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Reciprocal Causation and Biological Practice

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1. Introduction

Proponents of an "extended evolutionary synthesis" (EES) criticize standard evolutionary theory on the grounds that it overlooks the causal roles of developmental and ecological phenomena.[1] On this view, processes such as niche construction and phenotypic plasticity are as much causes of adaptive evolution as they are products. By generating variation, as well as biasing evolutionary processes themselves, these phenomena participate with natural selection in episodes of "reciprocal causation." To ignore the feedback between ecology, development, and evolution in our theoretical synthesis, proponents argue, is to impede biological progress.[2] The way we conceptualize evolution influences the way we investigate it—the questions we ask, the empirical tools we use, and the assumptions we take for granted. Therefore, according to the proponent of an EES, conceptual revision is warranted.

Proposed revisions frequently center on the concept of reciprocal causation. Buskell's analysis of reciprocal causation is a precursor to this paper (2019). The aim of Buskell's article is "to interpret and carefully distinguish those places where the positive epistemic value of reciprocal causation might be used to argue for changes to consensus practice" (p. 268). He

[1] Throughout the paper, I will use terms such as "standard evolutionary theory," "consensus practice," and "the modern synthesis" interchangeably. The first and third are often cited as the targets of the EES (e.g., Laland et al. 2016, p. 196). The second comes from Kitcher (1993) and is cited in Buskell's article on reciprocal causation (2019, p. 267). I take these terms for granted throughout.

[2] For example: the proximate/ultimate dichotomy introduced by Mayr (see below) "is now actually impeding progress in several areas in biology" (Laland et al. 2013, p. 721).

identifies three such lines of argument, but I will focus on one in particular: "empirical aptness" (p. 270). Buskell defines empirical aptness as "a relationship between a researcher's resources and the generation of epistemic goods" (p. 271). Causal frameworks are such resources. We may think of empirical aptness as describing a tight "fit" between a causal framework and the aims of a particular research program. Therefore, if we are to choose between competing causal frameworks in a nonarbitrary fashion, they must differ with respect to the advantages they confer on empirical progress. If a biological concept is empirically apt, then, among other things, models that employ the concept may make accurate predictions or reveal causal mechanisms more frequently than models that do not employ the concept.[3] Arguments for an EES often rely on its promise that treating reciprocal causation as a central feature of adaptive evolution is *empirically apt* relative to the mainstream alternative.

Given that empirical aptness concerns the practical power granted by a particular conceptual framework, I will characterize a second feature frequently found in arguments for an EES: "explanatory aptness." Let us define explanatory aptness as the relationship between a conceptual framework and its ontological implications—the map it draws of the natural world. Empirical aptness and explanatory aptness may come apart in interesting ways. A framework may be empirically apt if it is employed by models that enable reliable predictions of the relevant

[3] These are not the only ways for a conceptual framework to be empirically apt. Buskell says that "while 'fit' could be understood in terms of a propensity to make more accurate predictions," this is intentionally left open (personal communication). In the same spirit, I have used generic language in the survey to measure beliefs about empirical aptness (e.g., "adjust our models," "shift in our research practices," etc.). Additionally, an anonymous referee helpfully suggests that a broader notion of empirical aptness may be necessary since evolutionary biology, a historical science, notoriously struggles with predictions. Still, I believe there is adequate support in the EES literature for using predictive power as a metric (but not the only metric) for empirical aptness (see, e.g., Uller and Helanterä 2019, p. 369; Gawne et al. 2018, p. 6).

phenomena. However, while it is of practical value, the same framework may fail to be explanatorily apt if it implies a distorted picture of the world.

There is a substantial disagreement about the explanatory aptness of reciprocal causation. Proponents argue that the concept of reciprocal causation undermines the partitioning of biological processes into "ultimate" and "proximate" causes—a distinction first introduced to biology by Mayr in (1961). Mayr defined proximate causes as those investigated by developmental (or "functional," to use his term) biologists, whereas ultimate causes are those sought by evolutionary biologists. However, proponents of an EES criticize Mayr's distinction, arguing that it fails to appreciate the potential for developmental mechanisms to answer "why?" questions, not just "how?" questions, and therefore provide evolutionary explanations (Laland et al. 2011). The concept of reciprocal causation is said to provide a more verisimilar picture of the biological world, wherein any process that biases the outcome of natural selection is a bona fide cause of evolution. Arguments for an EES frequently emphasize that reciprocal causation entails an expanded and radically permissive class of evolutionary causes; as such, it is *explanatorily apt* relative to the mainstream alternative.

This paper deploys the tools of experimental philosophy to investigate biologists' beliefs about the practical and conceptual import of reciprocal causation.[4] The motivation and design of the study is in line with the empirical approach of Karola Stotz, Paul Griffiths, and Rob Knight (Stotz et al. 2004, Stotz and Griffiths 2004, but cf. Waters 2004). These authors argue that

[4] The *Stanford Encyclopedia of Philosophy* defines "experimental philosophy" (often abbreviated as "x-phi") as "an interdisciplinary approach that brings together…the kinds of questions and theoretical frameworks traditionally associated with philosophy [and] the kinds of experimental methods traditionally associated with psychology and cognitive science" (Knobe and Nichols 2017). For criticisms of experimental philosophy, see, e.g., (Kauppinen 2007; Deutsch 2015). For arguments in defense of experimental philosophy—and experimental philosophy *of science* in particular—see (Griffiths and Stotz 2008).

successful conceptual analysis in philosophy of biology demands that philosophers "examine what different biologists say and do" (Stotz et al. 2004, p. 648). Of course, there are several legitimate strategies that a philosopher may use when engaging with biological practice. However, by systematically sampling the beliefs of researchers and educators, the philosopher may "avoid the inevitable biases that come from having worked in one particular biological field before becoming a philosopher, from collaborating with some particular research group or simply from having a particular interest in one or more fields of research" (Stotz et al. 2004, p. 649).

In what follows, I present quantitative survey data from faculty members in biology departments at universities across the United States. The survey data are meant to give a preliminary answer to the question at hand: is the concept of reciprocal causation empirically and explanatorily apt? After reconstructing an account of reciprocal causation in adaptive evolution (Section 2), I present the methods and results of the study (Section 3). My presentation of the data is followed by a preliminary discussion of the results (Section 4). Finally, I discuss ambiguities that arise from the study in its present form, and how to correct them in future research (Section 5).

2. Reciprocal Causation

The debate about whether evolutionary theory is due for an "extension" is, among other things, a debate about causation. Participants in the debate are often portrayed as belonging to one of two camps: opponents who view adaptative evolution as the result of a unidirectional causal process (e.g., natural selection acts on organisms) and proponents who view adaptive evolution as the result of a bidirectional causal process (e.g., natural selection acts on organisms

and organisms, in turn, act on the direction and magnitude of natural selection). The former picture is frequently attributed to the neo-Darwinian "modern synthesis" (see, e.g., Laland et al. 2015). [5] According to this picture, selection pressures are imposed on organisms, which passively respond. That is, they survive, reproduce, and die in accordance with their ability to withstand the relevant pressures. This is adaptive evolution—populational change due to heritable variation in fitness—and, according to those who prefer this former picture, causal influence in this process flows in one direction. In other words, if we want to explain why the phenotype of an organism is adapted to its environment, we say that natural selection bears all the causal responsibility.

[5] This description of the modern synthesis is by no means intended to be exhaustive. For example, I have not mentioned that the modern synthesis is often accused of being "gene-centric," i.e., of systematically ignoring the importance of environmental and epigenetic factors in development and inheritance. I have also excluded critiques of "phyletic gradualism," which is said to be characteristic of the modern synthesis (Pigliucci and Müller 2010, pp. 13-14). This is because I am, in this paper, concerned with one specific point of contention: whether adaptive evolution is best modeled as a unidirectional or reciprocal causal process. For those interested in critiques of the "gene-centric" assumptions of the modern synthesis, some *loci classici* are found in (West-Eberhard 2003; Jablonka and Lamb 2014). For a recent discussion of the pitfalls of phyletic gradualism, see (Müller 2017).

Additionally, an anonymous referee encourages me to emphasize that the modern synthesis is gene-centric due to its eclipse of the organism. The referee writes, "MS is the articulation of Mendelian genetics and neo-Darwinian evolutionary theory, through the intermediate of population genetics. It clearly puts the organisms between brackets" (personal communication). This is certainly true if one defines the modern synthesis *just as* the combination of Mendelian genes and a Darwinian mechanism of evolution. However, I am personally reluctant to assign a rigid historical referent to "the modern synthesis." I am convinced by Gawne et al., who write, "The composite and varied nature of early twentieth-century research has been almost systematically ignored in recent exchanges, which tend to treat the modern synthesis as some sort of monolithic conception of evolutionary theory … Unfortunately, things just are not so simple" (p. 2). I therefore note that, for scholars such as this referee, as well as Huneman (2010) and Nicholson (2014), a significant drawback of the modern synthesis is, paraphrasing McClintock, its lack of feeling for the organism. As we will see, it is this focus on the organism, or lack thereof, that becomes the crux of the debate.

This picture is sometimes discredited as an uncharitable caricature of the neo-Darwinian synthesis, at least as it is currently understood (see, for example, Wray et al. 2014). Others, however, see this unidirectional causal story as being deeply embedded in the "consensus practice" of evolutionary biology. The consequence, they claim, is a theory of evolution that is impoverished with respect to its explanatory power (Scott-Phillips et al. 2014, p. 1238). Those who oppose the neo-Darwinian synthesis on these grounds call for its revision. On their view, the causal responsibility of adaptation is shared between natural selection and organisms themselves. Each is said to mutually influence the other in episodes of "reciprocal causation" (ibid.). This revised causal picture is a cornerstone of the proposed "extended" evolutionary synthesis (Laland et al. 2015).

To use the language of would-be synthesis extenders, reciprocal causation "simply means that process A is a cause of process B and, subsequently, process B is a cause of process A, with this feedback potentially repeated in causal chains" (Laland et al. 2015, p. 6). This causal framework is "taken to challenge causal assumptions embedded in the models and explanations of consensus practice" (Buskell 2019, p. 268). If we are to understand what gives this concept its pride of place, we need to understand (1) the relevant relata whose causal interdependence is supposed to challenge the models of consensus practice, and (2) the ontological implications of this causal relationship that are supposed to warrant a shift in evolutionary explanations. This section is dedicated to pursuing these two goals.

2.1. What are the causal relata?

Proponents of an EES argue that "reciprocal causation is a very general property of biological systems," and that it "should now be regarded as the norm, rather than the exception"

(Laland et al. 2013, p. 738). But several domains of evolutionary biology, such as population and quantitative genetics, have long recognized that genes can interact with each other and their environment in biologically significant ways. For this reason, some authors have doubted whether reciprocal causation—as it is understood by proponents of an EES—contributes anything new to evolutionary theory (Dickins and Barton 2013). For example, Svensson (2018) observes that frequency-dependent selection, eco-evolutionary dynamics, and Red Queen arms races (*sensu* Van Valen 1973) are all modeled as causal feedback loops. Considering these examples, Svensson concludes that reciprocal causation, while undoubtedly important, "is already well recognized within contemporary evolutionary biology research" (2018, p. 6). However, Baedke et al. have called Svensson's conclusion into question. These authors argue that, due to the conceptual difficulties of distinguishing organisms from their environments, the practice of modeling organism-environment reciprocity fell out of favor with biologists of the modern synthesis (Baedke et al. 2021, p. 7).[6] Meanwhile, they argue, "gene-environment reciprocity was increasingly considered in population genetics," and among the examples surveyed by Svensson in (2018), "the vast majority of these evolutionary models did not encompass organism-environment reciprocal causation" (Baedke et a. 2021, 7-8). Laland et al. make a similar observation in (2015): "Contemporary evolutionary biology does recognize reciprocal causation in some cases," but "many existing analyses of coevolution, habitat- or frequency-dependent selection, are conducted at a level (e.g. genetic, demographic) that removes any consideration of ontogeny" (p. 7). It is for this reason that Baedke et al., in their historical

[6] But see (Moczek 2015) for a recent example of the renewed interest in the conceptual inseparability of the organism from its environment.

analysis, "focus on organism-environment reciprocal causation, given that this has been the main target of recent heated discussions about…the so-called 'EES'" (2021, p. 3).

This observation is very important for understanding the fault line.[7] Proponents of an EES do not argue that reciprocal causation *per se* has been ignored in evolutionary biology. Instead, they insist that reciprocal causation between *organisms* and *environments* has been neglected by standard theory, and it is this omission that they aim to rectify. Proponents of the EES commonly propose two processes through which organisms impinge on their selective environments: developmental bias and niche construction. I discuss each below.

2.1.1. Developmental Bias

Developmental bias is "the bias imposed on the distribution of phenotypic variation, arising from the structure, character, composition, or dynamics of the developmental system…The concept of developmental bias thus captures the observation that perturbation (*e.g.*, mutation, environmental change) to biological systems will tend to produce some variants more readily, or with higher probability than others" (Uller et al. 2018, p. 949). An example of developmental bias is the process of canalization. The evolutionary response to selection may be constrained when the development of an organism is highly canalized—i.e., when the development of certain traits is insensitive to genetic or environmental perturbations (Waddington 1942). Since the phenotype, not the genotype, is visible to selection, and since the canalized development of the phenotype is insensitive to underlying genotypic variation, a population may accumulate "cryptic" genetic variation despite the uniform development of

[7] *Sensu* Uller and Helanterä: "That two such different interpretations of the theoretical status of selective niche construction co-exist points towards the existence of a fault line in interpretative understanding" (2019, p. 356).

organisms within the population (Gibson and Dworkin 2004). This process is argued to fit the model of reciprocal causation because homeostatic mechanisms of development impinge on selection by nonrandomly restricting the amount of genetic variation upon which selection may act. But restricting phenotypic variation is not the only way that canalization is believed to bias selection—it may also do so by augmenting phenotypic variation. This is *prima facie* unintuitive: how can canalization, which limits phenotypic variation, also augment it? The answer is that when canalization leads to the accumulation of cryptic genetic variation, it should be possible for novel environmental conditions to "release" this variation. Phenotypic expression of the hidden genetic variation provides new material for selection, and the population's evolutionary response will likely increase as result (Schlichting 2008).

In addition to buffering against genetic variation, developing organisms may play a more creative role in evolution via phenotypic plasticity (Pigliucci et al. 2006). Phenotypic plasticity is "the ability of a single genotype to produce more than one alternative form of morphology, physiological state, and/or behavior in response to environmental conditions" (West-Eberhard 1989, p. 249). Two genotypes, for example, may express the same phenotype in one environment, yet express two different phenotypes in another environment. When plasticity is continuous, it is described as a "reaction norm," i.e., the phenotypic expression of each genotype is plotted as a function of the environmental variable. When plasticity is discontinuous, it produces traits that are "polyphenic" (e.g., Suzuki and Nijhout 2006). Through the development of reaction norms and polyphenisms, plasticity will partially determine the range of phenotypic variation that is visible to selection. It has also been argued that phenotypic plasticity promotes diversification and speciation, given that it may lead to novel phenotypes, divergence, and adaptive radiation (Pfennig et al. 2010).

Why, though, does the EES frame these developmental processes as instances of entire organisms imposing a bias on selection? Should we not say that plasticity, for example, is a property of the genotype, given that we use genotypes to plot reaction norms? We find a likely rationale in (Nijhout et al. 2021, pp. 353-4): gene-centric models of plasticity fail to "capture the complexity and non-linearity of the processes that lead to phenotypes. Genes codes for proteins, not traits…Moreover, phenotypes are constructed through the interaction of many diverse and complex processes that operate at different levels of organization (molecular, biochemical, cellular, hormonal, physiological, organismal), most of which are contingent." We may ascribe plasticity to genotypes out of practical convenience, and we may find statistical correlations between some genotypes and plastic capacities, but it is important to remember that the map from genotype to phenotype is riddled with mechanisms across all levels of the organism.

But, the skeptic may reply, even if we treat canalization and phenotypic plasticity as organismal phenomena, there is still reason to doubt that organisms are impinging on their *environments*. After all, plasticity modifies the organism, not the environment. Aaby and Ramsey (2022) have an interesting response to this objection—namely, constitutive niche construction—which I discuss briefly below. In the same vein, Baedke et al. (2021) acknowledge that constitutive niche construction "is also related to phenotypic plasticity and 'constructive development'" (p. 10).

2.1.2. Niche Construction

Niche construction is a general term for the influence of organisms on the environment they experience, allowing them to alter the selection pressures to which they are exposed (e.g., Laland et al. 2016). Organisms can cause a change in the environments they experience by

modifying them (e.g., chemically, structurally, etc.) or by changing them altogether (e.g., migration) (Day et al. 2003). In doing so, organisms impinge on the selective environment that, in turn, impinges on them.

Taxonomies of niche construction have been offered by Aaby and Ramsey (2022) and Chiu (2019), and both are surveyed by Baedke et al. (2021). Aaby and Ramsey identify three kinds of niche construction: external (i.e., modifying external factors of the environment), constitutive (i.e., modifying the phenotype, such that the environment is differently experienced) and relational (i.e., modifying the relation between oneself and the environment, including relations with other organisms). Similarly, Chiu identifies physical niche construction (i.e., "external" in Aaby and Ramsey's terminology) and experiential niche construction, which can occur either through relocation (i.e., moving to a new physical environment) or mediation (i.e., modifying how the physical environment is experienced).

These taxonomies account for the impact that organisms have on the environments they inhabit. But modifying the environment is not itself sufficient for reciprocal causation. In modifying the environment, the organism must also modify the selection imposed by the environment. There are two ways that niche construction may modify selection—the first will be well known to anyone familiar with the literature, whereas the second is only recently being discussed. First, organisms may modify selection by either introducing or buffering against selection pressures. When organisms modify their environment such that a novel selection pressure is introduced, this is called "inceptive" niche construction. When organisms modify their environment such that an already existing selection pressure is neutralized, this is called "counteractive" niche construction (Odling-Smee et al. 2003, pp. 45-6). In such cases, organisms

directly modify the selective environment, and indirectly modify the strength and direction of selection.

But the strength and direction of selection are not the only factors that determine the evolutionary response of a population. The response is also determined by the amount of heritable variation. As I discussed above, when organisms are developmentally plastic, the phenotypic variance within a population may be partially determined by environmental stimuli. If organisms modify their environment via niche construction, and the environment partially determines how the organisms develop, then niche construction can bias the process of natural selection—not by introducing or neutralizing a selection pressure, but by altering the range of phenotypic variation that is visible to selection (D'Aguillo et al. 2021; Fogarty and Wade 2022).

2.2. What does reciprocal causation entail?

Why are the emphases on developmental bias and niche construction so crucial for extending the evolutionary synthesis? It is because of their relation to a subsequent claim, namely, that if a developmental or ecological process biases natural selection, it is itself a cause of evolution (see, for instance, Laland et al. 2013, p. 737; Laland 2014, p. 6; Laland et al. 2015, p. 8; Laland et al. 2016, p. 195). Hence, an "extended" evolutionary synthesis. It is extended because it has widened the scope of what we consider to be causally explanatory for adaptive evolution. Reciprocal causation is a key resource for an EES because it entails an explanatory expansion. It compels us to reject the causal hegemony of natural selection, turning the class of evolutionary causes into a big tent party.

I will call this first line of reasoning the "explanatory aptness argument" for an EES.[8] By "explanatory," I refer specifically to the ontic conception of scientific explanation as it is understood by Salmon (1984, 1990).[9] According to the ontic conception, scientific explanations should capture the causal structure of the world. Salmon holds that "explanatory knowledge is knowledge of the causal mechanisms…that produce the phenomena with which we are concerned" (1990, p. 128). Strevens also endorses this view, claiming that phenomena are best explained by "a set of causal facts. It is an aim of science to discover these sets of facts, these explanations" (2008, p. 6).

Proponents of the EES argue that reciprocal causation is explanatorily apt relative to the unidirectional causal framework embedded in standard evolutionary theory. By neglecting the mechanistic details that underlie the sources of selection or the generation of novel variation, proponents argue, the Mayrian picture of evolutionary causation is incomplete. Interestingly, reciprocal causation has been accused of the same shortcoming. For instance, Martínez and Esposito have argued that reciprocal causation does not go far enough in its causal expansion (2014). The causal model favored by proponents of the EES is unnecessarily restricted, they argue, given that it only accounts for causation in two directions, and it fails to relate different levels of biological organization across different times scales (p. 212). It therefore appears that the explanatory aptness of reciprocal causation is up for debate.

[8] Explanatory aptness should not be confused with the notion of "explanatory adequacy" from (Baedke et al. 2020, discussed below). Explanatory adequacy is evaluated according to four different criteria. Explanatory aptness, on the other hand, is one-dimensional: it only asks how well a candidate explanation captures the causal structure of the phenomenon.

[9] For our present purposes, I restrict the meaning of "explanation" to the ontic conception, but this is not to deny the legitimacy of other kinds of explanations. For example, perhaps there are distinctively mathematical explanations (*sensu* Lange 2013). It is beyond the scope of this paper to weigh in on the larger debate about scientific explanation.

A second line of reasoning is the "empirical aptness argument" for an EES. The concept of empirical aptness comes from Buskell (2019, p. 270). According to Buskell, proponents argue that "reciprocal causation is ubiquitous among causes that underpin evolutionary phenomena, and models that employ reciprocal causation are thus more likely to be empirically apt for investigating at least some aspects of these phenomena" (ibid.). Modeling adaptive evolution in a way that apportions shared causal responsibility to natural selection and organismic behavior should foster the development of hypotheses, predictions, or analyses that are not possible by assigning responsibility to selection alone. Additionally, treating ecological and developmental processes that bias natural selection as causes of evolution should have similar effects on biological practice. In other words, if we are to choose between competing causal frameworks in a nonarbitrary fashion, they must differ with respect to the advantages they confer on empirical progress.

In what follows, we will see that explanatory and empirical aptness of reciprocal causation come apart in interesting ways.[10] Moreover, I will argue that this decoupling can help us to understand the fault line between proponents and skeptics of the EES. That this decoupling is possible will not surprise readers who are familiar with the literature on scientific modeling. For example, Levins argues in (1966) that population biologists must face a tradeoff between

[10] It is hopefully evident that the notion of explanatory aptness assumes scientific realism. The reader may substitute whatever flavor of scientific realism they like—not much hangs on this. For example, explanatory aptness does not assume a mind-independent reality, natural kinds, or ontological monism. One could be a pluralist and still deem one framework explanatorily apt relative to another framework (depending, of course, on the explanatory target). On the other hand, empirical aptness need not assume scientific realism. It is compatible with realism, but it is also compatible with instrumentalism—i.e., theories may be evaluated by their predictive success, not whether they generate causal-mechanistic understanding. In fact, as I discuss below, empirically apt frameworks are sometimes intentionally instrumentalist, as in the case of highly idealized models.

maximizing precision, generality, and realism when building models. In the terminology of this paper, precise models that maximize predictive power are empirically apt, whereas realistic models that maximize understanding of causal structure are explanatorily apt.

Salmon, in his discussion of the ontic conception, recognizes that empirical virtues may obtain irrespective of explanatory shortcomings: "To the extent that causal mechanisms operate, they explain how the world works…A detailed knowledge of the mechanisms may not be required for successful prediction; it is indispensable to the attainment of genuine scientific understanding" (1984, p. 133). Often this decoupling of explanatory and empirical aptness will occur through "idealization," i.e., the process whereby scientists knowingly distort their models to render the target phenomenon empirically tractable (see, e.g., Weisberg 2007). In cases of idealization, we cannot always "read off" our ontology from the model. Some examples from population biology suffice to demonstrate this point—e.g., populations are not actually infinite (despite assumptions of the Hardy-Weinberg model), and a predator does not actually capture prey at a rate that increases linearly with prey abundance (despite assumptions sometimes made in Lotka-Volterra models) (see, e.g., Weisberg and Reisman 2008). Finally, to consider a more humorous example, empirically apt but explanatorily inapt models are like the physicist's spherical cow: the model may successfully estimate milk production, but in doing so, distorts the cow to unrealistic extremes. If the physicist privileges explanatory aptness, then this model is woefully inadequate, but if they privilege empirical aptness, spherical cows are inoffensive.

Another way that explanatory and empirical aptness may decouple is through the process of "screening off" (from Reichenbach 1956 and Salmon 1971; for an application to biological explanation, see Brandon 1982). One sense of "screening off" can be put as follows: A is a cause of B, and B is a cause of C. However, A is not a direct cause of C, because B screens off A from

C. Therefore, if we want to predict C, and B screens off A from C, then all we need to do is understand B. The value that A takes does not matter so long as it results in the right value of B.[11] The strategy of screening off is often empirically apt, given that it allows us to abstract away from the mechanistic details of a phenomenon, or it allows us to break up complex causal processes into practically manageable units. However, in performing this kind of abstraction or dissection, the resulting explanation of the phenomenon may be similarly abstract or absent of mechanistic detail. In the next section, I will argue that disagreements over the empirical aptness of reciprocal causation can be explained in part by disagreements about the role of idealization and screening off, as well as the importance of mechanistic detail, in evolutionary explanations.

3. Determining Explanatory and Empirical Aptness

Is the concept of reciprocal causation explanatorily and empirically apt? In this section, I present the methods and results for a pilot study designed in pursuit of an answer. The quantitative data were collected with a Likert scale, a psychometric tool used to situate the degree of a participant's accordance with a given statement within a range of possibilities (Likert 1932). In the study, each participant was asked to report their level of agreement with statements concerning the role of reciprocal causation in adaptive evolution.

3.1. Methods

[11] This is sometimes described as a "Markov process," where all that is needed to predict future states is the present state. Van Valen makes this connection when he writes that genic selection "depends only on the current distribution of frequencies of alleles and their interactions with each other and the environment. It does not depend at all on the process by which the current distribution was obtained. In formal language, it is a Markov process" (1973, p. 19).

This pilot study was approved by the Duke University Institutional Review Board (protocol number 2020-0507) as exempt on June 8th, 2020. The participant population for the study consisted of faculty members in biology departments at 19 universities across the United States. It must be noted immediately that a survey of biologists working only in the United States is an insufficient sample, given that the debate over the EES involves an international research community. However, I emphasize that this should be interpreted as a pilot study, and a precursor to similar studies on a much larger scale. In subsequent studies, biology faculty around the world will be surveyed.

Responses were solicited from 812 faculty members, and 91 responses were received. No demographic data were collected for this survey, including gender, race, education, career stage, etc. Therefore, this study does not account for such factors. This is because the survey is meant to reveal beliefs about evolutionary causation among its participants in their roles as researchers and educators of biology. It is not intended to probe for correlations among certain beliefs and genders or generational divides. While the possibility of such correlations is undeniably interesting, important, and worth investigating, it is not the purpose of this study.

The universities chosen for the sample population were selected based on the level of recognition of their graduate programs in biology. More specifically, 17 of these 19 universities are listed among the top 20 "Best Biological Sciences Programs" according to the 2018 U.S. News & World Report. At universities where there are multiple biology departments, requests for participation were only sent to faculty members of the departments that house evolutionary biology, or the most relevant subdiscipline: MIT (Biology); Stanford (Biology); University of California, Berkeley (Integrative Biology); California Institute of Technology (Division of Biology and Biological Engineering); Harvard (Organismal and Evolutionary Biology); Johns

Hopkins (Biology); Princeton (Ecology and Evolutionary Biology); University of San Francisco (Biology); Yale (Ecology & Evolutionary Biology); Cornell (Ecology & Evolutionary Biology); Duke (Biology); University of Chicago (Ecology and Evolution); Washington University in St. Louis (Biology); University of California, San Diego (Ecology, Behavior, and Evolution); Columbia (Biological Sciences); University of California, Davis (Evolution and Ecology); University of California, Los Angeles (Ecology & Evolutionary Biology); University of Wisconsin, Madison (Department of Integrative Biology); University of Michigan, Ann Arbor (Ecology and Evolutionary Biology). The study asked participants to indicate their level of agreement with each of the following statements (Table 1). Statements were presented to participants in the order in which they appear below.

#	**Statement**
1	Developmental and ecological processes (e.g., those that facilitate niche construction, phenotypic plasticity, developmental constraints, etc.) can modify the range of phenotypic variation that a population expresses and/or the selection pressures to which those variants are exposed.
2	In doing so, a population can systematically bias the strength and/or direction of natural selection.
3	It is appropriate to think of the aforementioned developmental and ecological processes as being able to "bias" or "impose direction on" natural selection.
4	Generally speaking, standard models assume that causation in adaptive evolution is unidirectional (i.e., selection acts on phenotypes) as opposed to bidirectional (i.e., phenotypes bias the strength or direction of selection to which they are exposed, as well).
5	Developmental and ecological processes that systematically "bias" or "impose direction on" natural selection should be considered alongside natural selection as evolutionary processes in their own right.
6	A failure to recognize the aforementioned developmental and ecological processes as evolutionary processes leads to an impoverished account of evolution.
7	Reframing the aforementioned developmental and ecological processes as evolutionary processes alongside natural selection would require that we adjust our models in order to accommodate them.
8	This conceptual shift (i.e., reframing some developmental and ecological processes as causes of evolution in their own right) would amount to a shift in our research practices, not just our explanations.

Table 1. Participants were asked to indicate their level of agreement with each statement on a Likert scale.

3.2. Results

The results indicate that a majority of the participants agree to some extent with each of the statements in Table 1, with the exception of Statements 7 and 8. Statement 1 is meant to establish a baseline. It is an uncontroversial statement in contemporary biology. The phenomena of niche construction, phenotypic plasticity, and developmental constraint are well established in the literature (see, for example, Darwin 1881, Bradshaw 1965, and Maynard Smith et al. 1985, respectively). Moreover, the effect of these phenomena on variation and selective environments is also well established. Most importantly, acknowledging the occurrence of these phenomena does not commit oneself to either of the competing conceptual frameworks. The responses to Statement 1 demonstrate this: all but 2 of the 89 participants who responded to Statement 1 (nearly 98%) agreed with it to some extent.

Statements 2-8 are not uncontroversial. The language used in these statements is lifted from several oft-cited articles on reciprocal causation. For instance, in several places, the development, behavior, and ecological interactions of organisms are said to be the "sources of selection" and the "immediate causes of selection" (Laland et al. 2013, p 725), "initiate and impose direction on selection" (Laland et al. 2015, p. 8), "modify selection" (Laland et al. 2016, p. 196; Laland et al. 2021, p. 722), and finally, "bias natural selection" (Laland et al. 2013, p. 727; Laland et al. 2015, p. 8; Laland et al. 2016, p. 192).

Nearly 60% of respondents agreed with Statement 2, and over 60% of respondents agreed with Statements 3 and 4. However, we see greater variance in the strength of agreement or disagreement. In Statements 2, 3, and 4, respondents who disagreed (regardless of the strength of

disagreement) make up less than 30% of the population. The consensus begins to recede with Statement 5. Nearly 53% of the participants agreed with this statement to some extent, while the proportion of the participants who disagreed surpassed 30% for the first time. Except for Statement 6, the trend continues. Statement 6 is meant to determine whether participants agreed with proponents of the EES that "a failure to recognize these factors as evolutionary processes leads to an inaccurate and impoverished account of evolutionary dynamics" (Scott-Phillips et al. 2014). The responses to Statements 7 and 8 see the smallest proportions of the participants who agreed to some extent: 50% for Statement 7 and just over 43% for Statement 8.

#	Strongly agree		Agree		Somewhat agree		Neither agree nor disagree		Somewhat disagree		Disagree		Strongly disagree		Total
1	56.18%	50	39.33%	35	2.25%	2	1.12%	1	0.00%	0	1.12%	1	0.00%	0	89
2	25.84%	23	20.22%	18	13.48%	12	16.85%	15	5.62%	5	10.11%	9	7.87%	7	89
3	21.35%	19	22.47%	20	19.10%	17	11.24%	10	3.37%	3	14.61%	13	7.87%	7	89
4	9.09%	8	34.09%	30	18.18%	16	15.91%	14	6.82%	6	10.23%	9	5.68%	5	88
5	13.48%	12	25.84%	23	13.48%	12	11.24%	10	11.24%	10	12.36%	11	12.36%	11	89
6	23.86%	21	20.45%	18	17.05%	15	11.36%	10	6.82%	6	9.09%	8	11.36%	10	88
7	6.98%	6	23.26%	20	19.77%	17	17.44%	15	6.98%	6	15.12%	13	10.47%	9	86
8	9.09%	8	11.36%	10	22.73%	20	19.32%	17	6.82%	6	17.05%	15	13.64%	12	88

Table 2. Responses to the statements in Table 1.

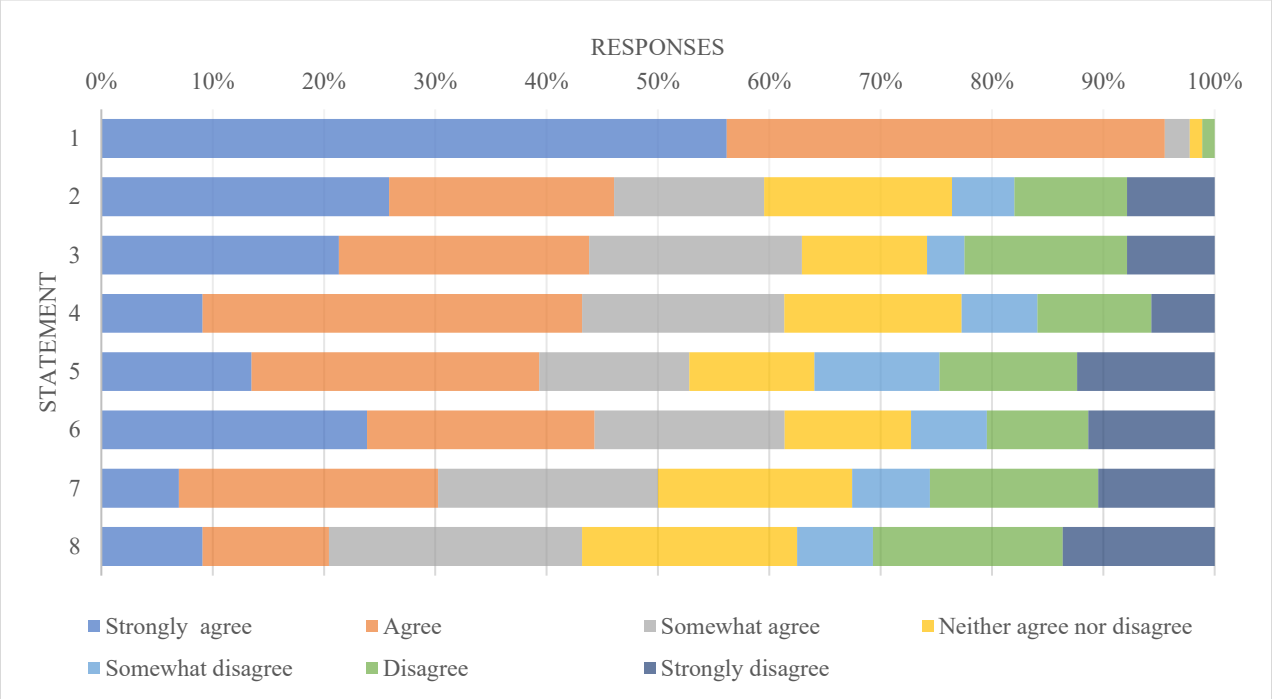

Figure 1. A stacked bar chart of the data in Table 2.

4. Discussion

Is reciprocal causation explanatorily apt? Nearly 60% of the participant population agreed that developmental and ecological processes can systematically bias the strength and/or direction of natural selection, while only roughly 24% of the population disagreed. Statement 5, "a process that biases natural selection toward a particular outcome is a cause of evolution," does not enjoy as much support, but is still affirmed by a majority of the participants. 53% of the population agreed that developmental and ecological processes that systematically "bias" or "impose direction on" natural selection should be considered alongside natural selection as evolutionary processes in their own right. In contrast, disagreement rose only to 36%. Over half of the participants, therefore, believe the causal structure of the EES is explanatorily apt.

Is reciprocal causation empirically apt? Only 50% of respondents agreed that expanding the class of evolutionary causes to include processes that bias natural selection would demand that we adjust our models to accommodate them. The proportion of respondents who disagreed exceeded 30%. The responses to the final statement, Statement 8, are even more telling. Just over 43% of respondents agreed that the proposed conceptual shift would amount to a shift in our research practices. Meanwhile, 37.5% of respondents see this conceptual shift as a shift in our explanations, but not our research practices. It is true that more respondents agree than disagree that an expanded class of evolutionary causes has practical consequences, but the degree of support is significantly less than that of previous statements. Pairing these results with our definitions of empirical and explanatory aptness (discussed in Section 2.2), we see that the proportion of participants who believe reciprocal causation is *explanatorily apt* is greater than the proportion of participants who believe it is *empirically apt.*

What could explain the decrease in support? One possible explanation is that natural selection "screens off" ecological and developmental processes from adaptive evolution.[12] In other words, if natural selection is a causal intermediary between the influence of individual organisms on their environment on one end and changes in trait frequencies on the other end, then we only need the strength and direction of natural selection to estimate the latter. Natural selection explains the evolutionary change regardless of the source of its strength and direction.

I do not mean to suggest that any of the participants responded to the survey with "screening off" explicitly in mind. I am suggesting, however, that an implicit practical commitment to screening off is a plausible explanation for the increase in opposition to reciprocal causation. Indeed, there are qualitative data to suggest that several participants had something akin to screening off in mind as they responded to the survey. At the end of the survey, each participant was invited to provide optional comments. One participant writes: "Niche construction and phenotypic plasticity can mitigate or alter the effects of natural selection, but that effect on natural selection is the *means* by which they affect the evolution of a population. They are not 'evolutionary processes in their own right' 'alongside natural selection' because they affect evolution only insofar as they affect natural selection." Another participant writes: "In my view developmental and ecological processes affect (cause?) natural selection which is a direct cause of evolution. I suppose that if they are in a causal chain they could be argued as causes, but these are indirect causes."

[12] In fact, Laland and colleagues have explicitly acknowledged this as a possible explanation, writing that, while "screening off certain processes will be necessary for model building and empirical tractability…acceptance of pluralism in conceptual frameworks and the recognition that exclusion of certain phenomena are pragmatic stances rather than inherent truths are vital" (Laland et al. 2013, p. 738).

We can see this kind of screening off in standard evolutionary models. For instance, consider the "breeder's equation" of quantitative genetics: $\Delta Z = h^2 S$ (Lush 1937). On the left-hand side of the equation, we have the evolutionary response to natural selection, represented as the change in the mean value of a given trait. On the right-hand side of the equation are two variables: heritability (h^2) and the selection differential (S). Heritability is a measure of the range of heritable variation within the population, and the selection differential is a measure of the strength and direction of selection. Notice that we do not need to know the *causes* of the strength and direction of selection to predict its effect. Infinite permutations of processes could have led to the value in S, but those processes are irrelevant to us. Assuming a constant h^2, the breeder's equation will enable the same prediction regardless of how the selection differential is realized.

In sum, the data suggest that participants recognize the ubiquity of reciprocal causation in evolutionary phenomena. They also recognize the bias toward unidirectional causation in standard models (such as the breeder's equation). However, we can reconcile these *prima facie* inconsistent findings with the notion of screening off. From a practical standpoint, the strength and direction of selection give us the information we need to predict an evolutionary response in one direction. It may be true that causal processes occur in pairs in biological systems, but that does not stop us from decoupling them and modeling them individually. This strategy allows us to abstract episodes of natural selection away from the particulars of the system. Thus, the skeptic may argue, if an extended causal framework makes no difference to our strategies of investigation, then it is not empirically apt.

How can the proponent respond? One available response is to deny that the strength and direction of selection, irrespective of their ecological sources, are sufficient for predicting

phenotypic evolution.[13] For instance, Laland et al. (2017) argue that knowing the sources of selection—i.e., whether the selective environment is constructed or non-constructed—will improve our ability to make qualitative predictions about the strength and variability of selection. They "propose that the selection generated by niche construction will be predictable, or at least more predictable than responses to environmental elements with little or no niche construction," because "niche-constructing activities generate consistent, reliable, sustained changes in environmental conditions" (ibid.). These predictions are supported by Clark et al., who—in a review of a large database of selection gradients estimated for quantitative traits in wild populations—demonstrate that "selection deriving from organism-constructed sources will exhibit reduced temporal and spatial variation in selection gradients and weaker (i.e., reduced intensity of) selection compared to nonconstructed sources" (2020, p. 25).

However, it is worth noting that the database analyzed by Clark et al. contains selection gradients that were estimated using the techniques developed in (Lande and Arnold 1983). That is, selection gradients were derived by comparing measures of trait frequencies within a population before and after episodes of selection. The estimation of a selection gradient from intergenerational changes in trait frequencies is *not* influenced by an awareness (or lack of awareness) of the sources of selection.[14] With this in mind, the skeptic may reply to the objection

[13] I am grateful to an anonymous referee for raising this objection.

[14] But cf. Svensson: "It is a very long time ago since evolutionary ecologists were simply satisfied by having quantified selection…Nowadays, evolutionary ecologists are busy understanding the ecological causes of selection…and few journals in evolutionary biology publish studies where selection coefficients are presented without any ecological context (and rightly so)" (2018, p. 6). This may be true, but there is still an important distinction to make between estimating selection and accounting for selection. Certainly, providing context for a selection coefficient helps us understand its strength, direction, or mode, and it helps us generalize to similar ecological systems, but if one's primary aim is to estimate the coefficient, this can be accomplished by screening off selection from the ecological context.

raised in the previous paragraph: whether knowledge of the source of selection will confer a practical advantage depends on our epistemic aims. If our aim is to make *a priori* qualitative predictions about the strength or direction of selection (i.e., predictions without longitudinal population data), then knowing the source of selection will be important. For example, if we know that population *A* modifies its environment while population *B* does not, we might predict *a priori* that selection on population *A* will be weaker than selection on population *B*. However, the skeptic is likely to reply, qualitative predictions such as these are arguably less valuable to the evolutionary biologist than quantitative predictions using empirically derived selection coefficients. If our aim is to make *a posteriori* quantitative predictions, then knowing whether the source of selection was constructed will not make a difference for our predictive capabilities, which depend primarily on the accuracy of our estimates of the strength and direction of selection. Such ecological knowledge will, however, help us *explain* the strength and direction of selection, even if it does not affect our ability to estimate these parameters. This comports with the analysis by Clark et al., who write that, "Consideration of the properties of the sources of selection potentially helps biologists *account for variation in selection*" (2020, 16, my emphasis). In other words, the skeptic might reply that knowledge of the sources of selection is *empirically* apt for qualitative predictions, but not for quantitative predictions. However, knowledge of the source of selection is *explanatorily* apt in both cases, as it provides a richer and more complete account of the variation in the strength, direction, and mode of selection between populations.

This discussion of screening off fits nicely with the "case-by-case evaluation of explanatory power" advocated by Baedke et al. in (2020). These authors argue that EES explanations will sometimes be superior, but at other times inferior, to standard evolutionary

explanations. How do we decide which explanation is best? It depends on which "explanatory virtues" are most important for understanding the relevant phenomenon. Baedke et al. identify four explanatory virtues that candidate explanations exhibit in varying degrees: precision, proportionality, sensitivity, and idealization (2020, p. 8). Precise explanations are specific in the details they provide; proportional explanations identify causes and effects that are similar in their degree of precision; sensitive explanations are precise but not robust; finally, idealized explanations are imprecise, and certain elements of the explanation are distorted for simplicity (pp. 8-9). EES explanations often privilege specificity but are very sensitive to changes in the underlying details. On the other hand, standard evolutionary explanations often privilege robustness, but in doing so, are imprecise and highly idealized. It is along these four axes that two candidate explanations may compete even when they agree on the evidence. This taxonomy of explanatory virtues helps us understand why some practitioners may gravitate toward EES explanations while others may privilege a standard theoretical framework: perhaps they prefer, or even require, some explanatory virtues over others. Practitioners who frequently abstract away from individual-level mechanistic details in their evolutionary explanations (e.g., by working with highly idealized models) will prefer explanations that are imprecise yet robust, while those concerned with developmental or ecological details are likely to prefer explanations that are precise yet sensitive. It would be unsurprising to learn that explanatory preferences are correlated with certain subdisciplines. I suspect that population and quantitative geneticists, whose models (such as the Hardy-Weinberg principle or the breeder's equation) are highly idealized, are more likely to view selection as a cause that screens off development and ecology, whereas biologists working within "eco-evo-devo" are more likely to view precision of mechanistic detail as paramount to successful evolutionary explanations. Testing this suspicion will be an objective of

future research.[15] In the next section, I consider other ambiguities that arise from the present study, and how these can be resolved with future studies.

5. Future Research

At least three ambiguities arise when interpreting the results of this study, all of which can be resolved by altering the methods in future studies. The ambiguities arise from (1) the inability to distinguish the participants who are trained evolutionary biologists from those who are trained ecologists, (2) the inability to determine which relata the participants believe are relevant, and (3) the inability to determine whether the reported beliefs of biologists genuinely guide their practice.[16] I will discuss each ambiguity in turn.

5.1. Indeterminate subdisciplines

As stated in Section 3.1, the pilot study was designed to survey the beliefs of the participants in their roles as researchers and educators of biology. When possible, requests for participation were only sent to faculty members of departments that house evolutionary biology. However, many of the solicited departments that house evolutionary biology also house ecology. I am therefore unable, in this study, to determine the proportion of participants whose research is primarily in evolutionary biology versus those whose research is primarily in ecology. This has potential consequences for my interpretation of the data, as it is possible that acceptance or rejection of reciprocal causation is positively or negatively correlated with one subdiscipline or

[15] I should, however, briefly mention a final possibility: perhaps biologists find reciprocal causation less empirically apt simply because they do not yet have (or have not yet applied) the right analytical tools. This is suggested in (Svensson 2018).
[16] I am indebted to an anonymous referee for suggesting these promising avenues for future research.

another. This ambiguity may be easily resolved in a future study by soliciting the participant's field of research, thereby enabling specification of the reference class.

5.2. Indeterminate relata

Similarly, the pilot study was designed to survey the beliefs of participants about reciprocal causation between developmental or ecological processes (broadly construed) and natural selection. The relata responsible for these developmental or ecological processes (e.g., genes, entire organisms, or populations) are left unspecified and open to interpretation, with two exceptions: Statement 2 mentions a *population* biasing selection, and Statement 4 mentions *phenotypes* biasing selection. This language was intended to be ambiguous—allowing researchers to substitute "organisms" if they like, while trying not to provoke worries about "organismal agency." I therefore avoided explicitly asking about organism-environment reciprocity with the goal of making the survey language palatable to, for instance, population geneticists as well as evo-devo biologists. There are, however, at least two reasons why a future study should explicitly use organism-centered language. The first reason is that organismal agency, as a conceptual framework, is amassing (or perhaps reclaiming) a considerable amount of attention and support in recent philosophy of biology literature (Walsh 2015; Sultan et al. 2022). A second and related reason is that, if Baedke et al. are correct, then we should be focusing on the "new views defending organism-environment reciprocal causation that are currently emerging in evolutionary biology" in our analyses of the EES (2021, p. 8; see also footnote 5). If, as Baedke et al. argue, organism-environment reciprocity is the crux of the debate, then it will not suffice to survey biologists about reciprocal causation *per se*. Instead, future studies will need to inquire about the organism's role in the evolutionary trajectory of its

own lineage. More specifically, future studies will need to inquire about different kinds of organism-environment reciprocity. For example, a participant may accept reciprocal causation when understood as developmental bias but balk in the context of experiential niche construction.

5.3. Indeterminate influence on practice

Finally, it is possible that a participant's beliefs, as recorded in the survey, do not really influence how they research or teach evolutionary biology. To some extent, we may have to accept this as a possible downside to experimental philosophy (and survey methods in general). It is always possible that participants answer questionnaires in ways that, for whatever reason, do not accurately reflect their actions. However, there are ways of mitigating this possibility. For example, in a future study, participants could be asked to provide information about the models they most commonly use in their research. We could then analyze the relationship between their beliefs about organism-environment reciprocity and their subdisciplines, as well as the empirical tools that are most important in their research.

6. Conclusion

The survey data I have presented indicate that a majority of the participants do not agree with a key claim of the EES—namely, that the concept of reciprocal causation is empirically apt relative to its unidirectional counterpart. However, over half of the participants agree that the causal picture drawn by proponents of the EES is explanatorily apt. When developmental and ecological processes bias the outcome of natural selection, those processes should be seen as genuine evolutionary causes alongside natural selection, even if this should not entail a shift in

research practices. These results demonstrate that the explanatory and empirical aptness of a framework can come apart in interesting and informative ways. It will be a goal of future studies to determine whether this trend holds for a much larger, and international, sample.

The data presented in this paper are preliminary, and the story that weaves the data together is one of several possible stories. It is also an incomplete story. Nevertheless, this kind of empirical engagement with biologists is a promising tool when we philosophers want to understand the practical utility of a conceptual framework such as an EES. Philosophers of biology often invoke the testimonies of practicing biologists as an authoritative resource. We rely on accounts from practitioners "in the trenches" to make inferences about the state of play in biology. However, we have two reasons to be pessimistic about the strength of such inferences. First, as it is presented in literature on the EES, the testimonial evidence is scant. Second, the testimonies are often from biologists who work in intellectual proximity to the relevant philosophers—sometimes even as collaborators on grant-funded projects. To incorporate the testimonies of biologists as evidence, we need to eliminate potential sources of bias. Experimental philosophy provides us with the tools to make such an improvement. By applying an empirical method to a philosophical dialogue with biologists, we can elucidate their understandings of organismal influence in evolution, as well as how these understandings guide their research, in a systematic fashion.

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References

Aaby, Bendik Hellem, and Grant Ramsey. "Three kinds of niche construction." The British Journal for the Philosophy of Science (2022).

Baedke, Jan, Alejandro Fábregas-Tejeda, and Francisco Vergara-Silva. "Does the extended evolutionary synthesis entail extended explanatory power?." Biology & philosophy35.1 (2020): 1-22.

Baedke, Jan, Alejandro Fábregas-Tejeda, and Guido I. Prieto. "Unknotting reciprocal causation between organism and environment." Biology & Philosophy 36.5 (2021): 1-29.

Bradshaw, Anthony D. "Evolutionary significance of phenotypic plasticity in plants." Advances in genetics. Vol. 13. Academic Press, 1965. 115-155.

Brandon, R. (1982). The levels of selection. In PSA: Proceedings of the biennial meeting of the Philosophy of Science Association (Vol. 1982, No. 1, pp. 315-323). Philosophy of Science Association.

Buskell, Andrew. "Reciprocal causation and the EES." Biological Theory 14.4 (2019): 267-279.

Chiu, Lynn. "Decoupling, Commingling, and the Evolutionary." *Evolutionary causation: biological and philosophical reflections* (2019): 299.

Clark, Andrew D., et al. "Niche construction affects the variability and strength of natural selection." The American Naturalist 195.1 (2020): 16-30.

D'Aguillo, Michelle, et al. "Genetic Consequences of Biologically Altered Environments." Journal of Heredity 113.1 (2022): 26-36.

Darwin, Charles. The formation of vegetable mould through the action of worms: with observations on their habits. Vol. 37. J. Murray, 1892.

Day, Rachel L., Kevin N. Laland, and F. John Odling-Smee. "Rethinking adaptation: the niche-construction perspective." Perspectives in biology and medicine 46.1 (2003): 80-95.

Deutsch, Max Emil. The myth of the intuitive: Experimental philosophy and philosophical method. Mit Press, 2015.

Dickins, Thomas E., and Robert A. Barton. "Reciprocal causation and the proximate–ultimate distinction." Biology & Philosophy 28.5 (2013): 747-756.

Fogarty, Laurel, and Michael J. Wade. "Niche construction in quantitative traits: heritability and response to selection." Proceedings of the Royal Society B 289.1976 (2022): 20220401.

Gawne, Richard, Kenneth Z. McKenna, and H. Frederik Nijhout. "Unmodern synthesis: developmental hierarchies and the origin of phenotypes." BioEssays 40.1 (2018): 1600265.

Gibson, Greg, and Ian Dworkin. "Uncovering cryptic genetic variation." Nature Reviews Genetics 5.9 (2004): 681-690.

Griffiths, Paul E., and Karola Stotz. "Experimental philosophy of science." Philosophy Compass 3.3 (2008): 507-521.

Huneman, Philippe. "Assessing the prospects for a return of organisms in evolutionary biology." History and Philosophy of the Life Sciences (2010): 341-371.

Jablonka, Eva, and Marion J. Lamb. Evolution in four dimensions, revised edition: Genetic, epigenetic, behavioral, and symbolic variation in the history of life. MIT press, 2014.

Kauppinen, Antti. "The rise and fall of experimental philosophy." Philosophical explorations 10.2 (2007): 95-118.

Kitcher, Philip. *The advancement of science: Science without legend, objectivity without illusions.* Oxford University Press on Demand, 1995.

Knobe, Joshua and Shaun Nichols, "Experimental Philosophy", The Stanford Encyclopedia of Philosophy (Winter 2017 Edition), Edward N. Zalta (ed.), URL = <https://plato.stanford.edu/archives/win2017/entries/experimental-philosophy/>.

Laland, Kevin N., et al. "More on how and why: cause and effect in biology revisited." Biology & Philosophy 28.5 (2013): 719-745.

Laland, Kevin N., et al. "The EES: its structure, assumptions and predictions." Proceedings of the Royal Society B: Biological Sciences 282.1813 (2015): 20151019.

Laland, Kevin N., et al. (2011). Cause and effect in biology revisited: is Mayr's proximate-ultimate dichotomy still useful?. science, 334(6062), 1512-1516.

Laland, Kevin, Blake Matthews, and Marcus W. Feldman. "An introduction to niche construction theory." Evolutionary ecology 30.2 (2016): 191-202.

Laland, Kevin, John Odling-Smee, and John Endler. "Niche construction, sources of selection and trait coevolution." Interface Focus 7.5 (2017): 20160147.

Lande, Russell, and Stevan J. Arnold. "The measurement of selection on correlated characters." *Evolution* (1983): 1210-1226.

Lange, Marc. "What makes a scientific explanation distinctively mathematical?." The British Journal for the Philosophy of Science (2013).

Levins, Richard. "The strategy of model building in population biology." American scientist 54.4 (1966): 421-431.

Lush, Jay L. 1937. Animal Breeding Plans. Ames: Collegiate Press, Inc.

Martínez, Maximiliano, and Maurizio Esposito. "Multilevel causation and the extended synthesis." Biological Theory 9.2 (2014): 209-220.

Mayr, Ernst. "Cause and effect in biology: Kinds of causes, predictability, and teleology are viewed by a practicing biologist." Science 134.3489 (1961): 1501-1506.

Moczek, Armin P. "Re-evaluating the environment in developmental evolution." Frontiers in Ecology and Evolution 3 (2015): 7.

Müller, Gerd B. "Why an extended evolutionary synthesis is necessary." Interface focus 7.5 (2017): 20170015.

Nicholson, Daniel J. "The return of the organism as a fundamental explanatory concept in biology." *Philosophy Compass* 9.5 (2014): 347-359.

Nijhout, H. Frederik, Anna M. Kudla, and Caleb C. Hazelwood. "Genetic assimilation and accommodation: Models and mechanisms." Current Topics in Developmental Biology 141 (2021): 337-369.

Odling-Smee, F. J., K. N. Laland, and M. W. Feldman. 2003. Niche construction: the neglected process in evolution. Princeton Univ. Press, Princeton, NJ.

Pfennig, David W., et al. "Phenotypic plasticity's impacts on diversification and speciation." Trends in ecology & evolution25.8 (2010): 459-467.

Pigliucci, Massimo, and Gerd B. Müller. "Elements of an extended evolutionary synthesis." Evolution: The extended synthesis (2010): 3-17.

Pigliucci, Massimo, Courtney J. Murren, and Carl D. Schlichting. "Phenotypic plasticity and evolution by genetic assimilation." Journal of Experimental Biology 209.12 (2006): 2362-2367.

Reichenbach, H. *The Direction of Time*, ed. M. Reichenbach. (1956): Los Angeles, University of California Press.

Salmon, W. C. (1971). Statistical explanation and statistical relevance (Vol. 69). University of Pittsburgh Pre.

Salmon, Wesley C. *Four decades of scientific explanation*. University of Pittsburgh Press, 1990.

Salmon, Wesley C. *Scientific explanation and the causal structure of the world*. Princeton University Press, 1984.

Schlichting, Carl D. "Hidden reaction norms, cryptic genetic variation, and evolvability." Annals of the New York Academy of Sciences 1133.1 (2008): 187-203.

Scott-Phillips, Thomas C., et al. "The niche construction perspective: a critical appraisal." Evolution 68.5 (2014): 1231-1243.

Smith, J. Maynard, et al. "Developmental constraints and evolution: a perspective from the Mountain Lake conference on development and evolution." The Quarterly Review of Biology 60.3 (1985): 265-287.

Stotz, Karola, and Paul Griffiths. "Genes: Philosophical analyses put to the test." *History and philosophy of the life sciences* (2004): 5-28.

Stotz, Karola, Paul E. Griffiths, and Rob Knight. "How biologists conceptualize genes: an empirical study." *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences* 35.4 (2004): 647-673.

Strevens, Michael. *Depth: An account of scientific explanation*. Harvard University Press, 2008

Sultan, Sonia E., Armin P. Moczek, and Denis Walsh. "Bridging the explanatory gaps: What can we learn from a biological agency perspective?." BioEssays 44.1 (2022): 2100185.

Suzuki, Yuichiro, and H. Frederik Nijhout. "Evolution of a polyphenism by genetic accommodation." Science 311.5761 (2006): 650-652.

Svensson, Erik I. "On reciprocal causation in the evolutionary process." Evolutionary Biology 45.1 (2018): 1-14.

Uller, Tobias, and Heikki Helanterä. "Niche construction and conceptual change in evolutionary biology." The British Journal for the Philosophy of Science (2019).

Uller, Tobias, et al. "Developmental bias and evolution: A regulatory network perspective." Genetics 209.4 (2018): 949-966.

Van Valen, L.M. (1973) A new evolutionary law. Evol. Theory 1, 1–30

Waddington, Conrad H. "Canalization of development and the inheritance of acquired characters." Nature 150.3811 (1942): 563-565.

Walsh, Denis M. Organisms, agency, and evolution. Cambridge University Press, 2015.

Waters, C. Kenneth. "What concept analysis in philosophy of science should be (and why competing philosophical analyses of gene concepts cannot be tested by polling scientists)." *History and philosophy of the life sciences* (2004): 29-58.

Weisberg, Michael, and Kenneth Reisman. "The robust Volterra principle." Philosophy of science 75.1 (2008): 106-131.

Weisberg, Michael. "Three kinds of idealization." The journal of Philosophy 104.12 (2007): 639-659.

West-Eberhard, Mary Jane. "Phenotypic plasticity and the origins of diversity." Annual review of Ecology and Systematics(1989): 249-278.

West-Eberhard, Mary Jane. Developmental plasticity and evolution. Oxford University Press, 2003.

Wray, Gregory, et al. "Does evolutionary theory need a rethink?." Nature News 514.7521 (2014): 161.