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The proximate-ultimate distinction and the active role of the organism in evolution

Grant Ramsey[1] · **Bendik Hellem Aaby**[1]

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Abstract

The validity and utility of the proximate-ultimate distinction in biology have recently been under debate. Opponents of the distinction argue that it rules out individual-level organismic processes from evolutionary explanations, thereby leading to an unfounded separation between organismic (developmental, behavioral, etc.) causation and evolutionary causation. Proponents of the proximate-ultimate distinction, on the other hand, argue that it serves an important epistemological role in forming different kinds of explanation-seeking questions in biology. In this paper we offer an interpretation the proximate-ultimate distinction not only as a means of forming explanation-seeking questions, but also as a distinction that can help highlight the way in which individual-level organismic processes can be evolutionary causes. We do this by interpreting the distinction between proximate and ultimate causes as a distinction between structuring and triggering causes.

Keywords Niche construction · Evolution · Selection · Reciprocal causation · Adaptation · Proximate-ultimate cause distinction · Structuring-triggering cause distinction

✉ Grant Ramsey
grant@theramseylab.org

Bendik Hellem Aaby
bendik.aaby@kuleuven.be

[1] Institute of Philosophy, KU Leuven, Leuven, Belgium

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Introduction

The distinction between proximate and ultimate causation was introduced by Mayr (1961) and has since become a staple of introductory chapters in biology textbooks. The distinction serves as a way of picking out two separate sets of causes in order to answer different explanation-seeking questions of biological phenomena.[1] These phenomena are typically organismic traits, such as morphological features or behaviors. Roughly speaking, proximate causes are invoked to explain *how* something works by accounting for the mechanisms underlying the relevant trait. Ultimate causes, on the other hand, answer questions concerning *why* the trait exists, or why it occurs in a particular way and not in any other way. For example, the question, "why did the dove take flight when it saw the fox?" can be answered in terms of proximate or ultimate causes. A proximate cause explanation could cite the bird's perception of the fox and the biochemical cascade that results in motor neurons firing in a pattern that leads to the wings flapping. An ultimate cause explanation could cite the heritable variation in how doves respond to foxes, and point to the unfortunate ones that failed to flee, that they were removed from the species, leaving only those disposed to flight. These appear to be parallel explanations citing distinct sets of causes. Thus, an initial view of the proximate-ultimate distinction might take proximate causes to consist in *biological mechanisms* working in individual organisms and ultimate causes to consist in *evolutionary processes* (principally natural selection) working on populations or species.

Recently, however, the validity and utility of the proximate-ultimate distinction (PUD) have come under scrutiny. Some argue that the PUD has outlived its usefulness and that retaining it might actually hamper the integration of novel perspectives and processes into evolutionary theory (e.g., Laland et al. 2009, 2011, 2013a, 2013b; Uller and Laland 2019).[2] Specifically, they argue that the PUD is inconsistent with, or categorically rules out, the causal—or active—role of the organism in the evolution of its species.

While some take the shortcomings of the PUD to be fatal, others hold that it is worth retaining if properly interpreted (e.g., Sholl and Piglucci 2015; Dickins and Barton 2013; Dickins and Dickins 2018). These interpretations, however, cast the PUD in interrogatory or epistemological terms—as a distinction among the different kinds of questions, explanations, or languages we use in understanding biological

[1] Mayr uses the German word 'fragestellung' (which roughly translates to something like "way of asking/posing a question") to emphasize that the distinction captures two different research methodologies (Mayr 1961, 1501).

[2] Calcott (2013) offers a different reason to discard the PUD than an appeal to the active role of the organisms and reciprocal causation. He argues that lineage explanations—the explanation of how individual-level developmental mechanisms can produce different phenotypic effects in individuals of a shared linage—points to a class of questions that address individual-level mechanisms (typical for proximate questions), yet are addressed in a diachronic temporal context (typical for ultimate questions). Lineage explanations point to an interesting challenge for the integration of evo-devo in evolutionary theory. However, as Calcott neither appeals to the active role of the organisms nor reciprocal causation to make his argument (in fact he argues that in many lineage explanations, an appeal to reciprocal causation is not part of the explanation), we have elected to leave the problem of lineage explanations in relation to the PUD unaddressed in this paper.

phenomena. While we agree that these are important uses of the PUD—they are what introductory textbooks highlight—there is something more at stake than what type of explanatory language is appropriate in biology under different circumstances. The opponents of the PUD are concerned not only with the epistemology of the biological sciences, but also its causal structure (Uller and Laland 2019). In fact, a key area of contention among contemporary biologists is what causal factors are involved in engendering evolutionary change.

Proponents of an extended evolutionary synthesis (EES) argue that the current causal structure of evolutionary theory needs to be supplemented by additional evolutionary causes hitherto "neglected" by standard evolutionary theory (Laland et al. 2015).[3] These additional causal factors include multilevel selection, niche construction, the role of development and behavior in evolution (especially adaptive plasticity), and non-genetic inheritance, to name the most predominant (Laland et al. 2014, 2015). Common to all of these is the introduction of reciprocal causation and an emphasis on the active role of the organism in evolution. As we will argue in more detail below, it is mostly proponents of the EES who are worried about the validity and utility of the PUD[4] as they argue that the active role of the organism is categorically excluded from the causal structure of evolutionary theory if we interpret the PUD as a distinction among different kinds of biological causes. In other words, we can say that the arguments leveled against the PUD are arguments to the effect that the PUD insufficiently (or perhaps fallaciously) captures the causal structure of evolutionary theory.

Thus, in this paper we will go further than providing an epistemological justification of the PUD. We aim to show that the active role of the organism in evolution—which the opponents of the PUD argue is inconsistent with an interpretation of the PUD as distinction between different kinds of biological causes—is in fact consistent with our preferred interpretation of the PUD. Thus, our aim is an ontological one. We want to show that there is a coherent ontological basis of the PUD that allows for the organism to play an active role in evolution. In order to accomplish this, we will use the distinction between structuring and triggering causes (e.g., Dretske 1988, 2004). On our view, proximate causes can be seen as triggering causes producing individual-level behavioral, developmental, and ecological outcomes, while ultimate causes can be understood as structuring causes producing population-level outcomes. In the example of the dove and fox above, the proximate causes are those that *trigger* the flight response in the dove (such as the biochemical and neurophysiological mechanisms responsible for the flight response), while the ultimate causes are those that have structured dove populations in a way that has

[3] 'Neglected' might not be an ideal word to use here, as it may carry the connotation that these additional evolutionary causes have not been researched or have otherwise been "swept under the rug." This is of course not true. Attention to these additional "nonstandard" causes and their relation to evolutionary theory have been pursued by evolutionary theorist in the twentieth century and earlier (e.g., Baldwin 1896a, 1896b; Waddington 1953; see also West-Eberhard 2003, ch. 2). 'Neglected' can here be understood as something along the lines of: "treated as non-standard or special causes of evolution.".

[4] Sholl and Piglucci (2015) and Pigliucci (2019) are notable exceptions. Pigliucci is one of those that have spearheaded the debate on an extended evolutionary synthesis (e.g., Pigliucci and Müller 2010), yet still defends the validity and utility of the PUD.

led to the flight response being a part of the behavioral repertoire of the population of doves. By interpreting proximate causes as triggering causes and ultimate causes as structuring causes, we can show how individual-level behavioral, developmental, and ecological causes can in fact operate as evolutionary (i.e., ultimate) causes in virtue of structuring population-level causes (e.g., natural selection or drift). On this view, the activities of organisms and the reciprocity that it generates can be treated as contributing causes of evolutionary outcomes. More on the structuring-triggering distinction later—first let's consider in more detail the PUD and its critics.

The origin and purpose of the proximate-ultimate distinction

When Mayr (1961) introduced the PUD, it was a supposed to illustrate three uses of causality in the biological sciences: (1) as a means of prediction, (2) as a means of historical explanation, and (3) as a means to explain apparent teleology—or goal directedness—in nature. A key motivation behind the PUD was its use in explaining goal-directed behavior.[5] Returning to the dove and fox example, the question, "why did the dove take flight when it saw the fox?" can be answered by citing either the *efficient* causes (the mechanisms responsible for the flight response) or the *final* causes (natural selection favoring a flight response in those circumstances). These are translated by Mayr from their Aristotelian guises into proximate and ultimate causes, respectively.

The PUD is especially important in the context of apparent teleology because, in contrast to a mason building a house or a cabinetmaker constructing a dresser, the why questions of biology cannot generally be answered by citing goals as antecedent causes. When asking the mason why she builds the house using Flemish bonding instead of the more common running bonding as the brick layering pattern, she could explain why by showing a design or blueprint that depicts a brick house with a Flemish bond pattern, or by showing how it will end up being cheaper using a Flemish pattern for the bond due to less leftover material after the construction process is finished. No matter what the actual answer will be, an experienced mason will be able to answer all the why questions, and especially all the why-this-and-not-that questions, by citing the final product (the brick house with a Flemish pattern for the bonding) or its implications (the construction costs, for example) as the reasons for which the particular means (the actual brick layering according to the principles behind the Flemish pattern) were utilized. That is, the *ultimate* causes can explain *why* the *proximate* causes obtain, and they do so by a standard sequential causal-historical explanation using the goal(s) to explain the means.

Such explanations are available in evolutionary biology only in special circumstances—like cases of learning—in which can we account for non-human goal-directed behavior through an explanation at the level of the individual. In most other circumstances, we need to account for the goal-directed trait exhibited by an individual through an explanation at the level of the population or species of which the

[5] We are not claiming that this is the only motivation behind Mayr's use of the PUD. See Beatty (1994) for an excellent discussion on the many different roles the PUD served for Mayr throughout his career.

individual is a member. In other words, in order to explain why the dove exhibits a flight response when it perceives a fox, and not a fight or freeze response, we need to provide an account of why the flight response is preferentially retained in the population. We commonly do this by showing how the flight response incurs a higher relative fitness than the fight or freeze response and is thus favored and retained in the population through the action of natural selection.[6]

For Mayr (1961), proximate causes can be separated into intrinsic and extrinsic physiological causes. Ultimate causes, on the other hand, are separated into *genetic* and *ecological* causes. Genetic causes refer to the roles genes play in creating and maintaining variation in the population, while the ecological causes concern the selection pressures on this variation. It might, however, be better to think of the separation between genetic and ecological causes as a separation between *variational* and *eliminatory* causes, where the variational causes are simply those responsible for creating and maintaining phenotypic variation in a population (e.g., mutation, recombination, plasticity), while the eliminatory causes are those that eliminate (or retain) phenotypic variants based on fitness differences or chance (i.e., natural selection and drift). On such a reading, the PUD will not be committed to the actual mechanisms responsible for the introduction and transmission of phenotypic variation, nor the origins and maintenance of selection pressures. As we shall see in the following section, many of the critiques leveled against the PUD stem from a literal interpretation of genetic and ecological causes as ultimate causes, instead of interpreting them as variational and eliminatory causes.

Reciprocal causation and the problem of the active role of the organism in evolution

While the PUD has become a canonical element of introductory evolutionary biology textbooks, it has not been without critics (e.g., Ariew 2003; Amundson 2005; West-Eberhard 2003; Laland et al. 2009, 2011, 2013a, 2013b; Uller and Laland 2019; Watt 2013). An overarching worry among the critics is that the PUD might inadvertently undermine the causal role of the organism in evolutionary theory. In particular, they argue that there are many cases in which developmental, behavioral, or other individual-level processes (i.e., organismic causation)—which are presumed to be proximate causes—play a role in evolutionary outcomes. These are cases of reciprocal causation, cases in which an organism's development or activities are both the *causes* and *products* of evolution (e.g., Levins and Lewontin 1985; West-Eberhard 2003). Such cases, they argue, are incompatible with a strict separation (that is, treated as distinct and non-overlapping) between proximate and ultimate causes (Laland et al. 2011, 2013a, 2013b; Uller and Laland 2019). In fact, some of the critics argue that the PUD could in some instances even be a misleading heuristic that glosses over important parts of the causal history of evolutionary outcomes (Laland et al. 2019; Uller and Laland 2019).

[6] In some cases, a trait may be present in a population through the action of drift, which could be invoked as an ultimate cause in the explanation of trait distributions (Ramsey 2013).

We outline two sources of these worries. First, the challenge to genetic ultimate causes. This challenge consists primarily in showing how the underdetermination of the phenotype by the genotype—i.e., a constructive view of development—allows for "plasticity-first" views of evolution, such that organismic plasticity can act as an evolutionary driver. Second is the challenge from niche construction. This challenge stems from cases in which the activities of organisms shape the selective environment they experience and thus modulate the selection pressures acting on them. In both cases, there is reciprocal causation that makes the evolutionary *products* (e.g., dispositions to perform niche constructing behaviors, plastic developmental responses to environmental perturbation, etc.) a *cause* of the evolution of these very products.[7] As we will argue below, these two challenges can be seen as one overarching challenge to the PUD—namely the challenge from reciprocal causation. Let's start by considering in more detail how reciprocal causation and a constructive view of development challenge the PUD.

Constructive development and the origins of phenotypic novelty

If we opt for a literal reading of Mayr's separation of ultimate causes into genetic and ecological causes, as well as a strict separation of proximate and ultimate causes, we end up with a view in which development can be seen as an execution of a predetermined genetic program that has been shaped by natural selection and, consequently, a view of evolution in which development—a kind of proximate cause—is only a product and not a cause in evolution.

> [A] strict exclusion of proximate causes in evolutionary explanations appears to confer on genes causal and informational privilege in development. Indeed, when Mayr described genetic causes as ultimate causes, despite that genes exercise their phenotypic effects through development, it reflected his metaphysical view of development as the execution of a genetic program (e.g., Mayr 1961, 1984). (Uller and Laland 2019, 5)

In this quote by Uller and Laland, they argue that Mayr takes development to be the execution of a predetermined genetic program. In other words, that Mayr gives causal and informational privilege to genes in development. This view is often referred to as genetic determinism or, in a less radical version, gene centrism. On this view, development (and sometimes even all phenotypic expression—even behavior such as nest building or migration, e.g., Dawkins 1976, 1982, 2004) is seen as predetermined by a genetic program that itself has been formed by generations of selection. On such a view, then, proximate causes—those that govern the individual's development, physiology, behavior, and

[7] Reciprocal causation is widespread and instances can be found in niche construction, cases of coevolution, sexual selection (e.g., mate-choice), frequency-dependent selection, social evolution, maternal effects, and so on. Even Darwin seems to have appreciated the importance of reciprocal causation in his work on earthworms (Darwin 1881). See Laland et al. (2009, 2011, 2013a, 2013b) and Uller and Laland (2019) for a more in-depth discussion of these examples. See also Svensson (2018) for a critical examination of these arguments.

environmental interactions—are understood as the products of a genetic program, which itself is a product of ultimate causes (primarily natural selection).

A consequence of this view of development and behavior is that proximate causes cannot be causally efficacious in bringing about evolutionary outcomes, as they themselves are solely the products of the prior action of selection on deterministic genetic programs that execute them. By interpreting proximate causes as executions of genetic programs, development and behavior are seen only as outcomes of evolutionary processes, and cannot be considered evolutionary causes in themselves. Indeed, we can find some affirmation that Mayr might, at least in some parts of his writings, endorse such a consequence:

> The clarification of the biochemical mechanism by which the genetic program is translated into the phenotype tells us absolutely nothing about the steps by which natural selection has built up the particular genetic program. (Mayr 1980, 9-10)

This view is problematic. Genetic determinism and other strongly gene-centric views have over the last decades been challenged. It is now well established that an organism's environment affects development via environmental conditions influencing pathways of gene expression, either directly or mediated through physiological effects (Nijhout 2003; Lewontin 2000; Gilbert 2012; Sultan 2019). This means that a genotype—or the genetic program—may produce different phenotypes in different environmental circumstances. Such phenotypic plasticity has been extensively documented (West-Eberhard 2003; Sultan 2015; Gilbert and Epel 2015) across many types of organisms and in relation to a diversity of environmental conditions (Sultan 2019).

The underdetermination of the phenotype, the multiple sources of developmental information, and the many different causal factors involved in development, was summarized by proponents of developmental systems theory in the *causal parity thesis* (e.g., Oyama et al. 2001; Griffiths and Gray 1994). Building on the insights of developmental systems theory, as well Lewontin's constructionist biology (Lewontin 1983, 2000), Laland et al. (2019) and others (e.g., Moczek 2019) offer an alternative account of the gene-development relation, which they label *constructive development*. According to this view, developmental processes are to be regarded as:

> Open and constructive through self-assembly, and a corresponding rejection of the idea that organisms and their activities are fully specified by genetic programs. Organisms are regarded as influenced, but not determined, by their genes, and their activities as shaped by developmental information-gaining processes as well as natural selection acting on genetic variation. (Laland et al. 2019, 132-133)

On a constructive view of development, the metaphor characterizing development as an execution of a predetermined genetic program is simply untenable.

Constructive development appears to pose the greatest threat to the PUD in cases in which organism-initiated phenotypic novelty acts as a source and driver of

evolutionary innovation and adaptation (e.g., Moczek et al. 2011)—when, that is, there is a reciprocal relationship between the organism-initiated phenotypic novelty and natural selection. This can, for example, occur when an adaptive plastic response to changing environmental conditions is subsequently refined and stabilized as a trait under strong genetic control through genetic accommodation or assimilation (Baldwin 1896a, 1896b; Waddington 1953; West-Eberhard 2003; Sultan 2015). On this view, often called "plasticity-first" evolution (Levis and Pfenning 2016), "genes are followers, not necessarily leaders, in phenotypic evolution" (West-Eberhard 2003, 158). Such cases appear to be problematic for the PUD, since in these instances, proximate causes are the drivers of adaptative evolution (Laland et al. 2013a, 2013b). The sources of evolution, according to the PUD, are genetic ultimate causes. Thus, we end up with a case in which organismic causation is simultaneously a proximate cause and an ultimate cause, which is inconsistent with a view of proximate and ultimate causes as separate and non-overlapping.

Niche construction and organismic control over selection

Another way that reciprocal causation can manifest itself is through the process known as niche construction. Niche construction occurs when organisms actively modify their environment—or their relationship to it—in such a way that the selective environment is changed (Odling-Smee et al. 2003; Laland et al. 2016; Aaby and Ramsey 2019). Recall that ecological ultimate causes are the causes responsible for the differences in relative fitness between individuals in a population—in other words, they are the *selection pressures*. But with niche construction, organisms (operating via proximate causes) can change selection pressures. In such a case, proximate and ultimate causes appear to be one and the same.

The soil-processing effects of earthworms provide a good illustration of the challenge to the proximate-ultimate distinction offered by niche construction. Through their burrowing and related activities (e.g., eating and excreting), earthworms alter the biological, chemical, and physical characteristics of the soil to the benefit of many other species—especially plants, since earthworms boost soil fertility. Earthworms are thus responsible for modifying some environmental variables that are parts of the selective environment of other species (Darwin 1881)—the proximate activities of earthworms can influence ultimate events (Laland et al. 2005, 2019).

In addition to affecting the selection pressures on other species, earthworm behavior affects the selection pressures acting on its own species, primarily through lowering the soil matric potential (the amount of energy it costs to extract water from the soil), which helps the earthworms avoid desiccation in their relatively dry terrestrial habitat (Turner 2000). The earthworm is physiologically quite poorly adapted to terrestrial life. The organs that serve the same function as kidneys in vertebrates, the *nephridia*, do not store water, leading to a daily water loss of 60–90% of its body weight. Daily water loss for humans, by contrast, is 2.5–10%. If earthworms resided in a freshwater aquatic habitat, this would not be a problem. In fact, it would be adaptive since the main challenge in such a habitat is to conserve internal solutes and other minerals during constant diffusion of water through the body. While there

is an advantage to urinating large amounts of diluted urine with low solute concentration in freshwater aquatic habitats, this is not the case for terrestrial habitats. Under such circumstances, organisms should produce low quantities of urine with a higher solute concentration to avoid a build-up of solutes (Turner 2000).

Earthworms thus transform the soil they are living in, making it more suitable for their own physiology by lowering the soil matric potential such that water is easier to obtain (and retain) from their physical surroundings. The altered soil is passed on to the subsequent generations through ecological inheritance. This is thus a form of niche construction that involves trans-generational adaptive modification of the environment, which in turn has selective effects.

An important consequence of niche construction for evolutionary dynamics is that it generates feedback between organismic activities and environmental conditions. The fact that organisms can inherit modified ecological conditions through ecological inheritance is what makes this feedback particularly interesting, as it constitutes a reciprocal relationship between the activities of an organism and the environmental states that are affected by those activities over generations. This reciprocal relationship can have strong effects on subsequent evolution (Laland et al. 2005).

In the earthworm example, the ultimate explanation of the trait of the earthworm—the retention of nephridia adapted for a freshwater habitat—is explained by the environmental conditions of the earthworm, in particular the low matric potential of the soil. However, the low matric potential is explained in part by the activities of individual earthworms over generations. Thus, an evolutionary explanation of the nephridia retention is causally incomplete without a reference to the proximate causes—the burrowing activities of individual earthworms—which maintain the selective environment over generations. In other words, an explanation of the nephridia retention in earthworms that excludes the activities of earthworms leaves an "explanatory gap" (Laland et al. 2019, 127–133). In this case, the burrowing of earthworms is simultaneously a proximate cause (the physiological, behavioral, and ecological mechanisms responsible for the burrowing activities of individual earthworms), as well as the ultimate ecological cause that explains why the earthworms have retained an osmoregulatory organ adapted for an aquatic and not terrestrial habitat.

Just as with the challenge to the PUD from constructive development, the challenge from niche construction is based on cases of reciprocal causation. In the case of niche construction, it is the *ecological* ultimate causes that are simultaneously proximate causes, while in the case of constructive development it is the *genetic* ultimate causes that are simultaneously proximate causes. But what is important about each case is the reciprocal causation, and that this causation is due to the active role of the organism in evolution: organisms perform actions that have evolutionary effects, making their actions simultaneously proximate and ultimate causes.[8] The challenge for proponents of the PUD is, therefore, how to maintain the distinction while nevertheless acknowledging the active role of the organism in the evolution of its species.

[8] See Buskell (2019) for an excellent discussion of the different ways reciprocal causation is used to challenge "standard" evolutionary theory.

Can Mayr's account overcome the problem of the active role of the organism?

It is clear that the challenge of the active role of the organism in evolution needs to be addressed. In this section, we will consider in more depth Mayr's approach and whether it can meet this challenge. And in the following section, we will turn our attention to other ways of reinterpreting the PUD.

When one examines Mayr's discussions of evolution, one sees that he is keenly aware of the importance of organismic activity in driving evolution. For example, in discussing evolutionary novelty, Mayr argues that behavior often plays the role of a "pacemaker," whereby behavior frequently exposes organisms to novel selection pressures that can result in relatively rapid subsequent evolutionary changes in life history, morphological, and physiological traits (Duckworth 2009):

> A shift into a new niche or adaptive zone is, almost without exception, initiated by a change in behavior. The other adaptations to the new niche, particularly the structural ones, are acquired secondarily […] With habitat and food selection—behavioral phenomena—playing a major role in the shift into new adaptive zones, the importance of behavior in initiating new evolutionary events is self-evident. (Mayr 1963, 604)

This quote shows that he saw how organismic behaviors can drive evolution. In fact, there are striking resemblances between Mayr's "pacemaker" model and much of the work citied to undermine the proximate-ultimate distinction often referred to as instances of reciprocal causation and "plasticity-first" evolution. As Svensson (2018) points out:

> Mayr's view of a crucial role of behaviour in the evolutionary process is clearly compatible with feedback between the organism and its environment. Mayr's surprisingly early insights on the issue has clear similarities with similar views expressed several decades later by West-Eberhard, Levins and Lewontin (West-Eberhard 1983; Levins and Lewontin 1985), albeit not developed in detail by him. (Svensson 2018, 6)

Even in Mayr's original example illustrating the difference between proximate and ultimate causes—the migration of the warblers of New Hampshire[9]—it was (presumably) individual warblers that initiated traveling north from their native tropical habitat of Central America during interglacial periods in search of seasonal resources (Curson et al. 1994). And the action of these warblers had evolutionary effects.

Another mechanism offered by Mayr (1974) for allowing development and experience to alter behavior is via what he termed *open behavioral programs*. The idea

[9] One quibble with Mayr's explanation: The New World warbler (*Parulidae*) is a family of tropical birds thought to have originated in Central America, which is where they reach their greatest extant diversity (Curson et al. 1994). Thus, it is best to think of them not as temperate birds that fly south to avoid starvation and freezing during winter, but as tropical birds that fly north to nest in regions with fewer predators and a seasonal abundance of insects.

is that selection can favor either open or closed behavioral programs, where open programs modify outcomes based on experience and closed programs change outcomes little or not at all based on experience. Thus, in open behavioral programs, the behavior (the execution of the genetic program) is not predetermined by natural selection, but rather sensitive to experience and environmental stimuli.[10]

In open behavioral programs and, indeed, in much of development and behavior, the phenotype is *underdetermined* by the genotype: there are more causal factors involved in shaping developmental trajectories and behavioral outcomes than mere gene expression. For example, developmental pathways can be triggered by specific environmental cues, such as the temperature-dependent sex determination in reptiles (Warner and Shine 2008) or environmentally induced epigenetic changes to gene functions (John and Rougeulle 2018). Also, behaviors that have commonly been treated as strongly determined by genes, e.g., nest building in birds, are now believed to involve a considerable amount of plasticity (e.g., Hansell 2007).

Given Mayr's insights into the active role of the organism in evolution, what are we to make of his PUD? One possible response is to switch the focus of the PUD from *causation* to *explanation*. In this way, organismic activities—plastic responses to environmental cues, niche construction, learning, etc.—might be cited in proximate or ultimate explanations, depending on the explanatory context. We could treat them as proximate causes when they are invoked to explain *how* an individual interacts with its environment (including ontogeny), while we might treat them as ultimate causes when they are invoked to explain *why* the organism interacts the way it does, and not in another way—i.e., in order to explain an evolutionary outcome (e.g., Scott-Phillips et al. 2011).

This shift from causation to explanation is one that has been attempted by a number of theorists who saw that trying to sort causes into mutually exclusive bins of proximate and ultimate causes is difficult, if not impossible. Let's now examine some of these accounts, before arguing that an epistemological rendering of the PUD, while tenable, leaves unanswered important questions about the causal structure of evolutionary theory. In particular, how does the PUD map onto a causal structure of evolutionary theory in which the organism can play an active role?

Alternative interpretations of the proximate-ultimate distinction

There are many who have defended the validity and utility of the PUD in the face of the problem of the active role of the organism in evolution. They have mainly done so by shifting focus from causation to explanation. That is, they take the PUD to be a distinction primarily concerned with what sort of explanation-seeking questions we can pose regarding biological phenomena and as indicating what kinds

[10] In cases of phenotypic underdetermination due to developmental plasticity, norms of reaction can play the same role as Mayr's open behavioral programs. The norm of reaction is a central concept in evolutionary theory, and it thus seems unlikely that Mayr, or indeed anybody else, would argue that the phenotype is predetermined, or overdetermined, by the genotype. See Dickins and Dickins (2018) for discussion.

of explanations are appropriate to provide in response to these questions. Haig (2013), for example, argues that the PUD is really just is a distinction between how-come questions (proximate) and what-for questions (ultimate). These questions are explained by citing either the relevant biological mechanisms or adaptive rationale. On this interpretation, there is no conceptual or logical commitment that excludes developmental processes, behaviors, or other individual-level ecological interactions to play a role in either proximate or ultimate explanations. It is rather that if these processes are to figure in evolutionary (i.e., ultimate) explanations, they cannot figure simply as biological mechanisms. Instead, they have to be related to a population-level process or outcome.[11]

In a similar vein, Scholl and Pigliucci (2015) argue that a "lean version" of the PUD can play a useful pragmatic and epistemological role in modeling causation in biology. On their view, proximate and ultimate explanations inform different *contrastive questions*. The PUD helps us in picking out causal factors that should be foregrounded or backgrounded in causal explanations of different biological phenomena. Proximate questions seek explanations in terms of *biological mechanisms*, while ultimate questions seek explanations in terms of *evolutionary processes*.

Birch (2017) goes a bit further and argues that if we carefully explain what constitute proximate and ultimate causes, we can avoid an apparent inconsistency between the PUD and developmental processes, behavior, and other ecological interactions playing an explanatory role in evolutionary explanations. On Birch's view, proximate causes should be understood as the causes that are responsible for the within-generation occurrence of and changes to a phenotype, while ultimate causes are understood as the causes that are responsible for the across-generation reoccurrence and changes to the phenotype. Thus, in cases where proximate and ultimate causes overlap, we can construe them as either proximate or ultimate causes depending on whether we are explaining within- or across-generation phenomena.

Finally, Otsuka (2014) argues that the main obstacle for the PUD to incorporate proximate causes into evolutionary explanations is that proximate and ultimate causes are not described in a similar theoretical language. That is, ultimate explanations are usually cashed out in statistical terms, while proximate explanations are cashed out in mechanistic terms. Thus, in order to incorporate individual-level and developmental processes into evolutionary explanations, one needs to translate the mechanistic talk of proximate causes into a statistical language representing ultimate outcomes. He uses causal graph theory to show how proximate mechanisms produce evolutionary responses through affecting one or more of the four components of the Price equation, i.e., the (1) rate and (2) direction of selection, (3) parent–offspring resemblance, and (4) fidelity.[12] The causal decomposition of the Price equation shows how variables such as epigenetic inheritance, niche construction, maternal effects, etc. can influence (1)-(4). Thus, if we find cases in which a variable (i.e.,

[11] See below. See also Dickins and Dickins (2018), Dickins and Barton (2013) and Scott-Phillips et al. (2011).

[12] This is a vastly oversimplified description of his account, which merits more attention than we can give it here. See Otsuka (2014, 2016) and Okasha and Otsuka (2020).

a proximate cause) affects (1)-(4), then we have a case in which proximate causes influence evolutionary outcomes.

All of these interpretations address the problem of the active role of the organism in evolution by highlighting the explanatory role of the PUD. Namely, the PUD is a distinction between different explanatory questions, contexts, or languages. While we agree that the PUD could be interpreted as a distinction among the kinds of questions and explanations that we utilize in biology, these interpretations leave unexplored whether the explanations or questions are separable because of a separation in underlying causes. PUD explanations are causal explanations, so even if one attempts to interpret the distinction as one among kinds of explanations, such explanations nevertheless carry assumptions about the causal structure of evolutionary systems. It is therefore important to consider how the causes behind these explanations are related to one another.

Furthermore, the ongoing debates surrounding the validity and utility of the PUD, as well as the need for an extended evolutionary synthesis, center on the causal structure of evolutionary theory—namely how inclusive we should be when elaborating this causal structure. The opponents of the PUD repudiate the causal structure it implies (if interpreted as a distinction between distinct and non-overlapping causes). Instead, they offer a unified view of causation in biology—reciprocal causation—in which proximate and ultimate causes sometimes are one and the same, just acting on different spatial (individual and population) and temporal (within and across generations) scales. While an explanation-based account of the PUD may circumvent the issue of reciprocal causation and the active role of the organism by arguing that it highlights different explanations that the same sets of causes can provide under different explanatory contexts, this does not provide an account of how such causes relate to the causal structure of evolutionary theory. Are all individual-level developmental, behavioral, and ecological causes (i.e., proximate causes) *possible* evolutionary (ultimate) causes? If so, what makes them *actual* evolutionary causes? When a proximate cause is cited in an explanation of a proximate event, can the same cause be cited in the explanation an ultimate event? Or are these different causes?

Answers to such questions do not straightforwardly follow from our epistemological commitments. We cannot answer them simply by claiming that cause and effect are to be identified with the *explanantia* and *explananda* of our explanations. Unless we also admit some ontological commitments as to what makes something a proximate or an ultimate cause, we get little bearing on such questions.

Thus, we hold that an account of the ontological implications of the PUD—that is, an ontological interpretation of the PUD—is needed to properly address questions concerning the causal structure of evolutionary theory that lie at the heart of the debate over the need for an extended evolutionary synthesis. In the following section, we suggest that a possible ontological interpretation of the PUD could be based on the distinction between structuring and triggering causes. We then show how this interpretation can accommodate the active role of organisms and the reciprocal causation they create.

Structuring and triggering causes and the proximate-ultimate distinction

The structuring-triggering cause distinction is borrowed from Dretske (1988, 2004). It is not a distinction he originated, but is nicely formulated by him through a thought experiment:

> A terrorist plants a bomb in the general's car. The bomb sits there for days until the general gets in his car and turns the key to start the engine. The bomb is detonated (triggered by turning the key in the ignition) and the general is killed. Who killed him? The terrorist, of course. How? By planting a bomb in his car. Although the general's own action (turning on the engine) was the triggering cause, the terrorist's action, wiring the bomb to the ignition, is the structuring cause, and it will surely be the terrorist's action, something that happened a week ago, that will be singled out, in both legal and moral inquiries, as the cause of the explosion that resulted in the general's death. (Dretske 2004, 169)

This distinction has already been used to analyze causes in evolutionary biology, in particular in the debates concerning whether fitness differences, selection, and drift can be causes of evolution (Ramsey 2016). In arguing that, say, fitness differences cause evolutionary change, it needs to be established what sort of a cause this is. Ramsey (2016) invokes the structuring-triggering cause distinction to argue that while fitness differences might not be triggering causes of evolutionary outcomes, they can be understood as structuring causes.

To better see how the structuring-triggering cause distinction translates to biological causes, it is helpful to consider organismic life histories. A life history is an entire life lived by an organism. It has various properties, some are common and periodic (being asleep), some persist until death once they arrive (being adult), others are ephemeral (eating a particular meal at a specific moment). What is most relevant to selection, fitness, and drift are special events along the life history, namely, acts of reproduction (and events influencing the prospects of reproduction)—in particular, the quantity and timing of reproductive acts distributed over life histories. In this framework, fitness can be understood as a disposition, which could be fleshed out in terms of the average[13] reproduction over the set of life histories, not just actual life histories but possible life histories. But of these possibilities, of course, only one life is lived. The life that is lived is *triggered* by the specific set of circumstances encountered by the organism. But the entire set of life histories is *structured* by the characteristics of the organism and the totality of the environmental variables.

Let us now consider how the structuring-triggering cause distinction could map onto the PUD. Picture again the set of possible life histories that an organism might follow. Take the example of a bird, a robin, say, beginning as an egg deposited in a nest. This robin has many possible lives before it. It may die young, perhaps just after fledging, or perhaps a year into its life. It might succumb to starvation, disease,

[13] An average—understood as an arithmetic mean—is not, it turns out, the best way of quantifying fitness. See Pence and Ramsey (2013) for a discussion of the mathematical foundation of fitness.

predation, or an accident. We can ask of an individual what triggered the life that it lived from among the possibilities. Why did it have the outcome it did instead of another? Why did it starve while its nestmates survived? Asking why an individual followed one among the possible life histories is to ask about the triggering causes.[14] Triggering causes are thus the causes that trigger one instead of another possible life history.

How are proximate causes related to triggering causes? To answer this question, consider again the terrorist example with additional complexities added by Ramsey (2016):

> It could be that the terrorist did not want to blow up the general's family, so she put a pressure sensor under the back seat that would make the key trigger only a secondary smoke bomb intended to warn and to terrorize him and his family. A single structuring cause (setting up the car with the pair of bombs, the pressure sensor, and the key switch) has set the world to have two possible outcomes (assuming, of course, that the general will definitely turn the key). (Ramsey 2016, 432)

In such a situation, we can ask about the possible outcomes for the general, and we can ask why one among the possible outcomes was triggered. These questions become more interesting if we have a population of like individuals, as we do in biological species. If we ask of the population why it evolved in a particular way over some stretch of time, we will be interested in structuring causes, since those are the causes that determine the characteristics of the set of possible life histories. It is this set of possibilities that is important, and the exploration of this possibility space by the population is what constitutes evolution. Let's flesh this out with an example.

The world is set up such that most robin life histories end prior to successful reproduction—indeed, most die in their first year (e.g., Sullivan 1989; Yackel-Adams et al. 2006). There is thus an incredible selection pressure during this first year on being able to procure sufficient food and avoid succumbing to parasites or predators. These selection pressures shape the possibility space; they are structuring causes. And it is these causes that we examine in understanding evolution.

Nevertheless, just as we can ask what it was that triggered the smoke bomb in the general's car instead of the fatal bomb, we can ask of an individual robin what it was that triggered the life outcomes that it realized. Why did this robin live only a week after fledging? What triggered this life history instead of other possibilities, such as bearing broods in two successive seasons before succumbing to predation?

[14] The reader might object here and argue that since we use contrastive questions to highlight the difference between structuring and triggering causes, our suggestion fares no better in uncovering the ontological implications and commitments than an epistemological interpretation of the PUD. However, we think it is important to highlight two different classes of questions one might pose: clarificatory and explanation-seeking questions. We are using contrastive *clarificatory* questions in order to uncover what makes structuring and triggering causes distinct kinds of causes, while the epistemological interpretation of the PUD takes the PUD to be a tool for formulating different contrastive *explanation-seeking* questions we may ask of biological phenomena. Of course, an answer to a clarificatory question might overlap with an answer to a related explanation-seeking answer, but they should not be treated as identical kinds of questions.

Thus, we have two questions: First, why does the set of possible life histories for this population (or species) have the characteristics that it does? Second, why did this individual follow this particular life history instead of another possibility? The first is answered in terms of structuring causes, the second in terms of triggering causes. Structuring causes thus do the job of ultimate causes, they explain why a population of organisms has the characteristics it does and not others, while triggering causes can do the job of proximate causes by explaining how (e.g., through which mechanisms) a particular organism interacts with its environment or how it develops.

Structuring and triggering causes and the active role of the organism in evolution

In the preceding section, we offered a rationale for interpreting ultimate causes as structuring causes and proximate causes as triggering causes. Now we want to show how on such an interpretation, ultimate causes can include the active role of the organism in evolution. Let us revisit the earthworm example. For earthworms, the effects of individual-level burrowing activities help to create a selective environment in which the population-level response is to retain a nephridia adapted to an aquatic, and not terrestrial, environment. As we saw above, a proper explanation of why the earthworm retains the nephridia invokes the action of selection, but the action of selection cannot be fully appreciated without an account of what generates and maintains the selection pressures that produce the adaptive response.

In this case, the ultimate cause of the nephridia retention is the action of natural selection. However, the action of selection is in part due to the structuring activities of the earthworms. As such, the joint effect of individual-level burrowing activities is what causes natural selection to favor nephridia retention. In Mayr's vocabulary, the individual-level burrowing is the (ultimate) ecological cause of the nephridia retention. While each individual worm's burrowing has only a minor effect on selection pressures, the collective effect of all the burrowing activities creates strong selection pressures on earthworm populations. In this case, the effect of collective burrowing plays the same structuring cause role as abiotic and biotic environmental factors do in other evolutionary systems. For example, why did melanism spread in peppered moth populations across Britain in the 1800s? The ecological ultimate cause is the increased presence of soot on resting surfaces that made the gray morph more conspicuous than the melanistic one. The presence of soot thus structured the action of selection to favor melanistic morphs. But the moths did not paint the soot on the tree surfaces and are thus not participants in the creation of this selection pressure. In the case of the earthworm, the worms themselves are active participants in creating the selection pressures and are thus simultaneously the producers as well as recipients of these pressures.

With earthworms, the selection pressures acting on them are modified by their behavior. The behavioral traits involved in this modification are a robust developmental outcome and not learned from conspecifics or based on socially transmitted behavioral innovations (Ramsey et al., 2007). Let us now consider how the active role of organisms, manifested through behavioral innovations, can be an ultimate

genetic cause in evolution. To illustrate this, let us imagine an individual Macaque who discovers that it can use a rock to crush the shell of clams it can easily forage at the seashore. Let us further imagine that the clam-crushing Macaque enjoys a significant fitness advantage in virtue of the extra nutrients the clams provide, relative to other non-clam-crushing members of its population. This behavior spreads through the population via imitative learning. If the fitness advantage is great enough and the fidelity of transmission is high enough, the clam-crushing behavior will spread through the population.

In this example, there is a behavioral innovation (a case of constructive development) that is transmitted via social learning. One consequence of this is that the behavioral innovation introduces novel variation for selection to act on. The novel variation *structures* the action of selection in virtue of providing new behaviors to select for. While it is socially transmitted, the clam-crushing behavior creates novel selection pressures on the capacities for learning, finding the right size and shape of rock, manual dexterity, and so on. There will be selection for the individuals to perform such behavior earlier in their life history, and more skillfully. Over many generations, this could lead to the behavior arising in the absence of a model needed to imitate. What was once a socially transmitted innovation could thus become a genetically transmitted trait. And the selective pressures that drove the genetic evolution arise from the active role of the innovative organism (Bateson 2004, 2017a, 2017b; Bateson and Gluckman 2011). The activities of organisms can thus be *structuring* causes of evolutionary outcomes.

By distinguishing structuring and triggering causes, we are not claiming that these causes are non-overlapping. The example here highlights such overlaps. Nevertheless, the sets of causes picked out by the structuring-triggering distinction play distinct roles and can be used to form distinct explanations of developmental and evolutionary phenomena. We thus hold that one way of making sense of the causes underlying the PUD is to associate proximate with triggering causes and ultimate with structuring causes. Such an interpretation allows the PUD to sidestep the problems of reciprocal causation and the active role of the organism in evolution, while at the same time acknowledging that there are distinct sets of causes that play important roles in evolutionary causation.

Conclusion

We have argued that the recent challenges to the PUD are chiefly based on the fact that organisms can play an active role in their evolution. Such activity can lead to reciprocal causation—i.e., cases in which a product of evolution is also a cause of that very product. We then showed how proponents of the PUD have attempted to accommodate the active role of the organism by providing an interpretation of the PUD focused on explanation, not causation—i.e., as a distinction between different explanatory aims, contexts, or languages. An explanatory rendering of the PUD, however, leaves unexplored whether the explanations or questions are separable because of a separation in underlying causes. Because these explanations

are citing causes, we argued that even if the main role of the PUD is explanatory, we should nevertheless scrutinize the causal basis of these explanations.

By invoking the distinction between structuring and triggering causes, we argued that there is in fact a way to interpret the PUD as an ontological distinction between different kinds of causes, and not just between the different kinds of explanatory contexts or languages. This account allows organisms to play an active role in evolution in virtue of organismic causation structuring population-level selective processes. In this way, organismic causation can be an ultimate cause.

If we focus not on the way causes structure the selection pressures, but instead on why one particular life history instead of another was realized, then we are focusing on triggering causes. On our interpretation of the PUD, organismic causation clearly plays roles as both structuring and triggering causes. There is a single causal nexus, and structuring and triggering causes are overlapping components of this nexus. Distinguishing among them allows us to better understand and explain the complexities of evolutionary causation.

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