Explanatory Independence and Epistemic Interdependence: A Case Study of the Optimality Approach

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Abstract

The value of optimality modeling has long been a source of contention amongst population biologists. Here I present a view of the optimality approach as at once playing a crucial explanatory role and yet also depending on external sources of confirmation. Optimality models are not alone in facing this tension between their explanatory value and their dependence on other approaches; I suspect that the scenario is quite common in science. This investigation of the optimality approach thus serves as a case study, on the basis of which I suggest that there is a widely felt tension in science between explanatory independence and broad epistemic *inter*dependence, and that this tension influences scientific methodology.

1 Introduction

1.1 The Optimality Approach and its Detractors

In evolutionary and behavioral ecology, the optimality approach is used to model the evolution of phenotypic traits—physical characteristics, behaviors, and life-history strategies—without representing the system of genetic transmission. Optimality models represent the evolution of a phenotypic trait as a function of the relative fitnesses of the possible

trait values. They are equilibrium models, as opposed to dynamic models. That is, optimality models are used to predict the phenotypic trait values present at an evolutionary equilibrium, not the series of individual changes that a population undergoes on the way to the equilibrium.[1]

For example, optimal foraging theory is used to predict what food sources organisms tend to select as a function of maximal energy intake for a given time investment (Stephens and Krebs [1986]). The redshank sandpiper (*Tringa totanus*), a bird that feeds on worms in mudflats, exhibits a preference for eating large worms over small worms. A simple optimality model accounts for this preference: if large worms and small worms are both readily available, then a redshank's energy intake is maximized when large worms are chosen, since they yield more ingested biomass (Goss-Custard [1977]). The evolution of the redshanks' preference for large worms is modeled as a function of the fitness-effects of this preference.

The optimality approach has been criticized by various biologists. Perhaps most famously, Gould and Lewontin ([1979]) argue that the optimality approach goes wrong by ignoring the possibility of constraints and other non-selective evolutionary factors. Marcus Feldman is further concerned that the intricacies of genetic transmission in particular undermine the legitimacy of the optimality approach (Schwartz [2002]; Feldman [private correspondence]). These are both versions of the concern that the optimality approach omits important parts of the evolutionary process and, as a result, is unreliable. Nowhere in the simple optimal foraging example above was there mention of, e.g., the genetic causes of foraging behavior. Yet foraging behavior could not evolve if it weren't heritable, and the particularities of how that trait is passed on may affect how it evolves.

In light of such concerns, Lewontin ([1979]) suggests a provisional role for the optimality

[1]The optimality approach includes game-theoretic models, which are used when the fitness of a trait depends on the trait values (strategies) of other members of the population. Game-theoretic models often do not predict a single optimal trait value, but an optimal distribution of trait values in a population, or even cyclical change (Hofbauer and Sigmund [1998]).

approach. He claims that optimality arguments are 'a form of reasoning that is a short-cut attempt to predict community evolution,' whereas a truly dynamical theory of evolution will 'predict the evolutionary trajectory of the community [...] on a purely mechanical basis' ([1979], p. 6). On this view the optimality approach is a crutch, to be used only until it can be replaced by a model that more fully represents the evolutionary dynamics. Optimality models—equilibrium models—are to be replaced by models that capture the step-by-step dynamics of evolutionary change. Dynamic models that predict evolutionary change 'on a purely mechanical basis,' would incorporate the details of genetic or epigenetic transmission, and perhaps myriad other evolutionary influences as well.

1.2 The Optimality Approach and Anitreductionism

This style of argument against the optimality approach is reminiscent of a reductionist view of science, like that espoused in the classic papers by Nagel ([1961]) and Oppenheim and Putnam ([1958]). According to that view, the existence of different fields of science may currently be a practical necessity, but science aims to eventually explain the successes of these various fields by tracing their laws back to a basis in microphysical laws. This form of reductionism seems to be motivated by the idea that any regularities discovered in higher-level fields must somehow arise from underlying physical dynamics. This is a palatable idea to most; vitalism and similar views have few present-day apologists. Much more controversial is the claim that information about these underlying physical dynamics is needed to explain higher-level regularities. If so, then higher-level fields of science are provisional: the discovery of underlying dynamics explains their previous successes and renders them obsolete.

Explanatory reductionism and Lewontin's ([1979]) criticism of optimality models have this in common. Both suggest that modeling approaches are at best provisional if they do not fully capture the dynamics that underly a pattern of change. Such approaches are shortcut measures, adopted because what Lewontin calls the 'mechanical basis' of change is

not yet known. This parallel suggests that an answer to criticisms of the optimality approach may be able to do double-duty. Philosophical grounds for rejecting the idea that optimality models are only of provisional value are, if widely applicable, also grounds for rejecting similar ideas about other modeling approaches in science.

The aim of this paper is to do just that. The optimality approach persists, despite the criticisms made against it, because it plays an important role in population biology. Optimality models are best suited to provide a particular form of evolutionary explanation. Contra Lewontin ([1979]), explanatory reductionists, and others (e.g. Strevens [2009]), information about underlying dynamics does not always improve explanations. Instead, explanations tailored to their circumstances often benefit from the omission of information about the underlying dynamics. It is in part because of their value as evolutionary explanations that optimality models continue to be important to population biology, and a similar rationale motivates the continuance of other modeling approaches that neglect some dynamics. I call this the **explanatory independence** of such modeling approaches, of which the optimality approach is one instance.

Yet this explanatory independence is not the whole story. Critics of the optimality approach are correct that some of the dynamics neglected by optimality models, including the dynamics of genetic transmission, can shape the course of evolution. This is why it is necessary to establish the applicability and the parameters of an optimality model on a case-by-case basis. The same is also true of many other modeling approaches. Many models only partially represent the dynamics responsible for their target phenomena. Accordingly, their success depends not only on the dynamics they represent, but also on the character of the dynamics they neglect. One cannot ascertain whether such a model is successful without looking beyond the model to get information about causes that do not appear in the model. This dependence results in a circumstance that I call the **epistemic interdependence** of modeling approaches.

In this paper, I argue that explanatory independence and epistemic interdependence account for the role of the optimality approach in population biology. I also offer some considerations that suggest these notions apply equally well to many other modeling approaches in science. Confirming models and formulating explanations generate quite different demands, and the tension created by these competing demands accounts for several features of scientific methodology. Explanatory independence and epistemic interdependence are investigated in turn below. I begin each discussion by focusing on my case study of the optimality approach; then I suggest reasons for believing that other modeling approaches fit into the same pattern.

As a final preliminary, I should distinguish my approach from a different type of antireductionism. A common rejoinder to explanatory reductionism is to grant the metaphysical point of supervenience, and then to defend the explanatory importance of distinct fields of science on the grounds of multiple realizability (Fodor [1974]; Putnam [1975]; Garfinkel [1981]; Kitcher [1984]; Jackson and Pettit [1992]). My approach is substantially different. In my view, the explanatory value of models that neglect underlying dynamics is not due to multiple realizability. Instead, I argue that these explanations are valuable because of the need to simplify explanations in the face of widespread causal complexity. Accordingly, I do not contrast independent, non-reductive explanations with metaphysical dependence in the form of supervenience. The salient contrast is instead with the *epistemic* dependence that exists among modeling approaches as a result of this widespread causal complexity. This epistemic dependence is of much more immediate relevance to scientific practice than the metaphysical question of supervenience.[2]

[2]My terminology is partially borrowed from Sterelny's ([1996]) distinction between explanatory independence and ontological dependence. Note, however, that Sterelny's distinction is a version of the classic antireductionist distinction between the metaphysical state of affairs ('ontological dependence') and the strategy for formulating explanations. As indicated, I have a different distinction in mind.

2 Explanatory Independence

2.1 Optimality Explanations

In Section 1, I described how optimality models shortcut the actual dynamics of evolutionary change, and I discussed the various criticisms of the optimality approach that result. Despite such criticisms, the optimality approach still enjoys widespread use. Optimality models have not been superseded by more comprehensive models, e.g., models that also represent genetic dynamics. Instead, they continue to play an important role in evolutionary ecology and population biology more generally. Lewontin ([1979]) suggests one reason for the continued use of optimality models: the optimality approach is a useful shortcut because genetic information about natural populations is in short supply. Further, little is known about the genetic causes of most phenotypic traits. For these reasons, it is often not feasible to model the genetic dynamics of evolutionary change.

This certainly is an important motivation for the continued use of the optimality approach. Yet there is another reason still: optimality models best explain an important class of evolutionary phenomena. The causal relationships on which optimality models focus are key to understanding why certain traits evolve. Optimality models represent how selection pressures influence the fitness of traits, which is crucial in determining the results of long term evolution acting in a constant environment.[3] For this reason, optimality models are well situated to explain traits that result from long term evolution.[4] Whenever selection's influence on traits is the crucial causal relationship for understanding the course of evolution,

[3]Several analyses support the conclusion that optimality and game-theoretic models can be reliably employed to model long term evolutionary change (Charlesworth [1990]; Hammerstein [1996]; Gomulkiewicz [1998]; Eshel et al. [1998]; Eshel and Feldman [2001]). The assumption that the optimality approach can reliably substitute for a full population genetic approach is known as the 'phenotypic gambit' (Grafen [1984]).

[4]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. The present issue is not how to test the accuracy of an optimality model, but the explanatory value of accurate optimality models. For discussion of issues related to the confirmation of optimality models, see Section 3.1.

an optimality model provides the best explanation. The continued use of the optimality approach thus is not solely the result of practical necessity due to epistemic limitations. There is a nonepistemic justification for optimality modeling, namely, that these models best explain an important class of evolutionary events.

Recall the redshank sandpiper example from above. Redshanks exhibit a preference for eating large worms over small worms. A simple optimal foraging model explains this preference: a redshank's energy per unit time is maximized when large worms are selected, since they result in more ingested biomass (Goss-Custard [1977]). Redshanks have evolved foraging behavior that includes a large-worm preference because redshank birds with this preference enjoyed a selective advantage.

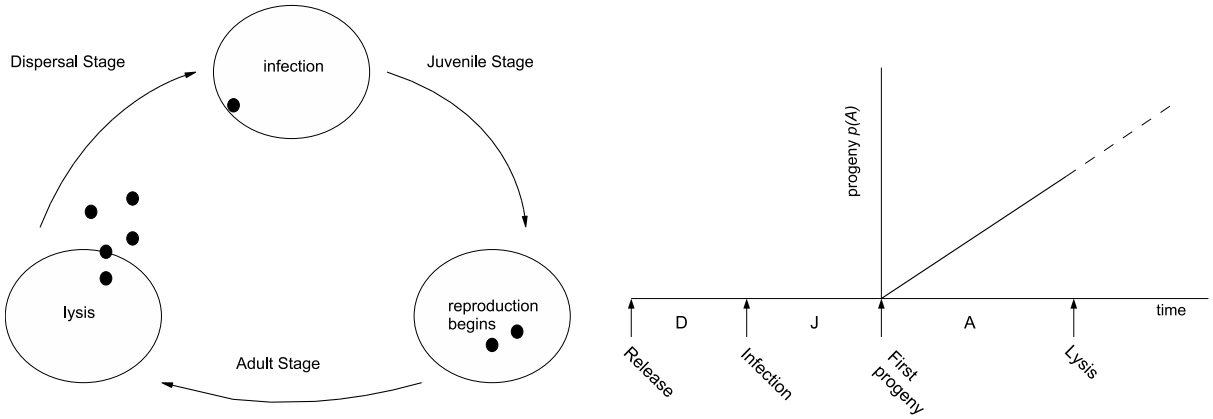
With enough genetic and developmental information about redshanks, a genetic model could, in theory, be constructed for the evolution of this foraging preference. The model would depict the series of genetic changes in the redshank population that eventually led to a population comprised of individuals with a taste for large worms. Suppose that this genetic model also represents the dynamics of selection, just as the optimal foraging model does. Would this more comprehensive model explain the redshanks' large-worm preference better than the simple optimality explanation?

For two reasons, the answer is no. The inclusion of genetic details would actually hinder the explanation. First, these details would detract attention from the causal relationship that is of central explanatory value, viz., the selection pressures that led to a redshank population full of large-worm eaters. This key explanatory relationship would be lost amongst less crucial details. Second, including information about the genetic causes of the large-worm preference would actually be misleading. The evolution of the large-worm preference is due to the consistent selective advantage enjoyed by this trait. Referencing specific genetic causes in an explanation of the trait's evolution suggests a level of sensitivity to the particular genetic details that is in fact absent. Optimal foraging theory accounts for a wide variety of foraging

behaviors in predators of all kinds. The means of transmission for this diverse group of behaviors across multiple species is inevitably quite varied. The optimal foraging explanation for redshank's preference for large worms identifies the preference as an instance of this broad pattern. Including genetic information in the explanation would suggest, contrary to fact, that the exact genetic causes of the preference were the reason the preference evolved.

Consider another example. Many bacteriophages (viruses that infect bacteria) reproduce by lysis, a process whereby new viral particles constructed inside the host bacterium burst out into the environment, killing the host in the process. Bull et al. ([2004]) consider an optimality model developed by Wang et al. ([1996]) for the length of time viral particles should be allowed to accumulate before the host is lysed in order to maximize reproductive potential. The model divides a bacteriophage's life history into three phases. The dispersal phase begins when a viral particle is released from a lysed host and ends when this phage infects a new host; the juvenile stage begins with this infection and ends when the first progeny (new viral particles) have matured inside the host; the adult phase begins when these first progeny have matured and ends with lysis. This life cycle is depicted in Figure 1(a).

There is a fitness tradeoff between early and late lysis. Early lysis shortens the time to the next generation, when the new viral particles begin to reproduce, but late lysis allows time for more viral particles to accumulate within the host cell. The optimal lysis time will balance these two factors. The relationship between life cycle and number of progeny is depicted in Figure 1(b). The optimal lysis time depends upon the overall length of the life cycle: the longer the average dispersal time (D) and the juvenile stage (J), the longer lysis should be delayed. This is an instance of a quite general pattern, according to which organisms with longer life cycles benefit from delaying the age at which reproduction begins (Wang et al. [1996]; Bull et al. [2004]). The optimal lysis time (A^*) in a bacteriophage population with average dispersal and juvenile stages of total length $D + J$ is shown in



(a) The life cycle of a bacteriophage depicted as three consecutive phases: juvenile, adult and dispersal stages. Lysis ends the adult phase.

(b) The length of the adult phase (A) results from a fitness tradeoff between number of progeny ($p(A)$) and length of the total life cycle.

Figure 1: Two representations of the life cycle of a lytic bacteriophage (Bull et al., 2004).

Figure 2.[5]

This study nicely illustrates the value of optimality explanations. Bull et al. ([2004]) have information about the specific genetic influences on lysis-timing in bacteriophages; the purpose of their paper is to explore the relationship between those genetic details and the optimality model. They do not employ the optimality model out of practical necessity. Yet Bull et al. do not suggest replacing the optimality model with a model that incorporates genetic dynamics. I suggest that this is because the optimality model is a superior explanation for the evolution of lysis-timing.

I claimed that replacing an optimality model with an explanation that cites genetic details would detract attention from the main explanatory causal relationship—namely, the

[5]Assuming a constant density of hosts and a constant average dispersal time, fitness is proportional to the expression $[p(A)]^{1/(D+J+A)}$, where $p(A)$ is the number of viral particles released with an adult phase of length A, and D and J are the average lengths of the dispersal and juvenile phases. Varying the length of A changes both the the number of phages released at lysis and the generation time of the phage life cycle. Maximizing this fitness function for A yields the same result as maximizing its log, $ln[p(A)]/(D + J + A)$. The optimum occurs where the graph of $ln[p(A)]$ is tangent to a line intersecting the horizontal axis at the beginning of the $D+J$ interval. All other lines through the origin that intersect $ln[p(A)]$ will have a shallower slope, indicating a lower population growth (Wang et al. [1996]; Bull et al. [2004]). This is shown in Figure 2.

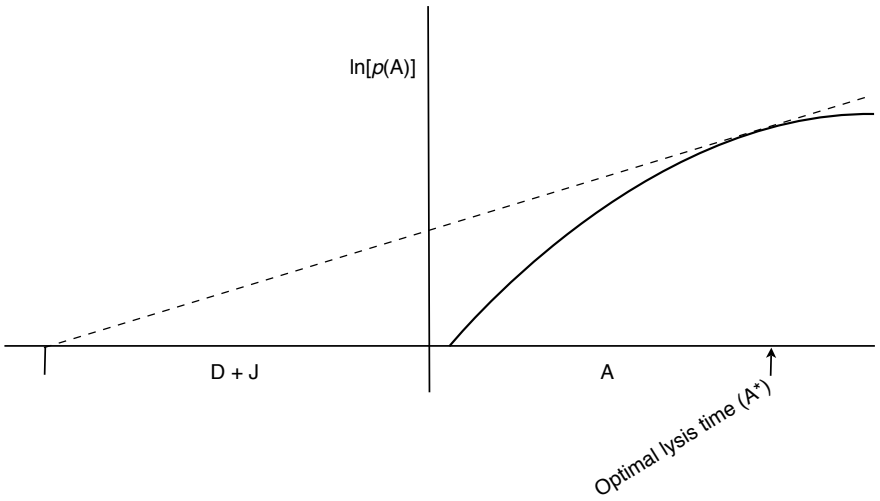


Figure 2: The optimal lysis time for a bacteriophage population with an average dispersal and juvenile period of the length D + J. See footnote 5 for the details of the model.

influence of natural selection on trait values—and would misleadingly suggest sensitivity to genetic details. So it is for explaining the timing of lysis in bacteriophages. Different types of genetic factors influence lysis-timing in phages with large genomes and in phages with small genomes (Bull et al. [2004]). Subtler genetic differences likely exist among different populations of phages as well.[6] Different phage populations thus exhibit different dynamics of generational genetic change, even though they face the same selection pressures on lysis-timing. Any model of lysis-timing that incorporated genetic dynamics would apply only to a narrow range of bacteriophage populations. Such an explanation would obscure the key determiner of bacteriophage lysis-timing: the time it takes to reproduce is balanced against the number of progeny in a way that maximizes reproductive potential. It would also suggest that lysis-timing would have evolved differently with different genetic dynamics. That suggestion is misleading, since in fact there is a single pattern of lysis-timing in bacteriophages, regardless of the genetic influences on the trait.

[6]To further complicate matters, all phages exhibit plasticity in lysis-time—that is, the expression of the genes controlling lysis varies with the environment—and different types of phages exhibit different degrees of plasticity (Bull et al. [2004]). This introduces another dimension of variability in the relationship between genetic factors and phenotypic traits.

In contrast, the optimality explanation of lysis-timing provides a single picture of the causal relationship between selection pressures (the tradeoff between shorter generations and greater number of offspring) and lysis-timing (delayed lysis when generations are long, accelerated lysis when generations are short). This picture is applicable to all phage populations, regardless of their genetic compositions and the size of their genomes. Moreover, as Bull et al. ([2004]) indicate, this is consonant with an even more general pattern whereby organisms with a long lifespan delay reproduction and those with a short lifespan hasten reproduction.

The explanatory value of this information is clear. For traits that result from long term evolution by natural selection, the sources of the traits' selective advantage is key to understanding why they evolved.[7] This is because such traits evolve in accordance with broad patterns of selective influence. Recall that the explanatory power of natural selection was one of the pivotal successes of Darwin's original theory of evolution. Darwin's insight was to explain many features of organisms by citing their contribution to survival and reproduction, that is, their selective advantage. Optimality models inherit this explanatory role.

2.2 Causal Patterns and Context of Inquiry

Assumptions about various features of scientific explanation lurk in the background of my argument for the explanatory value of optimality models. In this section, I make those assumptions explicit and briefly defend them. This further supports the value of optimality explanations, and it also suggests that optimality explanations fit into a style of explanation that is common in science.

I have argued that optimality models best explain some traits because they depict the

[7]See (Abrams [2001]) for a detailed discussion of the roles that optimality explanations play in population biology, and see (Rosales [2005]) for a defense of the explanatory value of game-theoretic models in particular. Also, note that for short term evolution and for traits that are not strongly influenced by selection, the optimality approach is not widely successful. In these situations, other causal influences tend to eliminate the possibility of an evolutionary pattern due to selection.

evolutionary role of the traits' selective advantage, and that including other details, such as genetic information, would actually detract from the explanation. Yet genetic transmission is indubitably a causal factor in evolution. This suggests that explanations can benefit from omitting some information about the causes of the event to be explained. I embrace this result. In my view, the best explanation of an event will focus selectively on some causes of that event, thereby neglecting other causal influences. This restricted focus enables the explanation of an event to depict a broad causal relationship that plays an important role in bringing about the event. An explanation that is focused in this way shows how the event in question falls into a pattern of cause and effect, and it indicates the conditions in which one can expect the occurrence of events like the one in question.[8] As optimality explanations illustrate, the omission of other causal factors is valuable because it directs attention to the causal pattern and implicitly or explicitly specifies the extent of that pattern.[9]

This is why the redshanks' preference for large worms is explained by the fact that preferentially eating large worms maximizes ingested biomass. This explanation identifies the redshanks' eating habits as an instance of a general foraging pattern, whereby predators evolve in a way that optimizes energy intake. The causal relationship between worm-size and evolved foraging habits is mediated by many intervening steps, but that information is immaterial to the explanatory causal relationship. For example, the particular genotypes related to the redshanks' worm preference are irrelevant to the pattern of optimal foraging. This is illustrated by the fact that the pattern is observed in predators of all kinds, with a

[8]Alternate formulations of this point capture the intuitions behind different accounts of scientific explanation. Focusing on a broad causal relationship crucial to bringing about an event shows why the event was to be expected (Hempel [1966]); how the event embodies a general pattern (Friedman [1974]; Kitcher [1981]); and what causal factors (in some sense) made a difference to the occurrence of the event (Woodward [2003]; Strevens [2009]). Indeed, I expect my points here to be separable from any particular account of explanation.

[9]I argue elsewhere that the neglect of causal information is valuable to an explanation when it makes the explanation more general in a desired way (Potochnik [2007]). That is one way to articulate the explanatory value of omitting some causal information, but there may be alternate formulations, perhaps in terms of unification or simplicity.

wide range of genomes.

My claim, then, is that explanations benefit from the omission of some causal information because this facilitates focus on a broad causal relationship crucial to bringing about the event. At first glance, this is similar to views articulated by Putnam ([1975]), Garfinkel ([1981]) and Strevens ([2009]), but there is an important difference. According to those authors, explanations should neglect causal information that is irrelevant to the occurrence of the event to be explained. Simply put, their idea is that if different underlying causal dynamics could have led to the same event, then the actual underlying dynamics are not relevant to explaining the event. I urge a further-reaching omission of causal information. Some of the causal factors that I suggest are to be omitted from an explanation in fact *are* relevant to the occurrence of the event to be explained. Consider the genetic causes of the redshank's foraging preference. Those genotypes are an essential part of the process of cumulative evolution that leads to the redshank's foraging habits. (Note that they would qualify as difference-makers for both Woodward ([2003]) and Strevens ([2009]).) Yet, as causally important as these genotypes are, their particular specification is not relevant to the causal *pattern* of optimal foraging. Despite their causal relevance, then, the genetic details do not belong in an explanation that focuses on the optimal foraging pattern.

This difference is important. The Putnam/Garfinkel/Strevens tack of eliminating irrelevant causal information does not sufficiently limit the information included in an explanation, especially when explaining an event that results from a complex causal process. Cumulative evolutionary change offers plenty of examples. Evolution is without fail a complex process, with diverse causal factors influencing fitness, inheritance, and development. Yet most evolutionary explanations focus on only a subset of those factors. As I see it, the neglect of causal factors facilitates a focus on a causal pattern into which the event in question fits.

Evolutionary explanations also illustrate a further complication. A single causal

process—especially a complex process like cumulative evolutionary change—may instantiate multiple causal patterns, and a causal factor may be relevant to some but not all of these patterns. For example, whereas redshank genotypes are not relevant to the optimal foraging pattern, they are relevant to the species- or population-specific pattern of generational genetic change that leads to the large-worm preference. As a consequence, factors that causally influence the event to be explained, like redshank genotypes, may not belong in *some* explanations of that event, but cannot be eliminated from *every* explanation of that event. The move to explanations that focus on a causal pattern creates the need to decide on a case-by-case basis whether a causal influence belongs in an explanation, taking into account which causal pattern the explanation features. As a result, the omission or inclusion of some causal influences depends on a contextual factor, namely, which causal pattern the explanation is designed to highlight.

This wrinkle was not made explicit in Section 2.1's argument in support of optimality explanations, so let's consider its import. Optimality models show how evolved traits contribute to survival and reproduction, and those causal patterns are explanatory when attention is focused on how the environment influenced the evolution of a trait. The intuitive appeal of the optimality explanation of redshank foraging preferences results from an implicit focus on the environmental sources of selective advantage. The same is true for explaining bacteriophage lysis-timing in terms of maximizing reproductive potential in the face of a fitness tradeoff. Focusing on other types of patterns can result in different styles of explanation. This is why genotypes belong in an explanation of redshank foraging preferences when generational genetic change is the pattern of interest. Similarly, genetic and developmental factors help explain lysis-timing when attention is focused on how the pattern of inheritance influences the evolution of lysis-timing. In general, which causal pattern is explanatory depends on the context of inquiry, that is, the research program in which the explanation is formulated.

Within the context of a particular research program, causal factors that are not relevant to the pattern of interest are bracketed off as background conditions. This often involves black-boxing those factors, i.e., providing only a functional specification of them. For instance, optimality explanations simply specify that a trait is heritable rather than depict its actual genetic causes, and genetic explanations use selection coefficients to stand in for how the environment acts on different phenotypes. Black-boxing some causal factors allows the focal causal pattern to take center stage in the explanation. Yet, as the coexistence of optimality explanations and genetic explanations illustrates, causes that are treated as background conditions in one research program might very well be focal in another.

We have seen examples of optimality explanations; let us consider a research program where a genetic explanation is sought instead. Curnow and Ayres ([2007]) develop two genetic models for the evolution of maternal behavior in the cowpea weevil (*Callosobruchus maculates*). A female weevil can lay her eggs in a single seed or in multiple seeds, and she can produce offspring with different levels of aggressiveness. Curnow and Ayres's models make simple assumptions about the fitness of these traits in order to focus on the genetic dynamics of their evolution. They develop two models to contrast the evolutionary process in different genetic scenarios: when the traits are each affected by a single gene and when each is affected by two genes (single-locus and a two-locus models, respectively). Curnow and Ayres justify their decision to develop genetic models for this behavioral trait by specifying that 'a full population genetic approach is needed if the rate of change of allele frequencies is of interest' ([2007], p. 68). The researchers seek a genetic explanation for the evolved behavior because the pattern of genetic change—the rate of allelic change—is of interest.

The context of inquiry is a pragmatic feature of explanation: it is an influence on explanations that goes beyond facts about the event to be explained and the causes of that event. I suspect that explanations are always local in this way, that it makes no sense to talk about *the* best explanation of an event, independent of a context of inquiry. Van

Fraassen assigns a similar role to context; he articulates the point as follows.

> It is sometimes said that an Omniscient Being would have a complete explanation, whereas these contextual factors only bespeak our limitations due to which we can only grasp one part or aspect of the complete explanation at any given time. But this is a mistake. If the Omniscient Being has no specific interests (legal, medical, economic; or just an interest in optics or thermodynamics rather than chemistry) and does not abstract (so that he never thinks of Caesar's death *qua* multiple stabbing, or *qua* assassination), then no why-questions ever arise for him in any way at all—and he does not have any explanation in the sense that we have explanations ([1980], p. 130).

Explanation is indelibly context-dependent. Research interests determine which causal relationships in an event's long and complex causal history are focal. The rest of the causal history is neglected, black-boxed as mere background conditions for the causal pattern featured in the explanation. But let me be clear about the extent of this context-dependence. Explanation is only context-dependent at the level of determining which of the many actual causal factors should be included in a particular explanation. If the process under investigation does not conform to some causal pattern, then that pattern cannot explain the outcome of the process. No amount of interest in a pattern can will it into existence.

This discussion clarifies Section 2.1's defense of optimality explanations. There I argued that optimality models best explain traits that result from long term evolution heavily influenced by natural selection. The more accurate claim is that this is so *in particular contexts of inquiry*, that is, when research interests are directed at understanding the selection pressures on traits, a common focus in evolutionary ecology. It must also be the case that the evolutionary process in question embodies the pattern represented in the optimality model. When these conditions are met, the inclusion of additional causal information—about the dynamics of genetic transmission, for example—would only detract attention from

the explanatory causal pattern: how selection shaped the evolution of the trait. The failure to abstract away from extraneous details would also misleadingly suggest that these causes cannot be bracketed, that they are an essential part of the focal causal pattern. Other contexts of inquiry result in a focus on different causal patterns. Curnow and Ayres ([2007]) seek a genetic explanation for the trait they investigate because they are interested in how genetic transmission influences evolution. In that context, information about the ecological sources of fitness would be unimportant, distracting details.

The coexistence of distinct explanations for a single event I call **explanatory independence**. The explanations are independent in the sense that each individually explains the event in question; indeed, each is the best explanation of the event in the context of certain research interests. Explanatory independence ensures the persistence of distinct genetic and optimality approaches to modeling evolutionary change. This example of distinct, independent explanations is particularly apt for two reasons. Evolution is a prime example of a complex causal process, and genetic and optimality explanations each focuses upon a part of the causal process that the other explicitly ignores.

I suspect that explanatory independence is found throughout much of science. Independent explanations, tailored to different purposes, are valuable whenever complex causal processes are encountered, and we live in a complex world. Even apparently straightforward events result from the combination and interaction of diverse causal influences, as part of causal processes that extend indeterminately far back in time. Examples of this abound in traditional fields such as economics and meteorology (consider the multitudinous influences on global warming). Collaborative ventures such as global systems science and biogeophysics provide further illustration. As for evolutionary explanations, so for explaining other results of complex causal processes. Events that are influenced by many different causal factors have multiple independent explanations, each tailored to a different context of inquiry. For instance, one may focus on the *atmospheric* causes of global warming (such as levels of

water vapor and carbon dioxide), the *sociological* causes of global warming (such as the industrial revolution), or the *economic* causes of global warming (such as the undervaluing of greenhouse gases on the global market).

3 Epistemic Interdependence

3.1 What Optimality Models Overlook

I have argued that the explanatory value of optimality models is due to their focus on natural selection's influence on traits, a focus that is achieved partially via black-boxing genetic dynamics. This also accounts for the tractability of the optimality approach, a point in the approach's favor that is not contentious. Yet optimality models' neglect of other evolutionary factors adds an element of unreliability to their use. Because optimality models use highly simplified assumptions as placeholders for complex dynamics, their successful use depends upon evolutionary dynamics that the models themselves do not explicitly represent. In other words, optimality models are epistemically dependent on unrepresented dynamics. Information about these unrepresented dynamics helps establish whether an optimality model's simplifying assumptions are problematic, and thus how successful the model is.

Genetic constraints are a prime example of how unrepresented dynamics can have unanticipated affects on evolution. Genetic transmission can involve a host of complications, such as epistasis (different genes with interacting effects) and pleiotropy (one gene with different unrelated effects). Such complicating factors may cause an evolutionary outcome to deviate widely from an optimality model's predictions. For this reason, optimality models are epistemically dependent on unrepresented features of genetic transmission. The success of the simplifying assumptions that optimality models use to stand in for genetic dynamics must be established on a case-by-case basis.[10]

[10]There is a related methodological question regarding the degree to which an optimality model's

Bull et al.'s ([2004]) investigation of lysis-timing in bacteriophages illustrates these points. Recall from Section 2.1 that Bull et al. evaluate an optimality model developed by Wang et al. ([1996]). In particular, they use genetic information to assess the success of the model's simplifying assumptions. One assumption of the model is that lysis is the phages' only reproductive option. This ignores the possibility of phage secretion—a nonlytic method of bacteriophage reproduction that is advantageous if it does not compromise fecundity. Bull et al. confirm that phage secretion is not an option by comparing the genetics of lytic and nonlytic phages. The genetic systems behind lysis and phage secretion are fundamentally different, so no small number of mutations would suffice to interchange the two systems of reproduction (Model and Russel [1988]). This shows that it is not possible for lytic bacteriophages to switch to phage secretion, which vindicates the assumption in question.

A second assumption of the model is that lysis-timing is a quasi-independent trait, i.e., that it can vary without simultaneously changing other traits of the bacteriophage.[11] Quasi-independence could fail if the genes that influence lysis-timing have pleiotropic effects (additional unrelated phenotypic effects), in which case selection on other traits may influence the evolution of lysis-timing in an unexpected way. Bull et al. establish quasi-independence by investigating the particular genes responsible for lysis-timing. In large-genome phages, lysis-timing is controlled by modular genes: mutations to those genes have few effects on other traits, and the genes retain their function when inserted into different phage genomes. Phages with small genomes have genes dedicated to controlling lysis (Loessner et al. [1998]; Vukov et al. [2000]; Ramanculov and Young [2001]; Bull et al. [2004]). This shows that genetic influences on lysis-timing do not have major pleiotropic effects, which confirms quasi-

assumptions must be corroborated for the model to be legitimately employed. That question is beyond the scope of this paper; the present epistemic point is just that information about unrepresented dynamics bears on the confirmation of an optimality model. I address the separate methodological question in (Potochnik [2009]).

[11] The idea of quasi-independence is due to Lewontin ([1978]). The independence is 'quasi' to allow for some genetic linkage between traits. For traits to be quasi-independent, linkage must be weak enough for mutations to change one trait value without also changing the other trait value (Lewontin, correspondence).

independence.

Bull et al.'s ([2004]) work demonstrates how genetic information can help confirm the viability of an optimality model's simplifying assumptions and illustrates the optimality approach's epistemic dependence on genetics. Yet the epistemic dependence of the optimality approach is broader than this. There are additional factors besides genetic transmission that are not represented in optimality models, but that have the potential to derail the models' successful employment. For instance, the space of evolutionary possibility is always shaped by background physical and biological constraints. The tremendous influence a simple constraint can have is exemplified by the jigsaw constraint in *Cerion*, a genus of West Indian land snail. Different populations of *Cerion* exhibit tremendous variation in the shapes of their spiral shells. Gould ([1989]) discusses how the combination of two constraints govern this variation. There is a geometrical constraint: for a given shell-size, bigger whorls automatically lead to fewer whorls, just like how fewer big pieces than small pieces fit into a jigsaw puzzle of a set size. There is also a developmental constraint: all members of *Cerion* exhibit the same three-phase pattern of shell growth, and this further limits the possible shell shapes. Together these comprise the jigsaw constraint that results in the covariance of whorl number, whole size and the overall shape of the shell (Gould [1989]). Any optimality model that assumed the independence of these features would be unsuccessful.

Constraints arising from geometry, physics, and background biology thus must be added to genetic constraints on the list of evolutionary influences upon which the optimality approach is epistemically dependent. Nor is this the extent of optimality models' dependence on dynamics they do not explicitly represent. Influences on evolution can arise from nearly any direction and can affect the success of optimality models via their specific parameters, such as the fitness function and the range of possible trait values, and the success of their simplifying assumptions.

3.2 Mutual Epistemic Dependence

The previous section depicts how the success of optimality models depends upon dynamics external to the models' explicit considerations. Information about these dynamics is imported from other subfields of biology and different fields of science entirely. Here I argue that the optimality approach is not alone in its epistemic dependence on other approaches. To begin with, population genetic models of evolution are reciprocally dependent on ecological information about selection dynamics, information that optimality investigations are well-suited to provide. Indeed, I propose that epistemic dependence on other approaches is commonplace in science.

In population genetics, evolutionary change is modeled as generational changes in the distribution of genotypes (the genetic causes of different phenotypes). These models explicitly represent the genetic dynamics, which is why critics of the optimality approach consider this to be a superior approach to modeling evolution. However, as with optimality models, the success of genetic models depends on the corroboration of simplifying assumptions about neglected dynamics. Genetic models assign selection coefficients to genotypes as a way to account for the dynamics of natural selection. Because little is known about the relationship between most genotypes and phenotypes, selection coefficients are often based on an empirical measure of one component of fitness, such as number of offspring reared to weaning (Lewontin [1974]). When the phenotypic effects of genotypes are known or postulated, selection coefficients instead may be based on theoretical considerations of selection's influence. In both cases, selection coefficients can fail to reflect the actual selection dynamics, thereby undermining the model's success. This is where ecological information comes into play.

Consider the two population genetic models developed by Curnow and Ayres ([2007]) that were introduced in Section 2.2. Curnow and Ayres base the parameters of their models on theoretical conjectures about the fitness affects of the maternal cowpea weevil behaviors

at issue.[12] They emphasize the provisional nature of these assumptions:

> These models, and more complicated versions of them, must remain speculative until there is experimental evidence from natural or synthetic populations confirming the necessary values of the selection parameters ([2007], p. 74).

Experimental evidence regarding the effects of selection is needed to confirm the models' assumptions, and that evidence would come from an ecological investigation of the sources of fitness differences between maternal behavior in cowpea weevils.

Population genetic models and optimality models are in parallel positions. Each partially represents the complex process of evolutionary change and neglects the details of some of the dynamics. For this reason, the success of each approach depends on model parameters and background assumptions, the accuracy of which can only be confirmed by looking to other approaches to investigating evolution. Both are epistemically dependent; indeed, they are **epistemically *inter*dependent**. By this I mean that the success of these models depends on diverse sources of information about causes not explicitly represented—information gathered with the help of other tools and other fields of science—and that this dependence is mutual. The genetic approach and the optimality approach each offers partial insight into the complex process of cumulative evolutionary change, and both forms of insight are valuable. This is a particularly nice illustration of epistemic interdependence, since each approach offers information about some of the dynamics that the other neglects.

I conjecture that epistemic interdependence is widespread in science. Science's specialized tools variously require external sources of information about causal influences they neglect and provide such information for other approaches. The basic reason for epistemic interdependence is the same as the reason for explanatory independence: many phenomena

[12]The maternal behaviors under investigation are how many seeds a weevil lays her eggs in and the level of aggressiveness of her offspring. Curnow and Ayres assume that selection favors females who lay eggs of nonaggressive progeny in a single seed and females who lay eggs of aggressive progeny in different seeds. Additional assumptions are introduced with the models' specific selection parameters ([2007], §2).

result from the combination and interaction of diverse causes. This leads to partial, independent explanations, but it leads to an opposite epistemic result, namely, the necessity of collaboration. Complex phenomena are encountered throughout science. Many modeling approaches only represent a small subset of the diverse causal influences on their target phenomena, for reasons of tractability as well as explanatory focus. The neglect of causal influences has its price, though, for the success of a partial model is subject to the success of its assumptions about neglected parts of the causal process. This results in the situation that I have dubbed epistemic interdependence.

4 Balancing Independence and Interdependence

I have made two claims about the optimality approach: optimality models are valuable as independent explanations, and yet they are dependent on other modeling approaches for help confirming assumptions made in the models. Indeed, I have argued that population genetics models are in a similar position. They sometimes explain, but they too exhibit epistemic dependence. Given the importance of these modeling approaches to population biology, it seems fair to say that that field is characterized by what I have termed explanatory independence and epistemic interdependence. I also have suggested that this situation is much more widespread in science. I suspect that, as in population biology, the tools of science are epistemically interdependent in the sense that their applicability can often only be established in a piecemeal way, in a process that involves gleaning evidence from external sources. Yet these same tools provide independent explanations of their target phenomena, explanations that are tailored to specific research interests.

This pairing of explanatory independence and epistemic interdependence grounds an antireductionist conception of science. This is not the classic antireductionism that defends non-reductive explanations in the face of metaphysical supervenience. The focus is instead on

the tension between independent non-reductive explanations and the difficulties of confirming those non-reductive models. The need for independent explanations tailored to different contexts motivates the persistence of partial modeling approaches, as does the tractability of those approaches. Meanwhile, the epistemic *inter*dependence of such modeling approaches compensates for each approach's inherent limitations. A raison d'être for these approaches is their explanatory value, while their legitimacy is secured via mutual evidential support.

If I am right that this scenario is common, a distinctive picture of science emerges. The targets of many scientific investigations are phenomena that result from the combination and interaction of diverse causal factors. This causal complexity leads to both epistemic interdependence and explanatory independence. Causal complexity creates the need for individual, simplified explanations that pick out the causal patterns important for specific research interests, and it makes this divide-and-conquor approach to science effective for practical reasons as well. Separate fields, subfields and laboratories persist, as do their distinct research programs. Yet collaboration among workers in these different fields is important. Evidential interrelationships do not respect field-boundaries; they require cross-disciplinary evidence-gathering. This is illustrated by the proliferation of interdisciplinary investigations in science. I suggest that the optimality approach is typical in these regards. The character of the diverse fields of science, as well as the relationships among them, are heavily influenced by the counterbalanced pressures of explanatory independence and epistemic interdependence.

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