1 Evolutionary Causation

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Introduction

Scientific inference typically relies on establishing causation. This is also the case in evolutionary biology, a discipline charged with providing historical accounts of the properties of living beings, as well as an understanding of the processes that explain the origin of those properties. Familiar phenomena that demand an evolutionary explanation include the fit of form and function (adaptation) and the evolution of reproductive isolation (speciation), but also many others, including the origin of novelty and the organization of biological systems, including genomes and ecosystems. As expected from this diversity of topics, the study of evolution is a quite loosely organized endeavor. Nevertheless, there is broad agreement that an explanation for why organisms have particular features commonly will involve a particular kind of evolutionary process, namely evolution by natural selection.

While the basic principles of evolution by natural selection are simple—variation among individuals, differences in survival and reproduction, and heredity—the biological reality that instantiates these principles is incredibly complex. Furthermore, evolutionary processes encompass causation at different levels of biological organization, from genes to ecosystems, and at different timescales. This complexity makes it necessary to find means to represent biological systems in ways that leave out almost all causal detail, while ensuring that models, observations, and experiments carry explanatory weight. What evolutionary researchers should incorporate into their explanatory accounts, and what they should leave out, is far from a trivial issue.

While biologists are typically content to leave the nature of causation to philosophers, there is scientific value in reflecting on core theoretical concepts. Different views on causation can lead to alternative explanatory frameworks, dictate what counts as legitimate difference makers in evolutionary processes, and shape perspectives on key concepts such as natural selection and adaptation. How causation is understood thus shapes the structure of evolutionary theory, with both historical and contemporary debates in evolutionary biology revolving around the nature of causation (Laland et al. 2011). Yet, these issues

are rarely addressed. This limits the opportunity for constructive exchange and risks confusing outstanding evolutionary problems with semantic disputes, both of which may hinder scientific progress.

This edited volume brings together biologists and philosophers of science to provide a comprehensive treatment of evolutionary causation. It is based on the workshop *Cause and Process in Evolution*, which took place at the KLI in Klosterneuburg in May 2017. The contributions clarify the nature of causation in the historical and contemporary representation of evolution, specify alternative perspectives and reveal their underlying assumptions, and seek ways of thinking about causation that will be helpful to formulate research programs in evolutionary biology.

Proximate and Ultimate Causation

Whereas biological and philosophical reflections on the nature of causation in living systems have a long history,[1] contemporary notions of evolutionary causation have been shaped by the idea that there are two fundamentally different types of causation in biology. The distinction, brought to prominence in a paper by Ernst Mayr (1961), is that one set of causes—the "ultimate causes"—provide historical accounts for the existence of particular features in extant organisms, explain the "goal-directedness" of living beings, and enable predictions about how populations will evolve in the future. The other set—the "proximate causes"—are not historical and they are considered to explain and predict biological systems by establishing how different components work, much like a chemist, physicist, or engineer would establish causation. Under this view, evolutionary biologists are concerned with ultimate causes, whereas molecular and developmental biologists, physiologists, and most other biologists are concerned with proximate causes.

Mayr clearly put his finger on an important distinction in biology. An explanation for, say, why a particular warbler migrates in autumn is different from an explanation for why warblers migrate at all, rather than staying and enduring winter. Whereas the former invokes "an immediate set of causes"—such as photoperiod and hormones—that modify the birds' physiology and behavior, the latter invokes "causes that have a history and that have been incorporated into the system through many thousands of generations of natural selection" (Mayr 1961, 1503). More specifically, Mayr lists "ecological causes" and "genetic causes" as the two categories of ultimate causes. In the case of the warbler, an ecological cause could be the limited food availability in winter that implies that warblers that do not migrate would starve. The genetic cause is "a genetic constitution [acquired] in the course of the evolutionary history of its species which induces it to respond appropriately to the proper stimuli from the environment" (Mayr 1961, 1503).

Mayr's partition of causation stems from his strong commitment to "population thinking" and variational explanations over "typological thinking" and transformational explanations in evolutionary biology. Sober (1984, 149) made the following helpful analogy to

contrast the two types of explanation. Consider that one wants to explain why all children in a class read at the third-grade level. One account takes the children one at a time and describes how he or she attained that level of reading proficiency. These stories are then aggregated to explain why the class is composed of children that all read at the third-grade level. This is a transformational explanation. It explains by accounting for changes that occur to individuals. But there is an alternative explanatory account that does not rely on aggregation. This is to note that only children who can read at the third-grade level are admitted to the class. This is a variational explanation. In contrast to the transformational account, it explains why the class is composed of children with a particular reading proficiency without referring to any individual causal detail. The variational explanation succeeds if children vary in their level of reading proficiency, allowing their proficiency to make a difference to admissions, and that those who have reached the third-grade level do not lose their ability after they are admitted.

According to Mayr, when evolutionary biologists explain why warblers migrate in autumn, their aim is to explain why the species is composed of individuals that migrate, rather than any other kind of individuals (e.g., those who overwinter). Ecological causes are those that "select" individuals on the basis of their characteristics. Genetic causes ensure that those characteristics vary between individuals before they are selected, and passing on those genes to descendants ensures that the variants, once they have been selected, persist and can accumulate in the population down generations. On this view, mutation and recombination introduce genetic variation and the distribution of these variants changes under natural selection, drift, and migration. From this perspective, there is no room for proximate causes in evolutionary explanations since only genes are inherited, effectively preventing nongenetic developmental causes from becoming evolutionary causes.

Causation and the Role of the Organism in Evolution

The proximate–ultimate distinction has provided conceptual unity for the evolutionary sciences. It has been a core aspect of a shared consensus as to which forms of causality constitute satisfactory evolutionary explanations (i.e., historical and functional accounts) and which do not (i.e., mechanistic and developmental accounts). To this end, the distinction has been used to detect "flawed" evolutionary reasoning—models that, in addition to fitness differences and genetic inheritance, invoke proximate mechanisms to explain the fit of form and function (e.g., Scott-Phillips, Dickins, and West [2011] for human cooperation and Dickins and Rahman [2012] for extra-genetic inheritance).[2] On the above interpretation of the proximate–ultimate distinction, this response appears sound. Since genetic mutations are assumed not to be directed, nor to occur when organisms need them, on this reasoning all of the sustained directionality in evolution comes from fitness differences between genotypes, or natural selection.

While few would contest the value of distinguishing between different types of causal explanation, philosophers and biologists have recognized that this is not the only way to describe causation in living systems (e.g., Calcott 2013; Laland et al. 2013; Watt 2013). Thus, the implementation of the proximate–ultimate distinction, while uniting evolutionary biology, may inadvertently be ruling out certain legitimate classes of evolutionary explanations, and hindering evolutionary biology's ability to draw from adjacent fields (e.g., West-Eberhard 2003; Laland et al. 2011). Particularly contentious has been the implication that developmental processes, which are responsible for bringing phenotypes into being, are considered irrelevant for explaining phenotypic evolution. Several debates within contemporary evolutionary biology concern the extent to which causal effects of environment on organism (e.g., plasticity, epigenetic inheritance) and causal effects of organism on environment (e.g., niche construction) also can be evolutionary causes (Laland et al. 2011, 2015). While the challenges are not new, the exclusion of development in evolutionary explanation is increasingly difficult to reconcile with biological knowledge and how biologists actually go about doing their research.

To illustrate, consider the observation that killer whales have locally adapted diet and specialized hunting techniques (Foote et al. 2016; Hoelzel and Moura 2016). These characters are stably inherited down generations, making phenotypic differences between lineages persist. In fact, sympatric groups with different foraging modes appear to be reproductively isolated (Moura et al. 2014; Moura et al. 2015). Researchers have revealed that the adaptive divergence in diet and hunting technique is not due to genetic differences but to social learning (Foote et al. 2016). It is likely that the origin of novel foraging behavior is due to behavioral innovation through, for example, trial and error rather than genetic mutation (Foote et al. 2016). It thus appears that social learning—traditionally considered a proximate mechanism—contributes to the historical explanation for the adaptive fit between the local environment and phenotype, as well as reproductive isolation of killer whale populations.

That developmental processes like behavioral plasticity and social learning impose persistent adaptive directionality on evolution appears inconsistent with the exclusive role of natural selection in adaptive evolution, as well as the heuristic stance that genetics is the proper mechanistic focus of evolutionary analysis. This inconsistency arises because the explanation for adaptive divergence in killer whales relies, in part, on a transformational explanation[3]. It explains how a particular population becomes composed of fish eaters by referring not only to the fitness differences between individuals with different hunting techniques but also to the acquisition of fish-hunting skills in ontogeny and its spread to other members of the population through social learning. In contrast, a variational explanation for why the population is composed of fish eaters would not invoke environmental influence on how traits originate and are inherited.

It is possible to develop this variational account by reformulating what needs to be explained. What requires an evolutionary explanation, the argument goes, is not why killer

whale populations acquired the ability to hunt for seals or salmon, but why they acquired a capacity for social learning. On this account, ecological conditions ("ecological causes") have favored genetic variants ("genetic causes") that enable individuals to adjust their food preferences to local conditions.[4] This avoids a transformational account of evolution by making the adaptive directionality imposed by the organisms themselves a function of long-term natural selection on random genetic variation. This "rescaling" of evolutionary and developmental processes is a very common strategy to protect the notion that there are two fundamentally different kinds of causes in biology. It allows the evolutionary effects of phenotypic plasticity, extra-genetic inheritance, and niche construction to be accommodated into the genetic instantiation of evolution by natural selection (e.g., Scott-Phillips et al. 2014). While this can provide a valid historical explanation, the explanatory shift comes with at least three features that appear problematic.

The first problematic feature is that, to some (but seemingly not all) researchers, the evolutionary explanation simply appears incomplete. Staying with the killer whale example, the traditional account provides only a partial historical analysis, as it explains the killer whales' general reliance on social learning but not why particular populations acquire or maintain particular dietary traditions (killer whale ecotypes are often sympatric). Moreover, the different ecotypes have diverged genetically (Moura et al. 2015) and exhibit morphological adaptations such as population-specific digestive enzymes (Foote et al. 2016). Here the absence of an evolutionary account of why particular populations possess particular feeding habits appears to render incomplete the explanation for the existence and properties of morphological specializations. The latter requires knowledge of the particular dietary traditions of the population and cannot be predicted with knowledge of local ecology.

The second problematic feature is that 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; Mayr 1984). This view was common in the 1960s and continues to be so among contemporary biologists (Moczek 2012), but it has been widely discredited (Oyama 2000; Keller 2010; Griffiths and Stotz 2013). If phenotypes are underdetermined by their genotypes (i.e., if not all adaptive responses are located in the genome as "programs," "blueprints," or "recipes"), it appears conceptually inconsistent to ascribe the adaptive directionality in evolution caused by social learning solely to past selection on genetic variation (e.g., Mesoudi et al. 2013; Jablonka and Lamb 2014).

A third concern is that the justification for the explanatory shift appears to rely on the assumption that variation, differential fitness, and heredity are autonomous processes (Badyaev 2011; Walsh 2015; Uller and Helanterä 2017). For example, it is usually assumed that pattern of selection does not affect the rules of inheritance; inheritance is merely the

passing on of whatever alleles were selected. The variation that fuels evolution is considered similarly autonomous. Mutations occur randomly with respect to their consequences for phenotypic variation and fitness, and the acquisition of new variants does not change how variation is transmitted down generations. Process autonomy ensures that all adaptive directionality arising in development in principle can be explained in terms of previous rounds of selection on stably inherited (genetic) variation. But the same logic need not apply when the processes are causally intertwined or entangled (Walsh 2015; Uller and Helanterä 2017). On this account, it does not matter to what phenotype, or how far back in time, we shift our focus; how the principles of evolution by natural selection are instantiated is ever changing, thereby compromising a strictly variational account of adaptation and diversification.

Identifying the consequences of alternative causal representations of evolution is an increasingly pressing task as genome, cell, and developmental biology are producing results that question how well biological systems are represented in gene-centric evolutionary biology (i.e., the framework that motivated the distinction between proximate and ultimate causation). Some argue that evolutionary biologists need to embrace these findings and see them as a means to devise a richer explanation of life's diversity, if evolution is to remain the central guiding principle in biology (e.g., Laland et al. 2015; Sultan 2015). Others point out the traditional framework's ability to generate causal explanation even if some of its core assumptions are relaxed (e.g., Wray et al. 2014; Futuyma 2017). Whereas it remains to be seen if and how evolutionary biology will be transformed by these debates, they will not go away without a concerted effort to resolve conceptual differences or, at the very least, demarcate the fault line(s) of interpretative understanding of the evolutionary process.

Despite the central role of the nature of causation in evolutionary biology, the outstanding issues are rarely addressed. Evolutionary biology textbooks, for instance, hardly ever cover this topic, and such analysis as exists is dominated by philosophers of science. To biologists, at least, the literature on causation in biological systems may appear idiosyncratic and poorly connected to evolutionary theory. This edited volume therefore brings together leading biologists and philosophers of science to focus on the causal structure of evolutionary theory from historical and contemporary perspectives, and thereby endeavor to shed light on current debates.

A Brief Summary of This Book

The opening contribution by Massimo Pigliucci captures the motivation for this volume. Pigliucci outlines four alternatives for the relationship between science and philosophy of science. He makes a case that there can be mutual benefit, and goes on to show—using four case studies of causation—how this benefit can be realized. Pigliucci's analysis emphasizes that, despite their different aims, philosophers and scientists can enrich their respective disciplines through active engagement.

plasticity as a property of the genotype is what maintains the causal privilege of natural selection in evolutionary explanation. Sultan thus points toward a fundamental dilemma facing evolutionary biologists—should they accommodate plasticity and extra-genetic inheritance using idealizations that maintain the contemporary explanatory agenda, or should they seek to ground alternative research programs in constructive views of development and inheritance?

The role of alternative research programs is of central concern to Kevin Laland, John Odling-Smee, and Marcus Feldman. Their survey of the origin of the concept of niche construction, its reception, and the continued debate surrounding its evolutionary implications illustrates how metaphysical and epistemological views shape scientific pursuits. Laland and colleagues single out three conceptual issues of particular importance in this regard: the notion of organismal agency, the concept of development as programmed *versus* constructed, and causal entanglement *versus* causal autonomy of variation, differential fitness, and heredity. They argue that grasping these points is essential to understanding what is new and important about niche construction theory. The message is that, rather than making niche construction fit the current conceptual framework, there is scientific value in actively pursuing alternative points of view.

That organisms co-create the niches in which they develop and are selected is also the theme of the chapter by Renée Duckworth. Building on insights from her work on evolutionary cycles of displacement between western and mountain blue birds, she explains how we can understand better how evolution works by determining how the behavior of biological entities at one scale influence patterns of variation at another scale. In particular, Duckworth is concerned with evolutionary stasis. To be robust at one level is to be dynamically flexible at another level, and Duckworth argues that to understand stasis we must first understand how ecological interactions evolve their robustness. This robustness, in turn, is to be found in the niche-constructing and developmentally plastic responses of the active organism.

The evolution of biological organization is also the focus of the two following chapters. Both are concerned with the evolutionary transition from a population of individuals to a population of collectives. This transition represents a challenge to traditional explanations since it is unclear how fitness benefits at the individual level can result in the formation of collectives that function as their own evolutionary units. Heikki Helantera and Tobias Uller discuss this problem by applying the Darwinian space framework of philosopher of science Peter Godfrey-Smith (2009) to the evolution of social insect colonies. They conclude that an evolutionary account of how collectives acquire evolutionary individuality requires a focus on the evolution of spatial and functional organization through causal feedbacks between reproductive fitness, plasticity, and niche construction.

Richard Watson and Christoph Thies take this further and ask if plasticity, niche construction, and extra-genetic inheritance are in fact necessary conditions for a transition in evolutionary individuality. Their starting point is that collectives that represent evolutionary

units, such as multicellular organisms, must exhibit heritable variation in fitness over and above that of the individual members of the collective. They argue that this is only possible if individuals within collectives exhibit plasticity, extra-genetic inheritance, and niche construction. In fact, Watson and Thies argue that it is these properties of parts that make collectives approximate the causal autonomy of variation, differential fitness, and heredity that the "standard model" assumes. Their conclusion is that the organism plays an active role in explaining transitions in individuality, which may argue in favor of historical explanations that combine variational and transformative accounts.

Philosophical Reflections on Evolutionary Causation

The final section of the book consists of five chapters that analyze evolutionary causation from a philosophical perspective. Denis Walsh begins by drawing attention to what he calls "the paradox of population thinking" in evolutionary biology. The paradox lies in that the causes of individual lives and deaths are what makes the population change, but these causes are precluded from the explanation of how evolution happens. According to Walsh, invoking natural selection or drift as causal explanations is to overpopulate the world with causes. On his account, changes in the composition of trait types in a population is an analytical consequence and higher order effect of the individual causes of evolution. The contention surrounding the role of development in evolution arises from the failure to realize that both "first-level causes" and "higher order effects" explanations are valid.

Jun Otsuka is also concerned with diagnosing the conceptual underpinnings of the debate over proximate causes in evolutionary explanation, in particular the current controversy over the "extended evolutionary synthesis" (EES; Laland et al. 2014). Otsuka concludes that there are different views on both the ontological units of evolution and on the proper methodology for studying their dynamics. Otsuka explains the former using causal graph theory and shows that, in the EES, the evolutionary dynamics arise, not from the genes, but from the entire causal structure of the biological system. Whereas this does not imply that all causes make an equal contribution, it does imply that there is generally no ontological justification to elevate particular causes to evolutionary causes. Otsuka also demonstrates how this ontology is accompanied by a shift in research methodology. He concludes that the challenge now is to demonstrate that this conceptual framework can provide a productive and unified research program.

Arnaud Pocheville turns his attention to the suggestion that causal entanglement of the processes of evolution by natural selection compromises traditional accounts of evolutionary causation. Making the point that entanglement occurs at both levels and timescales, he shows how entanglement is handled by evolutionary theory through "rescaling." In contrast to the interpretation in terms of process (or causal) autonomy, the timescale framework focuses on whether or not one can explain, for example, social learning in terms of imposing constraints on a slower evolutionary process. Pocheville's analysis suggests that this emphasis on local invariants, rather than causes, significantly influences

how one should interpret the challenge to evolutionary theory posed by plasticity, extra-genetic inheritance, and niche construction.

Lynn Chiu approaches the entanglement of causes and processes in evolution through an examination of niche construction. In niche construction theory, natural selection and niche construction are two coupled processes that share responsibility for the evolution of adaptive fit between organism and environment. A common strategy is to rescale these two processes, such that natural selection bears responsibility for the direction on evolution imposed by niche construction. Building on Lewontin's insights, Chiu makes the point that organisms do not only physically modify their world but also change how the world is experienced. She argues that this implies that organism and environment can "commingle" in ways that spread selective causes across organism and environment to make niche construction and natural selection inseparable. In these cases, natural selection still explains adaptation, but it is not the same externalist "natural selection" as in the traditional account. Chiu proposes a strategy to identify whether or not it is appropriate to assume that natural selection explanations are externalist.

In the final chapter, Karola Stotz discusses the relationship between causation and information. She points out that biological systems are unique in that they are informed. Whereas the standard interpretation is that this information resides in the genome, Stotz demonstrates how information is acquired, expressed, and stored in development and heredity through a variety of biological processes. This leads her to argue that biological information is a substantive causal factor in both development and evolution. Importantly, the contribution of different causes can be quantified in terms of their specificity, which provides strategies for how information from different sources can be quantified and compared. Stotz suggests that this allows establishing the explanatory relevance of proximate sources of information in evolution.

Conclusion

This collection of essays demonstrates that contemporary debates about evolutionary biology are not solely about new data or novel theoretical findings but also revolve around fundamental conceptual issues (albeit often promoted by new findings). A productive dialogue between biologists and philosophers of science offers the best prospect for resolution of these challenging issues, and we hope this volume makes a contribution toward that resolution.