Chapter 4
Causing and Composing Evolution: Lessons from Evo-Devo Mechanisms

Cristina Villegas

Abstract Evolutionary developmental biology (evo-devo) is often vindicated by theoreticians of the field as a mechanistic science that brings a mechanistic perspective into evolutionary biology. Usually, it is also portrayed as stressing the causal role that development plays in the evolutionary process. However, mechanistic studies in evo-devo typically refer to lineage-specific transformations and lack the generality that evolutionary explanations usually aim for. After reviewing the prospects and limits of a mechanistic view of evo-devo and their studies of homology and novelty, in this chapter I propose a way to combine the mechanistic view of evo-devo with the population-level inclination of more classical approaches to evolution. Such a proposal provides a philosophical framework for understanding the causal role of development in evolution both as mechanistic and as generalizable, population-level.

Keywords Evolutionary developmental biology · Innovation · Variation · Homology · Populations · Developmental repatterning

4.1 Introduction

If there is one type of explanation that has received the attention of most philosophers of biology in recent years, it is mechanistic explanation (Machamer et al., 2000; Glennan, 2002; Bechtel & Abrahamsen, 2013; Craver & Darden, 2013). So-called "new mechanicism" arose as a vindication of the non-nomological nature of many kinds of explanations in science, and it has been especially prolific in its application to biological phenomena. The mechanistic approach considers that there are scientific explanations without any appeal to fundamental laws of nature.

C. Villegas (✉)
Centro de Filosofia das Ciências, Departamento de História e Filosofia das Ciências, Faculdade de Ciências, Universidade de Lisboa, Lisbon, Portugal
e-mail: cvillegas@fc.ul.pt

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These consist in describing a system that is responsible for the *explanandum* by decomposing it into its component parts and the activities such parts are engaged in, and subsequently recomposing the system back in terms of the way the parts and activities are organized to produce the phenomenon under study (Bechtel & Abrahamsen, 2013). Unlike more classical types of mechanicism, the new mechanical philosophy does not intend to *reduce* phenomena to the components of a system, rather stressing the role of the organization of those component parts into a whole in bringing about phenomena. This feature makes mechanicism especially interesting for the life sciences.

Mechanistic explanations are particularly prominent to account for what Mayr once (1961) labeled *proximate* causes, namely the causes acting at the level of an organism, determining how it is and what it does. Unlike *ultimate* or evolutionary causes, proximate causes are responsible for the functioning and behavior of organisms. If a scientist seeks to explain *how*, say, hearts pump blood, then she needs to refer to the mechanism responsible for such pumping, which may include factors such as muscle contractions, regardless of the evolutionary history of the circulatory system. A well-known example is the mechanism for the circadian cycle, introduced by Bechtel and Abrahamsen (2010) to exemplify the features of mechanistic explanations. In this example, the relevant components are biochemical substances within cells, such as RNA, which act by regulating the presence of further biochemical components, such as proteins.

Evolutionary developmental biology (evo-devo for short) is often vindicated by theoreticians of the field as a mechanistic science (Wagner et al., 2000; Hall, 2003; Müller, 2007). This mechanistic aspect is important insofar as evo-devo is considered to bring a mechanistic perspective into the otherwise "mechanism-less" field of evolutionary biology. In particular, it is argued that taking development into account entails considering the mechanisms underlying phenotypic change, a precondition for evolution through natural selection that is assumed but not explained in classical approaches to evolution. From a classical perspective, evolutionary questions pertain to a separate domain of biological causes altogether than mechanistic ones: ultimate causes acting on populations throughout generations (Mayr, 1961). Some philosophers have argued that, in fact, evo-devo shows that there is no such separation between ultimate and proximate causes, and that, instead, there is a reciprocal causation between organismal and evolutionary causes (Laland et al., 2011). Nonetheless, combining these different kinds of explanation, "ultimate" or evolutionary, and "proximate" or mechanistic, is germane to this field. As Ron Amundson holds, the "difficulty of integrating population thinking with the mechanistic thinking of developmental biology" is inherent to evo-devo (Amundson, 2015, p. vii).

This scenario gets more complicated when further subtleties about biological causation are introduced. A more complete, still classical picture of biological causes is Tinbergen's (1963) categorization based on the four questions to be asked about the nature of traits: their survival value, their evolutionary history, their ontogenetic origin, and how they work in a *mechanistic* sense. Setting aside some ambiguities (see Conley, 2020), Tinbergen's schema is typically interpreted as specifying two distinct types of *proximate* causes *à la* Mayr: mechanistic explanations and

explanations of ontogeny (Bateson & Laland, 2013). Here, "mechanistic explanations" focus on physiological aspects independently of the way the traits are acquired during ontogeny, whereby this acquisition process represents a different type of proximate causal process. The most classical works on biological mechanisms seem to prove this association right, insofar as most mechanisms refer to the way some system works rather than how it came to be acquired during development, as in the classical biochemical example of the circadian cycle (Bechtel & Abrahamsen, 2010).

Paying attention to this schema, the abnormal situation of evo-devo becomes clear. Not only does it combine the proximate and the ultimate domain by bringing together organismal and evolutionary causes, but it does so through the consideration of *ontogenetic* causes rather than *physiological* ones. Some philosophers have pointed out the difficulty of applying the mechanistic schema to ontogeny, and thus to the domain of developmental biology (Mc Manus, 2012; Love, 2018; Baedke, 2021), which further complicates the task of applying it to evo-devo. Importantly, this difficulty concerns the causal nature of developmental mechanisms, and raises the question of whether developmental mechanisms are a cause of evolution, as argued by evo-devo theoreticians (Wagner et al., 2000; Müller, 2007). In the upswing of the new mechanicist philosophy, and given the apparent centrality of mechanisms in evo-devo (Baedke, 2021), it becomes important to analyze to what extent evo-devo is a mechanistic science and, moreover, what that says about the causal relation that development holds with evolution. That is the aim of this chapter.

The chapter is structured as follows. Section 4.2 reviews the prospects and limitations of considering evolutionary and developmental biology, separately, as mechanistic sciences. Section 4.3 addresses the mechanistic aspects of evo-devo studies of homology, arguing that they point at a causal and compositional[1] role of developmental mechanisms in phenotypic unity and diversity. Section 4.4 discusses the causal role of development in phenotypic changes, both in evo-devo studies of evolutionary novelties and within the broader domain of a mechanistic view of evolution. It introduces the idea of *developmental repatterning* (Arthur, 2011) as a population-level evolutionary mechanism responsible for biases in phenotypic change and innovations. Section 4.5 concludes with some final remarks about causation and mechanicism in evo-devo.

4.2 Two Unusual Kinds of Biological Mechanisms

Evo-devo is a highly interdisciplinary research area that combines insights and methodologies from developmental and evolutionary biology, both broadly construed. It brings a comparative and phylogenetic perspective to the study of developmental systems and, moreover, it sheds light into the developmental

[1] In this chapter, I use constitution and composition interchangeably.

processes underlying phenotypic change in evolution (Müller, 2007). Before assessing the mechanistic and causal aspects of evo-devo as a discipline, it is therefore convenient to review the scope and limitations of mechanicism for each of the disciplines that it intends to combine.

4.2.1 Mechanisms of Development

Developmental biology studies the process of organismal formation from zygote to adulthood (or some other developmental stage). Every multicellular organism goes through this process, which consists of a myriad of physical, molecular, cellular, tissue-level, and organism-level sub-processes. Despite the ubiquity of development within multicellular life, developmental biology largely lacks general laws (Minelli & Pradeu, 2014), instead focusing on species or taxa-specific processes, typically through the study of model organisms such as the fruit fly *Drosophila* or the weed *Arabidopsis*. Like many other branches of the life sciences, developmental biology was originally descriptive, providing phenomenological models of embryonic stages with little to no mechanistic content. But innumerable progresses have made developmental biology grow as an experimental science that identifies relevant components and causal factors in the formation of specific traits in organisms, making it a good candidate for being a field where mechanisms play an important explanatory role. For example, in the era of developmental genetics, there is a great deal of developmental biology that relies on experimental studies of correlative associations of genes. For instance, knockout experiments identify genes that *make a difference* in the adult phenotype by blocking their expression at a given developmental stage. Building mechanistic models of how such a difference is made is a crucial part of how developmental biology seeks to explain.

Let me now introduce an example that will guide us throughout the chapter. Flowers are the reproductive traits of angiosperm plants, a monophyletic group that separated from their closest extant relatives, gymnosperms (plants that generate unenclosed seeds, such as conifers), between 300 and 350 million years ago. They are not individual organs but a highly integrated set of them. While there is much flower diversity, a prototypic flower usually consists of at least four organs: sepals, petals, stamens, and carpels. The development of flower organs follows a pattern known as "the ABC model", which associates a specific combination of three to five homeotic genes (labeled *A*, *B*, and *C*; or *A*, *B*, *C*, *D* and *E* in more recent versions) to each of them (Theißen & Rümpler, 2021). Although only more complex versions of it seem to be currently accepted (Theißen et al., 2016), the classical model can serve for illustrative purposes. The basic idea of the model is that *A*, *B*, and *C* genes are expressed differentially in specific developmental stages, forming flower organs in a temporal and spatial sequence. For example, the original model states that expression of *A* genes forms sepals, while the combination of *A* with *B* genes forms petals. The sequential expression of, first, *A* genes and, then, *B* genes would then make sepals appear before and below petals in flowers. The model is not

mechanistic *per se*; rather, it identifies genes as *difference-making* causes for specific phenotypes through mutational experiments. However, mechanistic versions of the model identify the transcription factors that ABC genes code for, as well as the proteins that their target genes produce. It is the case for the "floral quartet" model (Theißen et al., 2016), where MIKC-type proteins derived from ABC genes form quaternary complexes that bind to specific DNA regions to control the expression of genes involved in the formation of the different flower organs. With this example in mind, let us see how to make sense out of developmental mechanisms.

The extent to which there is a defined set of entities that can constitute a developmental mechanism is not a simple question, but much work has been devoted to uncovering these entities in the last decades. Gene products within cells such as proteins and transcription factors are the main components of development at the chemical level, for they are responsible for important developmental factors such as signaling and gene-regulatory pathways that translate environmental and genetic inputs into phenotypic outputs such as cell differentiation. Extracellular and environmental components can also be crucial, as well as physical components such as mechanical stresses or bioelectrical potentials, all of these responsible for the behavior and expression patterns of cells at specific points of the developing organism. The number and kinds of components that are relevant for explaining a specific developmental phenomenon not only are vast but vary for each specific case. Still, the nature of these components is relatively well understood in model systems. In the model of flower organ development illustrated above, the entities specified are MIKC-type proteins that ABC genes code for, the sections of DNA where these proteins bind, and the gene products of those sections.

The complications arise when it comes to understanding the organization of activities carried out by developmental mechanisms (Jaeger & Sharpe, 2014). Developmental processes are dynamically complex. Explanations of developmental mechanisms shall count as *dynamic* mechanistic explanations, that is, as those in which the recomposition of the system involves the description of a nonlinear dynamic behavior (Bechtel & Abrahamsen, 2010). The regulation of specific variables such as gene expression is normally non-linear and allows, for example, for genes regulating their own expression, so that the variables and parameters of developmental systems tend to be time-dependent (Jaeger & Sharpe, 2014), turning the whole parametrization of the dynamics into a complex task. The result is complex behaviors such as threshold effects and feedback loops. For instance, in the ABC model of flower development, some protein complexes bind to two gene regions forming DNA loops (Wagner, 2014). In these cases, gene expression only results from a complex pattern of cooperativity and interdependency within the protein complexes and with their binding sites.

Some philosophers have pointed out that these complex developmental dynamics uncover some limitations of the mechanistic paradigm (Mc Manus, 2012; Love, 2018; Baedke, 2021). In explanations of non-developmental phenomena such as cell metabolism, the *explanandum* is extended in time but remains constant. Developmental phenomena, in contrast, are not only extended in time but of changing nature. When developmentalists seek to explain the development of a trait, such

as petals and sepals in flower plants, they need to account for profound changes in the nature and organization of its mechanistic elements, including in their hierarchical organization in levels. This is because the organ level, where we may situate *the flower*, is only the result of the process, but is not present in all the stages that precede its formation. For example, the floral meristem is a set of undifferentiated cells that precedes the flower but is not yet identifiable as a differentiated organ. Additionally, the flower forms out of the expression in meristem cells of E-class genes, which are a molecular rather than a physiological reality (unlike the floral meristem).

The conundrum of this problem relates to the causation/constitution distinction. When first introduced, the new mechanistic philosophy acknowledged a dual potential of the framework: in terms of *causal* explanations and in terms of *constitutive* ones (Craver, 2007; Bechtel & Abrahamsen, 2013; Romero, 2015). Causal explanations are those where *explanans* and *explanandum* are cause and effect, which demands that they are not the same entity and that there is an asymmetric temporal relationship between one another. A gene involved in the formation of the floral meristem can be a cause of the proteins responsible for meristem cell differentiation. In contrast, constitutive explanations are those where *explanans* and *explanandum* are different levels of description of the same system, which implies their ontological identity and temporal synchronization. A petal cell can be a constitutive part of the flower. Thus, a mechanistic explanation of a flower can mean two different things: a description of a *causal* mechanistic chain of events at the morphological level leading to the formation of the flower, or a description of a *constitutive* mechanism at a lower level such as the molecular. Developmental explanations, nonetheless, typically combine both (Mc Manus, 2012; Ylikoski, 2013). For example, cellular properties in the flower meristem can be both seen as causal factors and lower-level components in the formation of flower organs.

Given this complexity, other frameworks have been proposed for understanding both the nature of development and of developmental explanations. The processual view of development (Baedke & Mc Manus, 2018; Nuño de la Rosa, 2018; Bich & Skillings, 2023) argues that developmental processes are temporal parts of the life cycle, where dynamic organization plays a more fundamental role than the entities it gives rise to. For example, Baedke and Mc Manus (2018) contend that a better way of understanding hierarchical levels in development is to consider *time scales* instead of compositional relations. Similar arguments are found in the dispositional view of development (Hüttemann & Kaiser, 2018), where mechanisms are seen as the manifestation process of the dispositions of a system. By invoking a primordial dynamic developmental reality from which mechanisms can be abstracted away in scientific practice, these two positions avoid some of the complexities of understanding mechanisms as the fundamental ontology of development.

Nonetheless, and despite the complications derived from adjusting the complex science of developmental biology to the mechanistic framework, it is beyond doubt that developmental biologists explain by describing the components of developmental processes and their organizational properties, including their causal, spatial, and also temporal relations. The cruciality of these temporal relations, while

challenging, need not undermine a mechanistic framework, especially given that the new mechanistic philosophy constantly embraces new ways of accounting for complex dynamics (see Krickel this volume). For example, it has been recently argued that the distinction between causal and constitutive relations should be abandoned altogether since, at least in some sciences—including biology—, "diachronic causal constitutive relations" are instead the norm (Leuridan & Lodewyckx, 2021). According to this argument, the constitutive relations described in science are sometimes diachronic, implying some level of temporal extension where interlevel causation also plays a role. Such a consideration might help accommodate the fact that new levels of mechanistic organization are generated in development (e.g., the emergence in developmental time of the tissue level from pre-existing lower-level elements such as cells), a phenomenon that is sometimes considered to be out of the scope of mechanistic explanations (Baedke & Mc Manus, 2018; but see Austin, 2016 for a different view). In sum, whether or not there are currently enough philosophical tools to integrate developmental complexity, it seems that a mechanistic view has a strong potential to incorporate most of the developmental phenomena. How exactly to do so—and whether this view must be ontological or epistemological—remains an open question, which additionally complicates the task of accommodating these unusual kinds of biological mechanisms into the evolutionary domain.

4.2.2 Mechanisms of Evolution

Evolution is the historical process of transformation and diversification of the tree of life. The process itself is thought to follow similar rules of descent with modification throughout the whole tree, and finding general models on the basis of such rules is one classical target of evolutionary biology. One of its goals is thus to explain the phenotypic composition of species in terms of the rules governing the historical changes undergone by populations—and sometimes to predict short-term future ones. Unlike developmental biology explanations, these explanations are not usually mechanistic, in the sense that they don't consist of a decomposition (and recomposition) of evolutionary phenomena into entities and activities. Rather, they typically take the form of the application of statistical models involving population dynamics factors such as natural selection, genetic drift, and mutational rates.

As mentioned in the introduction, Mayr's (1961) classical picture labels these factors *ultimate causes*. However, some philosophers reject the idea that one can talk about causes at the level of population dynamics (Walsh et al., 2017, reviewed in Pence, 2021). Their viewpoint is that the classical population-level explanations of evolution do not reflect evolutionary causes, because the real causes of evolution act at the level of individuals and their relation to the environment (Walsh et al., 2017). From this position, "ultimate" explanatory terms such as genetic drift are statistical, and only organism-level explanations of the trends they represent provide causal explanations of evolution. This alleged lack of causal content in classical evolutionary explanations is sometimes referred to as a lack of *mechanistic* content

(Pigliucci & Kaplan, 2010). From this point of view, organism-level mechanisms are considered causally responsible for the changes that underlie the population-level trends we see in evolution. One task for the agendas extending the classical framework of evolution, including evo-devo, is therefore to study the interplay between those individual-level mechanisms and their population-level effects (Laland et al., 2011). For example, evo-devo studies how differences in the mechanisms of flower organ development may have resulted in patterns of selection (Mondragón-Palomino et al., 2009).

Other scholars defend that the causes of evolution are fairly described at the population level. Organism-level mechanisms such as developmental or ecological ones *compose* evolutionary causes at a lower level from this point of view. The rationale is that, since they are not difference makers of populational changes, these organism-level developmental and ecological mechanisms need not be considered in causal evolutionary explanations (Millstein, 2003). There are different ways of interpreting the population-level causes present in classical evolutionary explanations, including the mechanistic perspective. Thus, a number of philosophers have provided tentative analyses of what a mechanistic view of population-level evolutionary causes would look like (Skipper & Millstein, 2005; Barros, 2008; Illari & Williamson, 2010; DesAutels, 2016, 2018). From this position, organism-level developmental and ecological mechanisms can be considered components of the population-level evolutionary *mechanisms* referred to in models of population dynamics. One mechanistic task for evo-devo would therefore consist in specifying the role of development in population-level mechanisms of evolution.

The philosophical enterprise has nonetheless mostly been limited to natural selection, following the usual label in the scientific literature, where selection is often called a "mechanism" of evolution (e.g., Bell, 2008). In addition, most of the attempts have focused on pointing out limitations of the first new mechanistic views to account for selection, and considerations of more recent mechanistic developments are rare. For example, Skipper and Millstein (2005) first noticed that the irregularity of evolutionary phenomena made it unsuitable to be analyzed in terms of the early mechanistic views proposed by Machamer et al. (2000) and by Glennan (2002), and suggested that only a "stochastic" or probabilistic mechanistic approach could deal with such difficulties. However, while most later mechanistic views acknowledge the stochastic nature of mechanisms, the irregularity of natural selection may not entirely be captured by stochasticity. As Pérez-González and Luque (2019) point out, this irregularity is germane to the fact that natural selection always acts in conjunction with other evolutionary factors such as mutations and migrations, and is inseparable from its counterpart genetic drift. A population that does not change at the rate predicted by selection is, by definition, a population that undergoes drift. This irregularity problem applies more generally to any attempt to understand selection in mechanistic terms insofar as it is hard to identify the specific phenomenon that it produces as distinct from the results of other evolutionary factors (Beatty, 1984). This is why functional approaches identify the phenomenon that selection explains with a specific *process* rather than an outcome (DesAutels, 2016),

pointing at the events underlying the higher survival and reproductive success of fitter organisms in populations.

Similarly to the worries raised about dynamism for developmental mechanisms, the foundational article from Skipper and Millstein (2005) further argued that selection is better described as composed of stages or time-slices rather than parts. In addition, it stressed that the interactions that comprise it do not fit the standard criteria of a mechanistic view, since the relevant activities attributed to selection (such as those organisms engage in during reproduction and in their relation to the environment) are at the very least suspicious of lacking regularity. One attempt to solve this consists in relaxing the criteria of stability of both entities and activities. According to Illari and Williamson's (2010) view, these need only be *functionally stable*, that is, stable enough to produce the phenomenon of natural selection. Following their account, selection fits into a "functional hierarchy" composed of a myriad of mechanisms acting at different lower levels to produce the enhanced survival and reproduction of fitter individuals. These lower-level mechanisms are composed of very diverse types of entities such as populations, organisms or chromosomes, and activities like sexual selection, recombination, or reproduction, presenting organization insofar as they combine to produce selection.

It is in this sense that developmental and ecological mechanisms are perceived as components of population-level mechanisms, although there is no consensus about how to understand this compositional relation. For example, while Illari and Williamson (2010) advocate for selection as a highly complex multilevel mechanism, authors such as Barros (2008) stress that selection is a two-level mechanism: it acts at the level of the individual-environment interaction and at the population level.

However, there is no point in discussing the components of selection without a clearer view of how they relate to other causes of evolution, such as drift or mutations (DesAutels, 2018; Pérez-González & Luque, 2019). In particular, functionally individuating the lower-level mechanisms composing selection demands criteria for discerning when they compose other higher-level causal factors of evolution. Indeed, disagreements about the levels composing selection as a mechanism might indicate a disparity in the evolutionary phenomenon it is supposed to explain. Here is where non-classical agendas of evolution may be of help, since describing the complexity of evolutionary mechanisms and how they interact is part of their agendas. In the case of evo-devo, one goal is to understand how evolutionary mechanisms relate to developmental ones. The issue actually comprises two different aspects of evolutionary mechanisms. The first one is: are other population-level causes of evolution mechanisms too? As a matter of fact, very few attempts have been made to account for genetic drift, mutations or migrations in population-level mechanistic terms (DesAutels, 2018), and there seems to be no framework for understanding the way in which these putative mechanisms may interact with one another (Pérez-González & Luque, 2019). The second one is: are the lower-level mechanisms composing evolution *evolutionary causes* too? The mechanistic view of evolution seems to involve causes and mechanisms at different levels and, as such, needs to deal with how these different levels of causation relate to one another. With these questions in mind, let us turn to the mechanistic aspects of evo-devo.

4.3 *Evo* and *Devo*: The Mechanistic Composition of Variation

The main explanatory agendas where evo-devo clearly introduces a mechanistic perspective are the study of homology and of evolutionary novelty. These are the two sides of the same coin: novelty is present whenever a trait lacks an homologue in its ancestral lineage and is not homologous to a different body part of the same organism. In these studies, developmental mechanisms are seen in a comparative and phylogenetic framework, and the goal is to explain the mechanistic bases of phenotypic commonalities and diversity. This is an important task in the evo-devo agenda, for it brings a mechanistic aspect to the study of phenotypic variation, which is a necessary condition for evolution to take place, but mostly assumed without explanation in classical evolutionary approaches.

Intraspecific variation—the one that matters for classical, microevolutionary approaches—implies the existence of the same trait in different forms in a given population: for instance, bigger or smaller versions of the same wing in a bird species, brighter or darker color versions of the same petal in a plant species, etc. This phenotypic variation always implies variation in the operation of the same developmental mechanisms, be it a slightly different interaction among its components, a different concentration of some of its elements, or a different interaction between the mechanisms and their environment. But when one looks at the inter-specific level, the issue gets more interesting. Homology is the presence of the same unit (an organ, a cell type, a morphological trait, a behavior) in different species. The study of homology is central to evo-devo because it tells us about what has been preserved in evolution and how it varies. One central question for evo-devo is thus whether there is homology of the developmental mechanisms responsible for homologous traits. In other words, whether there is correspondence in homology across different levels of composition of phenotypic diversity. This question gets right at the problem of characterizing developmental mechanisms, for it concerns how much variation in the developmental mechanism corresponds to variation in the phenomenon it explains, i.e., the phenotype.

It has been argued that developmental mechanisms have a "hybrid nature" (Newman, 2014): molecular and physico-cellular. While much of developmental biology focuses on the molecular level, physico-cellular mechanisms can be described for most developmental processes, complementing the description in terms of molecular ones. For example, in the case of flower development, cell division in the floral meristem prior to the activation of ABC genes depends on the radial position of cells, a physical property, regardless of the specific gene expression profile of the cell in question (Alvarez-Buylla et al., 2010). This could at first sight simply be interpreted as concerning the nested nature of mechanisms, where molecular genetics mechanisms (lower-level) compose cellular-physical ones (higher-level), but it actually concerns a much deeper developmental problem (Love, 2018). Comparative studies at different levels of organization have made it plain that seemingly homologous traits may be realized by a multitude of

mechanisms at a lower level, while apparently diverse traits can be realized by similar developmental mechanisms (DiFrisco et al., 2020). Thus, many different genetic mechanisms are constitutive of the same higher-level, morphogenetic mechanism in different species. And the opposite also holds: many morphogenetic mechanisms of development, such as tissue-level mechanical forces, vary more across species than the molecular mechanisms supposedly composing (and causing) them.

This poses a serious challenge for evo-devo research: are conserved mechanisms responsible for the generation of homologous traits despite this pervasive diversity? Or is homology of traits acquired in other ways relatively independent of genetic and developmental conservation? The two positions illustrated by this (even if very simplified) dichotomy indeed represent a significant divide in approaches to the problem of homology in evo-devo (Nuño de la Rosa & Etxeberria, 2012). On the one hand, character identity views of homology postulate the high conservation of core developmental mechanisms that provide "identity" to traits inasmuch as they are involved in the production of variants of the same character under an array of developmental contexts (Wagner, 2014; DiFrisco et al., 2020, 2023). On the other hand, organizational views of homology hypothesize positions of phenotypic stability where different developmental mechanisms converge in virtue of the internal organization of body plans, and that it is this convergence what characterizes trait homology, rather than the sameness of mechanisms underlying phenotypes (Müller, 2003; Newman, 2003; Peterson & Müller, 2016). The idea of mechanism is central to both positions, but for different reasons. Let me explain.

The "character identity mechanisms" view of homology proposes that there are level-specific mechanisms that explain homologous traits, and are responsible for their traceable identity in evolution at different levels (DiFrisco et al., 2020, 2023). Character identity mechanisms are "mechanistic architectures" with specific causal profiles that are retained in evolution despite changes in the inputs activating them as well as in the realization of their phenotypic effects:

> [Character identity mechanisms] are less replaceable in evolution than their upstream signaling inputs and downstream effector mechanisms ... [A]s a result, ... [they] are more likely to be evolutionarily conserved than other developmental mechanisms. (DiFrisco et al., 2020: 8–9).

The key idea is that the persistence of traits in evolution is explained by the recurrent instantiation of mechanisms causing and composing them in virtue of their parts, activities and organization. As mentioned, the nature of these mechanisms can differ depending on the level of organization of each phenotypic trait. For example, for cell types, a character identity mechanism is postulated to be composed of a gene regulatory network with cross-regulatory and signaling activity, while for tissues it is composed of cell types, extracellular matrices, signaling molecules, and the inter-cell signaling complexes they all engage in (DiFrisco et al., 2020). Depending on which specific cell type or tissue identity the mechanism is responsible for, the specific nature of these entities and activities changes. In addition, while these mechanistic profiles remain stable in different species, the overall process they are part of may undergo several types of changes. For example, they

specify the identity of the trait but not its "character state" or specific realization (Wagner, 2014; DiFrisco et al., 2020). The ABC model of flower development could exemplify this idea, for it describes a pattern found across all angiosperms despite the overwhelming diversity in flower morphology (e.g., size, shape, color, number and arrangement of each of the organ types). According to this view, this points at the retention of the elements and organizational lower-level mechanistic structure responsible for the identity of flower organs, like ABC genes, but not of other mechanistic components that explain the specific features of particular, species-specific flowers. Interestingly, this retention is not always explicable in terms of natural selection, for the causal profile of character identity mechanisms is sometimes independent of function. For example, most flowers are bisexual, with both stamens and carpels—male and female organs, respectively. However, in the rare cases of unisexuality in flowers, stamens and carpels develop too, only that one of these organs is sterile. This suggests that the floral plan, which includes sepals, petals, stamens and carpels, and is explained by the ABC model of development, may be developmentally retained despite changes in function, and thus that selection is not the only responsible for the co-occurrence of floral organs (Wagner, 2014).

From the perspective of the organizational view of homology (Müller, 2003; Newman, 2003; Peterson & Müller, 2016), traits are retained because of their organizational role in development and inheritance. However, unlike in the previous approach, traits can be homologous independently of their mechanistic composition at any given level. That is, different developmental mechanisms can converge to produce homologous traits. Also, two lineages may retain a homologous trait even if the underlying mechanisms for it undergo severe, independent changes. Therefore, in this case, the hypothesis is that variation in developmental mechanisms does not correspond entirely to variation in phenotypes. From this view, one may argue that, instead, traits are individualized in virtue of their causal and compositional *role within the entire body plan*, the latter understood in mechanistic terms or not. That is, while the identity of a trait is independent from its mechanistic basis, it depends on its specific organizational role within the developing organism and in reproduction:

> Homology denotes constancy of constructional organization despite changes in underlying generative mechanisms ... Homologues act as organizers of the phenotype ... [and] as organizers of the evolving molecular and genetic circuitry (Müller, 2003: 64).

If we understand the organism and reproduction in mechanistic terms, then traits are individualized as specific mechanistic components engaged in specific constitutive and causal activities. I will use the ABC model of flower development for exemplifying this idea too. From the organizational view of homology, the developmental pattern found across angiosperms does not point at the retention of any specific lower-level mechanistic element (such as a particular protein type). Rather, it points at the organizational role of the pattern itself within the development of angiosperms. As in the case with the character identity view of homology, the retention of the pattern does not depend solely on natural selection. In this case, it depends on the whole developmental organization of flowering plants.

These two different evo-devo approaches to homology provide mechanistic pictures of what prevails and varies in evolution, partially explaining patterns of interspecies phenotypic variation. Taking the perspective of developmental mechanisms as both causal and constitutional (Ylikoski, 2013), they both give a causal and constitutive partial explanation of extant variation. On the one hand, character identity mechanisms (DiFrisco et al., 2020) stress what are the relevant causal and compositional *lower-level relations* that make a developmental mechanism instantiate a specific type. On the other hand, the organizational view of homology (Müller, 2003) emphasizes what are the relevant causal and compositional *organismic-level relations* that make a developmental system generate a specific phenotype.

4.4 Causing Phenotypic Change

As mentioned above, homology and novelty are the two faces of the same coin. Thus, having a criteria for what counts as homology is tantamount to having criteria for discerning what is an evolutionary novelty. However, evo-devo is not just concerned with what counts as a new trait. It also seeks for mechanistic explanations of the phenotypic changes raising novelties.

Traditionally, phenotypic change has been associated with factors external to the organism, such as the occurrence of mutations or recombination through directional selection. From an evo-devo perspective, however, what matters is that external factors trigger phenotypic changes that significantly depend on the properties of the developmental system. In the case of evolutionary novelties, external factors can be seen as "initiating conditions" of phenotypic change, while developmental systems act as the "realizing conditions" of those changes (Müller & Newman, 2005). Taking a mechanistic approach to evo-devo, it follows that the features of developmental mechanisms causally contribute to the directionality of evolutionary change. In this last section, I revise some mechanistic aspects of evo-devo studies of evolutionary novelty (Sect. 4.4.1), and I introduce the idea of developmental repatterning as a population-level mechanistic component of evolution (Sect. 4.4.2).

4.4.1 Mechanistic Views of Innovation

Evo-devo approaches to homology also postulate mechanistic views of how evolutionary novelties may arise. From the "character identity mechanisms" perspective, evolutionary novelties can be explained by a reuse (through co-option) of a mechanistic component of one trait into another identity mechanism (DiFrisco et al., 2023). Some genetic changes lead to the reuse of the same developmental components, often by duplication, into a different trait. This reconfiguration of the mechanism underlying the trait may give rise to an evolutionary novelty. On the other hand, the organizational view of homology sees the very process of innovation as a

mechanism based on developmental properties (Peterson & Müller, 2016). Here, innovation is depicted as a number of stages where new components and activities arise from the previous ones, giving rise to new homologues (Müller, 2003; Newman, 2003). The idea is that the origin of traits is mostly driven by the cell, tissue, and epigenetic-level processes in a first phase of generation, before being genetically accommodated through canalization or similar processes at a phase of integration. Finally, traits become increasingly independent from the mechanisms of innovation involved in their generation (Müller, 2003).

However, providing mechanistic details of how novelties arise requires a complex picture that is rarely attainable. Philosopher Brett Calcott pointed out that most explanations of novelty in evo-devo take the form of lineage explanations, which actually consist of series of independent mechanistic explanations (Calcott, 2009). An evo-devo lineage explanation gives a set of mechanistic models of a developmental system, each of which explains (constitutes and causes) a particular stage in an evolutionary series of phenotypic variants. The requirement for such a set to constitute a lineage explanation is that each mechanistic model is linked to the next one by a continuity requirement. This means that one mechanism must be similar enough to the next one so as to justify that the one could be the result of a minor modification in the other. Therefore, one shall point at the right components in decomposing a developmental mechanism, because it is continuity in these components that warrants the plausibility of lineage explanations (Calcott, 2009).

It is important to stress that lineage explanations don't provide mechanistic explanations of evolutionary change, but a series of plausible (gradual) evolutionary changes in mechanisms (Kaiser, 2021). For the origin of flowers, evo-devo models have proposed that small changes in gene expression, such as the accumulation of higher levels of protein concentration or the acquisition of new binding sites, could have gradually resulted in the emergence of the ABC pattern of flower development (Baum & Hileman, 2006). But the causes of such changes are typically left outside the lineage explanation (Calcott, 2009): what caused the incremental changes in protein concentration or binding sites number is external to the developmental mechanisms described, and therefore not included in the explanation of the origin of the developmental pattern. Additionally, many evolutionary novelties involve the emergence of new levels of mechanistic organization, meaning that continuity between mechanisms does not always imply graduality.

Following the continuity requirement involves knowledge about *potential changes* in developmental systems. Several authors have pointed out that individuating developmental systems indeed demands a consideration of this potential, understood in dispositional (Brigandt, 2015; Austin, 2017) or topological (Jaeger & Sharpe, 2014) terms, thus falling beyond the limits of what a mechanistic account can deliver. This is indeed a crucial aspect of the evo-devo research agenda: it combines mechanism-based knowledge with quantitative means of explanation in order to introduce development into the broader evolutionary domain (Brigandt, 2015). Here, evo-devo typically abstracts away from specificities of developmental mechanisms, often making use of dispositional or even mathematical means of explanation. Hybrid explanations, where dispositional and topological explanations are

combined with mechanistic insights, are actually the norm in most evo-devo cases (Brigandt, 2015; Huneman, 2018).

This is not just a methodological observation. In the context of evo-devo, the issue of what a developmental mechanism is remains naturally intertwined with the issue of what kind of phenotypic transformations it can undergo in evolution, which normally demands explanatory means beyond the decomposition and recomposition of a system. For instance, Jaeger and Sharpe (2014) point out that developmental mechanisms shall be identified by a particular way of bringing about phenotypic changes, based on topological similarity in a configuration space of possible phenotypes. Similarly, Austin (2017) stresses that what characterizes an evo-devo ontology is that:

> it is [developmental] systems' intrinsic generative capacities which are causally responsible for providing the morphological novelty which subsequently shapes the *evolutionary* (read: selective) *landscape*. (Austin, 2017, p. 377, stress added).

In other words, the internal properties of developmental systems are responsible for their own variational tendencies in evolution, i.e., for the way they can vary (Wagner, 2014). This is why the bridge between developmental insights and the evolutionary approach has been vindicated in terms of the *variational dispositions* (Austin & Nuño de la Rosa, 2021) or *propensities* (Nuño de la Rosa & Villegas, 2022) that a developmental system has insofar as it has a tendency to generate evolutionary variation in specific ways. Mathematical means for measuring these propensities are indispensable, as exemplified in the use of the genotype-phenotype map as a mathematical instrument for predicting phenotypic changes from genotypic ones, and the use of morphospaces for studying the feasibility of evolutionary transformations. These methodologies typically intend to bridge evo-devo approaches to population level studies of evolution. Studies in flower evolution also exemplify this evo-devo approach, where the use of floral morphospaces, or mathematical spaces of possible flower phenotypes, are tools for studying the evolutionary dynamics of flowers in terms of both selective and developmental factors (Chartier et al., 2014). Although introducing mechanistic knowledge into genotype-phenotype mapping is an emergent tendency for increasing their predictive accuracy (Pavličev et al., 2023), there is a trade-off between this accuracy and their generality. In sum, it seems that evo-devo studies of novelty and developmental innovation are not always improved by increasing the level of mechanistic content (Brigandt, 2015).

4.4.2 Innovation as an Evolutionary Mechanism

We are now left with the task of relating evo-devo explanations to a mechanistic picture of evolution. The previous sections summarized the mechanistic view that is mostly regarded in evo-devo: an analysis of how specific mechanisms of development vary or can vary in evolution. This section concerns another interest of evo-devo, namely how development *systematically* biases the production of evolutionarily

relevant variation in lineages and populations. Notice the difference here. We have seen that developmental mechanisms are involved in the causation and composition of the phenotypic variation available for other evolutionary factors to feed upon. What we have not seen yet is whether there is a way to integrate the role of developmental biases in population-level causal explanations. For this, we need to assess how developmental mechanisms relate to populational causes, including alleged evolutionary mechanisms such as selection, drift and mutations.

One way to do this is to adhere to the statisticalist view of evolution (Walsh et al., 2017), and consider that developmental mechanisms, acting at the level of organisms, are causally involved in the only process that matters to evolution: the life cycles of individuals. These mechanisms make a difference to the way the reproduction of organisms give rise to new phenotypes. In particular, a mechanistic understanding of development contributes to a finer-grained picture of evolution by providing a mechanistic (partial) description of the generation of new variants in a specific lineage. Mutational and recombination events would trigger the response of developmental mechanisms, which constrain the phenotypic outcomes those triggers can generate (Baetu, 2012). In this picture, there are no ultimate causes: there are only *proximate* causes, in this case *ontogenetic*, that engage in a relation of reciprocal causation with the environmental needs of organisms (Laland et al., 2011).[2] This process of reciprocal causation *explains* the statistical trends taking place at the population level.

One could be satisfied with this view, since standard evolutionary biology is clearly not a mechanistic science: it does not explain evolutionary changes by decomposing a system into its parts and activities and recomposing it back. Rather, it takes the form of a statistical explanation. However, claims about the causal impact of development in evolution must be taken seriously from a causalist position too. Thus, one shall consider that development has a role in these population-level causes supposedly represented in statistical models. Advocates of the mechanistic view of evolution try to understand population-level evolutionary causes better by fitting them into the mechanistic framework. Thus, the evo-devo mechanistic question is: is there a way in which development is relevant *qua* evolutionary mechanism?

In population-level mechanisms of evolution, specific causal chains are *instantiations* of the mechanism as a *type*. For example, specific ecological processes that explain particular adaptations are instantiations of natural selection as a type of mechanism (Skipper & Millstein, 2005). Similarly, we need to think of developmental biases and innovation not as the result of specific causal chains in lineages—as those explained in terms of lineage explanations (Calcott, 2009)—but as a *type* of evolutionary phenomena instantiated in different causal chains. Recall that the evo-devo agendas on homology provide general views of innovation, either by pointing at the co-option of a mechanistic component into a different character

[2] However, it is not clear that reproduction counts as an organismal process, as it necessarily involves at least two organisms. Although this might hinder a purely organism-centered view of evo-devo (see Villegas & Triviño, 2023), it does not affect the arguments of this chapter.

mechanism (DiFrisco et al., 2023) or by stressing the stages of generation, integration and autonomization of a new trait (Müller, 2003). These mechanistic structures may be general enough to qualify as a *type* of evolutionary mechanism in the population-level sense. For example, the generation of a new phenotypic component through epigenetic processes can take place in any given population. However, these proposals still refer to particular kinds of developmental bias, and particularly of innovation, but not all kinds of phenotypic change seem to be explainable in their terms.

Both classical and recent work on evo-devo mechanisms can cast light into the mechanistic picture of evolution through the broad notion of "developmental repatterning" (Arthur, 2011). Developmental repatterning is a term used to refer to generalizable changes in development that produce evolutionary changes, such as heterochrony, heterotopy, heterometry and heteronomy (or heterocyberny, see Moczek, 2019). These processes make reference to changes in timing, location, amount or nature, respectively, of a component in a developmental mechanism that produces a phenotypic change. Classical evo-devo work is interested in these phenomena as a developmental kind of *evolutionary mechanism* (Hall, 2003), although the literature on evo-devo mechanisms has not dealt in detail with this broader idea of mechanism. It is important to stress that these developmental phenomena are identified in the literature as mechanisms of evolution, as exemplified by heterochrony as "a mechanism for evolutionary diversification of flower form" (Endress, 2006, p. 5). For example, changes in the timing of sepal production in the floral meristem explain variation in the size and number of sepals in the flowers of *Dipsacoideae* species (Naghiloo & Claßen-Bockhoff, 2017).

The kinds of phenotypic changes produced by developmental repatterning can be gradual, such as the size and number variation in sepals just mentioned, but they can be evolutionary novelties too. For example, heterotopy of B-type and C-type gene expression seems to have been involved in the origin of the flower plan. These genes are associated with male and female organs, respectively, in the ancestral lineage of angiosperms, and their conjunct expression in the same axes may have resulted in the origin of the bisexual plan of flowers (Wagner, 2014). Both the small heterochronic changes producing sepal size variation and the greater heterotopic changes involved in the origin of flowers are specific instances of developmental repatterning.

In turn, developmental repatterning refers to an abstract mechanistic explanation of how phenotypic variants and novelties arise in populations. Therefore, it can be used in the same sense that selection, drift and mutations can be thought of as mechanisms: general mechanistic structures that are alluded to for explaining populational phenomena in evolutionary explanations, and that can be described mechanistically only in the context of other explanatory agendas with a focus on organism-level phenomena. In these organism-level mechanistic explanations, scientists are not explaining *the* mechanism of, say, heterochrony, just like ecological mechanistic explanations don't explain *the* mechanism of natural selection. Rather, they explain the mechanism for a particular heterochronic change in a lineage (such as the heterochronic change in sepal development), or a particular episode of selection in a population.

For developmental repatterning to count as a mechanism, it must be accountable in terms of entities and activities organized in a certain way. Here I provide a very minimal characterization. At the very least, two individuals forming a lineage must be involved. Importantly, their genotypes, phenotypes, and developmental mechanisms are constitutive parts of developmental repatterning, and so are their own mechanistic components at lower levels. The relevant activities for developmental repatterning are reproduction, development, mutations and recombinations. These entities and activities are organized in such a way that reproduction of organisms generates new phenotypic variation biased by the properties of the underlying developmental mechanisms. Biased phenotypic changes functionally individuate developmental repatterning as a mechanism, similarly to how enhanced survival and reproduction of a type individuate selection. Again, the specific phenomenon produced can be very variable, ranging from minor changes to phenotypic novelties[3], just like selection can lead to the stabilization of a trait or to the emergence of complex adaptations. The difference will lay in how reproduction combines the mechanistic elements present in the lineage given the inputs it receives from mutations, recombinations, or environmental elements.

How does developmental repatterning relate to other population-level mechanisms of evolution? In mechanistic views of selection, phenotypic variation has tended to be considered either as a temporal stage (Skipper & Millstein, 2005) or as a component entity in a lax sense of natural selection (Illari & Williamson, 2010). This reflects the fact that variation is a precondition for evolution by natural selection to occur. As such, it tends to be assumed whenever mechanistic accounts of selection are discussed: it is *variation* in the way individual organisms deal with the environment that allow for differential survival in populations. However, this treatment ignores another fact about variation, namely that it is *produced* in iterations of reproduction independently of natural selection. The multilevel mechanistic proposal of selection (Illari & Williamson, 2010) is an exception, interpreting phenomena such as recombination or epistasis (i.e., gene expression that depends on the presence of other genes) as part of what constitutes selection. Considering these development-related phenomena as part of natural selection is nevertheless problematic. They are not part of selection but of the phenotypic *response* of the population to an episode of selection. That is, they are part of the way the reproductive and developmental properties of the population provide new variation once an episode of natural selection has occurred. Here is where developmental repatterning enters the scene.

Responses to selection depend on the genotype-phenotype structure, namely the way genotypic variation maps into phenotypic one. Although there is debate over whether the genotype-phenotype structure has evolved such that it promotes or facilitates adaptation, developmental organization is involved in producing all kinds of variation, adaptive or not. A population that changes through drift or mutations

[3] Including novelties driven by co-option of lower-level mechanistic elements (DiFrisco et al., 2023) or by epigenetic processes (Müller, 2003).

will also generate (sometimes new) variation mediated by the genotype-phenotype structure. Thus, the developmental repatterning responsible for phenotypic responses cannot be solely a constitutive of any of the other population-level mechanisms of evolution such as selection or drift. Instead, it is one mechanistic component of evolutionary change acting in conjunction with other population-level mechanisms. Given the ongoing nature of cycles of differential reproduction and generation of variation that evolution consists of, developmental repatterning can be seen as a previous mechanistic step for natural selection or drift, or as the step following them and mutations. In any case, it is a distinct mechanism that shall not be conflated with other population-level evolutionary mechanisms.

4.5 Concluding Remarks

We have seen that a great deal of the evo-devo agenda is mechanistic, especially when it comes to individuating developmental systems phylogenetically through the study of homology and evolutionary novelties. Here, it seems that there is an inclination in evo-devo to regard causal and constitutive relations of developmental elements and activities as explanatory of phenotypic unity and diversity. In this sense, there is a clear causal aspect of developmental mechanisms in the production of evolutionarily relevant variation in specific lineages. However, bringing mechanistic components to general evolutionary explanations and predictions is a different story. Mechanistic knowledge of developmental systems needs to be used in combination with other means of explanation, mostly topological and dispositional, sometimes to the extent that there is little mechanistic content in some evo-devo explanations of phenotypic change (e.g., through statistical uses of the genotype-phenotype map). This is not a limitation of the evo-devo approach, but a very interesting point of connection with more classical approaches to evolution. While it is obviously concerned with the mechanistic aspects of development that are relevant to evolution, the field is growing significantly in its incorporation of developmental biases into population-level studies of evolution. In doing so, it makes a more general statement about the causal impact of development that is not always explained in terms of specific developmental mechanisms.

This situation may seem to imply that it is not the mechanistic aspect of evo-devo what justifies the causal role of development that it forcefully vindicates, and therefore that the two lemmas of evo-devo are not directly related (cf. Wagner et al., 2000; Hall, 2003; Müller, 2007). However, in this chapter I have provided an alternative view through the characterization of developmental repatterning as a population-level evolutionary mechanism. Previous views about evolutionary mechanisms failed to articulate the relation that variation holds with other evolutionary factors understood as mechanisms. Developmental repatterning provides a mechanistic view of the generation of variation that acts in combination with other mechanisms of evolution. The generality of developmental repatterning as a mechanistic structure means that it is not restricted to the impact of specific developmental

mechanisms in a particular lineage—as it is often thought to be the main contribution of evo-devo. Rather, it refers to the organizational properties of all lineages that, through reproduction, development, mutations and recombinations, channel phenotypic changes through the properties of developmental mechanisms. I believe that incorporating developmental repatterning as the mechanism for phenotypic change and evolutionary novelty into the broader picture of evolutionary mechanisms helps situate better the agenda of evo-devo and its vindications on the causal role of development into our philosophical discussions of evolution.

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