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PERSPECTIVE

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Developmental plasticity and evolutionary explanations

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Abstract

Developmental plasticity looks like a promising bridge between ecological and developmental perspectives on evolution. Yet, there is no consensus on whether plasticity is part of the explanation for adaptive evolution or an optional "add-on" to genes and natural selection. Here, we suggest that these differences in opinion are caused by differences in the simplifying assumptions, and particular idealizations, that enable evolutionary explanation. We outline why idealizations designed to explain evolution through natural selection prevent an understanding of the role of development, and vice versa. We show that representing plasticity as a reaction norm conforms with the idealizations of selective explanations, which can give the false impression that plasticity has no explanatory power for adaptive evolution. Finally, we use examples to illustrate why evolutionary explanations that include developmental plasticity may in fact be more satisfactory than explanations that solely refer to genes and natural selection.

KEYWORDS

developmental plasticity, explanation, reaction norm, idealization, plasticity-first evolution

1 | INTRODUCTION

A growing number of studies suggest that locally adapted phenotypes originated as environmentally induced variants (for reviews, see Ehrenreich & Pfennig, 2016; Kelly, 2019; Schlichting & Wund, 2014; Schneider & Meyer, 2017; West-Eberhard, 2003). This body of research on "plasticity-led" evolution could be a sign that development is finding its rightful place in evolutionary biology (Gilbert, Bosch, & Ledón-Rettig, 2015; Moczek, 2015; Moczek et al., 2011; Sultan, 2015). Intuitively, if development is the source of all phenotypes, phenotypic plasticity represents a developmental bias[1] (Uller, Moczek, Watson, Brakefield, & Laland, 2018) on phenotypic variation that may shape evolutionary trajectories. More specifically, if adaptive evolution proceeds preferentially where plasticity leads, *evolutionary explanations* for adaptation and diversification should invoke development, physiology, and behavior alongside natural selection; a conceptual change that some consider central for moving evolutionary research forward (e.g., Laland et al., 2011; Laland et al., 2015; Müller, 2017; see Lewens, 2019).

Nevertheless, in this essay, we suggest that evolutionary biology has accommodated plasticity largely by resisting, rather than embracing, conceptual change. Our intention is not to ruin the party; it is our party too, and we do not want to go home. On the contrary, we believe that it is only by understanding how developmental plasticity has been accommodated by different communities of evolutionary biologists that one can hope to

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[1]We may refer to *developmental bias* as the phenotype distribution produced by one developmental system relative to that of another, or relative to a theoretical distribution. Developmental bias is commonly considered only in the context of phenotypic responses to genetic variation, but may also include nongenetic factors because these are always necessary for the generation of phenotypes.

resolve the contention that surrounds the role of plasticity in evolution (e.g., de Jong & Crozier, 2003 vs. Badyaev, 2005; Laland et al., 2014 vs. Wray et al., 2014; Futuyma, 2017 vs. Müller, 2017).

We will proceed as follows. We first point out that scientific explanation relies heavily on representations that idealize away most of the complex reality. As a result, the choice of idealization also determines what counts as an evolutionary explanation. With this in mind, it is easier to understand why plasticity sometimes appears to be a cause of adaptive evolution and sometimes appears to be an optional "add-on" to genes and natural selection. We conclude with examples to illustrate when and why referring to the phenotypic biases produced by developmental plasticity can make explanations of adaptive evolution more satisfactory than explanations formulated solely in terms of genetic variation and natural selection.

2 | EXPLANATION

The world is immensely complex. Natural phenomena are produced by such a diversity of causes that it would be impossible to refer to them all. In fact, even if we could, this would merely be a description. But science should also deliver understanding, and this requires phenomena to be explained. The causal complexity of the world means that these explanations must rely on representations that not only leave out most features but also misrepresent others (Cartwright, 1983; Potochnik, 2017; Strevens, 2008). The latter are known as idealizations, "assumptions made without regard for whether they are true and often with full knowledge they are false" (Potochnik, 2017, p. 42). Idealizations are found throughout science, including in models, theories and even laws (e.g., Cartwright, 1983). Idealization plays important roles in scientific explanations because it makes phenomena appear as if they were produced by the focal causes alone. This makes it possible to foresee what would have happened if things would have been different, and thereby grasp how features of the world are connected (Potochnik, 2017). Different idealizations pick out different causal relations. The choice of idealization, therefore, determines what causes that are considered explanatory and the knowledge that can be constructed.

To illustrate, consider outbreaks of antibiotic resistance, an increasing threat to global health according to the World Health Organization (https://www.who.int/antimicrobial-resistance/en/). One reason for these outbreaks is the liberal prescription of antibiotic drugs, an attitude that is affected by societal values and the personal beliefs of doctors. Theories and models designed to explain how these factors affect antibiotic resistance may benefit from assuming that appearance of resistance is instantaneous, that the rate of spread is constant, and that resistance does not change following its appearance. As biologists are well aware, all of these assumptions are false; yet they are helpful to understand why antibiotic resistance is common in many countries. At the same time, representing antibiotic resistance as if there was no evolution effectively prevents knowledge about the role of, for example, the strength of natural selection on resistant bacteria. This does not mean that natural selection is unimportant for outbreaks of antibiotic resistance; in fact, the opposite is true. It simply means that idealizations designed to deliver explanations in terms of some causes will screen off other causes, thereby preventing any meaningful inference of how those causes contribute to the phenomenon of interest. If the aim were to understand the role of natural selection for outbreaks of antibiotic resistance, one would need a different idealization. But this idealization too would undoubtedly marginalize important causes of antibiotic resistance. An important consequence of this screening off is that one must remain silent on the importance of causes that are idealized away from being explanatory. This is the case also within evolutionary biology.

3 | IDEALIZATION AND EXPLANATION IN EVOLUTIONARY BIOLOGY

Evolutionary explanations are historical explanations; they explain why populations or lineages change in particular ways (or remain the same) over time. The evolutionary process is incredibly complex, and biologists must choose which of its components to study. The central problem for the past 100 years has been to understand how natural selection contributes to adaptation and diversification. Looking for explanations based on fitness differences ("selective explanations"; Lewontin, 1983; Sober, 1984), makes it desirable to screen off other influences on evolution, even when these are potentially significant. A selective explanation requires that there is something to select on and that the entities of evolution retain their properties after they have been selected. This can be achieved by treating the principles of evolution by natural selection—variation, differential fitness, and heredity—as if they were produced by fixed and separable processes (Pocheville, 2019; Uller & Helanterä, 2019; Walsh, 2015; Figure 1). The familiar genetic idealization of evolution by natural selection[2] does exactly that: Phenotypic variation is

[2]Not all selective explanations refer to genes. For example, evolutionary game theory or adaptive dynamics tend to ignore genes, which may make them appear more compatible with developmental perspectives (e.g., Metz, 2011). These methods are, however, also designed to explain adaptation in terms of selection. It is beyond the scope of this paper to address how these selective explanations differ. However, we do not wish to claim that the diversity of

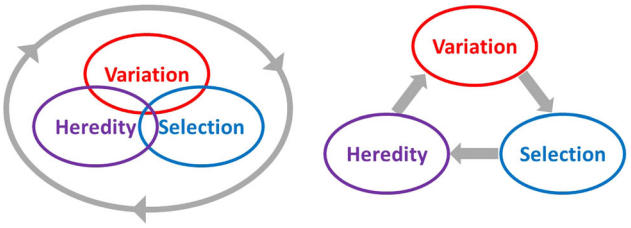

FIGURE 1 The three components of evolution by natural selection and their relationships. Evolution by natural selection requires variation, differential fitness ("selection") and heredity. The biological reality that makes populations of organisms fulfill these criteria is extraordinary complex, and the causes that sustain one criterion (e.g., variation) may be intertwined with causes of other criteria (e.g., selection), on both short and long time scales (left panel). For example, individuals may change their behavior in response to threats to their survival, and pass on those behaviors to their descendants. Over many generations, evolution by natural selection may change how behaviors develop, thereby influencing the relationship between genetic and behavioral variation. This causal complexity means that biologists must ignore and even represent falsely many aspects of reality to deliver evolutionary explanations. In evolutionary theory, it is common to assume that the three criteria for evolution by natural selection are separable (right panel). Further assuming that the sources of selectable variation are undirected with respect to its effects on fitness and heredity makes it possible to explain directional, adaptive, change solely in terms of fitness differences between reliably inherited types. These assumptions exclude developmental ("proximate") causes from explaining the adaptive change, even if all phenotypic evolution involves changes to development. While this idealization is appropriate for explanations in terms of fitness differences, it is of limited use if the aim is to understand the role of development in the evolution [Color figure can be viewed at wileyonlinelibrary.com]

assumed to arise independently with respect to fitness, and the acquisition and selection of particular phenotypes do not change how phenotypic variation is inherited. Thus, populations adapt because of selection of fit types, whose arrival is not only decoupled from the current environment but also from the types that were previously selected. As a consequence, any sustained directionality in evolution can be explained by referring to fitness differences between reliably inherited variants rather than to the introduction of variants (Mayr, 1961; see Stoltzfus, 2019; Figure 1). Development does not explain fitness differences, which makes it external to the explanatory agenda and therefore unable to account for the adaptive phenotypic change. Factors external to the explanatory agenda tend to be viewed as constraints, which is commonly the role that development is assigned in adaptive evolution (Antonovics & Vantienderen, 1991; Gould, 2002, Ch. 10).

To illustrate how idealization determines the difference between causes and constraints, consider transformational explanations of evolution (Calcott, 2009; Lewontin, 1983; Sober, 1984). Viewed from afar, individuals and populations are out of sight and adaptation and diversification appear as a transformation of lineages. From this perspective, the explanation for evolutionary change is to be found in the causes that transform one phenotype into another through developmental change, while natural selection limits evolution to phenotypes that can successfully survive and reproduce.

Transformational and selective explanations are both common in evolutionary biology. This is expected because adaptive evolution involves both the generation of heritable phenotypic variants and the differential fitness of those variants. This is well illustrated by the striking adaptive convergence in morphology and coloration between cichlid fish in Lake Tanganyika and Lake Malawi (Kocher, Conroy, McKaye, & Stauffer, 1993). Selective explanations for this convergence refer to the similarity in the ecology of the lakes, which made particular morphologies and lifestyles fitter than others. If the ecology of the lakes would have been less similar, present-day fish would have been less convergent. Transformational explanations instead refer to the similarity in the developmental biology of ancestral fish that colonized the lakes, which made particular morphologies and lifestyles arise more readily than others. If the development of ancestral fish would have been less similar, present-day fish would have been less convergent. Both ecology and developmental biology can, therefore, be called upon to explain why the fish are so similar in the two lakes.

While few biologists are likely to dispute the value of selective and transformational explanations in evolutionary biology, it can be easy to forget that both selective and developmental causes are causes of adaptive evolution. As explained above, idealizations that pick out one set of dependencies screen off others, and this determines which causes that are considered explanatory. In the following section, we show how this feature of scientific explanation helps to understand the different interpretations of the explanatory role of developmental plasticity in adaptive evolution.

4 | DEVELOPMENTAL PLASTICITY AND EVOLUTION

In her synthetic book, West-Eberhard (2003) encouraged evolutionary biologists to "put the flexible phenotype first," and to begin their evolutionary explanations with the origin of a novel phenotype. She broke down the

selective explanations can be safely lumped together. The reader may, therefore, wish to interpret the label "genetic idealization of evolution by natural selection" to refer to the conceptual underpinnings of selective explanations formulated on the basis of population genetics and quantitative genetics.

sequence of adaptive evolution into four fundamental aspects (abbreviated from West-Eberhard, 2003, p. 140):

(1) A novel input, genetic or nongenetic, affects a pre-existing responsive phenotype, causing a phenotypic change.
(2) Individuals respond by the mutual adjustment of their parts, resulting in a "novel" phenotype.
(3) Recurrence of the novel input, genetic or nongenetic, results in recurrence or initial spread of the novel phenotype.
(4) Selection on heritable variation in the regulation, form or side-effects of the novel phenotype in the sub-population of individuals that express the phenotype.

On this account "The causal chain of adaptive evolution begins with development... From these causal relations, it is clear that development, not selection, is the first-order cause of design" (West-Eberhard, 2003, p. 141). One implication of West-Eberhard's perspective is that environmentally induced phenotypes can initiate and direct evolution, captured in the slogan "genes are followers, not leaders, in adaptive evolution" (e.g., West-Eberhard, 2003, p. 157–158). In West-Eberhard's view, evolutionary explanations are neither selective nor transformational, but combine elements of both. This position arguably represents a break with tradition, and it has been enthusiastically embraced by some evolutionary biologists (e.g., Gilbert et al., 2015; Laland et al., 2015; Moczek et al., 2015; Sultan, 2015). In contrast, other evolutionary biologists appear to accept that plasticity can initiate and direct evolution, but oppose that this implies anything particularly important with respect to the explanation for adaptive evolution (e.g., Futuyma, 2017; Svensson, 2018; Wray et al., 2014; see also Charlesworth, Barton, & Charlesworth, 2017). To understand these different responses, it is necessary to look at the assumptions that underpin the evolutionary explanations that are on offer.

In evolutionary biology, plasticity is commonly represented by the relationship between environment and phenotype for a given genotype (i.e., a reaction norm). From a reaction norm perspective, West-Eberhard's sequence of events in adaptive evolution can be reformulated in terms of the expression of phenotypic and genetic variation in novel environments, followed by selection of particular reaction norms (Ghalambor, McKay, Carroll, & Reznick, 2007; Lande, 2009; Levis & Pfennig, 2016; Figure 2).

Like West-Eberhard's summary, the reaction norm account appears to grant that developmental plasticity and natural selection jointly explain adaptation. The sequence of events does begin with plastic responses to a

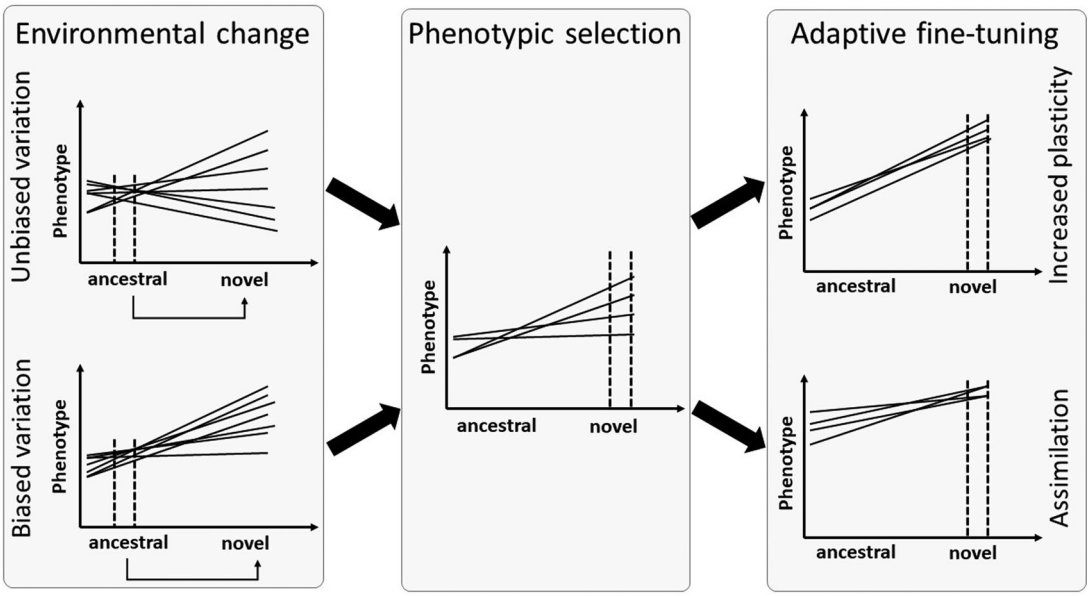

FIGURE 2 The reaction norm representation of the role of plasticity in evolution. Left: A group of genotypes adapted to an ancestral environment is exposed to a novel environment, which results in the expression of phenotypes that were not observed in the ancestral environment. The distribution of phenotypes may be unbiased (top left) or biased, perhaps in the direction of higher fitness compared to the ancestral phenotype (bottom left). Middle: Fitness differences between phenotypes results in the retention of a subset of the genotypes, which increases the average fit between phenotype and the novel environment. Right: Over evolutionary time, continued selection on standing genetic variation or new mutations can result in adaptive fine-tuning of reaction norms, which may exhibit increased (top right) or decreased (bottom right) plasticity compared to the ancestral population. The representation of plasticity in terms of genotypic reaction norms makes plasticity appear as an optional "add-on" to an explanation of the adaptive change that needs only cite natural selection and genetic variation

novel environment. However, because reaction norms are typically considered genotypes, they snugly fit within the genetic idealization of evolution by natural selection (Sultan 2019). The fitting of organism and environment happens because of selection of pre-existing genotypes (Figure 2), just as in any standard genetic model. Thus, natural selection and genetic variation do all the explanatory job. What if the shape of reaction norms makes individuals fitter in the novel environment than they would, had they not been plastic? This may indeed facilitate adaptation to the novel environment (e.g., Price, Qvarnström, & Irwin, 2003). But it does not change what counts as an explanation for adaptive change. The assumption that the principles of evolution by natural selection are separable means that those genotypes with the right kind of plasticity exist either because they had been selected in the past or due to chance (Figure 1). Representing plasticity by genotypic reaction norms, therefore, tends to make plasticity appear as an "add-on": information that could be omitted without loss of an understanding of the actual cause of adaptive evolution (i.e., natural selection; Wray et al., 2014).

This conclusion is expected; the genetic idealization of evolution by natural selection is designed to explain adaptive change solely in terms of fitness differences, not in terms of development, physiology or behavior. While the reaction norm representation of plasticity can identify important topics in evolution (e.g., the role of cryptic genetic variation; Dayan, Graham, Baker, & Foster, 2019; Levis & Pfennig, 2016), it will inevitably allow plasticity to be interpreted as interesting, but effectively superfluous, detail rather than as a cause of adaptive change. However, this interpretation (e.g., Wray et al., 2014) follows from assumptions made to deliver a selective explanation rather than being a fundamental feature of evolutionary causation.

Indeed, a different idealization of the evolutionary process can allow plasticity to play a more positive explanatory role. Consider a recent hypothesis for the evolutionary origin of decidual stromal cells, a cell type involved in embryo implantation in eutherian mammals (Wagner, Erkenbrack, & Love, 2019). Wagner et al. (2019) suggest that a generic stress response caused ancestrally by the invading embryo has evolved to become internalized and triggered by physiological signals before the stress is encountered. Once the stress response came under the control of physiological signals, the cellular phenotype could be further modified, eventually resulting in a distinctively novel cell type.

In contrast to the explanation offered by the reaction norm representation, plasticity takes on a fundamental role in this account; it explains the origin of the implantation response, and it is the modification of plasticity that transformed the generic stress response into the highly specific and adaptive mechanism of decidualization. The difference between the accounts can be understood by recognizing that Wagner et al.'s (2019) explanation is transformational; it explains decidualization through the developmental changes that transformed one phenotype into another rather than through fitness differences.

It thus appears that the idea that plasticity takes the lead in adaptive evolution can be productively embraced by different communities of evolutionary biologists. While doing so can enrich the field, it need not affect the kinds of explanation that evolutionary biologists are concerned with. This should not be surprising. The evolutionary process has many causes and different idealizations are needed to explore them: The multitude of causes of evolutionary phenomena calls for a multitude of research programs. But the focus on any particular causes means that others are marginalized or left unexamined, even when they contribute to the phenomena of interest. This can be confusing and make people talk past each other, in particular if some assumptions become so entrenched that they are no longer recognized as idealizations but considered to describe how biological systems really are.

5 | WHEN DOES "PUTTING THE FLEXIBLE PHENOTYPE FIRST" RESULT IN BETTER EXPLANATIONS?

One interpretation of this explanatory pluralism is that there is no genuine conflict with respect to the role of plasticity in evolution. Certainly, selective and transformational explanations are complementary and not mutually exclusive. However, appeals to pluralism may let us off the hook too easily. Biologists can agree that natural selection explains the convergent evolution of, for example, snail crushing cichlids in Lake Tanganyika and Lake Malawi. But they may also wish to know when an explanation for this adaptive convergence that includes developmental plasticity is better or *more satisfactory* than an explanation referring only to selection and genetic variation (see also Kovaka, 2019). Here, there appears to be a genuine controversy (Laland et al., 2014; Wray et al., 2014).

How to decide what counts as a satisfactory explanation is a fundamental problem in philosophy of science that we cannot hope to settle (see e.g., Lipton, 2004; Strevens, 2008; Ylikoski & Kuorikoski, 2010). But we can point towards reasons that, at least to us, suggest that an explanation for adaptive evolution that includes

developmental, physiological, or behavioral detail can be more satisfactory than explanations delivered by the genetic idealization of evolution by natural selection. Here, we propose three such reasons.

First, an explanation is satisfactory only if it matches up with the empirical evidence. Even if the adaptive match between organism and environment usually embodies natural selection, it may sometimes fail to do so, or only do so to a marginal extent. For example, killer whales appear to have adapted their hunting strategies to their local environments through behavioral innovation and social learning, rather than through natural selection of genetic variation (Foote et al., 2016). While biologists can agree that this is how locally adapted hunting strategies are established and maintained in killer whales, an idealization designed for selective explanations tends to disqualify such instances of adaptive divergence through social learning from being *evolutionary* phenomena (e.g., Dickins & Rahman, 2012; Mayr, 1961; Scott-Phillips, Dickins, & West, 2011), or perhaps imply that they ought to be explained by selection of preprogrammed, behavioral types copied from one individual to another (e.g., memes, Dawkins, 1976). But rather than a priori privileging a representation of evolving systems that deliver selective explanations, it may be more useful to let the biology of the system guide the assumptions.

Second, explanations refer to why a phenomenon is one way rather than another, and the appropriate explanation can, therefore, depend on the contrast set (Lipton, 2004, Ylikoski, 2007). To illustrate, consider an evolutionary explanation for why water fleas in ponds at high latitudes are heavily pigmented (e.g., Hebert & Emery, 1990). One interpretation of this question is that it demands an explanation for why water fleas are pigmented rather than transparent, because the latter is the norm at low latitudes. Assume that the difference between populations is greater than any direct effects of the environment (i.e., the difference is not accounted for by plasticity alone). Pigmentation protects the water fleas from UV damage and, because pigmentation is a heritable trait and UV irradiation is higher closer to the north pole, the incidence of dark water fleas at high latitude can be explained by survival differences between pigmented and transparent individuals. While one could also point out that pigmentation is an ancestrally plastic trait (Scoville & Pfrender, 2010), this does not appear to add much to the explanation: Pigmentation would have evolved adaptively (from transparency) at high latitude irrespective of its plasticity.

Pigmentation is not the only possible protection from UV irradiation. The question "why are water fleas at high latitude pigmented?" could also be answered by explaining why pigmentation evolved rather than, for instance, upregulation of repair proteins or behaviors that reduce UV exposure (Hansson & Hylander, 2009). An idealization designed for selective explanations will encourage a comparison of the fitness costs and benefits of alternative phenotypes or strategies. An idealization that recognizes developmental causes as causes of adaptive evolution (rather than as constraints) will also draw attention to the possibility that some traits are more likely than others to exhibit adaptive variation. For example, environmentally responsive phenotypes may be more likely to become selected, not because they are the fittest, but because they are the most recurrent under suitable ecological conditions (West-Eberhard, 2003), or because plastic traits generate more heritable phenotypic variation (Draghi & Whitlock, 2012; Noble, Radersma, & Uller, 2019)[3]. In this case, the fact that pigmentation evolved from a plastic response adds important information to the explanation: Pigmentation (rather than another trait) evolved adaptively at high latitude partly because pigmentation was a plastic trait[4].

Our third and final point extends this emphasis on the origin of adaptive phenotypic variation to instances where the evolutionary process modifies itself. Because selective explanations assume that there are multiple variants to select on in the first place, they are generally of limited value for explaining the evolution of novelties (Love, 2008; Moczek, 2019; Salazar-Ciudad, 2007). A particularly interesting case is the transition from single-celled to multi-cellular organisms. Explaining such transitions in individuality is challenging because the evolutionary process itself is changing (Godfrey-Smith, 2009; Watson & Szathmary, 2016). Selective explanations invoke fitness differences between individuals, whereas transformational explanations invoke the mechanisms of development and inheritance of those individuals. But what needs to be explained is the evolution of a different kind of individual, one with its own fitness and new mechanisms of development and inheritance. Both selective and transformational explanations struggle to handle this situation because during evolutionary transitions in individuality, the principles of evolution by natural selection are intertwined; they modify each other on the time scales relevant for the explanation (Laland et al., 2015; Pocheville, 2019; Uller & Helanterä, 2019;

[3]Plasticity can also enable a population to persist in a stressful environment until a beneficial mutation arises. For example, plasticity in pigmentation may have allowed water fleas to persist at high latitude until an appropriate mutation appeared. But in this case, there is (typically) no reason to expect this mutation to be more likely to fine-tune pigmentation rather than any other possible mechanism of protection to UV exposure. Thus, in this scenario, plasticity explains adaptation in virtue of its effects on population size in a particular selective environment rather than through the introduction of particular variants.

[4]The philosophically interested reader may wish to interpret the two contrast sets using Woodward's (2003) concepts of intervention and invariance.

Walsh, 2015; see also Woodward, 2003, p. 302–307; Figure 1). This is a good reason to believe that a satisfactory evolutionary explanation for transitions in individuality will require reference to how organisms respond to and construct their environments alongside fitness differences between individuals and groups (Watson & Thies, 2019; see also Laubichler & Renn, 2015; Helanterä & Uller, 2019).

These three reasons to look for idealizations that combine the virtues of selective and transformational accounts are likely not the only ones, nor do we anticipate that they represent three separate categories. Nevertheless, asking what makes an evolutionary explanation more or less satisfactory appears a fruitful way to sharpen intuition about the role of plasticity in evolution and may assist in choosing an appropriate idealization.

6 | CONCLUSIONS

Understanding how biologists make use of idealizations to explain adaptive evolution makes it easier to interpret different views of the explanatory role of plasticity in evolution (e.g., Laland et al., 2014 vs. Wray et al., 2014). The reaction norm representation of plasticity is a simple heuristic tool that is easily accommodated within the genetic idealization of evolution by natural selection. While this makes developmental plasticity appear as an "add-on" rather than as a prima facie cause of adaptive change, this has more to do with the choice of an idealization than with any fundamental feature of the evolutionary process. We suspect that those who argue for conceptual change will not be satisfied with accommodating plasticity in traditional accounts. Therefore, we have drawn attention to evolutionary problems for which neither selective nor transformational explanations appear fully satisfactory. Further understanding of the role of plasticity in evolution will rely on the formulation of idealizations that explicitly draw attention to the causal relations between the environmental responsiveness of development and adaptive evolutionary change.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

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