THE SCOPE OF RECIPROCAL CAUSATION

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Abstract

The role of reciprocal causation in Extended Evolutionary Synthesis (EES) is controversial. Proponents of EES argue that reciprocal causation is a key innovation, underpinning the necessity of EES. Conversely, critics of the EES maintain that Standard Evolutionary Theory (SET) adequately encompasses the concept of reciprocal causation, challenging the need for EES. This skepticism is rooted in two primary critiques. First, the mischaracterization of causal dynamics within SET by EES advocates leads to a *misrepresentation* of SET. Second, the oversight of how SET incorporates and acknowledges instances of reciprocal causation leads to claims about *the empirical inaptness* of SET. As a result, the debate has reached an impasse, with limited progress towards a constructive examination of reciprocal causation's significance to evolutionary explanations. This paper introduces the *scope argument,* which examines reciprocal causation through timescales and grain of explanations. This approach revitalizes the debate in two ways. First, reframing the debate in terms of scope clarifies the role of reciprocal causation by allowing research programs to specify targets of explanation. Second, the elements of scope (timescales and grain) elucidate the epistemic advantage of reciprocal causation in the respective research programs in question.

Keywords

Reciprocal Causation; Extended Evolutionary Synthesis; Standard Evolutionary Theory; Modern Synthesis; Evolutionary Theory; Scope; Niche Construction.

Statements and Declarations

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1. Introduction

Over the past decades, some biologists have claimed that evolutionary theory needs to be expanded to acknowledge evolutionary processes pertaining to development and ecology (Laland et al. 2014; Laland et al. 2015; Müller 2017; Pigliucci and Müller 2019). This proposal – the Extended Evolutionary Synthesis (EES) – claims to account for processes said to be overlooked in Standard Evolutionary Theory (SET). Such claims have generated a controversial debate in evolutionary biology. Some defend that EES is necessary to provide a more accurate picture of evolution. Others argue that EES may be superfluous given a charitable account of SET as a pluralistic framework that is open to accommodate empirical evidence from fields such as developmental biology and ecology[1]. Among other things, proponents of EES argue that reciprocity between organisms and environment is a key driver of evolutionary change. Such reciprocity is captured by the broader notion of reciprocal causation: the idea that organisms can be both causes and effects of evolution (Buskell 2019). Some accounts of reciprocal causation include relations other than reciprocity between organism and environment, such as gene-environment, gene-population or population-population relations (Baedke, Fábregas-Tejeda, and Prieto 2021). Examples of reciprocal causation include negative and positive frequency dependent selection, co-evolution (Svensson 2018), developmental plasticity and robustness (Schwab, Casasa, and Moczek 2019), and niche construction (Laland, Odling-Smee, and Feldman 2019). Reciprocal causation is a defining aspect of EES. Specifically, EES proponents argue that a major conceptual revision of SET is warranted on the grounds that SET neglects or sidesteps reciprocal causation in evolutionary explanations. Whether or not this is the case, however, remains unclear.

In this paper, my aim is to clarify the scope of reciprocal causation. Reciprocal causation can be considered a generic descriptor of causal relations between entities that alternate between being causes and effects. In the EES debate, the focus is often placed on the particular case of reciprocal causation between organisms and environments (henceforth referred to as "reciprocal causation" for simplicity). However, reciprocal causation also encompasses reciprocity between populations and environments as well as between genes and environments (Müller 2017). I argue that in either way one chooses to consider reciprocal causation, the scope of reciprocal causation can be

[1] An example of the debate and controversy can be seen in a debate in *Nature* between Laland et al. (2014) and Wray et al. (2014).

understood in terms of timescales and the level of grain of explanations. I show that timescales and grain receive different emphasis in the EES and in SET respectively, but that reciprocal causation is nonetheless compatible with both frameworks. While this paper reaches a somewhat deflationary conclusion, it nevertheless aims at providing useful tools to clarify what is at stake in the debate surrounding reciprocal causation. Clarifying the scope of reciprocal causation is an important step in encouraging greater integration between research fields in present-day biology, thus making the debate more constructive. What is novel about reciprocal causation in EES is its deployment within explanations that differ in scope from those in SET. This reorientation is vital if EES proponents wish to achieve a more defensible research program by clearly specifying the relevant areas where EES provides epistemic advantage over SET.

A few clarifications are key at this stage. First, the target of this paper is the ongoing *debate* about the need or not of an EES, and not the empirical *content* of the theories themselves. As such, the paper is aimed at clarifying the ideas and arguments that underpin recent debates about theory change in evolutionary biology. Second, the use of the term 'Standard Evolutionary Theory' may be seen as problematic since SET is a term often used by EES proponents to portray it as a monolith. Here I take SET to be an umbrella term that encompasses paradigms held by the Modern Synthesis (MS) and later on, synthesized in the advances in molecular biology and genetics of the second half of the 20th century. SET is the main target of criticism of EES proponents. To be sure, this paper will adopt SET as terminology to match existing literature on the topic (such as Buskell 2019; Baedke, Fábregas-Tejeda, and Vergara-Silva 2020; Hazelwood 2023), while being aware that it is an oversimplification. The term is thus used for pragmatic reasons, while acknowledging its limitations and pointing to the problematic aspects of this label.

The paper is structured as follows: in Section 2, I examine current criticisms of reciprocal causation; Section 3 analyzes how reciprocal causation and niche construction are intertwined; I explain the scope argument in Section 4 and show the relevance of timescales and grain of explanations to generate constructive debate surrounding reciprocal causation. I address challenges to the argument in Section 5, while Section 6 explains how the scope of reciprocal causation can help reorient the debate surrounding reciprocal causation. Section 7 presents concluding remarks.

2. Current Criticisms of the Necessity of EES

2.1. A primer on evolutionary causation:

Debate surrounding the necessity of an EES is frequently framed in terms of different causal accounts such as unidirectional causation, reciprocal causation and, more recently, multilevel causation (Martínez and Esposito 2014).

Mayr's (1961) account of causation relies on an initial distinction between two research agendas—functional and evolutionary biology—and two kinds of biological causes—ultimate and proximate. The functional biologist is concerned with how something operates and functions, while the evolutionary biologist is concerned with why it functions and operates as it does. The functional biologist is concerned with the study of proximate causes: an immediate set of developmental and molecular causes that comprise organisms, such as their physiological constitution, and which impact an organism's interaction with the environment over the course of its lifetime. The evolutionary biologist is concerned with "the causes that have a history and that have been incorporated into the system through many thousands of generations of natural selection" (Mayr 1961, 1503).

By contrast, "reciprocal causation" refers to the feedback interactions between organisms and their environment. The idea of reciprocity can be found, as Baedke and Fábregas-Tejeda (2023) show, as early as in twentieth century organicism. Later, Richard Lewontin (1985; 2000) further developed this notion, emphasizing its relevance for evolutionary explanations. Lewontin defends a view according to which there is a reciprocal relation between genes, organisms, and environment; and that each of these elements "can be both causes and effects" (Lewontin 2000, 100). His portrayal of reciprocity between organisms and environment contrasts with unidirectional causation, a view that EES advocates attribute to Mayr's proximate/ultimate distinction.

Mayr's account has been the target of criticisms on the grounds that proximate and ultimate causes do not take into consideration the reciprocity between organisms and environment (Laland et al. 2013). Additionally, critics argue that proximate developmental causes are not evolutionarily relevant because they are constitutive of the organism and as such, are not causally efficacious. Therefore, a satisfactory explanation of evolutionary causes requires a novel causal notion: reciprocal causation. Such criticism is a prelude to the debate surrounding the role of reciprocal

causation in EES; reciprocal causation has gained strength as a third type of causation beyond Mayr's (1961) proximate-ultimate causes. Consider the following definitions of reciprocal causation:

> (1) "Reciprocal causation captures the idea that developing organisms are not solely products, but also causes, of evolution. 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)
>
> (2) "Reciprocal causation is a common feature of both evolving systems (when the activities of organisms modify selective environments) and developing systems (where development proceeds through modification of internal and external environments)." (Laland et al. 2015, 6)

Broadly, reciprocal causation is a concept at the heart of arguments defending the need for an EES. As Gefaell and Saborido (2022) show, the four scientific principles upon which the EES is based are evolutionary developmental biology, developmental plasticity, inclusive inheritance, and niche construction. Transversal to those principles are two meta-scientific principles encompassed by the notions of reciprocal causation and constructive development. EES proponents claim that the explanatory emphasis that such processes receive in their research program provides EES with an epistemic advantage over SET. Most importantly, while some may concede that *some* instances of reciprocal causation do feature in SET (for example, between populations and selective environments), other cases are scarcely considered, such as reciprocal causation between organisms and environments.

By incorporating reciprocal causation in evolutionary explanations, the EES is said to represent a major innovation when compared to SET, which justifies the need for theory change. This claim, however, has been criticized by EES skeptics who highlight two flaws in the debate. Correctly identifying such flaws challenges the epistemic contribution of reciprocal causation to the EES. In what follows I discuss these two flaws separately for the purposes of clarity.

2.2. The flaw of misrepresentation:

A main source of skepticism towards EES arises from rhetoric that misportrays SET. This flaw has its origin in ambiguity about what the term 'reciprocal causation' encompasses. For instance, there seems to be a conflation between reciprocal causation *simpliciter* and the specific case of

reciprocal causation between organism and environment. Such ambiguity leads to defensive claims about reciprocal causation being adequately captured in SET. EES critics argue that it is not accurate to portray SET as a theory whose main causal accounts are unidirectional[2] and that it is a mistake to assume that SET is a monolith rather than a pluralistic framework accommodating different approaches and techniques to study causal interactions in evolution. But ambiguity remains as to whether this includes organism-environment reciprocal causal relations.

An example of such criticism coming from EES proponents can be seen in Müller (2017) who specifically states that causal reciprocity is a distinctive feature of EES in two domains: first, in the construction of phenotypic complexity (through DNA influencing cells, tissues and organs and through environmental effects on tissue-induced gene regulation) and second between *populations* and their selective environments. This characterization of reciprocal causation *simpliciter* encompasses relations at many different levels and is framed as a *general* criticism of SET as being excessively unidirectional in its causal explanations. Ambiguity about the target of reciprocal causation generates frustration and warrants further clarifications to allow for more fruitful debates.

As philosophers have clarified, however, some EES criticisms target a more specific blindspot of SET: reciprocal causation between organism and environment (Baedke, Fábregas-Tejeda, and Prieto 2021; Ramsey and Aaby 2022). The key point of contention in this particular case is the alleged epistemic irrelevance of organism-environment causal interactions in SET.

[2] Such misrepresentation has ignited controversy between theoretical biologists. For example, Lu and Bourrat (2018) argue that EES proponents' criticism of SET is based on a semantic confusion between the evolutionary and the molecular concepts of *gene*. Gupta et al. (2017) argue that it is misguided to claim that SET is narrowly gene-centric and ignores the richness and complexity of the living world. Similarly, Charlesworth et. al. (2017) demonstrate that evolutionary biology is not "dogmatically adaptationist" (2017, 9), despite too often being portrayed as such. Lastly, Dickins and Barton (2013) show that Laland misinterprets Mayr's claims about proximate and ultimate causes. In other words and according to these authors, EES proponents' interpretation of SET is uncharitable.

2.3.The flaw of empirical inaptness:

EES critics also argue that to point out the *empirical inaptness* of SET with respect to reciprocal causation is flawed. This claim is often supported by empirical evidence that reciprocal causation is a well-acknowledged mechanism in SET and that modelling reciprocal causal relations is a widespread practice in SET. The most comprehensive formulation of such criticism can be found in Svensson's (2018) critique of reciprocal causation in the EES. It amounts to an enumeration of instances in which SET does in fact capture reciprocal causal interactions in evolutionary processes. For example, negative and positive frequency dependence selection, cases of co-evolution, and eco-evolutionary dynamics are examples of SET incorporating reciprocal causation. Indeed, the research field of eco-evolutionary dynamics (Hendry 2016) explicitly acknowledges two kinds of unidirectional effects: the effects of ecological changes on evolutionary processes and the effects of evolutionary changes on ecological processes. Thus, eco-evolutionary dynamics theorists plainly acknowledge that an important goal "should be to elucidate bidirectional eco-evolutionary interactions," also known as eco-evolutionary feedbacks (Pelletier, Garant, and Hendry 2009, 1584).

Arguably, one may claim that the abovementioned examples essentially focus on reciprocal causation between populations and environment, without acknowledging the more specific case of organism-environment relationships. This creates an additional tension in the debate. While there is compelling evidence that SET acknowledges reciprocal causation between gene-environment and population-environments, it remains unclear whether the specific case of organism-environment reciprocal causation is adequately taken into account. In fact, Baedke, Fábregas-Tejeda, and Prieto (2021) review a thorough collection of quotes by architects of SET, showing their careful attention to reciprocal causation focusing on gene-environment and gene-population reciprocal interactions. However, it remains clear according to these authors that the specific case of organism-environment reciprocal causation encountered major explanatory challenges in SET which account for the neglect of this specific kind of reciprocal causation. Moreover, Cortés-García and Etxeberria Agiriano (2023) also show that the modern evolutionary synthesis disregarded organismic-level processes. However, as I will argue in Section 4, reframing the debate in terms of scope helps to explain why SET may not be empirically inapt, even if the processes mentioned focus on the level of populations.

EES proponents further claim that SET neglects niche construction as a major evolutionary process, as a result of SET's alleged disregard for organism-environment reciprocal causation, of which niche construction is a clear case. However, Gupta et. al. (2017) demonstrate that niche construction has not been neglected in SET. For example, note Fisher's (1919) conceptualization of the rest of the genome: niche construction was well-acknowledged at the core of SET. *Prima facie*, if SET is able to account for niche construction, then because of the central role of the organism in this process, it must – at least implicitly – account for some version of organism-environment reciprocal causation. However, as I will develop in Section 5, the scope of explanations might place emphasis on populations, rather than on organisms. Which is not to say that ultimately, an important role for organisms *qua* parts of populations is assumed in SET.

So far, the two flaws presented encompass points of tension in the theoretical debate surrounding the EES with respect to the originality of reciprocal causation more broadly and of reciprocal causation between organism and environment. A common strategy in both cases is to insist on the prominence of reciprocal causation in SET, whether as a general causal notion or when focusing on particular organism-environment interactions. To move beyond such conundrum, I suggest a shift in the strategy for analysing this controversy. Specifically, I advance the *scope argument* and suggest a perhaps more constructive avenue for the debate to unfold.

The scope argument provides an auxiliary description of the way the two frameworks describe and use reciprocal causation in explanatory capacity. The goal is to clarify (1) the explanatory targets of EES and SET proponents when using the concept of reciprocal causation, and (2) the scope of such explanations.

Ultimately, the scope argument is pluralistic: it demonstrates that reciprocal causation can be fruitful in explanations that vary widely in scope[3]. As I will argue in Section 4, this difference in scope can be understood in terms of timescales and grain of explanations under each framework. The following section discusses niche construction as an empirical example of reciprocal causation that helps illustrate the scope argument.

[3] This pluralist stance aligns well with how concepts and explanations in biology are understood (Love 2012; Mitchell and Dietrich 2006; Sterelny 1996).

3. Niche Construction and the Role of Reciprocal Causation

While reciprocal causation is a general term that can apply to many biological processes, I focus here on niche construction as a specific type of reciprocal causation. Niche construction encompasses processes of modifying the environment in ways that favor survival and selection of the niche-constructing species (Odling-Smee, Laland, and Feldman 2003, Sultan 2015). For example, ants, termites, and earthworms modify soil conditions, creating environments which are beneficial to their survival (Gupta et al. 2017). The soil-altering activities of earthworms, for example, result in feedback effects that shape earthworms' survival rates, thereby acting as a selective pressure in a feedback loop between earthworms and soil. Another example of niche construction is the damming activity of the North American beaver, *Castor canadensis.* Beavers use specific trees to construct dams, and their dams shape streams and wetlands. This reshaping leads to "changed patterns of sedimentation and nutrient cycling," altering the biological community and leading to greater diversity of species in the area (Sultan 2015, 96).

Niche construction can be described as a reciprocal causal relation between organism (O) and environment (E), as follows:

> *Niche construction* occurs whenever a population *O* changes its relativistic niche by changing a factor in *E* relative to its own features. If, by modifying a factor, *O* also modifies a natural selection pressure for itself, then subsequently the change in the niche caused by *O*'s prior niche construction may feedback to *O* either to select for a change in *O*'s features or to counteract an independent change in *E*'s factors that would otherwise have selected for a change in *O*. It may thereby either create, preserve, or destroy a synergy or matching relationship between *O*'s features and *E*'s factors. (Feldman, Laland, and Odling-Smee 2013, 43)

Niche construction therefore assumes that there is a reciprocity between organisms and environments and that, by virtue of this bidirectional interaction, an additional set of evolutionary causes comes about. Strong empirical evidence showing that niche construction is a significant cause of evolution supports the so-called *niche construction perspective,* which overlaps with claims about reciprocal causation put forward by EES proponents (Aaby and Ramsey 2022; Laland and Sterelny 2006; Laland, Odling-Smee, and Feldman 2019; Sultan 2015).

Consider, however, the *Castor canadensis* example from the SET perspective. SET explains the beavers' dam-building behavior as an adaptation that is beneficial for the beavers' survival. In turn, their survival impacts future dam-building and shapes beaver evolution. Dawkins explains the process as follows: "the *variations* in replicators have a causal link to *variations* in dams such that, over generations, replicators associated with good dams survive in the replicator pool at the expense of rival replicators associated with bad dams. [...]The beaver dam is as much an adaptation as the beaver tail" (Dawkins 2004, 379, emphasis in original). Dawkins' idea is that dam-building alleles were repeatedly selected over time, and such phenotypic adaptations—which he calls *extended phenotypes*—can be explained just like other adaptations. This adaptation process is ultimately random since its primary cause is random variation, rather than intentional beaver behavior. On this view, SET's conceptual tools—variation and differential fitness as components of selection—sufficiently explain how selective pressures cause beavers to modify their environment.

Dawkins is the most oft-cited critic of the niche construction perspective in EES literature. However, due to the pluralistic nature of SET, a fairer portrayal must take into consideration that many skeptics of EES are likely to accept empirical results coming from niche construction theory. In fact, a more nuanced alternative is to claim that EES skeptics are not skeptical of the importance of niche construction per se, but rather, of whether these empirical results warrant theory change. To avoid misportrayal of SET, views other than Dawkins' must therefore be briefly considered. Take, for example, Scholl and Pigliucci (2014) who analyze recent criticisms to the proximate/ultimate distinction. For these authors, the question is not whether certain types of causes exist, but rather, which causes will be foregrounded or backgrounded in any given explanation. Hence, criticisms that target the proximate/ultimate distinction loose pertinence once the issue is clarified in terms of explanatory salience. Depending on research agendas, some sets of causes will be deliberately favoured over others in explanations. For example, in Mayr's selectionist focus, developmental causes will be backgrounded while natural selection is foregrounded. In evo-devo research programs, by contrast, developmental causes are foregrounded. This view is compatible with the scope argument, which can be seen as an addition to what Scholl and Pigliucci label "explanatory salience" (p. 660). Similarly, Conley's (2019) clarification of the proximate/ultimate distinction highlights the different emphasis placed in developmental or evolutionary processes in different research agendas. In sum, nuanced critiques

of causal accounts tend to converge on the claim that it is simply a mistake for EES proponents to focus on causal incompleteness (reciprocal or otherwise) as a shortcoming of SET because it relies on mischaracterizing SET and ignoring the question of explanatory focus within different research agendas.

Niche construction is therefore an example of a situation in which different explanations address the same phenomenon. In the case of dam-building, EES proponents and niche construction theorists disagree with views such as Dawkins', wherein underlying genetic variation is the main cause of dam building, on the grounds that these extended phenotype views preclude a role for agential behavior (Aaby and Desmond 2021). They argue that this is an incomplete picture of all the causal forces at play: niche construction and natural selection are concurrent processes that both shape evolution (Uller and Helanterä 2019). Indeed, organisms co-evolve with and causally affect their environments; the causal arrow runs not only from E → O, but also from O → E (Laland et al. 2015). In other words, when beavers engage in dam-building activities, they are not only propagating "dam-building genes," but also transforming their environments, "acting on a host of beaver traits, influencing subsequent beaver evolution" (Laland and Sterelny 2006, 1752). Hence reciprocal causation is crucial: it captures the effect of an organism's modification of its environment on the organism's selection over time.

In what follows, and in the vein of the more nuanced critiques of causal incompleteness, I will argue that focusing on whether reciprocal causation is a feature of SET is errant. The substantial questions concern the scope of reciprocal causation under each framework and the ability of reciprocal causation at each scope to support research programs. The first pertinent issue, to which I turn in the next section, is to clarify the explanatory capacities in which each EES proponents and skeptics of the extension are using reciprocal causation.

4. The Scope Argument: Timescales and Level of Grain

While the explanatory power of EES compared to that of SET has been discussed in the literature (see Baedke, Fábregas-Tejeda, and Vergara-Silva 2020), the question of scope remains unexplored (recently, Wagner and Tomlinson (2022) addressed the scope of evolutionary theory in a broader context). Here, I focus on the scope of reciprocal causation within explanations, rather than the

overall scopes of SET and EES. Specifically, I argue that there are at least two significant aspects of the scope of reciprocal causation: timescales and level of grain[4]. Considerations of scope help generate constructive debate surrounding reciprocal causation, revealing the innovative aspect of reciprocal causation in EES by distinguishing it from its use in SET.

The notion of *explanatory depth* (Hitchcock and Woodward 2003; Strevens 2008) will be useful here. Hitchcock and Woodward argue that the depth of some explanations is inversely related to their degree of generality: there is often a trade-off between explanatory scope (i.e., generality) and depth (Strevens 2011, 190). A causal explanation can be more or less fine-grained according to its degree of generality and, accordingly, its scope. Where SET focuses on much longer timescales and more coarse-grained population-level explanations, EES is interested in shorter timescales and more fine-grained explanations. The goal of the scope argument is to show that debates surrounding reciprocal causation can benefit from greater clarity about the explanatory goals within each framework. While the debate is frequently framed in terms of evolutionary causation *per se*, a focus on causal explanations may bring additional clarity to the debate. Ultimately, a more constructive debate would address the ability of each framework to offer explanations that identify the *specific causal mechanisms* responsible for salient features of evolution, allowing the discussion to move away from pointing out flaws such as misrepresentation and empirical inaptness of SET.

4.1 Levels of Scope

There are at least three levels at which one can talk about the scope of reciprocal causation in EES and SET: the overall scope of a research program, the scope of reciprocal causation within a program, and the scope of explanations in which it is used. The three levels are hierarchically related: the scope of explanations that use reciprocal causation depends on the scope of reciprocal causation within each research program, which in turn depends on the scope of the research

[4] Discussion of scope can arguably be construed differently, ontically or epistemically. For example, the discussion can also focus on entities that can legitimately be said to be engaged in feedback processes (organisms vs. populations; genes vs. populations, and so on). The discussion presented here does not aim to exhaust all possibilities for construing scope, but rather to focus on two dimensions in which scope can be fruitfully spelled out and channeled to redirect the current debates surrounding reciprocal causation.

program itself. I present each level in turn to subsequently focus on the scope of reciprocal causation within explanations in each framework.

First, the scope of a research program concerns its *problem agenda*, defined as "a 'list' of interrelated questions (both empirical and conceptual) that are united by some connection to natural phenomena" (Love 2008, 877). Scope here refers to the kinds of questions that are and are not included within each problem agenda. Novick and Doolittle (2019), for example, characterize "scope claims" as claims that are "relative to the state of biological science at any given time: what problems are of most concern, what alternative resources are available, and what organisms are most centrally studied." (p. 1) For example, in evo-devo, a core research area of the EES framework, questions tend to focus on the causal mechanisms that explain development's impact on evolution. Examples include which developmental mechanisms account for the origin of novel traits, what role developmental plasticity plays in evolution, what forms of inheritance explain phenomena such as niche construction, and so on. Problem agendas thus help structure intellectual integration and scientific investigation (Neto 2020). A distinctive feature of EES is the heterogeneity of its agenda: it engages a variety of research questions that address phenomena at very different levels of organization—from genetic processes such as accommodation and assimilation to large-scale ecological processes—and its questions are both empirical and theoretical. Indeed, one criticism of EES is that it is too heterogeneous to represent a synthesis, being too broad in scope. Under such view, EES should rather be portrayed as a pluralistic framework (Craig 2010; dos Reis and Araújo 2020). Similarly, SET can also be described as a heterogenous research agenda for which a single scope might be too narrow (as per the misportrayal flaw described in Section 2.2). Comparing the scopes of two broad research programs such as SET and EES might be an unfeasible task given the heterogenous and pluralistic nature of the frameworks in question. This requires narrowing the discussion of scope to a second, more specific level, namely, the scope of reciprocal causation within each program, broadly construed.

The second level of scope relevant here is the scope of reciprocal causation within each program; in other words, how the notion of reciprocal causation is used within the context of a problem agenda. This is a matter of the kinds of causal relations that can be characterized as reciprocal, as well as the entities that interact in those causal relations. The scope of reciprocal causation is often discussed in the context of the respective research programs – SET and EES.

Within SET, these relations include negative and positive frequency dependence and cases of co-evolution (Svensson 2018). This points to the fact that reciprocal causation is, arguably, a process occurring between genes and environment and populations and environment, but less so between organisms as units and their surrounding environments. In EES, the main relations exemplified by reciprocal causation include niche construction and constructive development, where the focus is on reciprocity between organisms and their environments.

Finally, the lowest level concerns the scope of explanations making use of reciprocal causation, that is, the kinds of phenomena as well as the causal relata included in explanations that appeal to reciprocal causation. In other words, when using reciprocal causation as an explanatory strategy, what kinds of entities or processes are included in the explanation. It is this level that will be of most relevance to untangle the role of reciprocal causation within each research program. Specifically, the matter is one of *causal explanations*, i.e., the kinds of explanations that seek the cause of a phenomenon in order to explain said phenomenon. In the specific case of reciprocal causation, the matter seems to be finding causes have effects that in turn act as causes. Those effects-turned-causes vary according to the respective timescales to which they belong and their level of grain.

While the three levels are interrelated, the scope argument presented here will focus on the third level. To be sure, the goal of this analysis is to make claims about *debates* surrounding reciprocal causation, and not to evaluate the superiority of one theoretical framework over another. Moreover, as previously flagged, it might even be unproductive to try to characterize heterogenous and pluralistic research programs such as SET and EES under one single scope. However, in the ways in which the debate is portrayed and in the empirical examples that frequently appear to support claims about reciprocal causation, it is still nonetheless possible to evaluate the scope of explanations appealing to reciprocal causation explanations as they appear in the literature.

I hypothesize that there are at least two dimensions that can help delimit the scope of these explanations: the timescales they target and their granularity. These two factors are related; explanations that deal with longer timescales are more general and coarse-grained, and explanations that focus on shorter timescales are more particular and fine-grained. Timescales here refers to the characteristics of temporal processes typically included in the explanatory targets of

the EES. The level of grain of explanations refers to the level of detail and generality included in these explanations. Once the scope of an explanation is specified, it becomes possible to offer explanations using reciprocal causation which have the virtues of (a) having a specific target, which in turn helps to set empirical investigative agendas, and (b) being empirically relevant to the scope of the research program in question.

There are at least two reasons why scope is a fruitful way of understanding the role of reciprocal causation in EES in particular. First, EES proponents' criticisms of SET implicitly make claims about scope. Namely, EES's alleged necessity is grounded on the need to focus on developmental processes and their causal relevance to evolution (Laland et al. 2014; 2015). In the case of niche construction, EES proponents argue that SET is causally incomplete, since it disregards the developmental dimensions that are necessary for a thorough causal picture of evolution (Laland, Matthews, and Feldman 2016). Second, clearly delimitating the scope of a research program clarifies its research questions and thus differentiates its problem agenda from that of other research programs. Clarity about the scope of explanations within a research program prevents its critics from misrepresenting it (see Section 3.1).

While EES is and SET are heterogenous research programs, they share a similar goal of explaining adaptive evolution (Wagner and Tomlinson 2022). However, the two frameworks differ in their scope, and, consequently, in how reciprocal causation features in their respective explanations. Reciprocal causation is a core tenet of EES, so the scope of this notion should, ultimately, align with the scope of the research program. Indeed, the scope of EES explanations that use reciprocal causation is different than those of SET. Under SET, explanations of phenotypic change hinge on population-level effects such as the effect of environmental factors on allele frequency and the interplay between environment and fitness. If my assessment is correct, while the EES and SET share the same explananda, the respective explanans differ in scope.

4.2. Timescales

Broadly construed, scale is "the spatial or temporal extent across which observations span" (Potochnik and McGill 2012). The use of scale in biological explanations is not new (Baedke and Mc Manus 2018; DiFrisco 2017; Green 2018). Scale has been considered a viable conceptual alternative to levels of organization and a suitable framework for describing processes in evo-devo.

For the analysis of reciprocal causation, it will be most relevant to focus a specific kind of scale – timescales. More concretely, DiFrisco (2017) defines a timescale as "the characteristic amount of time it takes for system behaviours or processes to occur" (2017, 809). Timescale is an important component of explanations of biological processes, because such processes stabilize at certain timescales (Dupré 2012).

Emphasis on timescale dissolves some of the conceptual tensions surrounding the role of reciprocal causation in EES. Put simply, reciprocal causation is used to describe processes occurring at shorter timescales in EES than as in SET. The research programs within EES focus mostly, though not exclusively, on *constructive development*: the view that development results from organism-environment feedback interactions, rather than from genetic programming (Müller 2021). Studies performed within this research program focus on the environmental modification of organisms' development, and vice versa. Constructive development together with reciprocal causation is a distinctive feature of EES, and the timescale of reciprocal causation in EES places explanatory emphasis on development, understood here in the broadest sense of *ontogeny* (i.e., a description of events that occur over the course of an organism's life)[5]. This does not mean that reciprocal causal explanations in EES *only* cover development. Specifically, the explanations start from ontogeny but also encompass evolutionary processes such as population shifts in response to natural selection as well as the emergence and fixation fo phenotypic novelties (e.g., in cases of assimilation and plasticity-led evolution). Niche construction, for instance, is primarily conceptualized as a developmental process that shapes evolutionary trajectories while secondarily encompassing population shifts in responses to natural selection (e.g., biasing them through sustained rounds of niche construction). Even if the overarching goal is to explain longer-term evolutionary change, *explanatory emphasis* given to shorter timescales in claims about niche construction in EES is combined to other developmental mechanisms that in turn contribute to longer term phenomena. I further assess this concern in Section 5.

The *Castor canadensis* example provides a useful illustration. Here, the target of explanation is the mechanism of niche construction within a specific ecological system and the way niche-constructing behaviour is passed on. Niche construction modifies both selective environments and

[5] Note that this is much shorter than the time period captured by *phylogeny*, which covers the existence of a species and more (Gould 1977).

developmental environments, and changes in the developmental environment result in systematic changes to the phenotypic expression of inherited genes (Laland and Sterelny 2006, 1758). Proponents of the niche-construction perspective explicitly identify this restricted area as their explanatory target (Laland, Matthews, and Feldman 2016; Laland, Odling-Smee, and Feldman 2019). We can thus see that, under EES framework, reciprocal causation is mostly constrained to the developmental timescale.

SET's explanatory target, however, is to identify processes that occur at much longer timescales. Consider negative frequency-dependent selection (NFDS), which is one of the empirical examples used to illustrate that SET adequately models reciprocal causation (Svensson 2018). NFDS is measured by an assessment of the value of a variant and its abundance in a population, relative to other variants. The target of explanation here is to explain how the frequency of the variant affects selection (in the case of NFDS, the rareness of the variant is what explains its advantage, and not its commonness) (Brisson 2018). As such, the target of explanation is an evolutionary one, explanatory emphasis is a longer time scale. The crux of the matter here being to explain *how* the rareness of such variants affects fitness.

Clarifying scope in terms of timescales of these explanations produces a helpful distinction between explanations that use reciprocal causation at the developmental scale and those that use reciprocal causation at the evolutionary scale. This distinction means that one and the same notion need not fulfill competing or excluding roles within different research programs. Instead, reciprocal causation can be equally useful to explanations whose targets are processes and mechanisms at very different timescales. Distinguishing the two timescales is a heuristic strategy, but it nonetheless allows researchers to trace hierarchical distinctions between causal processes in a continuous manner (Baedke and Mc Manus 2018, 41). The upshot is that reciprocal causation happens at a continuum of timescales, and therefore clarifying the timescale of one's target—providing greater specificity to one's explanations—is critical to a fruitful debate.

4.3. Level of Grain

The second element of scope is the difference in the grain of explanations in SET versus EES. Here, I will draw from Strevens (2008) account of explanatory depth. Strevens argues that causal explanation can vary in terms of its level of grain. Fine-grained explanations distinguish between

"active" and "passive" causal dependence (Strevens 2008, 162). Coarse-grained explanations will not usually make this distinction and will provide a general account of causes including both active and passive dependencies. Strevens presents the examples of models in ecology or evolutionary biology where individuals are represented at the aggregate level, and as such, populations are described as a whole. Such models are characteristic of SET, whereby measures most often occur at the level of populations, thereby providing coarse-grained causal explanations. In the EES, however, the focus is not especially on populations but rather on the level of individual development[6]. One advantage of EES is therefore that it can provide more fine-grained explanations of how ontogeny causally influences phylogeny. Recall that a central claim of EES proponents is that evolutionary explanations must include developmental processes if they are to provide a complete picture of evolution. As such, EES is concerned with developmental explanations at individual or lineage level, while SET is concerned with population-level explanations and how variation in phenotypic traits correlates with changes in allele frequencies in populations (Kaiser 2021). Thus, though explanations offered by both theories aim to identify the mechanisms responsible for the emergence of novel phenotypic traits, EES's explanations are more fine-grained in that they provide a greater level of detail by focusing on what Strevens calls "agent-based models" (i.e., models that keep track of every state of an organism within a system) (Strevens 2008, 163). Such models capture the dynamics of individuals that cannot often be captured in population level-models.

For example, Müller (2021) argues that the innovation of EES is its demonstration that the properties of evolving developmental systems are as important as genetic variation in explaining phenotypic variation (2021, 1129). Indeed, most of the innovative elements of EES are tied to the inclusion of developmental processes, broadly construed. These include, but are not limited to, niche modifications, epigenetic inheritance, and constructive development. Developmental explanations are inherently more fine-grained than genetic ones because they deal with shorter timescales.

[6] One may rightly argue at this point that most EES models, especially those of niche construction, are population-level models mostly coming from population genetics. Even though such models may form the basis of the niche construction perspective, EES proponents seem to place explanatory emphasis on the developmental role of niche construction for evolution, which is compatible with the explanatory emphasis on ontogeny given in EES .

Importantly, more fine-grained explanations do not necessarily imply epistemic advantage. Rather, clarifying grain helps to identify the phenomena at which research programs are aimed, and the scope of their explanations of these phenomena. For example, a goal of eco-evolutionary dynamics (which Svensson (2018) labels as an emblematic case of reciprocal causation under SET) is to understand the contributions of ecological changes to changes in population dynamics; this is a coarse-grained, population-level explanation (Pelletier, Garant, and Hendry 2009). The research performed under EES, on the other hand, aims to understand how ecological changes impact the development of individuals in ways that change individuals' interactions with their surroundings (Sultan, Moczek, and Walsh 2021).

Consider for example, the forms of inheritance in niche construction as understood within SET versus within EES. SET's focus is genetic inheritance, measured through trait frequency, which can be applied globally to the evolution of a species. EES accepts genetic inheritance as the main form of inheritance, but additionally demonstrates the importance of the inheritance of organism-driven changes in an environment. On this view, "dam-building genes" are insufficient to explain niche-constructing behaviour. The need to include an additional form of inheritance at the level of ontogeny is supported by the fact that "organisms also transmit to their offspring altered physical and selective environments, both by physical action on their biological and non-biological environments and by habitat choice" (Laland and Sterelny 2006, 1758). These alterations will vary depending on the niche, so any given explanation will apply only to a particular niche. These explanations are less generalized and more fine-grained.

When evaluating EES's explanatory power, Baedke, Fábregas-Tejeda, and Vergara-Silva (2020) argue that explanations in EES are less idealized than those in SET because EES's explanations identify a range of causes. Thus, compared to those in SET's explanations, causal factors in EES's explanations individually play smaller causal roles. My argument is compatible with this view, and extends it by introducing two dimensions of scope. Table 1 below summarizes the differences in scale and grain of explanations under SET and EES.

Table 1. The scope of reciprocal causation and explanatory emphasis of research programs

Scope	*SET*	*EES*
Timescale of change	▪ Long-term change in allele frequencies ▪ Phylogeny	▪ Short-term, organism-driven ▪ Ontogeny
Level of grain	▪ Global, general, coarse-grained ▪ Selective environments ▪ Population-level	▪ Local, specific, fine-grained ▪ Developmental environments ▪ Individuals or lineages

5. Challenges to the Scope Argument

Timescales and level of grain are two of many potentially relevant elements with which one could analyze the scope of a research program. For example, spatial scale might also be a promising candidate for understanding both scope and the type of causal explanations invoked by various programs. Nonetheless, introducing timescales and grain of explanations in particular could raise concerns. Here, I discuss three such concerns.

First, one may argue against the use of timescales to describe processes occurring at different rates. Such use of timescales may introduce a problematic and artificial distinction in a similar manner to levels of organization: the so-called *levels problem* refers to the idea that the biological world is too messy to be organized into compositional, hierarchical levels (Brooks 2021; Potochnik and McGill 2012). Like hierarchical levels, timescales are an attempt to discretely classify a continuous property, and therefore it is unrealistic to attribute specific timescales to research programs. Furthermore, it may be too simplistic to attribute a specific timescale to developmental explanations, particularly as they arise in EES, because these explanations include a complex interplay between processes at different timescales. For example, as Baedke (2021) argues, phenomena such as morphogenesis and adaptation occur at very different timescales but nonetheless affect each other. Similarly, EES's explanations integrate development and evolution, because the goal is to arrive at comprehensive explanations across timescales. If this objection goes through, the scope of EES goes far beyond ontogeny, making the scope argument inappropriate to EES's explanatory targets.

While EES does integrate explanations across timescales, nevertheless there is methodological support for the scope argument with respect to timescales. In particular, the role reciprocal causation plays in EES is sufficiently specific and fine-grained to account for individuals or lineages. Consider niche construction. There is reciprocal causal feedback between organism and environment—and sometimes the surrounding species as well, as in cases such as *Castor canadensis*' dam-building. Thus, the scope of niche construction is, indeed, a short timescale. The implicit entailment of EES's explanation seems to be that niche construction is itself a cause of longer-term evolutionary processes: in EES, explanations of long-term phenomena are derived from explanations of short-term phenomena. To be sure, EES brings important contributions to evolutionary biology by *primarily* placing explanatory emphasis on shorter timescales with respect to reciprocal causation. This emphasis, however, does not exclude the possibility to combine niche construction, developmental bias and phenotypic plasticity to other classic evolutionary forces in a single evolutionary explanation. By comparison, under SET, the scope of enquiry is primarily the study of niche construction as an adaptation; by the nature of the concept of adaptation, this ranges over a much longer timescale. Reciprocal causation under SET is, therefore, an element of explanations at a much longer timescale than that of EES, even though in each case they can be integrated and are not mutually exclusive.

The second objection is that SET, in virtue of its focus on genes, likewise aims at a fine grain of explanation—not a coarse grain as I have argued. For example, the field of eco-evolutionary dynamics incorporates reciprocal causation with the aim of unravelling how genetic changes and ecological changes co-vary. Most lower-level changes are assessed at the genetic level, whereby small-to-modest-effects on genes are the key factors underlying the dynamics between organisms and environment (Hendry 2013). Explanations whose target is genetic variation's causal role in ecological changes, this objection argues, are therefore similarly or perhaps even more finely grained than explanations in EES.

In response to this challenge, I emphasize that the scope argument is specific to reciprocal causation. Areas of study that acknowledge reciprocal causation under the standard framework are assessing genetic variation, as in eco-evolutionary dynamics, but genetic variation is still measured at the level of populations, not individuals. Moreover, these population-level explanations support generalizations from one focal species to another, indicating that their scope is more coarse-grained than EES's. A core discipline of the EES is evo-devo, which, on the contrary, focuses on

genetic mechanisms such as gene co-option to explain mechanisms such as morphogenesis, but the goal is to understand how environment and organismal development are mutually shaped.

Third, and finally, one may argue that when reciprocal causation is conceptualized in terms of timescales and levels of grain, it boils down to the proximate/ultimate distinction so often criticized by EES proponents. As such, reciprocal causation, analyzed in terms of scope, does not add anything that hasn't already been captured by existing conceptual tools.

While I agree that there are similarities between reciprocal causation and the proximate/ultimate distinction, there is already fruitful philosophical literature focusing on this specific matter (Dickins and Barton 2013; Calcott 2013; Otsuka 2015; Ramsey and Aaby 2022). In fact, deflating reciprocal causation into the proximate/ultimate distinction is a common strategy in arguments that are skeptical towards the epistemic advantage of this notion. While deflationary accounts are a valuable source of criticism, they are not specifically aimed at *how* the concept is deployed within different research programs, which is the purview of this paper. Clarifying the scope of reciprocal causation in terms of timescales and grain shifts the focus from the alleged causal incompleteness of SET to how reciprocal causation is "empirically apt" within research programs (to borrow terminology proposed by Buskell (2019)). Morevoer, as Hazelwood (2023) shows, the disagreement surrounding reciprocal causation centers around its explanatory aptness, i.e., "the relationship between a conceptual framework and its ontological implications – the map it draws of the natural world" (p. 2). If this is correct, reciprocal causation maps the evolutionary story by placing explanatory emphasis in different timescales and levels of grain when used in explanations within different research programs.

6. Reciprocal Causation Revisited

So far, I have demonstrated that the focus on claims about the causal incompleteness of SET has led to stagnation in the debate, slowing down theoretical and empirical advances in understanding reciprocal causation. I have argued that we ought to reorient the discussion towards the *scope* of reciprocal causation under different research programs. Using the concept of scope, I described the different timescales and grain of explanations given by EES and SET.

The scope argument supports my claim that what matters to a productive debate is simply the abilities of SET and EES to support research programs that can identify specific causal mechanisms responsible for the salient features of evolution. Reciprocal causation is inarguably a core causal mechanism in EES's explanations and therefore ought to be scrutinized in order to clarify the research programs that EES supports. However, it is clear that both SET and EES identify reciprocal causation in their research programs; reciprocal causation is not unique to EES. Consequently, it is misguided to argue that an extension of SET is warranted on the basis of reciprocal causation representing a major innovation of EES. As a result, if the inclusion of reciprocal causation is epistemically advantageous, it remains unclear which framework has the epistemic upper hand. The critical, previously unnoticed difference is the scope of reciprocal causation within each framework.

This view is compatible with Buskell's (2019) analysis of the empirical aptness of reciprocal causation: reciprocal causation is useful when it structures and guides inquiry across a community of researchers. As a result, reciprocal causation plays different roles in each program's explanations by virtue of the scope in which it is used. The scope of its use, moreover, is a matter of different research programs' problem agendas. The question of whether reciprocal causation per se is an innovative aspect of EES, then, is simply misguided. Instead, we ought to consider whether or not and how reciprocal causation features in evolutionary explanations; the scope argument is meant to provide these answers.

While flaws of misrepresentation are a correct assessment of EES-based critiques of SET, overemphasizing such flaws provides no insights into the role of reciprocal causation. Neither does overemphasizing the empirical inaptness of SET. While identifying these flaws is an important clarificatory step, it detracts from assessing the actual role of reciprocal casuation in evolutionary explanations. These limitations mean that any EES response to the flaws of misrepresentation or empirical inaptness of SET will find it difficult to justify the need for an emendation of SET. If we wish to argue for an epistemic advantage of EES over SET, then we will need greater specificity about the role of reciprocal causation within the research programs. Moreover, when the debate is redirected to the scope of reciprocal causation, it becomes clear that natural selection and niche construction are distinct processes that are not on explanatory par, but that nonetheless capture evolutionary change from different (and likely coexisting) angles.

Thus, the epistemic advantage of a given research program is not a matter of which processes it can describe, but rather of the scope of the explanations it successfully uses in its descriptions. EES is novel and epistemically advantageous because it fosters empirical research at shorter timescales and provides fine-grained developmental explanations. It remains unclear, however, whether this innovative aspect is sufficient to motivate major theoretical revision. Thus, EES and SET can coexist insofar as their explanations differ in scope. As Mitchell and Dietrich (2006) argue, it is common in biology for similar phenomena to have different explanations, and an isolationist stance in response to competing research programs would be a mistake. I add that understanding the difference in scope between research programs is critical for integrating them when competing explanations arise. If the discussion remains anchored to the misleading question of whether SET incorporates reciprocal causation, this errancy will continue to motivate isolationism between the two research programs. Niche construction, which is a clear case of reciprocal causation, can be explained from different yet coexisting perspectives. More generally, we may find that once an explanandum is specified, one or the other framework may turn out to be more suitable for its explanation.

7. Concluding Remarks

Reciprocal causation is contentious, and has been the subject of much philosophical and scientific discussion, most of which focuses on overarching claims about the causes of evolution. The discussion of whether or not reciprocal causation represents a major theoretical innovation in EES thus motivating theory change is errant. It detracts from analyzing the capacity in which reciprocal causation is important to EES. The question ought not to be whether reciprocal causation is sufficiently new to motivate the emendation project, but rather, what differences there are in the explanatory use of reciprocal causation between EES proponents and skeptics.

By presenting an auxiliary argument that I called the *scope argument*, I have shown that this debate can and should be reoriented, in particular by using timescales and grain of explanations. Refocusing on the explanatory scope of reciprocal causation is necessary to generate constructive debate about reciprocal causation. Scope-specificity produces conceptual clarity, which allows the debate surrounding the role of reciprocal causation to move beyond theoretical claims and towards the specific empirical explananda and evidence relevant to each research

program. Reorienting our focus to scope rejuvenates the debate; in particular, it allows for a fruitful assessment of whether reciprocal causation sufficiently justifies the need for theory change.

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