating with one female is time lost searching for other mates. In addition, Parker observed diminishing returns on time spent copulating—that is, additional copulation time brings smaller and smaller increases in the number of eggs fertilized. Parker's observations are captured by the mathematical curve within the optimality model that represents average fertilization as a function of copulation time.

According to Parker's optimization criterion, the optimal value for c is the value that *maximizes the rate of eggs fertilized* across several iterations of this behavioral cycle. This optimal strategy occurs at the point where a line that passes through the origin and intersects the asymptotic curve with the steepest slope (line A–B above) intersects the curve. This optimal point occurs when c is equal to 41 min, which is fairly close to the observed value of 36 min. Given this predictive accuracy, and the fact that the parameters of Parker's model were based on detailed empirical observations, the model is often thought to have captured the major constraints and tradeoffs that were involved in how natural selection shaped this behavioral trait value (Sober 2000).

However, in order for this optimality model to provide an explanation requires some additional assumptions to show how this locally optimal strategy is related to the behavioral trait we observe in the real-world population. First, the explanation requires the assumption that natural selection will optimize the criterion of the model. In this case, Parker's model assumes that natural selection will maximize the rate of eggs fertilized. As a result, behavioral strategies with higher fertilization rates (on average) should increase in frequency within the population (since they will have higher fitness). In addition, the optimality explanation utilizes various idealizing assumptions, such as assuming that the population being modeled is infinite (thereby eliminating drift), that mating within the population is random, and that organisms within the population reproduce asexually such that offspring's copulation strategies are identical to their parents'. These idealizations are important to the optimality explanation because it is assumed that other evolutionary factors

would not be able to deter the population from reaching the optimal strategy favored by natural selection. These assumptions entail that the strategy that optimizes the criterion of the model is the equilibrium point of the evolving population. Consequently, Parker's optimality model may be used to give an equilibrium explanation for why dung flies copulate for approximately 36 min on average.

The censored causal model approach

The censored causal model approach contrasts biological optimality models with more comprehensive models that include additional causal factors; e.g. models that also represent genetic and epigenetic causes (Elgin and Sober 2002; Orzack and Sober 1994, 1996; Potochnik 2007, 2010). This approach maintains that optimality models' exclusive focus on the causal processes of natural selection and the omission of other causal factors is what allows them to be a valuable explanatory tool within biology.

The censored causal model approach characterizes the explanations provided by biological optimality models as causal explanations that ignore certain parts of the causal process that led to an evolutionary outcome (e.g. genetic causes) and emphasizes a modular part of that larger causal process—namely how natural selection shaped the trait in question. In other words, optimality models purposely ignore, or black box, the causal processes of genetic, epigenetic, and other evolutionary factors in favor of focusing on how the causal processes of natural selection influence the trait values in the population. For example, according to Potochnik, “An optimality model focuses on a particular modular part of the causal process leading to the observed phenotype” (Potochnik 2007, p. 688). This causal process is modular in the sense that it is dissociable from the other causal factors involved in a trait's evolution. Similarly, Orzack and Sober characterize optimality models as “censored” models, in which natural selection is the only evolutionary force represented in the model (Orzack and Sober, 1994, p. 363). According to the censored causal model approach, an optimality model provides a special kind of causal explanation by representing this dissociable component of the causal process that led to a phenotypic trait and ignoring other causal factors.

In order to demonstrate that these censored causal models (i.e. optimality models) are able to provide *adequate* explanations of phenotypic traits, the censored causal model approach appeals to the predictive accuracy of an optimality model when compared with a model that includes additional causal factors. For example, Orzack and Sober (1994) appeal to the predictive accuracy of an optimality model in order to establish that the process of natural selection (presumably the one described by the optimality model in question) is a satisfactory explanation of a phenotypic trait. They define the concept of a sufficient (i.e. adequate) explanation by reference to the predictions of a “censored” causal model; i.e. an optimality model in which the only evolutionary force is natural selection. They argue that if the predictions of the optimality model fit the observations according to standard statistical criteria, then natural selection can be regarded as a sufficient explanation of the evolution of the trait. They claim, “Natural selection here provides a sufficient explanation because taking other factors into account could not

significantly enhance the predictive accuracy of the optimality model” (Orzack and Sober 1994, p. 363).

This requirement for an optimality model to provide an adequate explanation is paralleled by Potochnik’s account. For Potochnik, the context of inquiry will determine what causal factors must be included, as well as the causal factors that should be left out of, the best explanation.⁴ According to her account, the best explanation of an event E is a model that:

1. represents the causes of E that figure into the causal relationship of interest in the particular context of inquiry at hand;
2. satisfies the criteria of explanatory adequacy; and
3. is maximally general within these constraints, where the generality that matters is the number of possible systems the model applies to (Potochnik 2007, pp. 683–685).

In order for an explanation to qualify as adequate on this account (i.e. condition two above) it must meet the following criteria:

1. $\Pr(E|C_{\text{expl}}) \approx \Pr(E|C)$
2. $\Pr(E|C_{\text{expl}}) \approx \Pr(E|C_{\text{expl}} \wedge C_k)$ for all C_k (Potochnik 2007, p. 684)

where C_{expl} is the set of causes cited in the candidate explanation of event E, C is the set of all causal factors that influence E, and C_k represents each event that is a causal influence on E. In short, an adequate explanation should not omit any causal factors that, if included, would drastically change the expected probability of the event to be explained.

Given that optimality models are often able to meet this criterion for being adequate explanations, Potochnik’s account goes on to argue that optimality models will often be *the best explanation available* because they provide a focused censored causal explanation that is *more general* than the explanation that would be provided by a model that included additional causal factors (Potochnik 2007, 2010). According to Potochnik, adding extra causal information to the censored causal explanation of an optimality model sometimes makes for a worse explanation because, “An optimality model highlights a modular part of the causal process that grounds certain phenotypic generalizations. Incorporating information about other parts of the causal process can only obfuscate the relationship between the instance to be explained and other, relevantly similar instances” (Potochnik 2007, p. 688). For Potochnik, optimality models highlight how the event *falls into a pattern of cause and effect*, which is independent of the causal factors omitted from the optimality model (Potochnik 2010, pp. 221–223). In other words, the censored causal explanations of optimality models highlight the causal processes of interest,

⁴ Furthermore, Potochnik argues, in some cases causal factors that are *causally relevant* to the occurrence of the explanandum (e.g. genetic causes) ought to be omitted from the best explanation of the event. This distinguishes her account from that of, say, Woodward and Strevens (Strevens 2009; Woodward 2003). For those authors, causes that make a difference to the explanandum must be included in the best explanation of an event. Potochnik’s account goes further in suggesting that even for causes that are required for the event to occur, the context of inquiry may dictate that they should be left out of the best explanation.

and are *more general* (in that they apply to more possible systems) than the explanations provided by models that include additional causal factors (Potochnik 2007, p. 687).

The main context in which Potochnik believes biological optimality models will often provide the best explanation is whenever traits *are the products of long-term evolution by natural selection*. In support of this claim, she again appeals to the predictive accuracy of optimality models by citing the results of Eschel and Feldman (Potochnik 2007, p. 687; 2010, p. 217; Eschel and Feldman 2001). What Eschel and Feldman demonstrate is that, “Phenotypic changes, when determined by long-term genetic dynamics, even with a multilocus genetic structure including recombination, tend to converge in the long term and, with probability 1, to local optima” (Eschel and Feldman 2001, p. 183). That is, over the long term, the results of dynamical genetic evolution by natural selection tend to converge to the predictions made by optimality models—either a local optimum or an evolutionarily stable strategy (ESS)—regardless of the kind of genetic system involved. Since more inclusive models would not greatly improve our predictive accuracy in this context (and we are interested in how natural selection influenced the trait in question), Potochnik concludes that optimality models will often meet her criteria for being the best explanation for traits that are the result of long-term selection. As a result, she argues, “the optimality approach has a long-term role assured in evolutionary study” (Potochnik 2007, p. 690).

Two problems for the censored causal model approach

In this section, I argue that the censored causal model approach is unable to establish a permanent explanatory role for optimality models within biology and mischaracterizes the explanatory virtues of biological optimality models. First, appealing to optimality models’ predictive accuracy (e.g. for traits that result from long-term selection) is insufficient to establish that they will be able to provide an adequate explanation. Second, a preference for explanatory generality cannot establish a preference for optimality explanations as they are characterized by the censored causal model approach, since models that represent fewer or different causal factors are not necessarily more general than models that include additional causal factors.

Predictive accuracy cannot establish that an explanation is adequate

To begin, arguments that appeal to the predictive accuracy of optimality models in order to establish that they will be able to provide an adequate explanation are unsuccessful. For example, Potochnik appeals to Eschel and Feldman’s demonstration of the predictive accuracy of optimality models whenever traits are the result of long-term evolution by natural selection in order to support her claim that optimality models will often be the best explanations in that context. In similar fashion, Orzack and Sober appeal to the predictive accuracy of an optimality model in order to establish that the process of natural selection is a sufficient explanation for the phenotypic trait in question. This reasoning follows directly from the censored

causal model approach's criteria of explanatory adequacy, which only requires that the censored causal model make the explanandum as probable as other models that include additional causal factors.

The problem, however, is that this requirement is too weak to establish that optimality models will be able to provide adequate explanations of phenotypic traits. Predictive accuracy of an optimality model is unable, on its own, to establish that the optimality model in question is an adequate explanation for the explanandum predicted.⁵ More generally, just because optimality models are predictively accurate for a class of phenomenon does not entail that an optimality model will ever provide an adequate explanation for *any* of those phenomena.

One and the same model can be understood with different scientific goals in mind. Under one construal, a model might be a mere predictive device, in which no claim of representational accuracy concerning the system's dynamics is made. According to another construal, the same model may be claimed to provide an accurate explanation about *why* the event occurred—even if its predictions are somewhat inaccurate (Godfrey-Smith 2006, p. 733). To be predictively accurate only one thing is required: the model's assumptions must entail the correct prediction(s).⁶ However, mere predictive accuracy is insufficient for establishing that the explanation being offered by a particular model is adequate.

The main problem is that predictive accuracy alone is unable to confirm whether any part of the explanans represented by the model is *true* of the target system.⁷ We require independent reasons for accepting the hypothesis represented within a particular optimality model before granting it explanatory adequacy. That an optimality model makes the correct predictions is unable, on its own, to establish any particular level of representational fidelity between the explanans represented within the optimality model and the real-world system that led to the evolutionary outcome. Although predictive accuracy (to some degree) is likely a *necessary* condition for a model to be an adequate explanation, it is not *sufficient*. In order for an optimality model to be an adequate explanation of some evolutionary phenomena—and thereby a candidate to be the best explanation—the model will also need to accurately represent features of the target system that are able to explain why the explanandum occurred.

What is more, how much representational fidelity is required to claim that a model “applies” to, or “accurately represents”, a target system will depend on the aims of the model builder (Matthewson and Weisberg 2009; Weisberg 2007). For modelers only interested in constructing predictively accurate models the level of representational fidelity required for success will be quite low.⁸ Some scientists will,

⁵ This point holds for models more generally. Predictive accuracy is unable to establish that the explanation provided by the model is adequate.

⁶ Meteorology is often a good example of this sort of modeling. Accurate predictions are paramount; explanatory accuracy is largely an afterthought.

⁷ For one thing, predictive accuracy makes a particular model no more likely than any other predictively accurate model.

⁸ Still others may be focused only on providing how-possibly explanations, say of how the evolution of altruism is possible. Here again, relatively low standards of representational fidelity are likely to be required.

however, be interested in having their model provide more detailed (how-actually) explanations of evolutionary phenomena and so are likely to require higher representational fidelity between the explanans of their model and the target system than in other contexts. Across all contexts, however, the fact remains that more than predictive accuracy is required to demonstrate that the explanans offered by a particular model is able to meet the standards of representational fidelity required to provide an adequate explanation.

In sum, the abilities of optimality models to predict the results of long-term evolution by natural selection are not the same as their abilities to explain those results. Accurately predicting a product (e.g. that a trait is locally optimal) is importantly different from adequately *explaining* why a population arrived at that product.⁹ To her credit, Potochnik does mention this distinction in a footnote when she says, “Of course, in order for an evolutionary outcome to be explained by an optimality model, the model in question must accurately represent the dynamics that led to the outcome” (Potochnik 2010, p. 217). Yet in both of her papers she goes on to argue that, given the results of Eshel and Feldman, optimality models will often be the best explanation of traits that evolve by long-term selection. However, Eshel and Feldman’s derivations showing that long-term evolution will converge to the predictions of optimality models are insufficient for establishing this claim. So although optimality models might meet the censored causal model approach’s criteria in this context, in order to establish this context as one in which optimality models will often be adequate explanations we require independent reasons for thinking that the dynamic relationships represented within particular optimality models are those that were present in the evolving system(s) under consideration. But this support will have to be provided on a case-by-case basis and will depend on changing standards of representational fidelity.

Consequently, the censored causal model approach’s appeals to optimality models’ predictive accuracy are unable to establish that optimality models will often (or ever) be adequate explanations. Therefore, more will have to be said in order to establish that optimality models will often provide the *best* explanation in any particular context. As a result, the censored causal model approach fails to secure a permanent explanatory role for optimality models within evolutionary biology.

Detail versus generality: are all optimality explanations more general explanations?

In discussing the generality of models it is important to distinguish between two kinds of generality. I will use an increase in *a-generality* to refer to a model’s ability to apply to a larger number of *actual* systems. Alternatively, a model will be more *p-general* if it applies to more *possible* systems.¹⁰ Potochnik’s account argues that

⁹ Brandon and Rausher (1996) make a similar complaint about Orzack and Sober’s (1994) claim that if a trait is locally optimal then natural selection can be regarded as a sufficient explanation of the trait in question. They object that Orzack and Sober have conflated a claim about product with a claim about process.

¹⁰ The terms “a-generality” and “p-generality” are borrowed from work by Michael Weisberg (Matthewson and Weisberg 2009; Weisberg 2003).

optimality models will often be preferred to models that include additional factors due to the p-generality yielded by their causal exclusions. What is problematic for the censored causal model approach is that including fewer or different causal factors in a model—which that approach maintains is what distinguishes optimality models from other evolutionary models—does not necessarily increase p-generality. Therefore, p-generality is not necessarily provided by optimality explanations as they are characterized by the censored causal model approach.

A highly detailed model may apply to fewer possible systems than a more abstract model that incorporates additional evolutionary factors. This is because p-generality does not trade-off with the number or type of causal factors included in the model, but with the specificity of the parameters of the model (Matthewson and Weisberg 2009, pp. 183–184). One way to potentially reduce a model's specificity is to remove parameters, but it is not the only way. What makes a model an optimality model, however, has nothing to do with the precision with which its parameters are specified. Instead, optimality models are distinguished by the kinds of information they leave out—e.g. according to the censored causal model approach there are no parameters in the model for genetic or epigenetic causes. However, having fewer or different parameters (or causal factors) does not entail that one model will apply to more possible systems than another model with more or different parameters (or causal factors). What matters for p-generality is *how detailed the model's specifications of the included parameters are*; i.e. how variable the included parameters are allowed to be. In the end, models that provide highly detailed descriptions of a few factors (i.e. their parameters are very precisely specified) may apply to fewer possible systems than a model that describes more factors but with less detail (i.e. its parameters are specified imprecisely). Therefore, whether an optimality model is more p-general than its more inclusive counterparts will have to be determined on a case-by-case basis. Optimality models understood as censored causal explanations are not necessarily more p-general.

In response, one might suggest that an optimality explanation is superior because it necessarily applies to more *actual* systems (Kitcher 1981). However, understanding optimality models as censored causal explanations does not entail that they will necessarily be more a-general either. The reason for this is that a-generality likewise does not trade-off with the *number* or *type* of causal factors included in a model, but again with the level of precision with which the parameters of the model are specified (Matthewson and Weisberg 2009, pp. 184–185). As with p-generality, sometimes models with only a few parameters that are very precisely specified will apply to fewer actual systems than imprecise models with additional parameters.¹¹ Consequently, if optimality models are to be distinguished by their representation of a modular part of a causal process and their omission of other causal factors, then the explanations offered by optimality models will not necessarily be more a-general than those offered by more inclusive evolutionary models. Very precise

¹¹ A-generality will also depend on how much heterogeneity is present among the proposed target systems. If the proposed target systems are largely homogenous, then specifying the model's parameters more precisely will have less of an impact on it's a-generality than if the proposed target systems are largely heterogeneous with respect to the included factors.

censored causal models may be far less a-general than imprecise models that incorporate additional causal factors.

In sum, censored causal models will not always be more general than models that include additional causal factors. This is because neither kind of explanatory generality exhibits a trade-off with the number or type of causal factors included in a model, but depends on the precision with which the model's parameters are specified. Therefore, we cannot conclude that if an optimality model represents *fewer or different* parts of a causal process then its explanations will consequently be able to apply to more real or possible systems.

The problem this raises for the censored causal model approach is that if not all optimality models provide more general explanations than more inclusive causal models then it is unclear whether they provide any explanatory virtues *solely in virtue of being optimality models*. However, if greater explanatory generality is not a necessary feature of optimality models then they are no more likely to be the best explanation available than other more inclusive models. Put differently, neither precision nor generality are essential features of optimality explanations as they are characterized by the censored causal model approach. Therefore, our preference for these explanatory virtues cannot establish a preference *for optimality explanations*—at least not if they are understood as censored causal explanations.

Suggestions for an alternative approach to optimality explanations

The previous section raised two objections to the censored causal model approach. In this final section, I argue that not all biological optimality explanations can be characterized as censored causal explanations. In response, I propose an alternative approach to understanding the explanations of biological optimality models—one that analyzes their reliance on synchronically representing constraints and tradeoffs as well as their essential use of various kinds of idealization to provide equilibrium explanations.

Optimality models do not represent a modular part of the causal evolutionary process

It is widely agreed that optimality models are distinguished by what they leave out. In this sense they are certainly a kind of “censored” model. One suggestion, then, might be that optimality models are simply models that censor information concerning various causal processes of genetic or epigenetic *biological* evolution. Yet optimality models are used in disciplines besides biology; e.g. economics. Therefore, what distinguishes optimality models from other kinds of evolutionary models is presumably something more generally applicable.

Perhaps, then, it is something about the mathematical techniques involved that distinguishes optimality models from other evolutionary models. Optimality models are distinguished by their use of *Optimization Theory*, which is applicable across varied scientific domains. In its most general form, Optimization Theory is just a mathematical technique that can be used to determine what values of some control

variable(s) will—given a set of tradeoffs and constraints—optimize the value of some design variable(s) (Beatty 1980; Maynard Smith 1978; Seger and Stubblefield 1996).

For example, if an engineer wants to construct a bridge, she may wish to optimize various design features; e.g. weight, cost, rigidity, width, etc. Not all of these can be optimized simultaneously however; certain tradeoffs (e.g. more width will mean more weight) and context-specific limitations (e.g. limited funds) will constrain the optimal design. One way to solve this design problem is to construct and analyze an optimality model in order to deduce the set of control variables that will result in the optimization of the design variables. An optimality model describes a function, which relates each possible set of control variables (i.e. the strategies) to values of the design variable(s) to be optimized. This function and the set of available strategies are determined by the constraints and tradeoffs of the particular design problem. Once the strategy set and objective function of the optimality model are specified, one can deduce which of the available strategies will yield the optimal values of the design variables (or the single currency on to which the various design variables are mapped). In sum, optimality models identify key *constraints and tradeoffs* that hold within a system and then utilize those constraints and tradeoffs to determine the locally optimal (i.e. best available) solution to the design problem. When studying evolutionary systems, this locally optimal solution(s) is then usually understood as the equilibrium state(s) of the evolving system.

This mathematical technique is replicated in biological optimality models as well. For instance, Parker and Maynard Smith describe a simple optimality model for a foraging lapwing population (Parker and Maynard Smith 1990).¹² Lapwings search for food by moving a few paces before pausing to look for their prey (insects), which they eat if they find. The available strategies in this case are the possible distances traveled in between each scan. The farther the lapwing moves, the less likely it will be scanning terrain already inspected (and thus more likely to find prey). However, once it has moved a distance equal to the diameter of its visual field, moving farther does not help since all of the new ground will already be outside of the previously inspected area. Furthermore, although moving increases the chances of obtaining prey, each step of movement *costs energy*. In the optimality model, curve B represents the benefit of adopting strategy x and curve C represents the cost of adopting strategy x . The benefit is the *average* caloric value of prey items obtained after moving a distance of x and the cost is the *average* energy utilized in moving distance x .¹³ By making the idealizing assumptions that $B(x)$ will increase (perfectly) asymptotically and $C(x)$ will increase (perfectly) linearly we get the following mathematical model (Fig. 2 below):

¹² Here I do not intend to endorse the accuracy of Parker and Maynard Smith's model. I utilize it only for the clarity with which it illustrates the structure of an optimality explanation of a biological phenomenon.

¹³ These averages range over the aggregates of individuals that play the various possible strategies across a range of decisions.

Fig. 2 Parker and Maynard Smith's model of the costs and benefits of lapwing foraging strategies (Parker and Maynard Smith 1990)

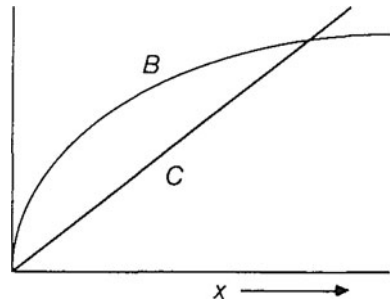
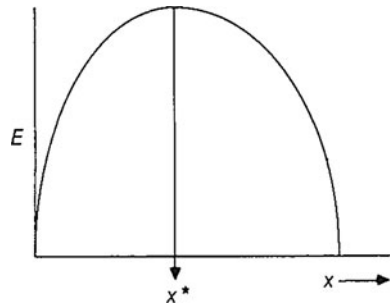


Fig. 3 Model representing the average energy gain for different lapwing foraging strategies. Average energy intake is maximized by the strategy, x^* (Parker and Maynard Smith 1990)



The proposed optimization criterion of this model is the average net energy gain per move, E , where, $E(x) = B(x) - C(x)$ (Fig. 3 below).

The optimal strategy, x^* , is that which maximizes E . This occurs when $dE(x)/dx = 0$ and $d^2E(x)/dx^2 < 0$. Now we add assumptions to show how this locally optimal phenotype is related to the real-world population. The optimality explanation makes the idealizing assumptions that the population being modeled is infinite (thereby eliminating drift), that mating within the population is random, and that organisms within the population reproduce asexually such that offspring perfectly resemble their parents. As a result, phenotypes with higher average energy intake should increase in frequency within the population (since they will have higher fitness) and the lapwing population is expected to (eventually) evolve the trait represented by x^* , or something close to x^* . This behavior is the locally optimal strategy given the tradeoff between the average caloric increase from the prey items obtained by moving and the average energy cost of moving, along with the other constraints of the foraging problem. Assuming that we observe actual lapwing populations exhibiting (approximately) this optimal strategy, the optimality model could explain the adaptive behavior by showing that it is the equilibrium point of the evolving population given the constraints and tradeoffs represented in the model.¹⁴ This basic type of cost-benefit analysis has been widely utilized to study optimal resource investment (Charnov 1976; Parker and Stuart 1976; Parker et al. 1999; Trivers 1974).

Most optimality models used within population biology are far more complex than the examples provided above. However, from these examples we can begin to

¹⁴ It is, of course, also important to test the assumptions of the model such as whether the assumed cost and benefit curves approximately represent the constraints within the real-world population.

see some of the features that make optimality models unique. Namely, optimality models represent various relationships that hold between the constraints, tradeoffs and the optimal strategy of a system. In the case of evolving systems, it is then assumed that this optimal strategy is an equilibrium point that will be arrived at regardless of the step-by-step dynamics of the system. What biological optimality models ignore, then, is not a specific subset of the causal processes included in other dynamical evolutionary models, but rather a particular *type* of information—specifically *all information about the step-by-step dynamics* of the evolving system.

This result is not surprising given that optimality models are a species of equilibrium models and it is precisely this feature that distinguishes equilibrium models from “dynamical” models (Sober 1983).¹⁵ This analysis does, however, show that the characterization of optimality models as censored causal explanations—i.e. explanations that represent a modular part of a larger causal process while omitting other causal factors—is quite misleading. For instance, Potochnik’s account suggests that optimality models differ from other evolutionary models only in *which* modular parts of the causal process that led to a phenotype they represent; namely, optimality models represent the causal processes that are involved in selection and omit the rest (those that have to do with other evolutionary factors). However, what optimality models actually ignore is all of the step-by-step dynamics of evolving systems in favor of specifying an equilibrium point of the system that results from the structural tradeoffs and constraints present in the evolving system. In other words, optimality models are simply a different *kind* of evolutionary model when compared to models that represent the dynamics of causal evolutionary processes. Optimality models do not represent, “a particular modular part of the causal process leading to the observed phenotype” (Potochnik 2007, p. 688). Rather, optimality models explain *by focusing on an entirely different set of relationships*: the constraints and tradeoffs that hold between the represented variables and those variables’ relationship to the equilibrium point of the system. Therefore, optimality models are not censored causal explanations, but instead offer a very different kind of explanation of evolutionary phenomena by showing how various population-level constraints and tradeoffs guarantee the evolution of an equilibrium state(s).

Synchronic representations of noncausal features

It is also interesting that there is nothing about optimality models that requires them to provide causal explanations.¹⁶ Indeed, the explanations offered by most biological optimality models receive essential contributions from *noncausal*

¹⁵ I do not intend to claim that optimality models are the only kind of equilibrium models in biology. Surely they are not; e.g. many population genetic models, such as the Hardy-Weinberg law, are also equilibrium models. Optimality models do, however, provide a unique kind of equilibrium explanation by utilizing optimization theory. It is this feature that distinguishes them as *optimality* explanations.

¹⁶ In fact, Elliott Sober has argued that all equilibrium explanations are noncausal explanations (Sober 1983). In contrast, recent advocates of causal accounts of explanation, such as Michael Strevens (2009) and James Woodward (2003), have argued that equilibrium explanations can be captured by accounts of causal explanation.

components.¹⁷ For one thing, the “heavy lifting” in most optimality explanations is done by mathematically representing tradeoffs among the variables within the model. These tradeoffs are vital to the explanation provided by the optimality model (change them and the predicted outcome changes), but it is often unclear how they can be understood as *causes* of the equilibrium point of the system (the model’s target explanandum).

First, the tradeoffs represented within optimality explanations are often not causal relationships between variables. For instance, in an economic optimality model, supply and demand may exhibit a tradeoff that is vital to the explanation of the market price, but it is unclear how we ought to understand supply as a *cause* of demand. Similarly, in the foraging lapwing model there is an important tradeoff between the probability of scanning terrain already inspected and the average energy expended on movement. However, it is misguided to claim that the probability of scanning terrain already inspected is *causing* average energy expenditure, or vice versa.

In addition, tradeoffs themselves are simply not the sorts of things that can enter into causal relationships—they are not events nor are they causal properties. Generally, causal explanations should provide information about the explanandum’s causal history. Yet as David Lewis explains, “a causal history is a relational structure. Its *relata* are *events*: local matters of particular fact, of the sorts that may cause or be caused” (Lewis 1986, p. 216). Other accounts of causal explanation appeal to causal properties. However, a tradeoff is not itself an event, nor is it a causal property within the population. Although the variables that exhibit a tradeoff may be causes, it does not follow that the tradeoff that exists between them is a cause. For instance, in the foraging lapwing model the explanation cites a population-level tradeoff between average caloric costs and benefits to different foraging strategies. However, it is unclear how to understand *this tradeoff* between population-level averages as a cause of the target explanandum: the equilibrium point of the evolving system (i.e. the population’s current trait distribution). There is a counterfactual dependence here that is key to the ability of these features to explain the biological phenomenon. However, this dependence is not a causal one.

What is more, in representing a tradeoff, a biological optimality model does not reference any causal processes of the biological population or any events within the population’s causal history. In the lapwing model, the key tradeoff is represented by the two (idealized) mathematical curves. The optimal strategy—the point at which average caloric intake is maximized—is then represented simply as the x-value at which the curve that represents the average net energy intake is maximized. Nowhere does the model describe a causal process (or causal trajectory) that unfolds over time or any events that occur prior to the explanandum—the model only identifies the optimal strategy by showing that average caloric intake is maximized at the point where $x = x^*$, given the model’s *synchronic* representation of population-level constraints and tradeoffs of the system. Moreover, none of the points along these idealized mathematical curves needs to be instantiated by the

¹⁷ I leave it open as to whether or not this makes them completely noncausal explanations or if they are simply explanations in which various kinds of noncausal information is essential.

population on its way to equilibrium in order for the optimality model to explain the evolutionary outcome. This is because the curves represent the population-level constraints and tradeoffs of the system, *not* its causal trajectory.

Consequently, the tradeoffs represented within many biological optimality models are not causes of the equilibrium point of the evolving population, nor does the model that represents those tradeoffs make any reference to the causal processes of the model's target biological population(s). It is, however, the mathematical representation of tradeoffs (and constraints) that does much of the explanatory work in a biological optimality explanation. Therefore, analysis of the above examples suggests that optimality explanations often rely heavily on synchronically representing noncausal relationships between a system's constraints, tradeoffs and the system's equilibrium point(s). As a result, it is unclear how causal accounts of explanation will be able to capture this essential part of optimality explanations.

The essential use of idealizations in optimality explanations

What is more, the explanations of several optimality models utilize various idealizations (e.g. idealized mathematical curves or assuming infinite population size), which ensure that the models themselves do not accurately represent the causal processes of any real-world biological population. The explanations of these models, therefore, cannot possibly provide a completely accurate description of the causal processes actually responsible for the phenomenon to be explained. Moreover, many optimality models utilize idealizations that appear to play *essential* roles within their explanations—i.e. the idealizations cannot be removed from the model without consequently eliminating the explanation. For instance, without introducing the assumption of infinite (or effectively infinite) population size, the laws of large numbers often used to deduce the equilibrium in optimality models are not applicable. As another example, the foraging lapwing model described above required the idealizing assumptions that the population is infinite, that organisms mate randomly, and that offsprings' phenotypic strategies perfectly resemble their parents'. Without these idealizations, the features represented in the mathematical model are unable to explain the target explanandum. Even more idealizations are often required in more complicated optimality models—e.g. game-theoretic explanations of highly general biological patterns. Therefore, for many biological optimality explanations it is unclear how the various idealizations could be removed from the model without consequently eliminating the explanation being offered.

These instances of idealized optimality models are similar to several cases in physics recently described by Robert Batterman (2002, 2009, 2010). Batterman calls explanations that introduce limits “asymptotic” explanations and argues that, “in many instances the various causal details need to be eliminated in order to gain genuine understanding of some phenomenon or other” (Batterman 2010, p. 2). In other words, these idealizations in physics take the model in precisely the *opposite* direction than that suggested by causal accounts of explanation by explicitly sacrificing accurate causal information in order to provide a more abstract explanation (Weslake 2010). In addition, on Batterman's view, idealizations that introduce limits are sometimes *essential* to an explanation because they allow for

certain mathematical operations to be performed that would not otherwise apply. The idealizations play an important (and sometimes ineliminable) role in the explanation in that they allow for the application of certain mathematical operations. In many cases, however, the explanation only applies to the idealized system—lose the idealizations and you lose the explanation.

Similarly, the explanations provided by at least many biological optimality models (e.g. game-theoretic models used to explain highly general biological patterns) seem to require that certain idealizations (e.g. infinite population size, random mating, constant payoff structure, etc.) be introduced in order to employ certain mathematical techniques. These optimality models make essential use of idealization in their explanations, but as a result they provide little (if any) accurate information about the causal processes operating in real-world biological population(s). Indeed, some of the most enlightening optimality models do not appear to describe any real-world causal processes at all, but instead utilize various kinds of idealizations in order to allow for the use of mathematical operations that apply only to the idealized population represented within the model. Therefore, important contributions to biological optimality explanations appear to come from the employment of various kinds of idealizations that explicitly sacrifice the models' ability to provide accurate causal explanations.

Conclusion

Given the considerations provided above, I conclude that it is incorrect to characterize all biological optimality models as providing censored causal explanations of biological phenomena. Optimality models do not represent a modular part of the causal process included in other dynamical evolutionary models, but instead focus on an entirely different set of explanatory relationships that hold between a system's constraints, tradeoffs and equilibrium point(s). In addition, it is unclear how many optimality models can be understood as providing causal explanations of biological phenomena since they often explain by using synchronic mathematical representations of noncausal features, which do not reference the population's causal history. Moreover, biological optimality models often make essential use of idealizations, which entail that they do not accurately represent the causal processes of their target system(s).

The discussion above does, however, provide some important suggestions for an alternative approach to understanding optimality explanations. We see that optimality explanations often rely heavily on mathematical representations of constraints and tradeoffs that hold within a system and use these mathematical representations to deduce the locally optimal strategy without referencing the causal history of the target system. Furthermore, many optimality models utilize various kinds of idealizations that remove or abstract away from various causal details, some of which appear to be necessary for the application of certain mathematical techniques. An alternative account of optimality explanations ought to provide a detailed analysis of these features and their contributions to optimality explanations of biological phenomena. These features demonstrate that optimality models do

offer a unique kind of explanation of biological phenomena. Therefore, an improved understanding of the explanations provided by biological optimality models is likely to provide important insight for debates concerning the nature of scientific explanation, the use of idealization in scientific explanations, and the practice of mathematical modeling more generally.

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