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Title: An Emerging Dilemma for Reciprocal Causation

Abstract: Among advocates and critics of the "extended evolutionary synthesis" (EES), "reciprocal causation" refers to the view that adaptive evolution is a bidirectional phenomenon, whereby organisms and environments impinge on each other through processes of niche construction and natural selection. I argue that reciprocal causation is incompatible with the view that natural selection is a metaphysically emergent causal process. The emergent character of selection places reciprocal causation on the horns of dilemma, and neither horn can rescue it. I conclude that proponents of the EES must abandon the claim that the process of natural selection features in cycles of reciprocal causation.

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1. Introduction

Despite the scientific, philosophical, and political disputes that have arisen from calls for an "extended evolutionary synthesis" (EES), at least one of its suggested revisions is gaining traction. Over the last decade, a sizeable subset of the literature in philosophy of biology has centered on the concept of "reciprocal causation." In this context, reciprocal causation is the increasingly popular view that adaptive evolution is a bidirectional process (Laland et al. 2011; Laland et al. 2013; Laland et al. 2015).[1] It is offered as an alternative to Mayr's distinction between proximate (i.e., developmental, individual-level) and ultimate (i.e., evolutionary, population-level) causes (Mayr 1961). Instead of privileging one kind of cause over the other in evolutionary explanations, EES proponents argue for parity between selection and development. During cycles of reciprocal causation, two different types of processes interact: the process of natural selection occurs when causation flows from the environment to the organism, and developmental processes such as niche construction, for example, occur when organisms causally impinge on their environment (Laland et al. 2015;

[1] Throughout the paper, I frequently cite Lala (formerly Laland) and colleagues as representatives of the EES. I recognize that proponents of the EES are not a monolithic community. However, I predominantly cite Lala and his coauthors because they have codified the tenets of the EES in a series of well-known papers.

Laland et al. 2016).[2] In this way, the processes of natural selection and niche construction are both causes and effects of each other.

The explanatory advantage of reciprocal causation has gone largely unnoticed due to the exaggerated rhetoric that is often used to defend it. For instance, some practitioners are quite skeptical about the EES (e.g., Gupta et al. 2017) and its calls for "a whole new causal structure for biology" (Buranyi 2022). Needless to say, we do not need a whole new causal structure for biology. What we need is to extract the concept of reciprocal causation from the rhetorically laden context of the EES and, instead, engage it with the vast preexisting literature on causation in biology. The aim of this paper is to begin bridging the literature lacuna. In doing so, I will present two claims and play them against each other. The claims are:

(RC) The process of natural selection is both a cause and effect of processes such as niche construction.

(EC) Natural selection is an emergent causal process.

[2] I use the example of niche construction throughout the paper. This is common practice in discussions of reciprocal causation (e.g., Svensson 2018; Baedke et al. 2021). Of course, EES proponents argue that other processes besides niche construction feature in cycles of reciprocal causation. Developmental bias is one such process. The omission of processes besides niche construction is merely for the sake of simplicity. You can replace niche construction with any proximate cause and the same conclusions will follow.

I will argue that EC poses a serious dilemma for RC. In Section 2, I provide support for RC, pointing to several places in the literature where such a view is defended. I similarly provide support for EC in Section 3. In Section 4, I argue that RC is incompatible with EC. I anticipate and respond to an interventionist defense of RC. In Section 5, I argue that RC is incompatible with the negation of EC. I conclude, in Section 6, that the proponent of reciprocal causation must abandon RC.

2. Motivating RC

Two claims, both central to the EES, will help to understand the motivation for RC. The first claim is that *any process that biases natural selection is itself an evolutionary process*. This claim is made by EES proponents in multiple places. For example, Lala et al. write, "In addition to accepted evolutionary processes that directly change gene frequencies, the EES recognizes processes that bias the outcome of natural selection, specifically developmental bias and niche construction" (2015, 8). This perspective, they argue, "entails that niche construction be regarded as a fundamental evolutionary process in its own right" (Laland et al. 2016, 195). This first claim highlights the importance of the actions of individual organisms and the impact of these actions on the evolutionary trajectories of their lineages. It is motivated by the perception that the "standard" evolutionary synthesis of the 20th century is guilty of privileging explanations at the level of the gene or the level of the population, meanwhile black-boxing (and thereby neglecting) the organism and its role in adaptive

evolution (see, e.g., Buskell 2019; Baedke et al. 2021; Hazelwood 2023). This claim will escape this paper uncontested.

What I will target, however, is the proposed causal mechanism by which organismal processes (such as niche construction) are argued to bias natural selection. This is captured by a second claim, namely, that *the process of natural selection is both a cause and effect of processes such as niche construction.* In other words, according to proponents of the EES, niche construction biases natural selection because they are reciprocal causal processes. Through this reciprocity, niche construction wields causal control over evolution, thereby cementing its place as an evolutionary process itself. This is the function that reciprocal causation is meant to serve in arguments for the EES: "reciprocal causation leads the EES to recognize several additional classes of evolutionary process … including processes that generate novel variation, bias selection and contribute to inheritance" (Laland et al. 2015, 7).

The reader may worry that this characterization of reciprocal causation is uncharitable. I have said that the relevant relata are two processes: natural selection and, in the present example, niche construction. But in many places, EES advocates argue for reciprocal causation between two *entities*, organism and environment, not two *processes.* Reciprocal causation between organism and environment is a relatively weak claim compared to reciprocal causation between two processes. By targeting the strong sense of reciprocal causation (between two processes) rather than the weak sense of reciprocal causation (between organism and environment), I may be accused of stacking the deck against reciprocal causation.

The weak sense of reciprocal causation is unobjectionable. No one doubts that organisms impact their environments and that environments impact their inhabitants. I can build a fire (organism causes change in environment) and the fire can warm and sustain me (environment causes change in organism). This organism-environment interaction is exactly the kind of reciprocal causation that Lewontin had in mind when he offered a pair of coupled differential equations in defense of his "constructionist" account of evolution (1983, 282). Any philosopher who rejects this works within a theoretical framework that is far removed from scientific practice.

However, organism-environment reciprocity is not the same as reciprocity between natural selection and niche construction.[3] The latter relationship is the strong sense of reciprocal causation. It is the strong sense because it is argued to elevate niche construction to the echelon of evolutionary processes. Furthermore, it is not an uncharitable reading. It is explicitly stated in arguments for the EES. For example, Lala and colleagues write that "the term '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, 6, my emphasis). Unless the authors are treating organisms and environments as processes sensu (Nicholson and Dupré 2018), and there is no

[3] Diagrams of reciprocal causation often represent natural selection as a causal arrow originating with the environment and terminating in the organism. This representation of natural selection is flawed for reasons detailed in (Lewens 2019, 712).

evidence to suggest this, then they are most likely referring to the causal efficacy of natural selection and ontogenetic phenomena such as niche construction. This is corroborated elsewhere in the literature. For example, Lala and O'Brien write:

> The key—and indeed subtle—distinction between the two perspectives [i.e., standard evolutionary theory and the EES] is that one views niche construction as a cause of evolutionary change as opposed to an effect of a prior cause (namely, natural selection) … The switch from treating niche construction as a cause rather than an effect cannot be dismissed as little more than a relabeling of already well-defined evolutionary phenomena … it emphasizes how the adaptive complementarity of organism and environment are the product of two reciprocal causes—selection and niche construction. (Laland and O'Brien 2011)

If these quotations are insufficient to convince the reader that the stronger interpretation is not only charitable but explicitly endorsed by proponents of reciprocal causation, then I am not sure what could convince them.[4]

The stronger reading of reciprocal causation is what I have labelled "RC"—the claim that will soon be subjected to an unforgiving dilemma. However, as I will demonstrate in Section 4.3, the failure of RC does not at all imply the failure of the first claim I introduced above, namely, that processes such as niche construction bias natural selection and are therefore evolutionary processes themselves. Niche construction does not cause natural

[4] Many thanks to an anonymous referee for encouraging me to forestall this objection.

selection, but as we will see, causation is not the only way for niche construction to make a difference.

3. Motivating EC

Over the last two decades, many philosophers have defended the view that natural selection is a causal process (see Bouchard and Rosenberg 2004; Stephens 2004; Reisman and Forber 2005; Millstein 2006; Brandon and Ramsey 2007; Sober 2013; Abrams 2015; Otsuka 2016; Bourrat 2018; Pence 2021). However, among the "causalists," as they are often called, there is some debate as to the level of biological organization at which the causal process of natural selection occurs—i.e., at the level of individuals or populations. [5]

[5] I wish to immediately head off a potential worry. I will use "levels" language throughout this paper. I recognize that talk of levels in philosophy of science is not without its discontents. Many have chosen to talk about "scales" instead (sensu Potochnik and McGill 2012). I am not an ardent defender of levels and am perfectly happy with this shift in terminology. I merely employ talk of levels because it is a familiar convention in literature on the metaphysics of biological systems. Nothing in the following argument hangs on the existence of mind-independent levels of biological organization. The argument merely depends on the existence of compositional relationships. An entity at the scale of a population is composed of entities at the scale of an individual. This relationship between

Early formulations of the population-level account come from Stephens (2004) and Reisman and Forber (2005), but the most perspicuous defense of selection as a population-level causal process is Millstein's (2006). Millstein argues that selection is a process whose cause "is variation in the population" and whose effect "is differences in reproductive success, a property of the population rather than the individual" (631-32). A similar conclusion is drawn by Sober (2013), who argues that, while trait fitness itself is causally inert, *variation* in trait fitness causes changes in trait frequencies within a population.

The views proposed by Stephens, Reisman and Forber, Millstein, and Sober share a striking feature: in the process of natural selection, the causal relata are properties of the aggregate, not properties of the individual. In other words, these authors depict natural selection as a novel causal process that arises at the level of the population from the interactions among individual organisms and their selective environment. It is for this reason that several philosophers have invoked the concept of metaphysical "emergence" to characterize the causal process of natural selection (e.g., Haug 2007; Pence 2021).

What does it mean for a causal process to be emergent? In a recent review, Wilson identifies two principal conditions: "nearly all accounts of emergence take this to involve both broadly synchronic dependence and (some measure of) ontological and causal autonomy" (2016, 346). Candidate emergent processes satisfy the first condition, synchronic

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scales is one of composition and not causation. So long as you accept this, then you may swap out talk of "levels" for "scales" at any point throughout the paper.

dependence, when they are implemented by processes at a lower level. In other words, the emergent process is not spatiotemporally distinct from the underlying processes. However, the emergent process *is* ontologically and causally distinct from the underlying processes. Candidate emergent processes may satisfy this second condition in a few ways—e.g., irreducibility of the process at the higher level to the lower level, novel laws that govern the process at the higher level, multiple realizability of the higher-level process, etc.

An aside: these are only a few of the possibilities for satisfying this second criterion as outlined in (Wilson 2016). I encourage the reader to consult Wilson's chapter (347) for a more exhaustive list. Metaphysicians disagree as to the contents of this list, and I leave it to them to work out exactly how the second criterion should be specified. It would be intellectually reckless of me to pretend to resolve such a vital debate in this paper. For Wilson's part, she argues that "much of this apparent diversity is superficial" (348). But even if Wilson is wrong, and there are substantive differences between these metaphysical positions, it will not impact my argument. I will account for the disjunctive nature of this second criterion with an argument by cases, demonstrating—as I do in the following paragraph—that each of the criteria I consider above (irreducibility, novelty, and multiple realizability) is satisfied by natural selection.[6]

[6] Further still, I have a minimal concept of emergence in mind. I am using the concept to refer to a higher-level process that is realized, not caused, by processes at a lower level. For the purposes of this paper, we need not wade into the niceties of the "emergence" debate

How, then, does a population-level account of natural selection satisfy the two principal conditions for metaphysical emergence? Natural selection satisfies the first condition because it is a population-level process that synchronically depends on the properties and interactions of the individuals that constitute the population. The emergent causal relata in this process are variation in trait fitness and the resultant change in trait frequencies—two population-level properties that are at any given moment determined by properties of the constituent individuals.[7] Likewise, natural selection satisfies the second condition because it is a population-level process that is ontologically and causally distinct from processes at the individual level. This second condition is satisfied on each of the readings of distinctness suggested by Wilson above, and I will take each in turn. First, natural

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(e.g., whether my sense of emergence is "weak" or "strong," sensu Wilson 2016). It will not affect my argument if there are stronger senses of emergence that do not apply to natural selection. I do not need natural selection to emerge from organisms as robustly as, say, diamonds emerge from carbon atoms. I merely need natural selection to emerge in a way that is irreducible, novel, and multiply realizable.

[7] I should note that, in these characterizations of natural selection, I assume a ceteris paribus clause sensu (Sober 1984): the process of natural selection will result in evolutionary change *provided that no countervailing process undermines it*. This is a conceptual possibility. It is not to suggest that there are ever real instances where natural selection is the only evolutionary process at play.

selection is made possible by population-level properties that are irreducible to those of their constituent individuals. The property of evolvability, for example, is a dispositional property of populations that "supervenes on many of the more concrete features of the individuals within populations and their interactions" (Brown 2014, 561). The process of adaptive evolution, moreover, is unavailable to an individual. Individuals have the capacity to engage in the processes of living, dying, mating, eating, developing, etc. They do not have the capacity to evolve. It is a category mistake to assert otherwise. Second, natural selection is governed by laws that do not apply to individual-level processes. For example, consider any "law" from population genetics, such as the Hardy-Weinberg law or Fisher's Fundamental Theorem of Natural Selection. These laws govern changes in the frequencies of traits and are sui generis at the level of biological populations. Finally, natural selection is multiply realizable. Say, for instance, that at a certain time a population has a ratio of 1 green beetle to 2 purple beetles. Later, due to differences in the relative fitness of each trait, the population has a ratio of 1 green beetle to 3 purple beetles. This change from 1:2 to 1:3 is multiply realizable—it can be instantiated by an infinite number of combinations of individual beetles (so long as they ground the right relata at the population level).

Haug (2007) is one of the first to explicitly offer a population-level account of natural selection in terms of emergence. Haug acknowledges that "there are powerful arguments that natural selection must be [a] population-level causal process, if it is a causal process at all," but that such a view faces a fundamental "grounding question," which he puts in the following way (432, emphasis in original): "How are population-level causal processes

related to the causal processes that occur at the level of individual organisms, and how is this relation compatible with the *causal* nature of population-level processes?" His answer to the grounding question comes in the form of a "realization relation," according to which "the population-level properties, *C* and *E*, which are the cause and effect in the process of natural selection, are realized by properties of and relations between the individuals in the population" (439).

Later, Pence picks up the mantle by clarifying Haug's suggested "realization relation." Pence cashes out this relation in terms of *composition*: "It is precisely the fact that individuals are arranged population-wise that enables the population to be causally effective" (48).[8] Specifically in the case of natural selection, he argues "that the causal interactions of individuals – eating, mating, dying, and so forth – produce the causal profile of natural selection precisely when those individuals compose populations of certain sorts with certain kinds of structures and relationships" (48-9). The picture of selection that Pence has sketched clearly satisfies the two principal conditions for metaphysical emergence listed above. First,

[8] Interestingly, Pence mentions niche construction explicitly in (2021): "recent discussions of niche construction, for instance, have inspired the examination of reciprocal causation in evolutionary theory . . . it is possible that the kinds of explanatory resources developed in our understanding of emergence will be useful in helping us come to grips with these cases" (26). But Pence's framework, I will argue, doesn't help us come to grips with reciprocal causation. It undermines it.

it satisfies the condition of synchronic dependence because, on his account, "the phenomena at the individual level, taken in the aggregate (and, hence, the population level), are sufficient to produce natural selection, itself a macro-level cause" (47). Second, it satisfies the condition of ontological and causal autonomy because, among other reasons, it is multiply realizable; "there are many sets of individual details that could give rise to the same selective change at the population level" (47).

Pence's account, I believe, gives us the strongest argument for EC. But a final note before moving on: since Pence's compositional account of emergence drives much of my argument, the reader may desire a more thorough exposition and defense. Due to constraints of space, I am unable to provide such a defense of Pence's compositional account of emergence. The reader is encouraged to explore the finer details of his argument in (2021, 44-53). However, I will briefly consider the possibility that natural selection is better captured by some non-compositional account of emergence. What if natural selection is *realized* by organismal processes without being *composed* of them? Perhaps the realization relationship should be cashed out some other way.[9] Would this make a difference for my argument?

My argument is shielded from this objection for two reasons. The first is that, according to many metaphysicians, you cannot have realization without composition because *realization is a species of composition*. For example, Aizawa and Gillett defend composition

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

as the best way of framing realization relations within the sciences tout court (2009). Wimsatt, similarly, writes that multiple realizability is "characteristic of any move from a lower compositional level to a higher one" (1994, 224). Wilson shares this commitment to composition, writing that "effectively all accounts of higher-level emergence take both material composition and modal covariation to be some part of emergent dependence" (2016, 363). For each of these authors, realization (multiple or otherwise) and composition are conceptually intertwined. Thus, in the context of this paper, the terms "realization" and "composition" are noncompeting and coextensive. They both hang together on a scientifically respectable account of emergence.

The second reason is that, while not all metaphysicians agree that realization entails composition, their reasons for this are immaterial in the case of natural selection. For example, Polger writes that "we should understand realization as distinct from composition" (2007, 249). For Gillett and Aizawa, realization relations are a subset of compositional relationships, whereas for Polger, they are two sets that overlap. Polger's skepticism about the compositional account derives from its inability to accommodate functionalist theories of mind. If mental states are *composed* of brain states, then this mereological relationship means that the realized whole is identical to the sum of the realizers. The problem, Polger writes, is that functionalist theories of mind individuate mental states by their function, not by the "stuff" they are made of (ibid., 257). This is an interesting objection, and it enjoys worthy, robust debate in the literature on philosophy of mind. However, the objection does not apply to emergence in population biology. For adaptive evolution, the underlying "stuff" matters.

While it is true that the selection for eyespots on butterfly wings can be multiply realized by an infinite number of combinations of butterflies, it cannot be realized by a population of platypuses. Because the underlying matter matters for the emergence of natural selection, then even in Polger's case, we should locate the relationship between individuals and evolving populations at the intersection of the two sets: composition and realization.

Having grounded both RC and EC in their relevant literatures, I now turn to the dilemma that EC poses for RC.[10] In the following sections, I will consider both horns of the dilemma (Figure 1). The proponent of the EES may either accept that natural selection is an emergent causal process or deny it, and I will outline what I understand to be the main costs of either choice. As we will see, each horn threatens the claim that the bidirectional model of natural selection and niche construction captures a reciprocally *causal* relationship.

[10] Given that niche construction is my example of choice for the dilemma, and that beavers are quintessential niche constructors, I exercise laudable restraint by resisting the urge to title this paper, "Dammed If You Do, Dammed If You Don't."

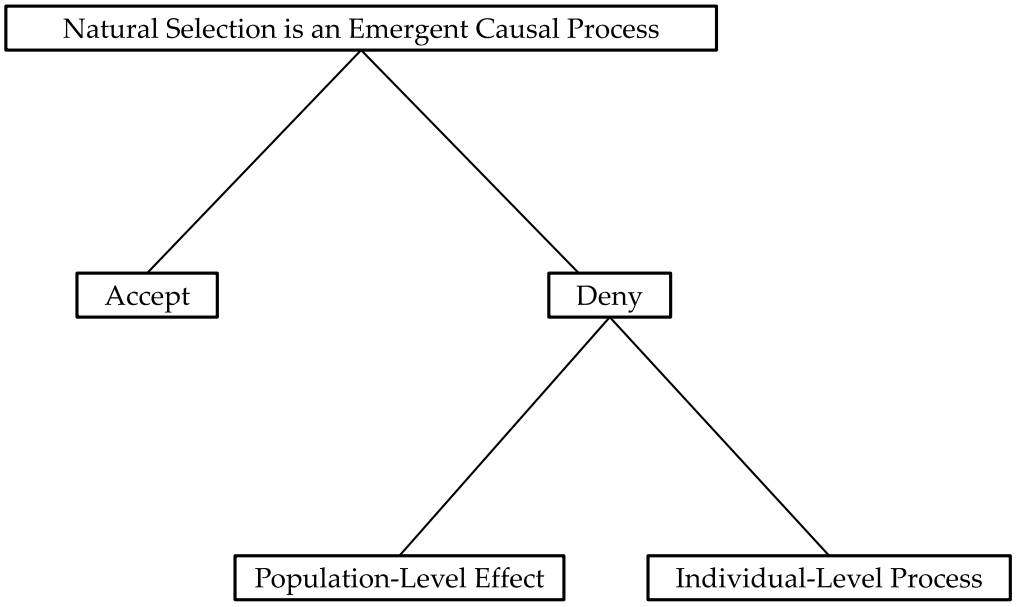

Figure 1. An emerging dilemma for reciprocal causation.

4. Affirming EC

Our dilemma begins with a simple, exclusive disjunction: either natural selection is an emergent causal process or it is not. I have argued for affirming the first disjunct: natural selection is an emergent causal process. Regardless of whether I am correct, the nature of this tautologous disjunction demands that any metaphysical picture of adaptive evolution must affirm one proposition or its negation. In the following section, I will assume that phenomena such as niche construction are individual-level processes—i.e., causal processes that are enacted by individual organisms, not entire populations. In Section 4.1., I will demonstrate that this assumption entails cycles of interlevel causation, which creates trouble for RC. In

Section 4.2, I consider an objection to this assumption and then attempt to discharge it. In Section 4.3., I consider whether Woodwardian interventionism rescues us from the dilemma.

4.1. A Kimian Problem for Reciprocal Causation

If we accept that natural selection is an emergent process and niche construction is an individual-level process, and we want to argue that these two processes both cause and are caused by each other, we are immediately hurled into the debate over interlevel causation. This problem will be familiar to anyone acquainted with Kim's rejection of downward causation (e.g., 1993). Indeed, the analogy between Kim's causal exclusion argument and the emergence of natural selection has been made by several others (Shapiro and Sober 2007; Haug 2007; Pence 2021), but a brief recapitulation will help us see the problem clearly.

Imagine that an episode of natural selection is counterfactually dependent on an episode of niche construction. If the niche had not been modified by the organism, then no variation in fitness would have been introduced to the population, and no selection would have occurred. It appears, prima facie, that a counterfactual theory of causation would deliver a clear verdict: the act of niche construction caused the episode of selection. But, in this case, the counterfactual dependence is deceptive. This is because counterfactual dependence of natural selection on niche construction does not reveal a *causal* relationship, but a *compositional* one. Processes at the population level are not caused by processes at the individual level—the former are composed of the latter. Because compositional relationships bear this counterfactual dependence, they superficially satisfy a key requirement for some

accounts of causation. For example, it makes some sense to say that had the mean kinetic energy of the molecules been different, the temperature of the system would have been different. Yet many would balk at the idea that mean kinetic energy *causes* temperature. Therefore, if proponents of reciprocal causation accept that natural selection is an emergent causal process, they are immediately faced with the problem of spurious interlevel causation.

Craver and Bechtel address the possibility of interlevel causation in (2007). On their view, we should reject causation between levels as the relata in question "are not distinct events, objects, or processes…They coexist with one another, and so there is no possibility of their *coming to* spatiotemporally intersect with one another" (552, emphasis in original). This point from Craver and Bechtel is especially relevant when we take seriously the emergent nature of selection: it is a population-level process that is coextensive with the interactions between the individuals that compose the population and their selective environment. In other words, on the emergent view, the process of niche construction does not cause the process of natural selection. Instead, niche construction is one of the many individual-level processes that *realize* the process of natural selection via composition. What does this mean for reciprocal causation? It means that causal reciprocity between the processes of niche construction and natural selection is conceptually incompatible with the emergent character of natural selection.

4.2. Objection and Reply

The reader may oppose my claim that niche construction is an individual-level process. If we

can demonstrate that niche construction is itself a population-level process, then we can deny that it stands with natural selection in a part-whole relationship. Perhaps natural selection and niche construction are two distinct processes that populations can engage in, each one begetting the other. This would allow us to sidestep the problem of spurious interlevel causation, perhaps thereby restoring compatibility between EC and RC. After all, niches may be occupied by entire populations, and they may likewise be constructed by entire populations. A group of beavers, for instance, may collectively build a dam, and they all profit from its shelter. This may be true. However, several responses to this objection suggest that it is overly restrictive in some cases and that it undermines key desiderata of the EES.

First, treating niche construction as a population-level process is overly restrictive in some cases—viz., those cases where an organism's *experience* of a niche is modified despite the lack of any physical modifications. Consider, for example, Chiu's discussion of "experiential niche construction" in (2019). In these cases, an organism develops in such a way that the environment presents it with new affordances (to borrow a term from Walsh 2022). The caterpillar that metamorphoses into a butterfly is a niche constructor—not simply because it creates its chrysalis, but because, once it ecloses, its new morphology enables it to experience an entirely new suite of environmental affordances. When we treat niche construction as a population-level process, we fail to account for cases such as these.[11]

[11] An anonymous referee objects that a similar argument could be developed for natural selection: if selection is an emergent phenomenon, then, on my view, it fails to account for

Second, treating niche construction as a population-level process may undermine key desiderata of the EES. A central tenet of the EES is that proximate processes have a rightful place in evolutionary explanations. In keeping with Mayr's distinction, these proximate processes are cashed out at the individual level, while ultimate causes are cashed out at the population level (e.g., Ariew 2003, Brown 2021). Consider recent arguments for the

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changes in the life history of an individual. The problem, the referee suggests, is that this precludes us from being able to invoke natural selection as an explanans for the life history of an organism. But this is exactly what I mean to show by endorsing an emergent account of natural selection. Since it is multiply realizable, selection *does* fail to account for individual life histories. Any number of combinations of life histories could give rise to the same higher-order process of selection, the same variations in trait fitness. It is helpful to recall Kitcher's defense of anti-reductionism, wherein he writes that higher-order explanations explain by appealing to robust phenomena, "not by rehearsing the gory details" (1984, 370). Natural selection explanations fail to account for individual life histories because they are, to borrow Kitcher's language, *autonomous levels of biological explanation* (ibid., 371). Sober is similarly committed to this kind of abstraction in (2001, 26): "evolutionary theory shows scant interest in individual organisms but prefers to talk about the fitness values of traits…Charlie the Tuna is not a particularly interesting object of study, but tuna dorsal fins are." Sober's point applies here mutatis mutandis: Heimlich the Caterpillar is not an interesting object of study for the evolutionary biologist, but butterfly metamorphosis is.

importance of "organismal agency" in the EES. This is definitionally an individual-level phenomenon, foundational to the "organism-centered perspective of the EES" which "captures the idea that organism-centered—rather than gene- or population-centered—explanations of evolution provide a perspective, often neglected since the mid-twentieth century, that would broaden our understanding of evolution" (Baedke and Fábregas-Tejeda 2023). Similarly, niche construction is described by architects of the EES as a *developmental* process that "depends on the environment-altering activities of phenotypes during ontogeny" (Laland et al. 2013, 737). The development of a phenotype is an individual-level process. That a set of token individuals exhibits the same type of process does not make it a population-level process. This is importantly distinct from the emergent account of natural selection, wherein the causally relevant features belong to the population, not its constituents.

Still, my interlocutor may reply that a pluralistic concept of niche construction is warranted. Despite the responses I have just provided, the fact remains that some examples of niche construction appear to be indisputable population-level processes. Robert Brandon has suggested that phenomena such as coal mining are good candidates for emergent processes of niche construction (personal communication). They satisfy the same two conditions that natural selection satisfies: the process of coal mining is synchronically dependent on the activities of the coal miners, and it has a distinct causal profile (e.g., atmospheric pollution) that is irreducible to any of the individual miners. In such cases, it seems I must accept that natural selection and niche construction are processes that occur on the same level of biological organization. Perhaps so. Nevertheless, where we locate niche construction on the

biological hierarchy is immaterial for the objection I am leveling against RC in this section. Why? Because the force of my objection comes from the claim that natural selection is an emergent process whose relata are variation in trait fitness and changes in trait frequency. Differences in trait fitness are realized by the interactions among a population of organisms within a common selective environment. Therefore, even when we consider cases of niche construction by entire populations, such as the community of coal miners, it does not change the fact that the variation in trait fitness (e.g., variation in the ability to withstand exposure to coal dust) is synchronously realized, not caused, by the niche-constructing interactions of the community within its environment. No matter where we locate niche construction on the biological hierarchy, it cannot *cause* natural selection.[12]

4.3. A Woodwardian Reply to the Kimian Problem

How can the proponent of reciprocal causation reply to the problem of spurious interlevel causation that I raised in Section 4.1? An appealing strategy is to invoke an interventionist theory of causation. James Woodward has, in several places, defended the possibility of

[12] In limiting cases such as these, it may be more precise to speak of realized processes (such as natural selection) and realizing processes (such as niche construction) rather than population- and individual-level processes. However, due to the ubiquity of individual-level instances of niche construction, I continue to use this terminology throughout the rest of the paper.

interlevel causation using an interventionist framework (2015; 2020; 2021). Although Woodward's arguments are concerned with Kim's "causal exclusion" problem—i.e., he is concerned with the possibility of interlevel interactions between brain states and mental states, not niche construction and natural selection—they are helpfully analogous to the issue at hand. Consider, for example, his remarks in (2015):

> …according to interventionism, a sufficient condition for a mental property (state etc.) represented by a value of variable M1 to cause some other mental property represented by value M2 (or a physical state P2) is that changes in M2 (or P2) be associated with changes due to interventions on M1, given satisfaction of the other conditions detailed in [Woodward 2003]…Looking at matters this way, it seems that it is appropriate not only to draw an arrow from M1 to M2 when interventions on M1 are associated with changes in M2, but also to draw an arrow from M1 to P2 in such cases, since P2 will also change under interventions on M1. (Woodward 2015, 321-22)

The objection to causal exclusion is simple: on an interventionist account of causation, X causes Y when "(i) there is a possible intervention that changes the value of X such that (ii) if this intervention (and no other interventions) were carried out, the value of Y (or the probability of some value of Y) would change" (Woodward 2003, 45). Therefore, if we—holding everything else fixed—intervene on a mental state at one time and observe a corresponding change in the underlying physical state at a subsequent time, we are justified in calling the mental state a cause of the physical state. Case in point (according to

Woodward): interlevel causation is not only possible but ubiquitous within an interventionist framework.

It appears that proponents of reciprocal causation have recourse to a similar defense, allowing them to accommodate my claim that natural selection is an emergent causal process. Following Woodward's example of mental causation, one might argue that even though the process of natural selection is composed of ecological and developmental processes such as niche construction, the latter may still causally impinge on the former. We can see how this response would play out by imagining a very simple case. Consider a population of organisms in a common selective environment E_1 at time t_1. The organisms are of three different types: A, B, and C. In E_1, all three types share the same fitness value. No selection is operating in our population in E_1 at t_1, as there is no variation in fitness between types. However, at t_2, a type A organism modifies the environment. This can happen in several ways—it might chemically alter the soil or physically alter the forest, etc. In any case, call this modified environment E_2. Suppose that E_2 completely replaces E_1, and that in E_2 our three types of organisms no longer share the same fitness value. In E_2, A is fitter than B, and B is fitter than C. As a result, there is variation in trait fitness in E_2 at t_2. Next, at t_3, imagine that all C organisms have perished; now, two-thirds of the population are A organisms and one-third of the population are B organisms.

With this albeit abstract example, it appears prima facie that we have just provided proof of concept that RC is compatible with an emergent characterization of natural selection. In one direction, we see an individual-level variable, niche construction, causing a

change to a population-level variable, reproductive success. By altering the environmental state from E_1 to E_2, a single organism has spurred an episode of selection. In the other direction, natural selection increases the probability that a future organism in the population will be a niche constructor (given that A, our niche-constructing phenotype, is fittest in the new environment).

Has interventionism rescued RC for an emergent metaphysics? I say no. But I must be careful in my response. I do *not* claim that interventionism fails to rescue RC because interventionism fails to legitimize interlevel causation per se. I do not hold such a view. Woodward is correct in claiming that interlevel causation is completely defensible under the right circumstances. Attempting to argue otherwise quickly devolves into a terminological dispute about causation—philosophers, replete with metaphysical zeal, banging their fists on tables (or, depending on who you ask, simples arranged table-wise).

What, then, are the right circumstances for a relationship to be characterized as interlevel causation on Woodward's account? His answer is clear: for variables to stand in causal relationships, they must be "independently fixable" (Woodward 2015, 2021).[13] Two

[13] "(IF): a set of variables V satisfies independent fixability of values if and only if for each value it is possible for a variable to take individually, it is possible (that is, 'possible' in terms of their assumed definitional, logical, mathematical, *mereological or supervenience relations*) to set the variable to that value via an intervention, concurrently with each of the

relata are independently fixable when it is possible to set the variable representing one relatum to one value without simultaneously wiggling the variable that represents the other relatum. The example Woodward gives is cholesterol: you cannot hold total cholesterol and high-density cholesterol fixed while intervening on low-density cholesterol. This is because total cholesterol is *realized by* the other two values. Therefore, since total cholesterol cannot be fixed independently of low-density cholesterol, these two relata do not stand in a causal relationship. Similarly, the relata in RC cannot be independently fixed. This is because one relatum (e.g., the activities of organisms) partially constitutes the other relatum (distributions of trait fitness). Indeed, Woodward says explicitly that "the feature of a part/whole relation that precludes causation is a failure of independent fixability" (2021, 230). Moreover, this insight has been previously demonstrated with respect to natural selection, specifically, by Shapiro and Sober:

> . . . while it is true that natural selection is not distinct from its supervenience base in a given token selection process, this is not a reason to deny that selection is a cause. In the same way, we regard the temperature, pressure, and volume of the gas in a container as causes even though they supervene on the states of the molecules making up the gas. Walsh demands that selection contribute something to evolution beyond the contributions made by the causal processes that impinge on individual organisms,

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other variables in V also being set to any of its individually possible values by independent interventions" (Woodward 2015, 316, my emphasis).

> just as Kim demands that mental properties have powers in addition to those of their supervenience bases. Of course selection cannot do this, but that is no argument against its causal efficacy. To assess whether X causes Y, you shouldn't try to hold fixed the micro-supervenience base of X while wiggling X. (2007, 251)

It is true that wiggling the bivalued variable "is a niche constructor" at one time will, under the right circumstances and controlling for the necessary background conditions, cause a change in the many-valued variable "differences in reproductive success" at another time (as demonstrated in Clark et al. 2020, for example). But such a relationship is not synonymous with the *process* of niche construction causing the *process* of natural selection. Why not? Because as soon as you intervene on the variable "is a niche constructor," you have *simultaneously* intervened on (or perhaps introduced) the emergent variation in fitness and have therefore intervened on (or perhaps begun) the process of natural selection.[14]

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[14] An anonymous referee objects that intervening on the *capacity* for niche construction alone (by introducing genes, for instance) is insufficient to impinge on a population's variation in fitness. This would also require the capacity to be actualized—for the niche constructing genes to be expressed, and for the environment to be physically modified. But if one assumes a propensity interpretation of fitness (sensu Mills and Beatty 1979), then this objection does not work. Understood this way, fitness is forward-looking; it is a probabilistic notion. Therefore, an intervention on niche constructing genes can indeed beget a corresponding change in trait fitness values. A brief example will help to make this point. Imagine that

Intervening on the process of niche construction intervenes on a *part* of the process of natural selection. Niche construction and natural selection *qua processes* are, therefore, not sufficiently distinct to stand in a causal relationship.

It appears that the intuitive force of the interventionist objection results from a conceptual ambiguity: equating natural selection with a difference in reproductive success is to conflate the *process* of natural selection with the *product* of natural selection (Millstein

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Heimlich the Caterpillar will develop into a Batesian mimic. He is not noxious to predators but will seamlessly imitate butterflies that are. Moreover, Heimlich is the only mimic among a population of caterpillars who will metamorphose into noxious butterflies. Is the mimicking trait fit in these circumstances? Very. The predator will probably learn to stay away from the aposematic warnings on the Heimlich's wings. Now, say I intervene on the genomes of every single caterpillar except Heimlich, and I swap out their noxious genes for non-noxious genes. Is the mimicking trait still as fit in this scenario as it was in the last? Certainly not. Why not? Because, in this scenario, the predator is far less likely to associate Heimlich's wings with danger. He is now much more likely to be eaten since his mimicking phenotype will afford him no protection. The fitness advantage conferred by his mimicry has decreased dramatically, but all I have done is swap out a gene in the surrounding caterpillars. A simpler way of putting the response is this: the genes of the members of my population *are part of my environment*, and therefore my fitness—which is indexed to my environment—depends in part on my neighbors' genes (see, e.g., Sterelny and Kitcher 1988, 345).

2002; Stephens 2004; Brandon 2005). Turning niche construction on or off can cause an evolutionary response—a generational change in trait frequencies. But it does not *cause* the process that results in such a response. Why? Because it *is part of* the process that results in such a response. That is, the individual-level process of niche construction causes an evolutionary outcome via its population-level counterpart—natural selection. This idea has received significant resistance from EES proponents, but I believe the resistance is misguided. Changes to fitness are the waystations through which an individual organism can impact the evolution of a population. A lone beaver can build a dam, but this act will not have adaptive consequences unless there is a population of beavers to vary in fitness.

Notice that a conclusion immediately follows from this argument, one that is quite friendly to proponents of the EES. If, as the interventionist reply suggests, building a dam at one time can cause an evolutionary outcome at another time, then niche construction (and, for that matter, *any* individual-level process that results in the same outcome) is an evolutionary process. It features in evolutionary explanations, it begets evolutionary outcomes, and it—along with natural selection—bears causal responsibility for adaptation. This is a significant desideratum of the EES, as shown in Section 2. Acknowledging niche construction as part of the supervenience base for natural selection satisfies this desideratum. Whether an organism is or is not capable of modifying its niche, and the degree to which it is capable of this, can be a bona fide cause of evolution—the *product* of natural selection—within an interventionist framework. It achieves this status through its ability to partially compose and thereby realize the emergent *process* of natural selection. However, this

requires the EES proponent to relinquish the claim that the two processes are causally interdependent. In other words, they must deny RC.

5. Denying EC

Of course, one way the EES proponent may respond is to simply deny my claim that natural selection is an emergent causal process. There are two ways they might do this. They can deny that natural selection is an *emergent* process, or they can deny that natural selection is a *causal* process. Denying that natural selection is a causal process amounts to the "statisticalist" position (Matthen and Ariew 2002; Walsh et. al 2002; Walsh et al. 2017). On the statisticalist view, the causal locus of evolution is at the individual level, and the only genuine causes of evolutionary change are those that introduce and remove individuals from a population (e.g., reproduction, birth, competition, death, etc.). Natural selection, it is argued, is a theoretical construct that describes a certain pattern of individual births and deaths within a population—a statistical trend and nothing more.

The advantage of the statisticalist view is that it allows EES proponents to privilege the causal feedback between organism and environment. Organisms modify the environment, and features of their modified environment (at least partly) cause the organisms to perish or survive and reproduce. The untenable cost of the statisticalist view, however, is its denial that natural selection is a causal process at all. Because it denies the causal efficacy of natural selection, statisticalism entails a rejection of RC.

The incompatibility between statisticalism and reciprocal causation is explored in a recent chapter by Denis Walsh, a prominent statisticalist (2019). However, Walsh is also at the forefront of the recent trend toward recognizing organismal "agency" in evolution—a position that is happily accommodated by the EES (Walsh 2015; Sultan et al. 2022). How are these two seemingly contradictory positions (pro-extension and anti-causalism) compatible? Walsh gives us an answer in (2019): "The error [of reciprocal causation] lies in construing individual-level causes and ensemble-level processes as somehow on an ontological par, as interacting causes of ensemble change…But this is a category error" (237). He continues, "The revisionists are right in insisting that there is no such thing as an ultimate cause as distinct from a proximate cause of evolution. All the causes of evolution are causes of individual living and dying. It is 'proximate' causes all the way down" (242). According to Walsh, while there are two levels of explanation in evolutionary biology (the individual and population levels), there is only one level of causation (the individual level). In other words, statisticalism is entirely compatible with elements of the EES, such as the emphasis on organismal agency and developmental bias. But statisticalism is not compatible with reciprocal causation—a "central" and "unifying" theme of the EES (Buskell 2019). This is because statisticalists not only deny the distinction between proximate and ultimate causes, but the existence of ultimate causes altogether. Therefore, the rejection of a bidirectional model between ultimate causes (such as selection) and proximate causes (such as organismal agency) follows *a fortiori* from the rejection of ultimate causes.

Therefore, if EES proponents wish to deny my claim that natural selection is an emergent causal process, they must do so by maintaining that natural selection is indeed a causal process, but one that operates at the individual level. Again, as briefly mentioned in the previous section, this view has been argued by several philosophers (see Bouchard and Rosenberg 2004; Pence and Ramsey 2015; Triviño and Nuño de la Rosa 2016; Bourrat 2018; Pence 2021). The various iterations of this view make unique contributions. At present, however, we are interested in the central claim that unites these positions under the individualist umbrella: in the process of natural selection, *individual fitness* is the cause. This view differs from statisticalism by affirming the genuine causal efficacy of natural selection, but it aligns with statisticalism by identifying the locus of causation at the individual level.

Reciprocal causation between niche construction and selection seems straightforwardly compatible with an individual-level account of selection since, on such an account, both processes occur at the same level of biological organization. By placing the causal locus of selection at the individual level, one might (again) conclude that we have sidestepped the problem of spurious interlevel causation. However, this conclusion would (again) be premature. As it turns out, the following objection gives us reason to doubt the conceptual coherence of an individual-level account of natural selection.

The objection, made by Millstein in (2006) and reiterated by Sober in (2013), may be put as follows: selection requires *relative* fitness—more specifically, a *difference* in relative fitness. But relative fitness is a relational property, meaning it requires more than one individual. In other words, an individual is not relatively fit or unfit until it faces the same

selection pressure as other individuals. Saying that natural selection is an individual-level process is like saying that I won (or, more realistically, lost) a race where I was the sole contestant—it is meaningless. Even in the most extreme example, in a population composed of two individuals, no selection can occur until there is *variation* in fitness. This variation is not a property of either individual, but a property of the pair. Therefore, an individual-level account of selection is conceptually incoherent because selection requires relative fitness and relative fitness requires a population.[15]

In sum, denying EC means we must either endorse a non-causal account of selection or a conceptually incoherent account of selection. Neither can rescue RC. If the analysis in this section is correct, then an individual-level account of selection must ultimately yield to a population-level account. It therefore seems that if we wish to affirm the causal efficacy of natural selection, which RC requires, then we cannot deny EC. We find ourselves back at the first horn of the dilemma.

6. Conclusion

No available metaphysical account of natural selection is compatible with the view that selection is both a cause and effect of developmental processes such as niche construction. If EES proponents accept that natural selection is an emergent causal process, then they must accept that selection is realized by—because it is composed of— the interactions between

[15] My rejection of individual-level selection is a notable departure from Pence (2021).

individuals and their environment, but it is not caused by them. If proponents wish to deny that natural selection is an emergent causal process, then they have two options: deny that natural selection is an *emergent* process or deny that natural selection is a *causal* process. Of course, denying the causal efficacy of natural selection is a non-starter for the proponent of reciprocal *causation.* And, as Millstein has shown, an account of selection as an individual-level process is really no account of selection at all.

My goal in this paper has been to put the concept of reciprocal causation in conversation with the broader literature on causation in biology. That the discussion about reciprocal causation has gone this long without explicitly considering statisticalist, emergentist, and interventionist arguments demonstrates the disconnect between the EES and the broader field of philosophy of biology. In this paper, I have attempted to begin bridging that gap. Of course, there is much more work to be done.

In sum, I have argued that the proponent of the EES must deny RC. However, I wish to be unequivocal in saying that such a denial by no means undermines a very important goal of the EES—to privilege the role of niche construction (and other developmental or ecological processes) in adaptive evolution. This goal can still be achieved without insisting that the processes of natural selection and niche construction are *causally* interdependent. Niche construction is undoubtedly an evolutionary process and, as such, has a rightful place in evolutionary explanations. But we must not introduce such processes into our explanations in a way that undermines the contributions philosophy has made to the metaphysics of biology.

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