

THE PRODIGAL GENETICS RETURNS: INTEGRATING GENE REGULATORY NETWORK THEORY INTO EVOLUTIONARY THEORY

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Aaron Michael Novick

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This dissertation was presented

by

Aaron Michael Novick

It was defended on

September 05, 2018

and approved by

James Woodward, Distinguished Professor, History and Philosophy of Science

Mark Wilson, Distinguished Professor, Philosophy

Sandra Mitchell, Distinguished Professor, History and Philosophy of Science

Mark Rebeiz, Associate Professor, Biological Sciences

Dissertation Director: James Lennox, Emeritus Professor, History and Philosophy of
Science

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The aim of this dissertation is to show how gene regulatory network (GRN) theory can be integrated into evolutionary theory. GRN theory, which lies at the core of evolutionary-developmental biology (evo-devo), concerns the role of gene regulation in driving developmental processes, covering both how these networks function and how they evolve. Evolutionary and developmental biology, however, have long had an uneasy relationship. Developmental biology played little role in the establishment of a genetic theory of evolution during the modern synthesis of the early to mid 20th century. As a result, the body of evolutionary theory that descends from the synthesis period largely lacks obvious loci for integrating the information provided by GRN theory. Indeed, the relationship between the two has commonly been perceived, by both scientists and philosophers, as one of conflict. By combining historical and philosophical analysis, I consider four sources of tension between evo-devo and synthesis-derived evolutionary theorizing in order to show how those tensions can be resolved. I present a picture of the conceptual foundations of evo-devo that reveals the potential for integrating it with existing evolutionary theorizing. In chapter two, I argue that a major historical source of tension between evolutionary and developmental biology was the debates, in the first half of the

20th century, about the possibility of explaining development in terms of gene action. I show that the successes of GRN theory put these worries to bed. In chapter three, I argue that, rather than conceive of evo-devo as typological, we should see it as resting on Cuvieran functionalism. I argue that Cuvieran functionalism complements the Darwinian functionalism of the modern synthesis. In chapter four, I present a picture of the fine structure of the concept 'homology'. This picture shows how accounts of homology that have traditionally been taken to conflict are in fact compatible and complementary. In chapter five, I analyze the nature of structure/function disputes in terms of types of answers to contrastive why-questions. On the basis of this analysis, I show how the structure of evolutionary theory requires both structuralist and functionalist approaches.

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PREFACE

This dissertation bears my name, I suppose to signal my responsibility for its faults, but it is really a joint production, and many people deserve praise for its virtues, should it be found to possess any. I hope to credit them all below.

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Chapter four of this work, “The Fine Structure of ‘Homology’”, is reprinted with permission from Springer: *Biology & Philosophy*, “The Fine Structure of ‘Homology’”, Aaron Novick, © 2018.

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1.0 INTRODUCTION

1.1 FIRST THOUGHTS

Evolutionary developmental biology, or evo-devo, is a flourishing and multifaceted field of research, but its place in the broader landscape of evolutionary theorizing is difficult to determine precisely, for a number of reasons. For one, the organizational structure of many biology departments separates ecology and evolution from molecular biology, with evo-devo falling on the molecular side. Institutionally, then, evo-devo is separate from most evolutionary theorizing. A second reason, reinforcing the first, is that evolutionary theory today descends from the modern synthesis period of the 1930s-1960s, during which a number of biological subfields coalesced around a genetic approach to solving evolutionary problems (Jepsen 1963; Mayr and Provine 1981; Dobzhansky 1982; Mayr 1982; Simpson 1982). Somewhat notoriously, however, developmental biology was excluded from the synthesis, though what exactly caused this exclusion remains a subject of debate (Amundson 2005; Davis, Dietrich, and Jacobs 2009; Love 2009b). Third, evo-devo is itself a complex science, and these complexities can be seen both diachronically

(evo-devo has changed dramatically since its inception in the late 1970s) and synchronically (multiple traditions within evo-devo co-exist and sometimes come in conflict).

My aim in this dissertation is twofold: (i) to analyze the conceptual foundations of evo-devo and (ii) to clarify its relationship to other kinds of evolutionary theorizing. The upshot, if I have succeeded, is an account of evo-devo's place within evolutionary theory that minimizes its tensions and maximizes its harmony with other evolutionary research programs.

I focus on one particular strain of evo-devo: that centered around developmental genetics. Developmental genetics has been a central part of evo-devo since the latter's inception (a crucial early discovery was of the deep conservation and developmental role of hox genes), but it is especially in the 21st century that it has come to be the dominant strand, with the flourishing of gene regulatory network (GRN) theory (Prud'homme, Gompel, and Carroll 2007; Wray 2007; Carroll 2008; Peter and Davidson 2015). It is my sense – which I try to substantiate in the chapters that follow – that many philosophical analyses of evo-devo have failed to adequately grapple with the central role of GRN theory in the field.

1.2 WHY EVO-DEVO? WHY GRN THEORY?

Why be worried about the place of evo-devo within the broader landscape of evolutionary theorizing? The most basic reason is that it seems to continuously come into conflict with older traditions in evolutionary biology. These conflicts span the gamut, from difficulties with interpersonal communication to large-scale theoretical disagreements.

At the small scale, practitioners of evo-devo often have a difficult time talking to population geneticists: their explanatory aims and strategies diverge widely, and accordingly it's difficult for either to see how the other is engaged in a common project (Mark Rebeiz, personal communication). While such difficulties may simply reflect a difference in interests, there is reason to think that the conflict lies deeper. While some practitioners of evo-devo take a conciliatory stance toward evolutionary theorizing that derives from the synthesis (Wray 2007; Carroll 2008), others see the two approaches as fundamentally in conflict (Davidson 2011). At a still larger scale, evo-devo is often lumped in with other new approaches to thinking about evolution as part of a package known as the "extended evolutionary synthesis," which claims to be a more adequate approach to evolution than the gene-centric approach derived from the synthesis (Pigliucci 2007; Pigliucci and Müller 2010; Pigliucci and Finkelman 2014; Laland et al. 2014, 2015; Müller 2017). On the flip side, many biologists have questioned the relevance of developmental biology to evolutionary theory and have challenged evo-devo's core claims (Wallace 1986; Reeve and Sherman 1993; Hoekstra and Coyne 2007).

In consequence of these conflicts, a great deal of philosophical attention has been focused on evo-devo and the possibility of integrating it into evolutionary theorizing more broadly (e.g., Burian 1986; Gilbert, Opitz, and Raff 1996; Amundson 1998; Gilbert 2003b; Lewens 2009b; Brigandt and Love 2010; Walsh 2013; Winther 2015). While many of these approaches are conciliatory, emphasizing the potential for fruitful integration and cross-talk between evo-devo and other approaches to evolution, the most detailed, developed, and thoroughgoing analysis of evo-devo concludes that evo-devo is incommensurable with the population-genetic-centric evolutionary theorizing that descended from the synthesis (Amundson 2005). On Amundson's view, the exclusion of development from the synthesis was not an accident but a conceptual necessity: the structure of synthetic theorizing rendered developmental biology irrelevant to answering questions about evolutionary causes. The relationship between evo-devo and population genetics, Amundson (2005, chap. 11) claims, possesses all the hallmarks of Kuhnian incommensurability: different (and competing) ontologies, different exemplars, different explanatory aims, and different explanatory standards. Thus far, Amundson's arguments stand largely unchallenged.

But, while the philosophical outlook may seem rather grim, the facts on the ground are somewhat more encouraging, and suggest that integration should be possible. First, there is the simple fact that, for over three decades now, both fields have co-existed and flourished. That suggests that neither is going to give way to the other: whatever the end result will be, it probably won't be a classic Kuhnian case of replacement of one paradigm by another. Both are here to stay. Second, there is some evidence that the exclusion of

development from the synthesis was less severe than it is often portrayed (Waisbren 1988; Smocovitis 2008; Davis, Dietrich, and Jacobs 2009).

Third, one of the barriers to integration concerns time scale. Evo-devo tends to focus on deeply conserved body plans (Raff 1996; Davidson and Erwin 2006) and on evolutionary novelties that arose in the distant past (Moczek 2009; Shubin, Tabin, and Carroll 2009) – on macroevolutionary phenomena. Population genetics, by contrast, focuses on microevolutionary phenomena: changes in the genetic composition of interbreeding populations (Cheverud 1984). While the architects of the modern synthesis argued that macroevolutionary phenomena can be explained by extrapolating microevolutionary causes (Dobzhansky 1982; Mayr 1982; Simpson 1982), actually making the connection is difficult, simply because it is hard to get information about microevolutionary causes in the deep past. However, recent work in evo-devo has investigated the evolution of development at smaller time scales, where population genetic resources can be applied (Nunes et al. 2013; Glassford et al. 2015). It remains to be seen how the subfield of micro-evo-devo will contribute to the integration of the two fields, but the prospects are promising.

My aim is to present a picture of the conceptual foundations of evo-devo that makes room for such integration. To do so, I focus on the role of GRN theory in evo-devo. GRN theory is a theory of animal development that focuses on the role of gene regulatory networks in driving developmental differentiation (Peter and Davidson 2015). The basic idea is that genes are not automatically expressed. Each gene contains, in addition to its protein-coding regions, regulatory regions called enhancers. A special class of proteins (transcription factors) bind to specific DNA sequences within these enhancers and either drive

or repress expression. Transcription factor genes are regulated in the same way as other genes, and so the genome encodes deep, hierarchical networks of transcription factors regulating the expression of other transcription factors (finally bottoming out in genes that carry out non-regulatory functions). These networks allow for the precise control of gene expression in space and time, which is essential for coordinating the precisely spatiotemporally localized events that characterize normal development. Evo-devo based on GRN theory studies how these networks evolve (or fail to evolve) to generate evolutionary change and evolutionary stasis (Davidson and Erwin 2006; Carroll 2008; Davidson and Erwin 2010; Peter and Davidson 2015, chap. 7; Glassford et al. 2015).

Why give GRN theory pride of place? Two reasons. First, much of the philosophical literature on evo-devo has focused on its structuralist, typological aspects (Amundson 1998; Brigandt 2007; Lewens 2009a; Love 2009a). This is especially true of Amundson's (2005) treatment. Structuralist tendencies have indeed been present in evo-devo from the beginning and are still present today (Webster and Goodwin 1982; Alberch 1989; Kauffman 1993; Newman 2014b). However, emphasizing them to the exclusion of all other tendencies misses out on the diversity of approaches present in evo-devo. Much of evo-devo, as I argue in my third chapter, adopts a functionalist approach, though one distinct from standard neo-Darwinian functionalism (Davidson and Erwin 2006; Wray 2007; Carroll 2008). Understanding the divisions within evo-devo, rather than treating it as a unified field, can help to understand how it can be integrated with evolutionary theorizing. It can also help us to understand disputes within the field (Carroll 2008; Newman and Bhat 2008). Second, and relatedly, I argue (especially in my second chapter) that GRN

theory's central role in evo-devo actually helps to resolve some of the historical sources of tension between evolutionary and developmental biology. When GRN theory-based work in evo-devo is lumped in with the structuralist approaches, the conflicts come to seem more than they are. When the two are separated, the possibility of integrating both strands of evo-devo into evolutionary theory is easier to see.

1.3 METHODS AND BACKGROUND

My aim in this dissertation is to understand the conceptual foundations of evo-devo and, in doing so, to show how it can be integrated with other aspects of evolutionary theory. Each chapter takes on a particular locus of conflict discussed in the philosophical literature surrounding evo-devo and attempts to show how it can be resolved. Chapter 2 focuses on the exclusion of development from the synthesis (Wallace 1986; Amundson 2005; Love 2009b) and its ramifications for understanding the place of evo-devo today. Chapter 3 considers the claim that evo-devo is perniciously typological (Amundson 1998; Ghiselin 2005a; Jenner 2006; Lewens 2009b; Love 2009a; Ereshefsky 2010; Rieppel 2010). Chapter 4 discusses developmental approaches to the 'homology' concept, which have come under intense criticism from multiple camps (Wagner 1989; Cracraft 2005; Ramsey and Peterson 2012; Currie 2014; Wagner 2014). Finally, chapter 5 considers the relationship between

structuralist and functionalist approaches to evolution, both within and beyond evo-devo (Rieppel 1990; Amundson 2005; Boucher 2015).

Finding these points of tension is easy (they're all widely discussed in the literature); resolving them is more of a challenge. Here, I adopt the phylogenetic approach to history and philosophy of science, as described by James Lennox (2001). The basic premise underlying the phylogenetic approach to history and philosophy of science is that problems that vex contemporary science and philosophy of science can often be understood and resolved by studying their historical origins. As Lennox notes, even well-confirmed theories can be formulated and revised in many different ways, and which of these possible ways are actualized shapes the further development of the theory. By understanding these contingent features of theories within the broader space of possibilities, we can come to see cases where problems are a product of features that the theory might easily do without.

For instance, we will see in chapter two that the exclusion of development from the synthesis was a product, in part, of two key factors: the immature state of developmental genetics in the first half of the 20th century, and the skepticism on the part of many developmental biologists that any genetic theory could explain the main phenomena of development. The theorizing of the synthesis coalesced in this context, and the absence of developmental biology from that theorizing reflects the antagonism between developmental biologists and Mendelian geneticists more than it reflects any principled irrelevance of development to evolution. By understanding the bases of that antagonism, we can see that GRN theory provides us the resources to resolve them. Insofar as the history of

exclusion continues to generate conflict between evo-devo and synthesis-derived approaches to evolution, it doesn't need to do so. Evo-devo has developed in a way that puts those issues to rest.

The phylogenetic approach to history and philosophy of science runs throughout my dissertation. In three of the chapters, it is on the surface. We have already seen its role in chapter two. In chapter three, I address the charge that evo-devo is perniciously typological by breaking down Ernst Mayr's unprincipled notion into two more respectable notions: natural state thinking and Cuvieran functionalism. Only the latter, I argue, plays a role in evo-devo. Here, it's crucial that it's Cuvieran functionalism, as opposed to Darwinian functionalism. By understanding the nuances of functionalist reasoning across the history of biology, we can understand how evo-devo fits into a functionalist tradition, though not the same functionalist tradition that informed the modern synthesis. Placed in this historical context, the problem to be solved changes: not how structuralist, typological evo-devo fits in with synthetic theorizing, but rather how the varieties of functionalist reasoning interrelate. Similarly, in chapter five, I try to understand both the structuralist and functionalist traditions in evo-devo in the context of the long history of structure/function disputes in biology. In trying to capture what is common to structuralist (or functionalist) positions widely separated by both time and theoretical commitment, I aim to show both why such disputes continuously arise and how structuralist and functionalist approaches to evolutionary theory can and must be integrated.

Only chapter four is an outlier. There, I aim to detail the fine structure of the 'homology' concept. My contention is that, when this fine structure is understood, the

interminable debate between genealogical and developmental approaches to understanding homology can be dissolved. This is the most thoroughly “contemporary” of the chapters: history plays little role in the argumentation (though historical research played a key role in shaping my understanding of the issues).

My approach in chapter four owes much to Mark Wilson’s (1982, 1994, 2006, 2015, 2018) work on the complex behaviors of scientific (and other) concepts. Wilson argues that scientific concepts are very often not characterized by an essential “core” that binds together all proper uses of the term. Instead, concepts have a patchwork structure, with local threads tying together various patches. For instance, temperature cannot simply be defined as mean kinetic energy per degrees freedom, since such a definition would be inapplicable to many of the materials whose temperature we aim to measure (rubber bands, for instance). Instead, localized patches of application develop and are coordinated at their points of overlap. Thus, predicates often do not “meet” properties in a simple, one-to-one fashion (Wilson 1982). The same, I argue, is true of ‘homology’. Disputes arise when a single account of homology is asked to do the work of multiple accounts, which must be stitched together in the right way.

The emphasis of my dissertation is fundamentally integrative: we need multiple approaches to evolutionary theory that can coexist and complement one another. Rather than ask for a single, unified evolutionary theory – which in this context means asking whether evo-devo or synthesis-derived theorizing is “right” – we should ask how different regions of evolutionary theory (including both population genetics and evo-devo) interrelate. In this, I see my work as addressing issues raised by Sandra Mitchell’s (2002,

2003; Mitchell and Gronenborn 2017) work on integrative pluralism. As Mitchell argues, new theories or modes of representation often co-exist for surprisingly long periods of time, even though one initially seemed to promise to replace the other (Mitchell and Gronenborn 2017). The key question to ask, then, is how these co-existing resources are integrated to provide scientific explanations (Mitchell 2002). That question lies in the background of much of this dissertation. In chapter three, for instance, I look at the role of Cuvieran functionalism in explaining deep conservation and show how this explanation of evolutionary stasis complements neo-Darwinian explanations of evolutionary change. Similarly, in chapter five, my aim is to develop a framework for understanding not just what's at stake in structure/function disputes, but how structuralist and functionalist approaches can both help to explain the evolution of a single part.

My work also owes much to James Woodward's (2013) careful disentangling of the types of causal claims that scientists make. In chapter two, for instance, it turns out that one of the issues leading developmental biologists to mistrust genetic research was the two fields' divergence over the kinds of causal claims they sought (Amundson 2005, chap. 7). Where the developmental biologists wanted "connecting process" type causal stories that showed how developmental stages arose from prior stages and gave rise to future stages, Mendelian geneticists offered "difference-making" (Waters 2007) causal stories that explained phenotypic differences in terms of genetic differences. With the help of Woodward's work, we can see that these two types of explanation are both legitimate, and that providing one does not preclude providing the other. In the terms of chapter two, providing a transmission genetic account (difference-making) does not preclude

providing a developmental genetic account (connecting process). Woodward's influence is also seen in my attempt to disentangle the precise causal questions at issue in structure/function disputes (chapter five). Here, again, needless disputes may arise if multiple distinct causal questions are condensed into a single question.

In each case, these philosophers' work has pushed me to analyze the conflicts between evo-devo and synthesis-derived evolutionary theorizing at a local level. I began this project thinking to tell a story about how to resolve the large-scale theoretical conflict between the central theory of the modern synthesis and the central theory of evo-devo – the two entities I took to be potentially incommensurable on the basis of Amundson's (2005) work. But this proved to be fruitless: that is not the scale at which actual disputes arise. On both sides, there is no central theory to be found. In the case of the modern synthesis, there is a broadly genetic approach to evolutionary theory shared by its proponents (but also by the figures it marginalized; see Waddington 1957; Goldschmidt 1982), but the details of how this worked out varied widely from theorist to theorist. There are shared themes running through the work characteristic of the synthesis (Lennox 2015), but what resulted was not so much a theory as a cluster of theories in partial harmony and partial tension (for a sample, see the papers collected in Jepsen, Mayr, and Simpson 1963). Moreover, these theories have undergone more or less constant revision over the six decades since the "hardening" of the synthesis (Gould 1983; Ishida 2017), despite recent claims to the contrary (Pigliucci 2016). For this reason, I speak throughout of "synthesis-derived evolutionary theorizing" (or similarly ugly descriptive phrases) rather than of "the synthetic theory", which does not exist.

The same is true of evo-devo: it's a multifarious research program with many parts that, while they generally complement each other, give rise to internal disputes. In trying to compare evo-devo with synthesis-derived evolutionary research programs, then, looking for the paradigm or theory that underlies each program and then trying to understand how these paradigms/theories relate proved to be a misleading and unhelpful strategy. How, then, are we to compare these complex research programs?

What I have done is attempt, in each chapter, to take a conflict that has been attributed to disagreements between the evo-devo position on the one side and the synthesis-derived position on the other side and to identify the relevant, smaller units between which the tension actually arises. Usually, these units are a particular type of explanatory strategy. In chapter two, for instance, I focus on a historical dispute over the relationship between genetics and development and show how this interacts with the different ontologies of two sorts of evolutionary explanation (one common in evo-devo, the other in population genetics). I argue that when we understand how GRN theory resolves the historical dispute, we can see how the two explanatory strategies can be related. In chapter three, the focus is once again on competing explanatory strategies, this time concerning deep conservation and the problem of evolutionary stasis (Wake, Roth, and Wake 1983; Rieppel 2010). In chapter four, I focus on different accounts of homology and the particular purposes for which they are used. In this case, the dispute turns out not to be between evo-devo and synthesis-derived theorizing, but between evo-devo and particular traditions in phylogenetic systematics (Hennig 1966; Wiley and Lieberman 2011). In chapter five, my focus is once again on explanatory strategies, in this case strategies for

explaining the evolution of form. Here my direct target is a view that we should understand these disputes as between philosophical stances, which are even more abstract and tenuous than theories (Boucher 2015). Instead, I argue that the dispute becomes more comprehensible the more we focus in on local explanations of the evolution of particular forms.

One of the virtues of this approach is that local integration within particular explanations often cuts across large-scale theoretical differences. Beckett Sterner and Scott Lidgard (2018) have recently furnished a very nice example of this in their study of the systematics wars that rocked systematic biology in the latter half of the 20th century. In that case, they show that, even as cladists and numerical taxonomists fought bitterly over the theoretical foundations of the discipline, systematists on both sides of the dispute fruitfully shared key aspects of their daily workflow. Even as theoretical conflict raged above, practical integration occurred below. A similar moral – or so I hope to show – applies to evo-devo in relation to synthesis-derived evolutionary theorizing.

1.4 DISSERTATION STRUCTURE: OVERVIEW

In presenting the aims and methods of my dissertation, I've said a fair amount about the content of particular chapters, but only in scattered form, picking details relevant to the point at hand. I'd like to end these introductory remarks with an overview of the

argumentative structure of each chapter of the dissertation. In each chapter, I get into the weeds (historical and contemporary); here I hope to provide the skeletal frame of each chapter, into which the details can be fit. First, however, a few more general remarks on how the whole thing hangs together.

As noted above, the most thorough treatment of the conceptual foundations of evo-devo and of its conflict with synthesis-derived evolutionary theorizing is Ron Amundson's *The Changing Role of the Embryo in Evolutionary Thought* (Amundson 2005). In that work, Amundson tells two stories about the sources of the conflict, in terms of two exclusions of developmental biology from the modern synthesis. The first exclusion (Amundson 2005, chaps. 7-9) occurred during the early years of the synthesis, and largely stemmed from the split between heredity and development that occurred with the rise of Mendelian genetics (this narrative of how the split arose has been challenged, see Meunier 2016). The second exclusion occurred later, in the aftermath of the Darwin centennial in 1959. Around this time, Ernst Mayr, one of the architects of the synthesis, re-wrote the philosophy of the synthesis in terms of a set of distinctions, most notably between population thinking and typological thinking and between proximate and ultimate causation (Mayr 1959, 1976). Though not initially designed to exclude developmental biology from evolutionary relevance, they came to be used in that way in the early days of evo-devo (Scholl and Pigliucci 2014).

The second and third chapters of my dissertation take up these two exclusions in turn. The second chapter revisits the split between heredity and development, arguing that the split existed from two perspectives (developmentalist and synthetic). Only the

developmentalist perspective generates an incommensurability between population genetic and evo-devo approaches to evolution. Happily, however, evo-devo vindicates the synthetic perspective. And so the first exclusion proves not to be a barrier to integrating evo-devo with synthesis-derived evolutionary theorizing.

The third chapter addresses the second split, focusing on the proper way to understand (a) the common claim that evo-devo is typological and (b) Amundson's claim that synthesis-derived theorizing rests on what he calls "exclusive population thinking". I argue that evo-devo's use of Cuvieran functionalist reasoning accounts for many of its allegedly "typological" characteristics and that Cuvieran functionalism is compatible with a defensible interpretation of what exclusive population thinking actually excludes.

In chapter five, I expand outward to consider the nature of structure/function disputes more generally. It is common to see evo-devo as a structuralist alternative to the functionalist synthesis (Resnik 1994; Amundson 2005; Wagner 2014; Boucher 2015). The conclusion reached in chapter three – that evo-devo relies on Cuvieran functionalism – should already suggest that this is too simplistic an approach. My aim in this chapter is thus to make sense of functionalism and structuralism as explanatory strategies in order to understand both (a) why both approaches to evolutionary theory are required and (b) why structure/function disputes nonetheless persistently arise (and can be expected to continue to do so), including within evo-devo.

I skipped over chapter four (on the 'homology' concept) in that brief overview because chapter four fits somewhat awkwardly with the other chapters. On the one hand, it shares a few key themes with the other chapters. Most notably, my aim is to understand the role

of developmental genetic approaches to understanding homology in relation to other approaches to understanding homology. In that regard, it fits with the dissertation's emphasis on how GRN theory helps us to understand the status of evo-devo. Moreover, my aim in this chapter, like in the others, is integrative: I aim to show how developmental genetic approaches to homology can be integrated with genealogical approaches.

But there is a glaring difference: the conflict I discuss is not between evo-devo and synthesis-derived evolutionary theorizing. Rather, it is between evo-devo and phylogenetic systematics, itself a field with a tumultuous relationship to the synthesis (the work of Hennig 1966 directly conflicted with the evolutionary taxonomy approach favored by Ernst Mayr; see Sterner and Lidgard 2018). When I first envisioned the chapter, I saw it as involving a conflict between approaches that treat homologs as the causally inefficacious residue of shared history and approaches that treat homologs as causal agents that shape evolutionary history (Amundson 2005, 238–44). But it turns out that the arguments I offer in chapter three resolve that issue, which is in any event not the main source of conflict between the different approaches to understanding homology. In following out those sources of conflict, this chapter was dragged somewhat away from the main line of the dissertation. It is more substantive for the departure.

I turn now to brief overviews of each of the four chapters.

1.5 CHAPTER TWO

The aim of this chapter is twofold: (1) to reconsider the nature of the historical exclusion of developmental biology from the modern synthesis, showing that this exclusion looked quite different from the different perspectives available at the time, and (2) to place evo-devo in relation to this historical conflict in order to show how evo-devo gives us the resources (GRN theory) to resolve it.

Regarding the first point, my central contention is that the split between heredity and development existed from two perspectives: that of developmental biologists and that of the proponents of the modern synthesis. From the perspective of developmental biologists, Mendelian genetics had severed the problems of heredity (transmission of traits between generations) from the problems of development (how traits are actually produced in an individual organism (Morgan 1923, 1926). Mendelian genetics concerned itself solely with transmission/heredity, ignoring development.

Moreover, developmental biologists argued that the Mendelian approach could not be used to explain the central phenomenon of development: the precisely spatiotemporally localized differentiation of the embryo (Lillie 1927; Russell 1930; Just 1936). One of the main reasons for pessimism was the fact that each cell in the embryo was known to possess the same genetic material (the Mendelians themselves showed this). How could a cause that was the same everywhere explain local differentiation? This problem, known as the developmental paradox (or Lillie's paradox), suggested that the genetic material

did not have the requisite properties to explain development, and thus that a genetic theory of development was an impossibility.

In consequence, many developmental biologists looked to the cytoplasm for the factors responsible for key developmental processes (Sapp 1987). Thus, from the developmentalist perspective, the split was ontological: the factors that explain heredity (the genes) lie in the nucleus, while the factors that explain development lie in the cytoplasm.

By contrast, from the synthetic perspective the split was merely methodological. This can be clearly seen in the case of Theodosius Dobzhansky (1982). Dobzhansky distinguished three types of genetic study: transmission genetics, developmental genetics, and population genetics. Transmission genetics studied the passing of traits between generations, correlating genetic differences (localizable to chromosomes) with phenotypic differences. However, transmission genetics said nothing about the developmental processes that produced these effects. That was the task of developmental genetics. Developmental genetics would study the same entities as transmission genetics, but, by using different methods, would reveal different aspects of their function. In this way, the split was only methodological, not ontological.

What does this tell us about the relationships between evo-devo and synthesis-derived theorizing? Amundson (2005, chap. 11) characterizes the incommensurability between them in terms of their divergent evolutionary ontologies. Where evo-devo sees evolution as a process of modifications of ontogeny, and thus sees ontogenies as the units of evolutionary change, synthesis-derived evolutionary theorizing sees evolution as a process of modification of the genetic composition of populations. Now, if the

developmentalists were right, this would generate a serious incommensurability. Without a genetic theory of development, such population processes could not be connected to the evolution of ontogeny.

Evo-devo, and in particular GRN theory-based evo-devo, however, vindicates the synthetic perspective as against the developmentalist perspective. GRN theory is precisely a developmental genetic theory of development—one that, moreover, explains how a globally constant genetic material can have locally specific effects (and so resolves the developmental paradox). It thus provides a bridge that allows for the integration of studies of the evolution of development with studies of the evolution of populations. That is not to say that such studies always arrive at compatible conclusions (they don't; see Hoekstra and Coyne 2007; Peter and Davidson 2011), but it does mean that those differences don't rest on incompatible ontologies.

1.6 CHAPTER THREE

This chapter addresses the concern that evo-devo is perniciously typological. This project runs into an immediate difficulty: the notion of typological thinking was developed by Ernst Mayr (1959), but Mayr's understanding of it was notoriously unprincipled (Witteveen 2015, 2016). As a result, when one surveys the range of discussions of evo-devo that attribute to it typological thinking, one finds a striking diversity of ways of

understanding the notion (Amundson 1998; Jenner 2006; Brigandt 2007; Lewens 2009a, 2009b; Love 2009a; Riegner 2013).

Accordingly, the first aim of this chapter is to understand in a principled way what it might mean for evo-devo to be typological. I argue that one of the functions of Mayr's notion of typological thinking was to understand what was common to all critics of gradualistic evolutionary theories. Typology, for Mayr, was closely connected to anti-gradualism: a typologist could only be either a saltationist or against evolutionary theory altogether. Moreover, one of the reasons that evo-devo is often considered typological is its central concern with the deep conservation of animal body plans. But the problem of explaining deep conservation is precisely that of explaining why something has failed to evolve. It's thus a promising place to look for "typological" thinking in evo-devo.

Mayr's project, however, was a failure: there is no one strand of thought common to all anti-gradualists. In the chapter, I distinguish two distinct threads: natural state thinking (given a precise account by Sober 1980) and Cuvieran functionalism. Natural state thinking involves modeling systems as having a natural state from which they can be deflected by an interfering force, but to which they will tend to revert when the force is removed. Many early critics of Darwin opposed his theory on such grounds (Jenkin 1867; Mivart 2009).

Georges Cuvier, by contrast, opposed gradualistic evolutionary theories on other grounds (Coleman 1964; Burkhardt 1977; Russell 1982). His argument had three steps: (1) show that organisms are highly functionally integrated, (2) argue on that basis that intermediates between existing forms would lack such integration and therefore be non-viable,

and (3) conclude that gradual evolution was therefore impossible. Though functionalist (it emphasizes the functional integration of organisms), this argument is not functionalist in the same sense that Darwin's reasoning was functionalist (Russell 1982). Specifically, where Darwin focused on organism-environment relations, Cuvier emphasized relations between parts internal to the organism. Cuvieran functionalism marks a second path to anti-gradualism, distinct from natural state thinking.

The second aim of this chapter is to show that Cuvieran functionalism plays an important role in evo-devo's explanation of the deep conservation of animal body plans. The task of explaining deep conservation is challenging: it requires explaining why parts that have (a) varied and (b) been exposed to numerous distinct environments have nonetheless failed to undergo evolutionary change. Evo-devo does this by applying a localized form of Cuvieran functionalism. According to this localized Cuvieran functionalism, there are certain regions of the genome, called GRN "kernels", that (a) underlie the development of deeply conserved elements of body plans, (b) are themselves deeply conserved (their topology is identical or nearly so in widely diverged taxa), and (c) are characterized by extensive positive feedback (Davidson and Erwin 2006; Peter and Davidson 2015, chap. 7). They are integrated in the sense that any disruption of the linkages in the kernel destroys overall kernel function, leading to the production of organisms that lack, e.g., a heart. This combination of functional integration and essential developmental role explains why they (and their associated body plan elements) have failed to evolve for hundreds of millions of years.

The third and final aim of this chapter is to show that Cuvieran functionalism is compatible with exclusive population thinking. The basic idea behind exclusive population thinking is that evolutionary causes are fundamentally causes that occur within interbreeding populations (there are exceptions to this, e.g., Erwin 2000, 2011; Doolittle 2017, but they do not help to vindicate Cuvieran functionalism). Once populations diverge (once gene flow ceases), they pursue independent evolutionary trajectories. Exclusive population thinking, on this basis, forbids invoking evolutionary causes that transcend populations. I argue that exclusive population thinking has a strong and a weak interpretation, based on how one understands “transcends”. On the strong interpretation, exclusive population thinking forbids any explanation that unifies what is going on in independently evolving lineages. Each population requires its own, unique explanation. But this, I argue, rules out explanations well beyond evo-devo, such as explanations of ecological trends (which by definition transcend the single species). Such explanations unify by showing how independently evolving populations are responding similarly to similar causes. A weaker, more defensible understanding of exclusive population thinking should permit such explanations, which respect that populations without gene flow are following independent (but similar) evolutionary trajectories.

This weaker understanding of exclusive population thinking allows for Cuvieran functionalist explanations. Precisely because GRN kernels are preserved, and because disruption of them leads to developmental catastrophes that are fatal in all naturally occurring environments, they will give rise to similar selection pressures in all members of a lineage. Shared ontogeny can thus explain the similar trajectories of these different

lineages, but only because the same causes is repeated within each individual lineage. Exclusive population thinking does not forbid Cuvieran functionalist explanations. Recasting the role of typological thinking in evo-devo as the role of Cuvieran functionalism in evo-devo thus allows us to see how evo-devo can complement, rather than conflict with, synthesis-derived evolutionary theorizing.

1.7 CHAPTER FOUR

The aim of this chapter is to present a detailed account of the fine structure of the ‘homology’ concept. By “fine structure” I mean a picture of how different accounts of homology relate to each other, showing their regions of overlap and non-overlap. On the view I defend, understanding homology requires two types of accounts: (1) an abstract, formal genealogical account that unifies the application of the term to all kinds of characters, and (2) a number of locally enriched accounts that specify how the genealogical account applies to particular kinds of characters. I argue that enriched accounts overlap with, but are not nested within, the genealogical account.

Why think that ‘homology’ has a fine structure? Richard Owen’s classic definition of ‘homologue’ sets out the key problem that accounts of homology should solve. Owen (1843, 379) defined homologs as “the same organ in different animals under every variety of form and function.” That is, homologous parts (such as the bat’s wing and the

dugong's front fin) are in some sense the same, despite being potentially quite dissimilar. An account of homology should explain this sameness.

There are two major traditions for understanding homological sameness: genealogical and developmental. Genealogical accounts, based on Darwinian evolutionary theory, say that homologous parts are the same in virtue of descending from the same part in a common ancestor (Darwin 1964; Hennig 1966; Cracraft 2005; Ramsey and Peterson 2012; Currie 2014). Developmental accounts, by contrast, understand homological sameness as the result of shared developmental processes or constraints (Roth 1984; Wagner 1989; Roth 1991; Owen 2007; Wagner 2014). This basic disagreement over the nature of homological sameness leads to further points of tension between, discussed in detail in the chapter.

As a result of these disagreements, many biologists and philosophers treat the two accounts as competing accounts of the same phenomenon (Amundson 2005, 238–44; Cracraft 2005; Ramsey and Peterson 2012; Currie 2014). A number of philosophers, however, have argued that the accounts are compatible, and simply serve different functions (Jamniczky 2005; Brigandt 2007; Griffiths 2007; Laublichler 2014).

I agree with the compatibilists, broadly speaking, but it's a tricky position to defend. Given the disagreements and apparent contradictions between the two accounts, how can they be both (a) accounts of a single phenomenon and (b) nonetheless compatible? It would be easy to render the accounts compatible by accepting that the two types of account are simply talking about different things — then we have a case of mere polysemy. Compatibilists, however, have correctly recognized that there is a deep relationship

between the phenomena described by the two types of account, and thus this easy solution cannot work. My attempt to delineate the fine structure of 'homology' is intended as a compatibilist position that satisfies both desiderata.

The basic view is this. Genealogical accounts of homology play a unifying role: they show how all different kinds of biological entity can be homologous. They are able to do so in virtue of their abstract, formal character. They are formal in that they define homology in terms of the topology of phylogenetic trees: two parts are homologous if they can be traced back, via a continuous path, to an ancestor that also had that part. They are abstract in that they ignore the details of how it is that parts can be inherited. Thus they can apply to different kinds of characters, despite differences in the mechanisms that allow them to be mapped onto phylogenetic trees.

Although the abstract, formal character of genealogical accounts is a boon in allowing them to apply widely, it leaves them importantly incomplete. Specifically, they presuppose the very sameness relation they were supposed to explain. Parts in different species are homologous if they can be traced back to the same part in a common ancestor. Now the problem is to understand what makes parts the same in ancestors and descendants. In the limiting case, this becomes the problem of understanding what makes parts the same in parents and offspring.

To address this issue and complete the genealogical account, we need enriched accounts that explain parent-offspring sameness. This requires looking at how parts are inherited. But parts are inherited in different ways (in the chapter, I give details for body parts and genes). Thus, multiple enriched accounts are needed, each of which completes

the genealogical account in a particular domain. I further show that, once we have these enriched accounts, we can see that they cover phenomena that fall outside the genealogical account (such as serial homology and paralogy—the repetition of body parts and genes, respectively, within a single organism). Enriched accounts thus overlap with, but are not nested within, the genealogical account.

This view, I argue, resolves the tensions between the two kinds of accounts with which we began. Developmental accounts, on my view, should be understood as enriched accounts of body part homology. In their regions of overlap, genealogical and developmental accounts provide compatible descriptions at different levels of abstraction. The developmental account elucidates the mechanisms that make it possible to apply the genealogical account to body parts. In their regions of non-overlap, they are simply talking about different things and so do not conflict with one another.

1.8 CHAPTER FIVE

The aim of this chapter is to provide an account the nature of structure/function disputes that satisfies three desiderata. First, it should help make sense of why structure/function disputes have been a persistent feature of biological science across multiple rounds of radical theory change. Second, it should show how structuralist and functionalist

explanatory strategies can be fruitfully integrated. Third, it should capture the diversity of approaches to evo-devo, which includes both structuralist and functionalist traditions.

A natural strategy for understanding the nature of structuralism and functionalism, given their persistence across hundreds (arguably thousands) of years of biological inquiry and radical theory change, is to treat them as abstract enough to avoid commitment to any particular belief. Often, this involves characterizing them in terms of views about explanatory priority: structuralists say structure is explanatorily prior to function, and vice-versa for functionalists (Russell 1982; Asma 1996; Amundson 2005). The details of how structure explains function (or vice-versa) may then change dramatically over time, while structuralism and functionalism are still recognizable views. Sandy Boucher (2015) has taken this approach to its extreme, arguing that structuralism and functionalism are philosophical stances *sensu* van Fraassen (2002).

In this chapter, I advocate a different approach. I argue that more concrete content can be given to structuralism and functionalism if, instead of focusing on them as supra-theoretical positions, background commitments, or stances, we understand them as explanatory strategies. That is, rather than talk about structuralism and functionalism, we should talk about structuralist and functionalist explanations. Relatedly, rather than focus on conflicts between structuralism and functionalism as abstract commitments, we should focus on local structure/function disputes, where structuralist and functionalist explanations provide competing answers to different contrastive why-questions.

Because my interest is in understanding evo-devo, my focus in this chapter is on understanding structure/function disputes in the context of evolutionary theory (though I

do sketch some arguments to show how my analysis could be extended to pre-Darwinian structure/function disputes – first desideratum). The starting point for my analysis is the two-part structure of evolutionary theory: evolutionary change requires both (i) the generation of novel variants by mutation or other processes and (ii) the spread of variants through a population by selection or drift. Analyzing several examples of classic structure/function disputes in an evolutionary context, I show that the structuralists in each case explain structure by invoking limits to the variants that are actually generated. By contrast, the functionalists in each case explain structure by assuming that variation is relatively unlimited (in pragmatically limited respects), and that the processes of spread therefore play a key role in explaining structure.

On this basis, I develop a view according to which structuralists and functionalists can be distinguished by the kinds of answer they give to particular contrastive why-questions, of the form, “why did form X evolve rather than form Y.” Structuralist explanations explain this in terms of limits to the generation of variation (variants in the direction of Y were not generated), while functionalist explanations explain this in terms of selection favoring the spread of one variant over another (variants were produced in both X and Y directions, with selection favoring those in the direction of X).

This view shows how structuralist and functionalist explanations can be integrated (second desideratum). In any case of evolutionary change, processes of generation and spread must both be involved. A complete explanation of any particular bit of organic form (were such a thing possible) would therefore require both structuralist and functionalist aspects. Contrastive why-questions, however, can take pure structuralist or pure

functionalist answers (among other possibilities, discussed in the chapter). Structure/function disputes arise when competing structuralist and functionalist answers are given to particular contrastive why-questions.

Finally, this view accounts for the internal complexity of evo-devo (third desideratum). In contrast to accounts that treat evo-devo monolithically as a structuralist challenge to functionalist neo-Darwinism (most notably Amundson 2005), my view does justice to the substantial functionalist strain in evo-devo (discussed in chapter three). In doing so, it helps make sense of evo-devo's internal complexity. Structure/function disputes arise entirely within evo-devo (e.g., Carroll 2008; Newman and Bhat 2008), precisely because evo-devo has always mixed structuralist and functionalist approaches.

2.0 THE PRODIGAL GENETICS RETURNS

The progress of genetics and of physiology of development can only result in a sharper definition of the two fields, and any expectation of their reunion (in a Weismannian sense) is in my opinion doomed to disappointment. (Lillie 1927, 367)

Although a large amount of work still remains to be done in this field, it is indeed fair to say that the genetics of the transmission of hereditary characters is, by and large, understood now. But the problem of heredity is much wider. (Dobzhansky 1982, 10)

2.1 DEVELOPMENTAL GENETICS AND EVOLUTIONARY THEORY IN TENSION

The modern synthesis of the 1930s and 1940s provided the foundation of contemporary evolutionary theorizing. It ranks as one of the major accomplishments of 20th century biology. Yet, in a recent article, Eric Davidson (2011, 35–36) had this to say about a particular “Neo-Darwinian” (i.e. synthesis-derived; cf. Hoekstra and Coyne 2007) approach to evolution:

Mechanistic developmental biology has shown that its fundamental concepts are largely irrelevant to the process by which the body plan is formed in ontogeny. In addition it gives rise to lethal errors in respect to evolutionary process. Neo-Darwinian evolution is uniformitarian in that it assumes that all process works the same way, so that evolution of enzymes or flower colors can be used as current proxies for study of evolution of the body plan. It erroneously assumes that change in protein coding sequence is the basic cause of change in developmental program; and it erroneously assumes that evolutionary change in body plan morphology occurs by a continuous process. All of these assumptions are basically counterfactual.

Davidson is a partisan of gene regulatory network (GRN) theory.¹ Animal development begins with a relatively undifferentiated embryo and ends with a fully differentiated adult organism. GRN theory states that differentiation results from the precise spatiotemporal localization of gene expression, controlled by regulatory architecture encoded in the genome. For Davidson, the study of this regulatory architecture has revealed the falsity of many of the assumptions of the modern synthesis.² For instance, he sees in the synthesis a uniformitarian commitment to the belief that body plan evolution is a continuous process. According to GRN theory, however, the process is discontinuous in fundamental ways.

How are we to understand the tensions between GRN theory and synthesis-derived evolutionary theorizing? On one view, their conflicts are merely empirical, to be decided by the weight of the evidence. But from another vantage, the tension lies at a deeper,

¹ For a comprehensive review of GRN theory, see (Peter and Davidson 2015). I provide a brief summary of some of the key points below (Section 2.7). It bears mention that GRN theory primarily concerns animal development. How well its insights apply to plant development remains an open question. GRN theory was first articulated by Britten and Davidson (Britten and Davidson 1969).

² Davidson is not the only biologist to have been struck by a seeming tension between developmental genetics and the modern synthesis. Others include John Gerhart and Marc Kirschner (2007), Gerd Müller (2010), and Stuart Newman (2014a).

conceptual level. Ron Amundson (2005, chap. 11) has argued that evolutionary-developmental biology (including GRN theory) is “incommensurable” with synthesis-derived evolutionary theorizing. On this view, the conceptual framework of evolutionary theory, as descended from the modern synthesis, renders developmental biology irrelevant to evolutionary theorizing. If so, then GRN theory, which draws evolutionary implications from the findings of developmental biology, conflicts with the synthetic theory at the level of this basic conceptual framework.

I aim to show that there is no incommensurability between synthesis-derived evolutionary theorizing and GRN theory, and thereby to illuminate the shared ground that makes their empirical conflicts possible. Amundson traces the conceptual exclusion of development from the synthesis to the split between heredity and development that emerged from the Morgan school. While Amundson is right to connect the two, their relations are more complicated than he recognizes.

The argument to follow can be summarized:

1. The split between heredity and development looked very different to developmental biologists than to proponents of the modern synthesis (Section 2.2, Section 2.4).
2. From the developmentalist perspective, the split was ontological: genes play a crucial role in determining hereditary phenomena, but not in determining developmental phenomena. Developmentalists offered two primary arguments for this view (Section 2.2).

3. From the synthetic perspective, the split was methodological: transmission genetics studies the role of genes in heredity, developmental genetics the role of genes in development. The two disciplines accordingly have distinct methodologies (Section 2.4).
4. Amundson's incommensurability arises only if one adopts the developmentalist's perspective on the split between heredity and development (Section 2.3).
5. Of the two arguments for the existence of an ontological split between heredity and development, one is based on a misunderstanding of the Mendelian genetic research program and its relationship to developmental genetics (Section 2.5). This misunderstanding persists in Amundson's account (Section 2.6).
6. GRN theory provides the resources to address the other argument favoring the developmentalist perspective. GRN theory thus vindicates the synthesis perspective and heals the split between heredity and development (Section 2.7).

In consequence, there is no incommensurability between evo-devo (at least those parts based on GRN theory) and synthesis-derived evolutionary theorizing. The tensions between them are not so deep and intractable as has been thought (Section 2.7).

2.2 LILLIE'S DOUBTS

In "The Gene and the Ontogenetic Process," the embryologist Frank Lillie asserted the existence of a split between development and heredity and pessimistically assessed the prospects for a reunion. Lillie (1927, 361) noted that, in 19th century theories like those of Charles Darwin and August Weismann, "the theory of development included the theory of heredity." Since that time, however, "physiology of development and genetics have pursued separate and independent courses." Lillie argued that the split was the result of Mendelian geneticists isolating the problems of development from those of heredity, addressing only the latter, and that Mendelian genetics lacked the resources to explain development. The split could therefore be expected to be permanent.

Lillie's paper is worth investigating in detail, for several reasons. The arguments he made concerning the limited scope of genetic explanations were accepted by a number of developmental biologists.³ These arguments characterize the developmentalist position that Amundson claims is incommensurable with the synthetic position. In order to understand the nature of this purported exclusion, we thus need to understand the nature

³ Lillie was not alone in his views. For instance, E. S. Russell levied similar criticisms of Mendelian genetics (Russell 1930, ch. 5). Unlike Lillie, however, Russell insisted that heredity was a subsidiary problem to the larger problem of development. Thus the Mendelians could not, in his view, even explain heredity. This difference between Lillie and Russell is, in my view, at least partially semantic. To utilize a distinction defended below (Section 2.4 and Section 2.5), Russell and Lillie both allowed for genetic explanations of transmission but not of development. They differed in that Russell rejected, while Lillie accepted, the equation of heredity with transmission. This is not meant, however, to deny the possibility that there are further disagreements between them that explain this semantic disagreement. See also Just (1936).

of the split between heredity and development. Furthermore, Theodosius Dobzhansky (1982, chap. 1) explicitly denied Lillie's claims. Dobzhansky's rejection of Lillie's claims is closely tied to the role that he expected developmental biology could play in evolutionary theory. Finally, Lillie's arguments are worth considering because they establish clear criteria as to what would count as a reunion of heredity and development. Lillie believed that no genetic theory could meet these criteria, but, as I will argue below (Section 2.7), GRN theory does meet them.

In Lillie's view, the disciplinary split between heredity and development rested upon a physical split: the factors relevant for understanding development were located in the cytoplasm of the fertilized egg, while the factors relevant for understanding heredity were located in the nucleus.⁴ This immediately suggests two strategies for healing the split: get heredity into the cytoplasm or get development into the nucleus. Lillie's gloomy assessment of the prospects of reunion was due to his belief that neither strategy could succeed. The first had been pursued by proponents of cytoplasmic inheritance and opposed by the Mendelians (Morgan et al. 1915, 235–39; Dobzhansky 1982, 68–72). The reasons for the failure of this program do not concern us here (see Sapp 1987). Lillie's arguments in his 1927 paper, if sound, showed that nuclear (i.e. genetic) factors could not explain development.

Lillie proposed two criteria that any unified theory of heredity and development must satisfy:

⁴ "The radical distinction between Mendelian segregation and embryonic segregation is, I believe, that the former is an affair of the nucleus, the latter of the cytoplasm" (Lillie 1929, 515).

First criterion: The theory must be able to explain the differentiation of the embryo over the course of development.

Second criterion: The theory must be able to recognize the distinction between “open” (pluripotent) and “closed” (fated) terms in development.

Lillie argued that a genetic theory of heredity and development could satisfy neither criterion. Therefore “any expectation of their reunion (in a Weismannian sense) is in my opinion doomed to disappointment” (Lillie 1927, 367). I take up his reasons for pessimism about each criterion in turn.

Lillie’s first criterion is based on the fact that development is a process of differentiation.⁵ During development, a relatively undifferentiated germ (the fertilized egg) produces a fully differentiated organism (Lillie 1927, 362). The “essential problem” that a theory of development must solve is to determine the cause of this increase in differentiation over time (Lillie 1927, 367).

This requirement can be clarified further. Lillie (1927, 362–63) distinguished two sorts of differentiation. The first, “embryonic segregation”, involves the “progressive genetic restriction” of developmental potencies (i.e. the possible end states into which any particular part of the embryo can develop) in a definite, precisely controlled sequence in

⁵ Lillie’s focus was on animal development. GRN theory is likewise primarily a theory of animal development, though GRNs play a role in plant development as well. Developmental phenomena also occur in the microbial world (O’Malley 2014, chap. 4), but they fall outside the scope of this debate.

space and time.⁶ Once segregation is complete, a second form of differentiation follows, which involves “the realization of the potencies isolated in the final terms of the segregation process, thus involving histogenesis and definitive functional development.” I will call this second process “realization.”

Lillie’s first criterion requires that a unified theory of development must explain both embryonic segregation and realization. Lillie (1927, 366) allowed for a genetic explanation of realization:

Geneticists have, however, brilliantly demonstrated that genes are concerned in phenotypical realization at different stages of the life history, and it is therefore a reasonable postulate that this is true of all phenotypic realization.

Regarding embryonic segregation, however, Lillie argued that a genetic explanation was impossible. The source of this impossibility was the fact, accepted by the geneticists themselves, that each cell possessed the same complement of genes. The problem is that, if each cell of the embryo is genetically identical, there cannot be a genetic explanation of embryonic segregation. Such an explanation would require explaining regional differences in the embryo in terms of something constant across all regions. But that is impossible. Thus, as Lillie (1927, 366) put it, “so long as the theory of the integrity of the entire gene system in all cells is maintained [...] the phenomena of embryonic segregation must,

⁶ E. E. Just (1936) criticized Lillie’s notion of “embryonic segregation” because he thought “segregation” implied a particular (incorrect) mechanism by which the restriction of developmental potencies occurred. However, as I read him, Lillie clearly intended ‘embryonic segregation’ to refer to an experimentally determined developmental phenomenon without presupposing any particular mechanism by which the phenomenon was produced (see especially Lillie 1929, sec. VIII).

I think, lie beyond the range of genetics.” This is known as the “developmental paradox” (Amundson 2005, 177–80).

Turning to the second criterion, Lillie pointed to a fundamental limitation of genetic analysis that, in his view, scuttled any attempt to develop a genetic theory of development. Lillie’s distinction between embryonic segregation and realization implicated a further distinction between open and closed terms in development (Lillie 1927, 363).⁷ Open terms are pluripotent, i.e. they have multiple potential fates that remain open to them, depending on the conditions under which they develop, while closed terms are limited to a single fate, regardless of developmental context (Lillie 1927, 364). The process of embryonic segregation involves taking wholly open (totipotent) terms and progressively restricting their potencies until they become closed terms, at which point all that remains is realization.

Any adequate theory of development must preserve this distinction. But, Lillie (1927, 367) argued, genetic analysis inherently treated all developmental stages as if they were closed terms:

At whatever stage of development a character may be selected for examination, and whatever the nature of the character, it must always, so far as genetic method is concerned, be treated as a finality. It has no past, except the genes postulated as a result of their appearance in previous generations—and no future. The genetic method reveals, *alpha*, the gene, and *omega*, the final term.

⁷ A “term,” for Lillie, is a stage of the ontogenetic process. Because different regions of the embryo develop at different rates, terms are local and not global: “final terms are scattered all along the life history from a very early stage indeed” (Lillie 1927, 364).

Even if one can show that genetic changes impact developmental processes, genetic methods (i.e. crossing experiments) only establish correlations between genetic changes and changes in particular aspects of development considered outside the context of the entire developmental stream. Genetic analysis thus could not distinguish between open and closed terms. For the purposes of genetic analysis, all terms were closed. Crossing experiments treated the gene as “alpha” and the trait as “omega,” skipping over the entire intervening process. The consequence, for Lillie, was that any genetic theory of development must *in principle* fail the second criterion of adequacy.

In sum, Lillie viewed the disciplinary split between heredity and development as the product of an *ontological* split between the nuclear, genetic processes responsible for realization and the cytoplasmic, non-genetic processes responsible for embryonic segregation. Because of this ontological split, no unified genetic theory of heredity and development was possible. Lillie proposed two criteria any such theory must meet – it must explain embryonic segregation, and it must recognize the distinction between open and closed terms – but he raised powerful arguments against the possibility of a genetic theory meeting these criteria.

2.3 AMUNDSON ON THE CONCEPTUAL EXCLUSION OF DEVELOPMENT FROM THE SYNTHESIS

Lillie's diagnosis of a split between heredity and development underlies Amundson's claim that the modern synthetic theory of evolution treated development as irrelevant to evolution. Here we must be careful, for Amundson actually provides an account of not one but two exclusions of development from the synthesis. The first exclusion was based on the Mendelian separation of heredity and development. This exclusion characterized the synthesis from its inception in the late 1930s to the "hardening" (Gould 1983) of the synthesis during the 1940s and 1950s (Amundson 2005, chaps. 7–9). The second exclusion rested on conceptual distinctions (typology versus population thinking; proximate versus ultimate causation) developed by Ernst Mayr and others. These distinctions crystallized around the *Origin* centennial of 1959 and played a major role in debates (beginning in the 1970s) about the relevance of development to evolution (Amundson 2005, chaps. 10–11).

Only the first exclusion is relevant here, for it is this exclusion that purportedly rests on the divergent conceptual framework of the synthetic theorists and their developmentalist opponents. The second exclusion is the product of conceptual distinctions superimposed on the synthesis after the fact. They are, moreover, additions of dubious cogency (Scholl and Pigliucci 2014; Witteveen 2015, 2016). While historically important for understanding post-1959 disputes in biology, they are irrelevant to my concerns here.

Taking his cue from Lillie, Amundson argues that Mendelian genetics gave rise to a split between heredity and development. The Mendelians explained parent-offspring resemblances in a manner that entirely skipped over ontogenetic processes. Amundson (2005, 139) argues that two contemporary “truisms” about heredity are in fact stipulations that grew out of Mendelian genetics:

Truism 1. “Heredity is the passing of traits (or representatives of traits, such as genes) between generations.”

Truism 2. “Heredity is independent of development.”

It is easy enough to see how both truisms relate to classical Mendelian genetics, understood as the study of heredity. Mendelian genetics uses crossing experiments to trace parent-offspring resemblances (the passing of traits) back to genes transmitted from the parents to the offspring. Thus we get the first truism: Mendelian genetics explains the passing of traits between generations in terms of the passing of genes between generations. Moreover, Mendelian genetics furnishes this explanation by entirely bypassing development. It relies solely on correlations between genes and traits, saying nothing about how genes produce those traits. From this we get the second truism: Mendelian genetics can explain heredity without recourse to development.

From here, it is a short step to the exclusion of development from the synthesis. The synthetic theory is fundamentally a genetic theory of evolution. As Dobzhansky put it, “evolution is a change in the genetic composition of populations” (Dobzhansky 1982, 11–12). Thus “the mechanisms of evolution constitute problems of population genetics.” Population genetics is based on classical Mendelian genetics. Mathematical population

genetic models of the early 1930s, which helped lay the groundwork for the modern synthetic theory, showed how natural selection could act on Mendelian genes to accumulate phenotypic change. As Mendelian genetics ignored development, so did population genetics, which equally required only gene-trait and parent-offspring correlations, skipping over the intervening ontogenetic processes. In short, the synthesis furnished a genetic theory of evolution, development is irrelevant to genetics, and so development is irrelevant to the synthetic theory. Importantly, this exclusion was conceptual. It is not merely that the synthetic theory failed to include input from developmental biology, but that it in principle could not include such input.

On this picture, the architects of the synthesis disagreed with their critics (e.g. Lillie) not only about empirical questions, but also about the very criteria of adequacy for evolutionary explanations. Amundson argues that developmental biologists accepted, while the synthesis rejected, what he calls the “causal completeness principle” (Amundson 2005, 175–89). According to the causal completeness principle, understanding evolution requires understanding development, on the grounds that evolution can only modify adult form by modifying the ontogenetic processes that produce that form. Because the modern synthetic theory excluded development, its proponents were committed to rejecting the causal completeness principle. It is this disagreement that is the basis for Amundson’s claim that evolutionary developmental biology and the modern synthetic theory are incommensurable.

Amundson (2005, 255) summarizes the incommensurability between evo-devo and the synthesis, including their respective positions on the causal completeness principle, as follows:

[Synthesis]: Individuals don't evolve. Populations do. Populations evolve by natural selection.

[Evo-Devo]: Individuals don't evolve. Ontogenies do. Ontogenies evolve by modifications of ontogeny.⁸

Lillie's position on the split between heredity and development generates incommensurability between these views. The evolution of populations is (by definition) a change in their genetic composition. If a genetic theory of development is impossible, then in principle the evolution of populations cannot be connected to the evolution of ontogenies. Moreover, this hypothetical incommensurability between the synthesis and evo-devo was in fact actualized between the sciences of genetics and embryology at the time of the synthesis. As Gilbert (1998, 174) notes:

By the 1930s, genetics and embryology had their own rules of evidence, their own paradigmatic experiments, their own favored organisms, their own professors, their own journals, and, most importantly, their own vocabulary.

At the same time, this way of characterizing the incommensurability makes clear how it can be overcome. A genetic theory of development would allow biologists to connect

⁸ Amundson describes this as a broader conflict between adaptationists and structuralists. Since I do not here concern myself with the broader structure/function dispute Amundson discusses (I discuss it in chapters 2 and 4), I have changed the names of the positions to represent my narrower concerns. This change is not unfair to Amundson, for the synthesis is for him an adaptationist program, while evo-devo is a structuralist program.

the evolution of populations to the evolution of ontogenies, and so would bring the two perspectives together. This, I will argue below (Section 2.7), is precisely what has happened.

Thus far, I have explored the issue of the exclusion of development from the synthesis from the perspective of the developmentalist critics (old and new) of the synthesis and its genetic foundations. How does the matter appear from the other side? As we have seen, Amundson's argument for the incommensurability of evo-devo (and *a fortiori* GRN theory) and synthesis-derived evolutionary theorizing rests on Lillie's ontological reading of the split between heredity and development. Lillie's defended this reading by arguing that no genetic theory of development could meet his criteria of adequacy. But what if a genetic theory satisfying those criteria were possible? The architects of the synthesis certainly believed him to be wrong. As we will see, their optimistic view about the relationship between genetics and development leads to a very different picture of the conceptual exclusion of development from the synthesis: from the synthetic perspective, *the exclusion does not exist at all*.

2.4 DOBZHANSKY'S THREE GENETICS

The publication of Theodosius Dobzhansky's *Genetics and the Origin of Species* in 1937 was a pivotal moment in the early history of the modern synthesis. Drawing upon work in

classical Mendelian genetics and important results obtained by mathematical population geneticists (especially Ronald Fisher and Sewall Wright), Dobzhansky attempted to provide a genetic analysis of evolutionary problems, most notably the problem of the origin of discontinuity in nature. In doing so, he helped to articulate the theoretical framework around which disparate biological disciplines could be unified.

Dobzhansky's accomplishments were conceptual as much as empirical. Though he provided important answers to many of the questions he raised, the real novelty of his book lies in the way he framed those questions, drawing on the resources of Mendelian and population genetics. For present purposes, the most important conceptual division of the work was a threefold division of the science of genetics into transmission, developmental, and population genetics (Dobzhansky 1982, 8–12). Dobzhansky placed this division within a larger framework that is worth presenting briefly.

Genetics and the Origin of Species begins by establishing the central problem Dobzhansky wished to solve: “the nature and the origin of the discrete groups into which the living world is differentiated” (Dobzhansky 1982, p. 5).⁹ The diversity of life is not a continuous array; rather, it forms nested clusters. Dobzhansky's goal was to provide a causal analysis of the origin of these clusters that could explain why they were clusters at all, rather than a continuous array. Dobzhansky differentiated between causal and

⁹ Treating this as the most pressing problem in evolutionary biology was not at all a trivial move on Dobzhansky's part. Two decades earlier, Henry Fairfield Osborn (1916, p. 504) had argued that the problem of the origin of species had become “an incidental issue” and that the central problem was “the origin and history of single characters.” Dobzhansky's position marks a complete reversal of this: the problem of the origin of individual characters gets no discussion in his book.

descriptive approaches to evolution. Descriptive disciplines such as systematics and morphology were tasked with illuminating the historical course of life (Dobzhansky 1982, p. 6). By contrast, genetics was an experimental, “nomothetic” science. A genetic study of evolution promised to provide “an analysis of the conflicting forces tending to increase or to level off the differences between organisms,” and in doing so would furnish an explanation of organic diversity (Dobzhansky 1982, p. 7).¹⁰

Each of the three types of genetic study had a role to play in this analysis. The first subdivision, *transmission genetics*, studied “the mechanisms of transmission of hereditary characteristics from parents to offspring, that is, on the architectonics of the germ plasm of the sex cells.” Transmission genetics offered a genetic theory of heredity, showing how “the character of organisms are determined by the genes carried in the sex cells” (Dobzhansky 1982, 9). By 1937, Dobzhansky could confidently state that “the genetics of the transmission of hereditary characters is, by and large, understood now” (Dobzhansky 1982, 10).

Dobzhansky explicitly admitted, however, that transmission genetics was limited. It did not explain “the mechanisms of gene action in development” (Dobzhansky 1982, 10). This formed the subject matter of *developmental genetics*, also called “physiological genetics” (Goldschmidt 1938). Transmission genetics established correlations between

¹⁰ It is worth noting that this distinction between causal and descriptive sciences, with morphology falling on the descriptive side, contributes to what Scott Gilbert (1998) has called the “supersessionist” rhetoric with which geneticists portrayed their science as the completion and perfection of the allegedly dead-end science of embryology. I do not mean to deny that there was a rhetorical exclusion of developmental biology from the synthesis. I mean to deny only that a conceptual exclusion underlay this rhetorical exclusion.

particular genetic mutations and particular phenotypic effects. Developmental genetics sought to explain the complex process by which the genes produced these effects. In stark contrast to his happy assessment of transmission genetics, Dobzhansky lamented that developmental genetic study had thus far furnished little by way of established knowledge and, even worse, that “no reliable methods have been devised for investigations in this field” (Dobzhansky 1982, 10–11). This pessimism was widely shared (Morgan 1926, 290; Sinnott 1937, 62; Goldschmidt 1938, v–vi).

Both transmission and developmental genetics were “concerned with individuals as units” (Dobzhansky 1982, 11). By contrast, *population genetics* studied how the genetic composition of a population changes over time. Population genetics, as its name suggests, concerned itself with genetic phenomena at the population level, in particular the dynamics of changes in allele frequencies. Mathematical models of these dynamics elucidated the forces (selection, drift, mutation, migration) that drove these changes, though population genetics was not, for Dobzhansky, limited to these models.

Relying on this threefold division of genetics, Dobzhansky (1982, 11–12) defined evolution as “a change in the genetic composition of populations,” with the result that the problems of evolution – in particular the problem of organic diversity – were population genetic problems. Dobzhansky (1982, 13) recognized three basic types of population genetic problem: (a) the problem of how mutations enter a population (mutation, gene flow); (b) the problem of how mutations spread or fail to spread once present in a population; and (c) the problem of how the diversity “attained on the preceding two levels” is fixed. This last level concerned itself with the processes that create barriers between populations,

barriers that cause two populations to proceed upon independent evolutionary trajectories. Whereas the first two levels dealt with phenomena occurring within given populations, the third level explained how those populations became units in the first place.

Such was the conceptual framework of Dobzhansky's evolutionary theorizing. Nothing in this framework precludes the inclusion of developmental genetic discoveries into evolutionary theorizing. Dobzhansky conceived developmental genetics as an important but unfortunately immature part of the broader science of genetics. And it was genetics as a whole, not specifically transmission genetics or population genetics, that was to inform evolutionary theorizing.

Lying in the background of Dobzhansky's inclusive attitude toward developmental genetics is his conception of the split between heredity and development, which differed in essential features from Lillie's.¹¹ For Lillie, as we saw, there was an ontological split between *genetics* (heredity) and *development*. For Dobzhansky, in stark contrast, there was no such ontological split. Instead, there was a methodological split between *transmission genetics* and *developmental genetics*.¹² They were different subfields of genetics, with different methods (which, by 1937, had reached markedly different stages of development). There was, however, no principled barrier to incorporating their findings into unified explanations, including explanations of evolutionary phenomena.

¹¹ This issue is discussed further below (Section 2.5).

¹² R. A. Brink (1927, 280) drew the even stronger conclusion that the demonstration that genes were the hereditary material entailed that they must control developmental processes – that an ontological split such as Lillie imagined was impossible.

It is nonetheless true that *Genetics and the Origin of Species* incorporates very little developmental genetics in its explanations of evolutionary phenomena. Dobzhansky himself offered a perfectly innocuous reason for this: there was little to include. Later works of the synthesis did begin to include developmental genetic theories in their evolutionary theorizing. For instance, Julian Huxley (2010, 73–74) incorporated some of Goldschmidt’s developmental genetic conclusions in his explanation for why wild type characters are less modifiable by environmental changes than mutant characters. Similarly, Curt Stern (1963, 19–20), in his contribution to a volume edited by Mayr and Simpson, offered a speculative explanation of the evolution of sibling species in terms of the phenotypically silent remodeling of genetic reaction networks. G. Ledyard Stebbins, the foremost plant biologist of the synthesis, developed an entire research program in plant developmental genetics (Smocovitis 2008). Finally, it is worth noting that, after Britten and Davidson (1969) published their landmark paper introducing an early form of GRN theory, both Mayr (1970, 183) and Dobzhansky (1970, 413–14) cited the paper (in particular its evolutionary implications) sympathetically: Mayr with considerable enthusiasm, Dobzhansky rather more cautiously but still hopefully. For Mayr, understanding the structure of the “complex epigenetic systems” of organisms was important for understanding the amount and kind of genetic variability they could tolerate (Mayr 1969b, 316). Britten and Davidson’s proposal promised to help elucidate this problem. Mayr thus called for a developmental genetic contribution to answering one of the core problems of the modern synthesis: the origin of discontinuous variation in nature.

The case of Richard Goldschmidt is similarly instructive. Like Dobzhansky, Goldschmidt believed in the possibility of developmental genetics. Unlike Dobzhansky, Goldschmidt actually worked out a sophisticated theory of developmental genetics, from which he drew substantial evolutionary conclusions (Goldschmidt 1938, 1982). These conclusions were profoundly at odds with the synthetic theory: Goldschmidt denied that macroevolution is compounded microevolution, denied that geographic races are incipient species, and allowed only a reduced role for natural selection in driving evolutionary change. The architects of the synthesis responded, not by arguing that his work was irrelevant, but rather by assimilating it where they could (as in the cases of Huxley and Stern, mentioned above), while denying the aspects of his theory that led to his problematic evolutionary conclusions (Davis, Dietrich, and Jacobs 2009).

What the case of Goldschmidt shows is that the proponents of the synthesis made quite substantial assumptions about what developmental genetics would look like when mature. Namely, they assumed that it would not have implications like those of Goldschmidt's developmental genetics. Were development truly *irrelevant* to the synthetic theory, such assumptions would be unnecessary.

As these examples show, the synthesis included from start what developmental genetic information it could. Thus we can see that the conceptual exclusion of development from the synthesis exists only from the developmentalist perspective that regards developmental genetics with suspicion. From such a perspective, developmental genetics can only get at the most superficial part of development: realization. The major phenomena of development (Lillie's "embryonic segregation") must be left out of a genetic approach.

In this sense, the developmentalists excluded themselves.¹³ From the vantage of the synthesis, which was committed to the relevance of genetics to all of development, there was not and could not be any such exclusion.

My analysis thus far shows that the conceptual exclusion of development from the synthesis, insofar as it was real, rested on an empirical question: how much of development can be explained genetically? I have shown that participants in the synthesis answered this question optimistically, and accordingly included developmental genetic findings in their evolutionary theorizing. But nothing I have presented thus far resolves Lillie's powerful worries that the question is to be answered pessimistically: very little of development admits of a genetic explanation. Was Lillie right? We will see that neither of his arguments for the impossibility of developmental genetics succeeds. One was problematic from the start (Section 2.5). The other raised a more pressing issue, but even this has recently been resolved (Section 2.7).

2.5 LILLIE'S ERROR

We have seen that where Lillie saw an ontological split between heredity (genetics) and development, Dobzhansky saw only a methodological split between transmission

¹³ As Wimsatt puts it, "Developmental genetics proved largely intractable and lacked any generalizable organizing machinery comparable to that provided by linkage mapping. Because of this, embryologists had gone their own way" (Wimsatt 2015, 368).

genetics and developmental genetics. Dobzhansky explicitly opposed reading into this methodological split any general split between heredity and development. Heredity, he insisted, covered both transmission and developmental genetics. On this point, Dobzhansky (1982, 9–10; emphasis added) really could not be clearer, and is worth quoting at length:

The elegance and precision of methods devised by genetics to control the results of experiments involving crosses of individuals differing in many hereditary characteristics have led to claims that the problem of heredity has been solved. Although a large amount of work still remains to be done in this field, it is indeed fair to say that the genetics of the transmission of hereditary characters is, by and large, understood now. *But the problem of heredity is much wider.* Knowing the rules governing the distribution of hereditary characteristics of an organism among the sex cells, one is in a position to predict what constellations of genes are likely to be present in the zygotes coming from the union of such sex cells. Between the genes of a fertilized egg and the characters of the adult organism arising from it there lies, however, the whole of individual development during which the genes exert their determining action.

In this passage, Dobzhansky rejected both of the “truisms” that Amundson sees as lying behind the first exclusion of development from the synthesis.¹⁴ Dobzhansky rejected the view that heredity is the passing of traits or representatives of traits as opposed to “the passing on of developmental processes” (Amundson 2005, 148). For Dobzhansky, these developmental processes were as much a part of heredity as the transmission of genes. With this, the second truism equally falls by the wayside. Development was part of heredity.

¹⁴ Dobzhansky’s views on this point persisted unchanged into the third edition of *Genetics and the Origin of Species* (Dobzhansky 1951, pp. 12–13).

There nevertheless exist modified versions of both truisms that Dobzhansky accepted, and understanding these will help us to see the error in one of Lillie's arguments against the possibility of a genetic theory of development. These modified truisms are:

Modified Truism 1. Transmission is the passing of traits (or representatives of traits, such as genes) between generations.

Modified Truism 2. The study of transmission is independent of the study of development.

The first truism is basically a definition of the subject matter of transmission genetics. The second truism is a hard-won accomplishment. The development of crossing experiments in the Mendelian tradition made it possible to study transmission without studying development as well. In other words, it enabled the isolation and independent study of one aspect of heredity. Whereas Amundson claimed that his two truisms were controversial stipulations, these two truisms are rather innocuous. Even Lillie could accept them, and indeed did accept them, for they are the very basis for his diagnosis of a split between heredity and development.

We are now in position to see why Lillie's argument that no genetic theory could respect the difference between open and closed terms suffered from a fatal flaw. The argument, recall, was that the methods of genetic analysis precluded genetic theories from treating developmental stages in their developmental context. As an analysis of the methods of transmission genetics, Lillie's argument is perfectly good. Transmission genetic analysis does indeed look only at the correlations between genes (alpha) and traits (omega), skipping over the intervening processes. These methods could show genetic

effects on development, but only by treating developmental stages as if they were final, closed terms, as if they were fixed “traits” like any adult trait. Starting from the second, hard-won truism, that the methods of studying transmission can ignore development, Lillie correctly concluded that those methods cannot furnish a theory of development.

This warrants only the modest conclusion that there cannot be a transmission genetic theory of development. Lillie, however, attempted to infer that there cannot be a genetic theory of development at all. That conclusion follows only on the assumption that all genetic study must be limited to the methods of transmission genetics. We have seen that Dobzhansky explicitly rejected that assumption. While Dobzhansky could not say what the methods of developmental genetics would be, since they had by his own admission not yet been developed, he did not expect them to be subject to the same limitations as those of transmission genetics, since it was precisely those limitations that led to the need for a distinct arena of developmental genetic study in the first place.

It is important here to be clear about what Lillie got wrong and what he got right. Lillie’s argument rests on the assumption that all genetic methods will be subject to the same limitations as transmission genetic methods. Dobzhansky rejected this assumption, but this does not show that the assumption is false. It might have turned out that all forms of genetic analysis would indeed fail to preserve this distinction. But Lillie offered no good reason (indeed, no reason at all) in defense of this assumption. Lillie therefore failed to provide a principled reason why genetic analysis *must* fail to preserve the distinction between open and closed terms.

None of this suggests that Lillie was wrong to insist that a genetic theory of development must preserve the distinction. Lillie's criterion of adequacy still stands as legitimate. Only his argument that this criterion cannot be met fails. In 1937, it was an open question whether or not a genetic theory could satisfy the criterion. I will argue below (Section 2.7) that it is no longer an open question, for GRN theory satisfies it. But first there is another matter deserving attention, concerning Amundson's use of Lillie's argument, for Amundson's analysis repeats Lillie's error.

2.6 GENETICS AND CAUSATION

Amundson's argument that the architects of the synthesis rejected the causal completeness principle rests on his account of how Mendelian geneticists (and their synthesist followers) understood causality. Amundson (2005, 150) writes:

If a single allele can be regarded as the cause of pink eye color, then it is possible to causally explain adult characteristics without any reference to the embryological processes that actually brought them about.

This is just a consequence of the modified second truism about heredity (Section 2.5), but Amundson sees in it a direct conflict with developmental approaches to heredity. Commenting on a passage from Morgan et al. (1915, 209) in which they refer to a particular genetic factor as "the cause of pink," Amundson (2005, 149) writes: "Prior to this assertion, 'the cause' of any adult body characteristic could potentially include the entire

embryological history of the organism.” Whereas a developmental biologist would have thought that a trait could not be causally explained without referencing this embryological history, transmission genetic analysis seemed to obviate the need for reference to such processes.

Amundson’s argument here rests on a mischaracterization of the transmission genetic understanding of causality. Transmission genetics does not license the claim that a single allele causes a particular trait. The claims that transmission genetic experiments license are claims about how *differences* in particular genes cause (against a particular shared genetic and environmental background) *differences* in particular traits (cf. Waters 2007). A gene is never the cause of a trait, but a difference at a genetic locus may be the cause of a difference in a trait (Huxley 2010, 63).

Transmission genetic analysis elucidates the input-output relations between the allelic values of genes and the phenotypic values of traits (given a particular genetic and environmental background). What it cannot yield is knowledge concerning the mechanisms responsible for the existence of those input-output relations—i.e. the entire process of development. This is compatible, however, with the existence of other methods (those of developmental genetics) that do elucidate the processes underlying these input-output relations. As Woodward argues, there is no incompatibility between “difference making” and “connecting process” explanations (Woodward 2013). Transmission genetics skips over development, but it does not provide explanations that compete with developmental explanations. It doesn’t preclude the need for a connecting process explanation

invoking features of ontogeny. Like Lillie, Amundson takes the limitations of transmission genetics to be representative of the whole of genetics. They are not.

This has implications for how we understand the purported rejection of the causal completeness principle by the synthesis (recall from Section 2.3). Though he gets the precise diagnosis wrong, Amundson is right that transmission genetic analysis claims for itself a certain kind of causal self-sufficiency. Transmission genetic analysis provides knowledge about the differences in phenotypic traits caused by differences at genetic loci, and this knowledge did not require any understanding of the intervening developmental process. This is, again, the modified second truism concerning heredity.

In consequence, the population genetic theorizing that informed the modern synthesis is similarly self-sufficient. That work showed that a great deal of evolutionary analysis is possible in the absence of any knowledge of development. You can go a long way, evolutionarily speaking, with genotype-phenotype correlations. This amounts to a rejection of a strong form of the causal completeness principle, according to which the principle is taken to claim that no understanding of evolution is possible without understanding ontogeny. But, in that strong form, the principle is simply false. Indeed, showing that it is false can be taken as one of the major accomplishments of the synthesis.

That this work is self-sufficient, however, does not require denying a milder form of the causal completeness principle, one that claims that because evolution can modify adult phenotypes only by modifying ontogeny, understanding development is important for some kinds of evolutionary analysis. It is this milder form of the principle that is all

that is required for development to be relevant to synthesis-derived evolutionary theorizing.

In this milder form, Dobzhansky and other proponents of the synthesis accepted the causal completeness principle. Dobzhansky specifically set out to offer a causal, genetic theory of evolution, and he included developmental genetics as one of the branches of genetics that could contribute to this theory. If Dobzhansky did not incorporate such contributions into *Genetics and the Origin of Species*, this was for lack of anything to include. It was not due to any intractable conceptual barriers. This is further confirmed by the examples of Huxley, Stern, and Mayr (discussed above, Section 2.4), all of whom did draw on developmental genetic research to address evolutionary questions.

Thus, where Amundson sees deep-seated incommensurability between the synthesis and evo-devo, based on the two programs' relation to the causal completeness principle, what is actually going on is milder. The synthesis offered a route to the inclusion of development in evolutionary theorizing, a route running through developmental genetics. It was the developmental biologists – not the architects of the synthesis – who argued that this route excluded development proper. In this regard, the synthesis did not reject the causal completeness principle: some evolutionary problems did require input from developmental genetics for their solution. What they did reject was an overly restrictive form of the principle that required that all evolutionary problems require input from developmental genetics. They showed that a great deal can be done without such information.

Evolutionary-developmental biology rests largely, though not exclusively, on developmental genetics. It emerged as a field following the discovery of important roles played by *Hox* genes in development, and has since given rise to a well-developed form of GRN theory, which places these roles in a broader theoretical framework concerning the role of genetic regulatory networks in development. Amundson recognizes that evolutionary-developmental biology is drawing evolutionary implications from developmental genetics (Amundson 2005, 4–9). What Amundson fails to see is that insofar as evolutionary-developmental biology is a flourishing field, this is a vindication not of Lillie but of Dobzhansky. It is Lillie, not Dobzhansky, who would be surprised by the flourishing of this developmental genetic research program.

2.7 LILLIE'S DOUBTS ASSUAGED

The modern synthesis thus left conceptual space for input from developmental genetics and even, as we saw, began to include this input. But we have also seen that Lillie proposed criteria of adequacy that any developmental genetic theory must – but no contemporary developmental genetic theory could – satisfy. He argued that this failure was due not to human frailty, but rather to the impossibility of the task.¹⁵ Though one of his

¹⁵ Lillie explicitly criticized Goldschmidt's developmental genetic theory for failing to meet his criteria. Though Goldschmidt (1938) published *Physiological Genetics* eleven

arguments held no water (Section 2.5), his invocation of the developmental paradox provided powerful reason to worry about the prospects of developmental genetics.

In this section, I argue that GRN theory meets both of Lillie's criteria of adequacy: it explains how embryonic complexity increases over developmental time, and it preserves the distinction between open and closed terms in development. GRN theory thus accomplishes the reunion of heredity and development that Dobzhansky expected and Lillie thought impossible. It therefore provides a conceptual reconciliation of developmental genetics and synthesis-derived evolutionary theorizing that would be acceptable to both Lillie and Dobzhansky.¹⁶

GRN theory has both a developmental and an evolutionary component. The developmental component concerns the action of gene regulatory networks during animal development. The major aim of the developmental component is to explain the increasing complexity of the embryo over developmental time. This is apparent from the very first sentence of Peter's and Davidson's (2015, 1) book expounding the theory: "Considered from an informational perspective, the most interesting and defining feature of animal development is that it generates a continuous increase in complexity of spatial organization."

years after Lillie's article, on my reading, Goldschmidt's 1938 theory was vulnerable to exactly the same criticisms that Lillie raised in 1927.

¹⁶ Though Dobzhansky was right and Lillie wrong about the possibility of developmental genetics, this does not mean that developmental genetics as it actually developed must be compatible with synthesis-derived evolutionary theorizing. If this is not intuitive, consider that if Goldschmidt's developmental genetic theory had been correct, it would have vindicated Dobzhansky's view of the relation between developmental genetics and evolutionary theory while contradicting most of Dobzhansky's substantive conclusions about the nature of the evolutionary process.

The evolutionary component concerns the principles of how these networks evolve, with implications for issues ranging from the origin of animal body plans during the Cambrian explosion (Davidson and Erwin 2009; Erwin and Valentine 2013) to microevolution observed in contemporary populations (Erwin and Davidson 2009; Nunes et al. 2013). In drawing evolutionary conclusions from their account of development, Peter and Davidson (2015, 4) explicitly endorse the causal completeness principle in its weaker form:

Development produces the genomically encoded body plans of animals, and evolution is the process by which body plans arise *de novo*, and heritably alter in deep time. Since causality in development must at root be considered in terms of information processing, and how spatial control of gene expression is mandated by genomic regulatory information, so also the evolution of the body plan. At the informational level of the genomically encoded program for development, evolutionary process is equivalent to the time derivative of developmental process: evolution is change in this program over deep time.

Because understanding how GRNs evolve depends on understanding how they operate during development, the developmental component of GRN theory is the core of the theory, with the evolutionary implications as downstream consequences.

The developmental core of GRN theory hopes to explain increasing embryonic complexity over developmental time in terms of a regulatory architecture built into the genome and so equally present in every cell. It must thus face up to the developmental paradox. At the most abstract level, GRN theory dissolves the paradox by showing how this regulatory architecture is able to produce increasingly finely specified patterns of gene expression over time. To see how this works, we must delve into the details. The account below is condensed from Peter and Davidson's (2015) book, except where otherwise indicated.

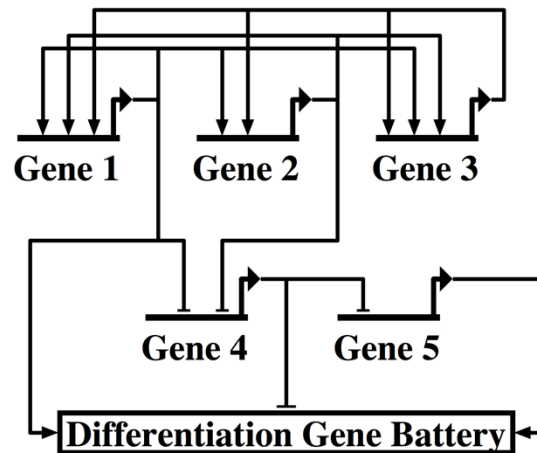


Figure 1. Example gene regulatory network.

The basic claim of GRN theory is that major aspects of development result from the action of intricately structured, hierarchical genetic regulatory networks (GRNs) that control gene expression during development (Figure 1).¹⁷ These networks consist of genes and their regulatory elements. The regulatory elements consist of clusters of transcription factor binding sites known as enhancers. Transcription factor proteins bind to these sites in order to recruit (or block) the complex molecular machinery that is required to begin

¹⁷ Figure 1 was created using BioTapestry (Longabaugh, Davidson, and Bolouri 2005).

transcription, thereby driving (or repressing) gene expression. Different transcription factors bind to different binding sites. Regulation of gene expression is therefore sequence-specific.

A gene, in the sense relevant here, is a stretch of DNA that may be transcribed to RNA and translated into a protein. Three types of gene are especially important in GRNs: transcription factors, signaling genes, and effector genes. Transcription factors, as noted, directly bind to regulatory sequences and drive or repress expression. Signaling genes enable communication between cells, which is important in patterning gene expression. Effector genes encode proteins that are not involved in gene regulation, but instead provide cells with their specific physical properties.

A particular GRN is defined by “the physical and functional relationships among regulatory genes” (Peter and Davidson 2015, 43). Regulatory interactions are combinatorial, with multiple transcription factors required to initiate transcription. Thus, each gene in a GRN requires multiple inputs. Their outputs are likewise multiple, as each transcription factor targets multiple downstream genes. There is a one-way flow of information through a GRN. For example, in Figure 1, the arrow from Gene 1 to Gene 2 is unidirectional: it indicates only that the former regulates the latter. It is true that Gene 2 also regulates Gene 1, but this is a separate, equally unidirectional interaction. This gives GRNs a hierarchical character, with upstream elements regulating downstream elements. This allows for a statically encoded regulatory architecture (as depicted in Figure 1) to be successively deployed over time.

GRNs are modular assemblages of subcircuits. These subcircuits fall into several distinct types, e.g. positive feedback loops and double negative gates. Subcircuit types are defined by their topologies and not by the particular genes they contain. Different types of subcircuit accomplish distinct developmental tasks. For instance, one common function of positive feedback loops is to take states of gene expression installed by transient inputs and stabilize them, so that they persist after the input is no longer present (see Gene 1, Gene 2, and Gene 3 in Figure 1). Double negative gates, by contrast, often serve to ensure that a particular set of genes is expressed in one region and not expressed anywhere else (see Gene 1, Gene 2, Gene 4, and Gene 5 in Figure 1).

Subcircuits are organized into a hierarchical network, with distinct types of subcircuits prominent at different levels of the hierarchy. These networks are determinate, in the sense that upstream circuits determine the activity of downstream subcircuits (Erwin and Davidson 2009). A developmental GRN terminates in a differentiation gene battery (Figure 1), a collection of effector genes. In Lillie's terms, the upper levels of the hierarchy account for embryonic segregation, while the differentiation gene batteries account for the realization of the segregated potencies.

With these materials, GRN theory resolves the developmental paradox and explains localized differentiation. The solution has two parts. The first part involves maternally deposited factors. Because GRN theory relies on a regulatory architecture encoded in the genome, that regulatory architecture is the same everywhere in the embryo. In order, therefore, for it to operate differently in different parts of the embryo, there must be some initial imbalance. This imbalance is furnished by materials in the egg cytoplasm, which

is already highly differentiated, though its level of differentiation pales in comparison to the adult organism. How exactly this imbalance is produced varies by organism. In amphibians, for instance, the dorsoventral axis is established by the localization of maternally deposited factors opposite the point of sperm entry (Weaver and Kimelman 2004). By contrast, in *Drosophila*, “the spatial cues for the embryonic dorsoventral axis originate during oogenesis in the follicle cell layer” – the point of sperm entry is irrelevant (Moussian and Roth 2005). In both cases, the initial imbalance produces regions of differential gene expression.

At this point, the regulatory architecture of the genome takes over, progressively increasing this initial differentiation – the second half of GRN theory’s solution to the developmental paradox. This regulatory architecture takes one regulatory state as input and produces a new regulatory state as output. Regulatory states are defined as “the collective sum of specifically coexpressed transcriptional regulators in a given place at a given time” (Peter and Davidson 2015, 48). GRN theory shows how the regulatory genome can produce output states that are more finely differentiated than the input states. The primary means by which this occurs is cell-cell signaling. This allows GRN theory to explain localized differentiation. The regulatory genome takes a relatively slight initial imbalance and amplifies it in precise, repeatable ways. Put a little differentiation in, get a lot of differentiation out.

Furthermore, GRN theory preserves the distinction between open and closed terms, for the abstract hierarchical structure of GRNs translates into definite spatiotemporal patterns of gene expression during development. In GRN theory, these regulatory states are

treated as open: depending on what signals they receive, they can give rise to multiple downstream regulatory states (explaining pluripotency). Moreover, they are recognized as having a past (the prior regulatory states that produced them) and a future (the subsequent regulatory states that they produce), eventually terminating in the activation of a differentiation gene battery. The activation of such a battery is responsible for realization (in Lillie's sense), at which point the development of a particular region is "closed."

GRN theory thus meets Lillie's criteria for a unified theory of heredity and development. Contemporary developmental genetics has advanced into Lillie's (1927, 366) "unconquered territory" and has done so without giving up "the theory of the integrity of the entire gene system in all cells." Dobzhansky was right to conceive the split between heredity and development not as an ontological split between genetics and development but as a methodological split between transmission genetics and developmental genetics, a methodological split that allows for explanations that incorporate the results of both subfields of genetics.

Insofar as the apparent conceptual (as opposed to contingent historical) exclusion of development from the synthesis rested on the existence of a split between heredity and development, that split existed only from the perspective of those who, like Lillie, regarded a genetic theory of development as an impossibility. Recall Amundson's (2005, 255) characterization of the incommensurability between the synthesis and evo-devo: whereas synthesis-derived theorizing focuses on the evolution of populations, evo-devo focuses on the evolution of ontogeny. As we saw, it is only if a genetic theory of development is impossible that these two perspectives are fundamentally incompatible.

Though Lillie and his sympathizers had legitimate reasons to believe in the impossibility of a genetic theory of development, GRN theory fully resolves their worries. It thus provides a theory that explains development in a language commensurable with the language of the modern synthetic evolutionary theory: the language of genetics.

2.8 RECAPITULATION

In the foregoing, I have defended a number of interlinked claims. First, I have shown that developmental biologists and the architects of the modern synthesis understood the disciplinary split between the study of heredity and of development in different ways. Developmental biologists argued for the existence of an ontological split between genetics and development, while the synthesists argued for the existence of a methodological split between transmission genetics and developmental genetics.

Second, I have argued that, from the developmentalist perspective, developmental biology is incommensurable with the synthesists' approach to evolutionary theorizing and so is conceptually excluded from the synthesis. However, this incommensurability and exclusion do not exist from the synthesists' perspective, and the synthesists did attempt to incorporate developmental genetic findings into their theorizing.

Third, I have presented and evaluated two primary arguments favoring the developmentalists' perspective on the split between heredity and development. One of these

arguments rested on a confusion about the nature of genetic methods. The other (the developmental paradox) was more compelling. However, contemporary GRN theory has the resources to resolve the developmental paradox in a manner that vindicates the synthesisists' view about the possibility of developmental genetics. GRN theory thus shows that developmental biology and synthesis-derived evolutionary theorizing are not incommensurable.

All of the work done here has been concerned with showing that there is conceptual space within synthesis-derived evolutionary theorizing for incorporating the consequences of GRN theory. This does not mean that such integration will always proceed smoothly. Indeed, the Davidson quotation with which I began reveals clearly that it will not. Plenty of work remains to be done. I have shown that this work can be done on friendlier terms than has sometimes been thought.

3.0 CUVIERAN FUNCTIONALISM: 500 MILLION YEARS OF STASIS

Cuvier believed that the radically different organizations of the four embranchements could not change from one to another without a fatal loss of integrated function during the transition. This argument is still a forceful one. (Raff 1996, 6)

3.1 ANTI-GRADUALISM, THEN AND NOW

The overarching aim of this chapter is to make a contribution to a broader project of understanding just how evolutionary-developmental biology (evo-devo) fits into the landscape of contemporary evolutionary theory. The focus here will be on the sense in which evo-devo involves what Ernst Mayr (1959) called “typological thinking.” To achieve this overarching goal, the chapter has three subsidiary aims: (1) to distinguish two ways in which Mayr’s notoriously unprincipled notion of typological thinking can be made precise, (2) to show that one of these ways—Cuvieran functionalism—plays an important role in evo-devo’s developmental genetic explanations of the deep conservation of animal body plans, and (3) to argue that Cuvieran functionalism is compatible with mainstream

evolutionary theorizing, and in particular with what Ron Amundson (2005) has called “exclusive population thinking”. The remainder of this introduction is concerned with making the relations between these three aims more salient.

As Ernst Mayr tells the story, one of Darwin’s major achievements was to replace typological thinking with population thinking (Mayr 1959; see also Hull 1965a, 1965b). Typological thinking, rooted in Platonic philosophy, had dominated pre-Darwinian biology, which treated species as fixed types, limited in their ability to vary. Darwin's *Origin* stood this way of thinking on its head, making variation fundamental and unlimited, relegating “types” to the status of purely statistical epiphenomena. From this point on, according to Mayr, all major disputes in evolutionary biology – encompassing both opposition to evolutionary theorizing of any stripe and opposition to particular Darwinian evolutionary theories (such as those of Mayr and Dobzhansky in the 1930s and 1940s) – could be fruitfully analyzed as pitting typological thinking against population thinking. The core of these disputes lay in the incompatibility of typological thinking with the acceptance of gradual evolution. A typologist, for Mayr, was necessarily an anti-gradualist.

Evo-devo, as it has matured over the past several decades, has frequently found itself at odds with mainstream evolutionary theorizing. In attempting to understand these tensions, scholars have latched onto Mayr’s framework. On one side are friends and practitioners of evo-devo who have argued that typological thinking is essential to, and therefore vindicated by, evo-devo’s successes (Amundson 1998; Brigandt 2007; Love 2009a; Wagner 2014). As every *modus ponens* has its *modus tollens*, so some critics have suggested that evo-devo is vitiated by its typology (Jenner 2006). Ron Amundson (2005, chap. 11)

has even suggested that evo-devo's typology is one factor (among several) that renders evo-devo incommensurable with mainstream evolutionary theorizing, with its commitment to exclusive population thinking.

A major impetus for understanding evo-devo as typological stems from its emphasis on deep conservation. This emphasis manifests at several levels, as evo-devo biologists study (i) animals' deeply conserved genetic toolkit (Carroll 2008); (ii) gene regulatory network "kernels" that have persisted unchanged for over 500 million years (Davidson and Erwin 2006); (iii) the conservation of complex phenotypic arrangements (Wagner and Schwenk 2000; Schwenk and Wagner 2001); and (iv) the deep conservation of animal body plans (Levin et al. 2016). Raff (1996, chap. 1) has suggested that this is a major difference between evo-devo and mainstream evolutionary theorizing: where the latter emphasizes the diversity of the organic world, the former emphasizes universality.¹⁸ The focus on what is constant as opposed to what is changing makes an analysis of evo-devo as typological – as a science of fixed types – alluring.

The difficulty with analyzing evo-devo as a typological branch of evolutionary theory is that Mayr never offered a coherent account of typological thinking. As Joeri Witteveen (2015, 2016) has shown, Mayr's initial introduction of the concept, justified as a "temporary oversimplification," was unprincipled from the start, merging two distinct concepts

¹⁸ The focus on diversity can be clearly seen at the start of Theodosius Dobzhansky's *Genetics and the Origin of Species*, a foundational work of the modern synthesis, from which contemporary evolutionary theorizing descends (Dobzhansky 1982, chap. 1). There, Dobzhansky establishes the diversity of the organic world as the key explanandum of evolutionary theory, with no mention of conserved features as a comparably important explanandum.

in a haphazard way, and only got worse with time. I am convinced by this work that “typology” and “typological thinking” are terms beyond repair, and that the analysis of evo-devo as typological is more apt to create confusion than illumination.

I do not, however, think that the Mayrian project was entirely hopeless. In introducing the notion of typological thinking, one of Mayr’s aims was to isolate a common pattern of thought that gives rise to opposition to gradualistic evolutionary theories. Though he failed in his attempt to carry out this project, the question itself is a good one: are there common paths to anti-gradualism, repeated throughout the history of biology? This question is especially pertinent for understanding evo-devo. In order to explain deep conservation, the biologist is tasked with explaining why some part of an organism has, over long periods of time and in multiple distinct lineages, failed to evolve. By understanding the various possible routes to anti-gradualism, we may thereby come to better understand the conceptual foundations of evo-devo.

In what follows, I argue that there are at least two distinct paths to anti-gradualism, one of which plays a crucial role in evo-devo’s explanation of deep conservation. I begin by presenting Mayr’s notion of typological thinking and demonstrating its close connection with anti-gradualism (Section 3.2), after which I show that anti-gradualism plays a key role in evo-devo’s developmental genetic explanation of deep conservation (Section 3.3). I then distinguish two paths to anti-gradualism, *natural state thinking* (Sober 1980) and *Cuvieran functionalism* (Section 3.4). I show that Cuvieran functionalism plays a central role in the developmental genetic explanation of deep conservation, while natural state thinking plays no role (Section 3.5). Having established the role of Cuvieran

functionalism in evo-devo, I argue that standard objections to typological thinking do not apply to Cuvieran functionalism (Section 3.6, Section 3.7, Section 3.8). I conclude by reflecting on how evo-devo's emphasis on universality/conservation fits together with mainstream evolutionary theorizing's emphasis on diversity (Section 3.9).

3.2 TYPOLOGICAL THINKING AND ANTI-GRADUALISM

Mayr believed that the division between typological thinking and population thinking rested primarily on metaphysical assumptions about the nature of variation. For the typologist, there exist "a limited number of fixed, unchangeable 'ideas' underlying the observed variability" (Mayr 1959, 2). These ideas are what is truly real, while variation is ephemeral. For the population thinker, by contrast, unique individuals are the ontological bedrock, and any collective description of a group of individuals (e.g. a species) can only be a statistical summary of the properties of those individuals. For the population thinker, the type is ephemeral, and variation is what is real.

Typological thinking, Mayr claimed, was incompatible with accepting gradual evolution:

Since there is no gradation between types, gradual evolution is basically a logical impossibility for the typologist. Evolution, if it occurs at all, has to proceed in steps or jumps. (Mayr 1959, 2)

Accepting typological metaphysics precluded accepting gradual change between types. Because types constrain variation, only two possibilities are open to the typological thinker: (i) the complete absence of evolution beyond the boundaries set by the underlying types, or (ii) evolution by saltational (single step) changes from one type to another. Gradual evolution, understood by the typologist as the change from one type to another by the gradual accumulation of small, within-type variants, is impossible, since the role of the type is precisely to constrain such variants within fixed limits. For the population thinker, by contrast, no such obstacles impede the acceptance of Darwinian evolutionary theory. Because variation is fundamental and unconstrained by any underlying type, natural selection can accumulate small variations indefinitely.

For Mayr, typological thinking was not merely one path to anti-gradualism among many. It was *the* path, repeated throughout the history of evolutionary theorizing:

Indeed, all saltationists have been typologists, and most typologists have been saltationist of one sort or another. Genuine variation and gradual change are, of course, incompatible with the typological viewpoint. (Mayr 1960, 354)

Opposition to gradualism has indeed played an important role in the history of evolutionary thought and, as the next section shows, a form of localized anti-gradualism may play a role in explaining deep conservation. However, Mayr's attempt to explain this in terms of a single, sweeping mode of thought—"typological thinking"—was a failure (Witteveen 2015, 2016). This is well-exemplified by the notorious saltationist Otto Schindewolf (1993), certainly among those Mayr intended his account to capture. At first glance, Schindewolf seems like a paradigmatic typological thinker. He explicitly tied his anti-gradualism to an account of the nature of variation, and he argued at length for the

reality of types (Schindewolf 1993, 410–15). However, he explicitly disavowed idealist metaphysics, both in general and specifically as applied to types (Schindewolf 1993, 330, 410–15). Mayr’s account fails to fit even this seemingly easy case. In the end, perhaps the only important figure who answers to Mayr’s notion of typology is Louis Agassiz (Winsor 2006).

Even if Mayr’s attempt to explain all opposition to gradualism in one fell swoop was a failure, however, the basic project of searching for common themes underwriting anti-gradualism in its myriad incarnations is worthwhile. Valuable work has been done in this area by Elliott Sober (1980), who showed that at least one important source of anti-gradualism, which I call *natural state thinking*, can be made philosophically precise. We will see below that natural state thinking does not account for the role that anti-gradualism plays in evo-devo, but before the underpinnings of contemporary anti-gradualism can be disentangled, I must show that such a thing as (respectable) contemporary anti-gradualism exists at all. To that task, I now turn.

3.3 EXPLAINING DEEP CONSERVATION

Deep conservation sets Darwinian evolutionary theory a difficult task: explaining why certain features of organisms have failed to evolve, despite the passage of extraordinarily long periods of time. This challenge is especially difficult if one accepts, as neo-Darwinian

evolutionary theory does, that genetic and phenotypic variation is widely available and that selection pressures are environment-relative (Rieppel 2010). The difficulty becomes even more acute when we realize that we are trying to explain a phenomenon that involves multiple independent lineages in terms of causes (mutation and selection) that operate strictly within individual populations.

To put these abstract points into a concrete context, consider the gene regulatory network that underlies endomesoderm specification in echinoderms. A crucial portion of this network, involving positive feedback between five genes, is identical in both sea urchins and starfish, which diverged over 500 million years ago (Davidson and Erwin 2006). Over those hundreds of millions of years, sea urchin and starfish populations have been exposed to a wide variety of environments, and they have almost certainly confronted these environments with a range of variations in the structure of this network. Moreover, since their divergence, they have been evolving independently, exposed to different selection pressures (and confronting them with different variations) both diachronically (within individual lineages over time) and synchronically (across contemporary populations). Yet, for some reason, none of these variations persisted to the present day. Nor is this an isolated case: major features of animal body plans have been conserved since the Cambrian radiation (Erwin and Valentine 2013).

Eric Davidson (a developmental geneticist) and Douglas Erwin (a paleontologist) have offered an explanation in terms of the genetic underpinnings of development (Davidson and Erwin 2006; Erwin 2015). Anti-gradualism plays a crucial role in this explanation. In subsequent sections, I will draw out the underlying pattern of thought

involved in this explanation, but for now I simply wish to present it with as little philosophical baggage as possible.

According to gene regulatory network (GRN) theory, the orderly progress of animal development (from a single cell to a fully differentiated organism) depends on the action of hierarchically structured GRNs. These networks precisely control the spatiotemporal expression of genes in the developing organism, allowing the embryo to differentiate. This process involves different types of developmental tasks, and one of the important results of GRN theory is that similar tasks tend to be accomplished by the activity of similarly structured GRN subcircuits (Peter and Davidson 2015, chap. 6). For instance, in order to develop a body part, an embryo must (a) specify the domain in which that part will develop, (b) determine the morphological structure of the part, and (c) actually build the part. Different GRN subcircuits underlies each task (Peter and Davidson 2011).

Davidson and Erwin's explanation of deep conservation concerns the first of these tasks: domain-specification. They argue that deep conservation of body plan features is the result of deep conservation of GRN subcircuits, called "kernels," essential for domain-specification. Kernels have four key properties (Davidson and Erwin 2006, 796):

1. They "execute the developmental patterning functions required to specify the spatial domain of an embryo in which a given body part will form." Accordingly, they are high in the GRN hierarchy, i.e. the network downstream of a kernel is much larger than the upstream network.
2. They are dedicated to serving that function alone; they are not reused elsewhere in development.

3. They are recursively wired (i.e. involve a great deal of positive feedback), with the result that “the products of multiple regulatory genes of the kernel are required for function of each of the participating cis-regulatory modules of the kernel.”
4. Because of this recursive wiring, “interference with the expression of any one kernel gene will destroy kernel function altogether,” resulting in the complete failure to develop the body part in question.

Together, these four properties of kernels can be used to explain a fifth feature: they are deeply conserved. Davidson and Erwin (2006) offer two examples of kernels: one for endoderm specification in echinoderms, and one for heart-field specification conserved between *Drosophila* and vertebrates (see Peter and Davidson 2015, chap. 7 for more examples). Both of these kernels originated when Bilateria first diversified and have been conserved since (>500 million years). What explains this? Davidson and Erwin (2006, 799) write:

We think that change in [kernels] is prohibited on pain of developmental catastrophe, both because of their internal recursive wiring and because of their roles high in the developmental hierarchy.

Because kernels are recursively wired, each gene in the kernel is essential to its function. Eliminating any one gene disrupts the functioning of the entire kernel. Because kernels are high in the developmental GRN hierarchy, such disruption has substantial downstream consequences. Disrupting the heart progenitor field specification kernel, for instance, prevents the development of a heart. Thus, any change to the linkage in a kernel

is likely to have catastrophic phenotypic effects.¹⁹ This generates strong selection against mutations that cause such changes – heartless animals don’t leave many offspring. That leads to the deep conservation of kernels, and thus to the deep conservation of those body plan elements that depend on them.

Three features of this explanation of deep conservation deserve mention. First, it is not an explanation of the origin of GRN kernels. It is an explanation of the conservation of GRN kernels that have already originated. Davidson and Erwin have little to say about the formation of kernels. They simply assume that they are produced by gradual, Darwinian evolution of a mundane sort (Davidson and Erwin 2006, 799). As such, their explanation is explicitly not saltationist (Erwin and Davidson 2006; contra Coyne 2006).

Second, a crucial premise of their argument is anti-gradualist, albeit in a local and not a global sense. They claim that there is no possibility of gradual phenotypic evolution through the modification of GRN kernels, because no changes to the linkages of GRN kernels produce small phenotypic effects. GRN kernels can either evolve by saltation or not at all. Since large phenotypic changes tend to be detrimental, the result is that GRN kernels, once formed, do not subsequently change. This is not a rejection of gradual evolution altogether – as we have just seen, they allow for the gradual origin of kernels – but

¹⁹ In fact, there are some differences between the heart-field specification kernel in *Drosophila* and mouse. These changes are the exceptions that prove the rule, however. Each of the changes is of the same type: what is a direct linkage between two genes in one species is an indirect linkage in the other. For instance, in mouse, *Gata4* activates *Nkx2.5* directly, whereas in *Drosophila* *Pnr* (*Gata4* homolog) activates *Mid*, which activates *Tin* (*Nkx2.5* homolog). The regulatory result is the same: gene A activates gene B. The only difference concerns the exact route by which this result is achieved.

an argument that a particular type of GRN subcircuit cannot evolve gradually once formed. It is precisely localized anti-gradualism, not global anti-gradualism.

Third, the anti-gradualism involved in their explanation of deep conservation is not absolute. GRN kernels represent one extreme in a graded hierarchy of variability and conservation. Davidson and Erwin (2006, fig. 3) suggest a general picture according to which different levels of the GRN hierarchy tend to correspond (roughly) to features conserved at different levels of the Linnean hierarchy: kernels underlie characters conserved at or above the phylum level, while differences at the periphery of the network tend to underlie differences between species. Intermediate portions of the GRN hierarchy are conserved at intermediate levels (this picture is developed further in Peter and Davidson 2011, 2015). Thus, while anti-gradualism plays a crucial role in explaining deep conservation, it is a part of a broader explanatory strategy for explaining why some parts of the genome evolve more readily than others.

3.4 TWO PATHS TO ANTI-GRADUALISM

Given that contemporary evo-devo explains deep conservation through a localized form of anti-gradualism, we may ask whether this anti-gradualism is *sui generis* or whether it rests on any broader pattern of thought. Here, I distinguish two bases of anti-gradualism: *natural state thinking* and *Cuvieran functionalism*. In this manner, I replace Mayr's sweeping

unification of all forms of anti-gradualism with a piecemeal approach that nonetheless allows us to recognize shared patterns of thought in temporally separated biologists. I stress that these two options are non-exhaustive: neither, for instance, will fit the arch-saltationists Richard Goldschmidt and Otto Schindewolf. I select them because they are useful for understanding contemporary evo-devo: what it is, what it is not, and how it fits together with mainstream evolutionary theorizing.

3.4.1 Natural State Thinking

Elliott Sober, in an attempt to recover something of philosophical value from Mayr's diatribes against "typology" and "essentialism", identified a type of model that he called a *natural state model*. On such a model, one distinguishes "between the *natural state* of a kind of object" and those non-natural states "produced by subjecting the object to an *interfering force*" (Sober 1980, 360). Evolutionary versions of natural state models treat species as having natural states around which individuals vary. Imposing selection on a population of such individuals can draw the population away from its natural state, but, once the selection is removed, the population will revert to the natural state. Moreover, there is only so far that selection can modify the population away from its natural state: variation is constrained.

Two phenomena could be invoked to support such a view in the nineteenth century. First, it was well-known that one could rapidly produce large changes in domesticated species, but that such efforts could only proceed so far before hitting limits. Second, it

was equally well-known that, as soon as such selection was relaxed, domesticated species showed a marked tendency to revert to their wild forms. Together, these phenomena suggested that artificial selection was best understood as an interfering force that can modify species only to a limited extent.²⁰

Natural state thinking played an important role in early opposition to Darwin, as exemplified by Fleeming Jenkin's (1867) review of Darwin's *Origin*. Jenkin's review is most famous for raising the swamping problem that arises when natural selection is combined with blending inheritance. In fact, however, he raised the swamping problem only as a challenge to the possible role of large, single-step changes in Darwinian evolution. Jenkin's argument against the possibility of natural selection producing new species by accumulating small variations is rather different, and rests on his endorsement of a natural state model.

Jenkin argued that natural selection cannot accumulate small variations indefinitely because, as the experience of breeders shows, such accumulation soon hits limits beyond which it cannot proceed further. Thus it appeared to Jenkin that there exist empirically determinable limits to the variability of species, and that these limits never exceed species boundaries. Domestication had never split one species into two. Species appeared to have an inherent tendency to stability. Jenkin captured this by describing variation within a species as contained within a sphere, with the average individual at the center.

²⁰ Today, we explain the limits to domestication by invoking standing genetic variation (once this is exhausted, selection becomes limited by the availability of new mutations), and we explain reversion as the result of the renewed operation of the same selection pressures that originally produced the wild form.

Individuals could vary in any direction, but were more likely to vary toward the center of the sphere. Artificial selection could push a population toward the edge of the sphere, but no further, and as soon as selection was relaxed, the population would start moving back toward the center. Importantly, this tendency to revert was not explained by the reimposition of selection pressures in a direction contrary to the artificial selection regime. It was explained entirely in terms of the variational properties of the species: variation predominantly trends toward the natural state.

As each species had its own natural state, speciation required that some individual be produced with a distinct natural state from the original species. This could not occur by the accumulation of small variants; it had to occur in a single step. Jenkin thus, on the basis of his natural state model, denied the possibility of gradualism. Speciation, if it occurred at all, must occur by saltation. Jenkin in fact allowed for such saltational evolution as a possibility, though he correctly noted that it was not Darwinian.

I've described this example at some length in order to make apparent just how distinct it is from the explanation of deep conservation considered in the previous section. That explanation does not treat GRN kernels as natural states. Genetic variants can arise anywhere in a GRN kernel, in any direction, and show no internally driven tendency to revert back to the conserved state. Nor are there any principled limits on how much a GRN kernel can vary. In principle, the DNA sequences on which a given kernel is based could accumulate point mutations indefinitely. What prevents them from doing so are strong selection pressures.

If, therefore, we are to find a more general pattern of thought underlying this particular explanation, we must look elsewhere than natural state thinking. This in no way undermines Sober's analysis of the role of natural state models in underwriting anti-gradualism. It just shows that Mayr's hope to find a single basis for all opposition to gradualism cannot succeed. There are multiple paths to anti-gradualism.

3.4.2 Cuvieran Functionalism

The work of Georges Cuvier illustrates a second path to anti-gradualism. Though Cuvier never published a detailed rebuttal of Lamarck's transmutationist theory, preferring instead to let it die of being ignored (Burkhardt 1977, 195–96), his work nonetheless provided a coherent rationale for rejecting Lamarck's theory. In reconstructing Cuvier's view, I draw on the work of E. S. Russell (1982, chap. 3), George Coleman (1964), and Richard Burkhardt (1977, 191–201). Coleman's account is especially interesting, since he explicitly argues that Cuvier was "the high priest of typology" and that this underwrote his opposition to transmutation (Coleman 1964, 98). His own excavation of Cuvier's reasoning, however, shows that Cuvier's opposition rested on a basis quite distinct from Platonic metaphysics.

At the heart of Cuvier's thought was his principle of the "conditions of existence." This principle began from the truism that viable organisms must be constructed in such a way that they can survive in the environment(s) in which they are found. For Cuvier, this notion had both an internal and an external face (Coleman 1964, 68; Burkhardt 1977,

194). Internally, the parts of an organism must be functionally integrated: they must work together to form a coherent whole. Externally, the particular form this functional integration takes must be suited to a particular environment.

E. S. Russell (1982, 34) has argued that the internal face is the more important of the two in Cuvier's thought, and that "the idea of the external conditions of existence, the environment, enters very little into his thought." It was the emphasis on internal functional integration, for instance, that was the root of Cuvier's important notions of the correlation and subordination of parts (Coleman 1964, 67). I shall therefore follow Russell (1982, 238–40) in treating emphasis on functional integration within the organism as the core of specifically *Cuvieran* functionalism, in contrast to *Darwinian* functionalism, which stresses adaptation to particular environments. I claim neither that they are incompatible nor that Cuvier and Darwin stressed one to the complete exclusion of the other. I merely claim that they are different, and that they need names.

Cuvier's functionalism underwrote his arguments (a) in favor of the view that every taxonomic group was a sharply defined, morphologically stable unit (Coleman 1964, 99) and (b) against transmutationist hypotheses. In particular, he thought that this necessity for functional integration limited variability. Central organs (e.g. the heart and nervous system) could not vary as much as less essential features (e.g. hair and color), as variations to these organs were more likely to upset the overall integration of the organism (Coleman 1964, 143). Furthermore, modification in any one part necessitated modifications in many others, again to preserve functional integration. Boundaries between taxonomic groups were sharply defined because the intermediates were not viable (Coleman

1964, 90; Burkhardt 1977, 193). The non-viability of intermediates equally ruled out the possibility of gradual transformation, which would require proceeding through such intermediate stages.²¹

The line of reasoning summarized above can be condensed into a three-step argument against the possibility of gradual evolution. First, organisms must be functionally integrated in order to survive (principle of the conditions of existence). Second, this need for functional integration makes intermediate forms non-viable. Third, the non-viability of intermediate forms precludes the possibility of gradual transformation, and so explains the existence of sharp boundaries between taxonomic groups. Note that no metaphysical commitment to the underlying reality of types is presupposed here. It is not the invariability of types (i.e. group characters) that explains the impossibility of transmutation. Rather, the invariability of types and the impossibility of transmutation receive a common explanation in terms of the non-viability of intermediates.

It is also worth noting that this Cuvieran functionalist argument against the possibility of transmutation does not rest on beliefs about the nature of variation.²² It rests entirely on the non-viability of intermediates: if intermediates are not viable, gradual evolution

²¹ Cuvier also offered empirical evidence against transmutation, including: (i) that Egyptian mummified animals showed that animal forms remained stable over long periods of time (Burkhardt 1977, 149); (ii) that species whose range included multiple distinct environments showed that animal form remains stable in different environments (Coleman 1964, 145); and (iii) that the fossil record shows a lack of the intermediate forms a transmutationist theory would predict (Coleman 1964, 150). None of these arguments, however, turn on his functionalist commitments.

²² To look ahead, this is in keeping with the analysis of structuralism and functionalism developed in chapter five.

cannot bridge the gaps between natural groups. This says nothing about whether or not such variants can or do arise.

Cuvieran functionalism therefore offers a path to anti-gradualism that departs quite radically from Mayr's account of typological thinking, despite Coleman's (1964, 98) diagnosis of Cuvier as "the high priest of typology." Mayr's notion of typological thinking implied a close connection between anti-gradualism and the metaphysics of variation. This connection is maintained in Sober's account of natural state thinking, but is lost in my reconstruction of Cuvieran functionalism.

We can therefore see that Cuvier's three-step argument against transmutation, based on his functionalist commitments, provides a second path to anti-gradualism, distinct from the path relying on natural state thinking.²³ In the next section, we will see that it plays an important role in the explanation of deep conservation.

3.5 CUVIERAN FUNCTIONALISM TODAY

The explanation of deep conservation presented above (Section 3.3) repeats Cuvier's three-step argument point-for-point, albeit in a quite different argumentative context. In

²³ Though distinct, the two are compatible, as is exemplified by St. George Jackson Mivart's *On the Genesis of Species* (Mivart 2009). In that work, he both reiterated Jenkin's arguments based on natural state thinking (Mivart 2009, 118) and challenged the ability of Darwin's theory to account for "the infinitesimal commencement of structures" on Cuvieran functionalist grounds (Mivart 2009, 23, 52).

particular, where Cuvier applied his functionalist argument globally, considering the functional integration of the entire organism, Davidson and Erwin apply the argument in a locally restricted setting: they apply it to particular GRN subcircuits.

One of the key features of GRN kernels is their recursive wiring: kernels are characterized by extensive positive feedback. This allows them to play a crucial developmental role: they can convert transient inputs into a stably maintained regulatory state (Wagner 2014, chap. 3; Peter and Davidson 2015, chap. 6). Because they are functionally integrated to such a degree, loss of a single gene in the kernel can destroy kernel function entirely, resulting in failure to develop the body parts under their developmental control (Davidson and Erwin 2006). Combined with the importance of these parts to the organism, this makes gradual evolution of kernels extremely difficult.

In this argument, we can see each step of Cuvier's three-part argument. Davidson and Erwin begin with an emphasis on the recursive wiring of special GRN subcircuits (step 1: functional integration). Note that functional integration here is inward-looking. If the wiring of kernels is disrupted, then they fail to serve their developmental function and so fail to produce a viable offspring. This non-viability is largely environment-independent. A fly without a heart will not survive in any naturally occurring environment. Nor will a sea urchin that fails to properly develop its endomesodermal layer in early development. This leads to the conclusion that small changes to the linkages in these subcircuits will lead to developmental catastrophe, not in some particular environment, but in any environment the organism is likely to encounter (step 2: the non-viability of

intermediates). These subcircuits therefore cannot evolve in a gradual manner (step 3: anti-gradualism).

A word is required about what it means for kernels to evolve gradually. Gradual change is most readily understood at the phenotypic level, where it implies that large phenotypic changes are produced by the continuous accumulation of smaller such changes. As all genetic changes are discontinuous, the notion of gradual evolution is effectively meaningless at the genetic level. Though changes to GRN structure are technically phenotypic changes, as they are the consequences of (rather than identical to) changes in DNA sequences, the best available models of GRN structure are nonetheless discrete. Either a gene activates/represses another, or it does not, and so a linkage is either present in a GRN, or it is absent (Peter and Davidson 2015, chap. 1). In this regard, GRN evolution, like genome evolution, is essentially discontinuous, and the idea of gradual evolution does not straightforwardly apply. Provisionally, however, we might understand gradualism at the GRN level as involving the change of a single regulatory linkage at a time, in contrast to large-scale rewiring in a single step.

Understanding GRN-level gradualism in this way, we can expand on the sense in which Davidson and Erwin argue for the non-viability of intermediates as applied to kernels. The recursive wiring of kernels makes it difficult for them to evolve gradually, i.e. one linkage at a time, once they are established. Any one change would require compensatory changes to preserve the kernel's function. This is a GRN-level analog of Cuvier's view that single parts cannot change independently: change to any one part requires multiple coordinated changes. As acknowledged above (footnote 2), some changes to kernel

structure are possible, but they are of a special type: they convert a direct linkage to an indirect linkage.²⁴ Thus they preserve kernel structure, considered at the appropriate level of abstraction, and are therefore the exceptions that prove the rule. Since no mechanism is known that could simultaneously produce coordinated changes to multiple linkages, saltatory evolution of kernels is off the table. The only remaining option is non-evolution: once formed, kernels cease evolving.

As my aim in this chapter is ultimately to show how evo-devo fits into the broader landscape of evolutionary theorizing, I have focused on a developmental genetic explanation of deep conservation. Now that the Cuvieran functionalist argument pattern is on the table, however, we can see that it is in fact applied rather widely. In particular, it is the argument pattern that underlies Wimsatt's notion of generative entrenchment (Schank and Wimsatt 1986; Wimsatt 2015). Generative entrenchment is a relational property of entities: the more the activity or structure of a complex system depends on the presence of a given entity, the greater that entity's generative entrenchment, and the stronger the constraints it imposes on the evolutionary process. The emphasis is on diachronic dependencies: traits are generatively entrenched "to the degree that they have a number of later developing traits depending on them" (Wimsatt 1986, 198). Cuvieran functionalism, by contrast, also covers synchronic dependencies (cf. Wagner and Schwenk 2000; Schwenk and Wagner 2001). Models of generative entrenchment thus provide one way of locally applying Cuvieran functionalist reasoning: they show that

²⁴ Or vice versa. I do not know if the polarity of the changes between the *Drosophila* and vertebrate heart kernels has been determined.

gradual evolution is difficult in highly entrenched parts of an organism, and thus explain why evolutionary change is displaced onto other parts of an organism.

Mayr's notion of typological thinking was meant to capture a pattern of thought inherently at odds with Darwinian evolutionary theory – a pattern of thought he believed was pervasive enough to explain centuries' worth of opposition to gradual evolution. We've seen that at least two distinct patterns of thought can generate anti-gradualism: natural state thinking and Cuvieran functionalism. We know that the former is incompatible with Darwinian evolutionary theory (Sober 1980). The question is whether the latter fares any better. If it does not, the problems are more extensive than the loss of a particular explanation of deep conservation.

3.6 INTEGRATING DEVELOPMENTAL GENETICS INTO EVOLUTIONARY THEORY

As we saw above (Section 3.3), neo-Darwinian evolutionary theory has difficulty explaining cases of deep conservation. Olivier Rieppel (2010, 668; cf. Wake, Roth, and Wake 1983) expresses the tension as follows:

Stable developmental stages preserved through geologic time create a puzzle for Darwinian evolutionary theory, based as it is on fundamental variation, natural selection, and continuity of transformation.

If small variants are produced for all parts of the organism, and if natural selection accumulates small variations over time, how can anything remain constant over long

periods of time? Selection can favor phenotypes optimized for a particular environment, but body plans are conserved across numerous environments. The difficulty lies in explaining something stable across many environments and populations (body plans) in terms of a cause that's relative to a particular environment and population (mutations and selection pressures).

In addressing this issue, caution is required. Darwin's evolutionary theory is not identical to the neo-Darwinian theorizing of the modern synthesis. The synthesis involved conceptual innovations – most crucially in this context, the genotype-phenotype distinction – that help to resolve the tension Rieppel diagnoses. In particular, the genotype-phenotype distinction allowed biologists to reconceive each of Rieppel's three bases of Darwinism (variation, selection, and continuity). The explanation of deep conservation provided by contemporary developmental genetics depends essentially on these reconceptions.

Evolutionary change, as understood during the modern synthesis, is first of all genotypic change: "a change in the genetic composition of populations," as Dobzhansky (1982, 11-12) put it. This can be connected to phenotypic change by studying genotype-phenotype correlations (transmission genetics) and the causal processes that produce these correlations (developmental genetics). Developmental genetics thus has a role to play in explaining how the population genetic processes that alter the genetic composition of populations manifest in evolution of the phenotype.²⁵

²⁵ This picture is defended in chapter two.

The synthesis was Darwinian in emphasizing variability, selection, and continuity, but it understood each of these against the background of the genotype-phenotype distinction. Individual variability, i.e. the variations that randomly arise in particular organisms, is first and foremost genetic, though genetic variants may have phenotypic consequences. Selection concerns both genotype and phenotype. On the level of phenotype, selection may be understood both in terms of the causal interactions between organisms and their environments that determine reproductive success. On the level of genotype, it may be understood as the genetic response to phenotype-level selection (Endler 1986, chap. 1).

Finally, continuity concerns the phenotype. The synthesis insisted that alleles with large phenotypic effects were usually disadvantageous, and thus played only a minor role in evolutionary change. Alleles with small effect sizes were the basic matter of evolutionary change, with the result that the phenotypic would evolve gradually (though possibly rapidly; see Simpson 1982). At the genetic level, there is no question of continuity. All genetic change is discontinuous. Even if some genetic changes (e.g. point mutations) can be semi-arbitrarily considered “smaller” than others (e.g. large deletions), such “small” genetic changes are not more likely to have small phenotypic effects than are “large” genetic changes. Thus, even if a measure of genetic continuity can be given, there is no reason to think that selection will favor genetic continuity in the way it favors phenotypic continuity (Wright 1963, 366).

The crucial takeaway from all this is that the synthesis, in basing itself on Mendelian genetics and the genotype-phenotype distinction, separated variability from continuity.

Variability is genetic (though it may have phenotypic effects), whereas continuity is phenotypic (with no application to the genotype). This separation eases the tension with deep conservation. The GRNs that pattern stable developmental stages are not any less variable, genetically, than any other region of the genome. However, genetically unremarkable changes affecting these GRNs have quite remarkable phenotypic effects: large effects that are negative in all environments (Davidson and Erwin 2006; Erwin 2015). By revealing the processes connecting genotype to phenotype, developmental genetics can explain how the fact that selection favors continuous phenotypic change insulates certain regions of the genome (and associated aspects of the phenotype) from evolutionary change over long stretches of time.

Thus, contemporary Cuvieran functionalism, so far from being in tension with the modern synthesis, rests precisely on the synthesis' commitment to genetic variability, selection, and phenotypic continuity. By themselves, these commitments do not furnish an explanation of deep conservation. To get such an explanation, the gap between genetic variability and phenotypic continuity – that is, the gap between genotype and phenotype must be bridged. That is what developmental genetics does: it shows how alterations to different regions of the genome have predictably different phenotypic effects. By applying Cuvier's anti-gradualist argument locally rather than globally, it complements the theorizing of the modern synthesis and augments its explanatory power.

3.7 DEFINITION VERSUS DIAGNOSIS

Perhaps because Mayr's notion of typological thinking was so nebulous, biologists and philosophers have offered quite varied criticisms of exactly what's wrong with it. In this and the following section, I consider two such criticisms, both connected to the issue of deep conservation, in order to show that neither challenges Cuvieran functionalism.

Davidson and his colleagues' account of the deep conservation of GRN kernels, and their more general picture of GRN evolution, helps to explain why body plans are important theoretical constructs in evo-devo (Raff 1996; Erwin and Valentine 2013). The very idea of a "body plan" has, however, been criticized as perniciously typological (Ghiselin 2005a; Jenner 2006). Here, I alleviate this worry.

Both Ghiselin and Jenner raise this worry in the context of the view that species are individuals rather than classes. This is not the place to re-litigate that long-standing debate. It is enough to recognize that lineages are defined by shared ancestry. An ancestral species plus all of its descendants constitutes a lineage. No amount of subsequent evolutionary change can remove a particular species from its ancestral lineage (Jenner 2006, 387). If a future vertebrate lost every feature that characterizes the vertebrate body plan, it would still belong to the vertebrate lineage, i.e. it would still be a vertebrate. No genetic or phenotypic trait is essential for a given species to belong to a given lineage. Only descent matters. A consequence of this is that characters can only ever be diagnostic of systematic groups. They can never be definitive.

Both Ghiselin and Jenner worry that evo-devo's notion of body plans falls afoul of this important point. For instance, Ghiselin (2005a, 132–33), critiquing Stephen Jay Gould, writes:

He tried to make essences be not transcendent, but immanent, by incarnating them in the genome. In effect, he confused what are often called “conservative characters” or ones that evolve very slowly and perhaps for a while not at all, with ones that are unrestricted with respect to space or time.

Davidson and his colleagues also make “essences” (by which Ghiselin here means body plans) immanent in the genome (discussed further below, Section 3.8). The worry is that, in doing so, they treat them as features definitive of classes, about which laws of nature (or something like laws of nature) can be formulated (e.g. a claim about the evolutionary possibilities of “the tetrapod limb”).

This is a misplaced worry, however. Not only do Davidson and his colleagues not erroneously treat kernels as definitive of taxonomic groups, their position would hardly be sensible if they did. They are, after all, trying to explain the deep conservation of body plans. They do this by showing that the developmental genetic bases of body plan elements are themselves deeply conserved. The problem thus changes to explaining the conservation of the relevant GRN subcircuits. Crucially, these regions of the developmental GRN are no less susceptible to mutation than any other genomic region. Lineages containing these GRNs will continuously produce individuals that lack the supposedly “essential” kernel. Evolutionarily speaking, the regions of the genome that underlie GRN kernels *might* change. That is precisely why it's interesting that they *don't*.²⁶ Thus, were

²⁶ See also Wagner and Schwenk (2000, 188), who are very clear that evolutionarily stable configurations (ESCs) – whose stability they explain on Cuvieran functionalist grounds –

Davidson and Erwin truly typologists in the way that worries Ghiselin and Jenner, their very explanandum would disappear.

Davidson and Erwin take on board the insight that lineages are determined by shared ancestry and no more. Even a GRN kernel is merely diagnostic of a particular taxon, not definitive. At the same time, so long as those regions of the GRN are conserved, general claims can be made about the evolutionary possibilities of the body plan elements that depend on them. These claims are not spatiotemporally unrestricted: they extend (in space and time) exactly as far as conservation extends.

3.8 BODY PLANS AND POPULATIONS

Ron Amundson (2005) raises a quite distinct worry about the relationship between evo-devo, with its emphasis on deep conservation, and synthesis-derived evolutionary theorizing. Though he brings together numerous diverse threads in making this case, to which I can't do justice here, the culmination of his argument posits an ultimately simple basis for the continuing tensions between the fields. Mainstream, neo-Darwinian evolutionary theorizing is committed to something Amundson (2005, chap. 11) calls *exclusive population thinking* (described below). According to Amundson, evo-devo's reliance on the notion

are representative but not definitive of monophyletic clades: "Because both origin and escape from an ESC are relatively rare, ESC character states should be representative of a higher clade or significant parts of it."

of animal body plans as causal factors in evolution (which Amundson diagnoses as typological) falls afoul of exclusive population thinking.

I have argued that Cuvieran functionalism underwrites evo-devo's appeals to conserved body plans by explaining their persistence over deep evolutionary time. If Cuvieran functionalism can be shown to be compatible with exclusive population thinking, then the tension Amundson details can be dissolved. Here, I make the case that Cuvieran functionalism is compatible with any defensible interpretation of exclusive population thinking.

Exclusive population thinking, for Amundson, is the view that all evolutionarily relevant causes must operate within and not across populations. Amundson (2005, 256) writes:

Let us suppose that exclusive population thinking is generally accepted by neo-Darwinians. In this view, adaptive radiation is the way of evolution. Once speciation occurs, no causal force can unify distinct populations. [...] As long as development is conceived as a unified process that is shared among reproductively isolated groups, it is irrelevant to selection within populations. Therefore it is irrelevant to evolution. As long as evo-devo involves developmental types, it is perniciously typological. From this perspective, the only way for evo-devo to form a synthesis with neo-Darwinism is for evo-devo to [...] relinquish entirely the view that ontogeny is a thing that can be shared.

Amundson characterizes the conflict between population thinking and typological thinking as a disagreement over whether anything can unify the evolution of distinct lineages. Exclusive population thinking takes the negative position: once one lineage diverges into two daughter lineages, the daughter lineages follow independent evolutionary courses. As Reeve and Sherman (1993) put it, the ancestor does not reach up through time to clutch its descendants by the throat. Historically conserved characters are a mere

residue that cannot constrain further change. On this view, evo-devo's invocation of conserved body plans as causal factors in evolution is misguided: it treats body plans as more than the mere residue of history.

Amundson considers two possibilities for harmonizing evo-devo and mainstream (for Amundson, "neo-Darwinian") evolutionary theorizing. Either evo-devo can give up its reliance on the notion of conserved body plans as causal factors in evolution, or neo-Darwinian theorists can give up their commitment to exclusive population thinking. Neither option is especially attractive. There is something basically right in the idea that populations, once they have diverged, evolve independently. And yet the contemporary developmental genetic explanation of deep conservation of body plan elements is central to evo-devo, despite spanning lineages that diverged hundreds of millions of years ago. Both are crucial to contemporary evolutionary theorizing, so any attempt at harmonization that favors one at the expense of the other is *ipso facto* inadequate.

Happily, however, the conflict that Amundson diagnoses only exists if one adopts an overly restrictive interpretation of exclusive population thinking, one that is routinely and justifiably violated by evolutionary explanations offered outside of evo-devo. There is a less restrictive version of exclusive population thinking justified by the successes of mainstream evolutionary theorizing, but this interpretation is compatible with evo-devo's Cuvierian functionalism.

What does it mean to say that lineages evolve independently, not unified by any causal force? Lineages diverge when gene flow between two populations is eliminated. Once gene flow is eliminated, alleles in one population no longer compete with alleles in

the other. Variations that arise in one population are limited to that population and cannot spread to the other. No population genetic cause unifies the population: selection, drift, gene flow, mutation, etc. are all localized within a single population. And since population genetic processes drive evolutionary change, that means that lineages evolve independently once diverged.

The more restrictive interpretation of exclusive population thinking claims that, because distinct lineages evolve independently in this way, the evolution of each individual lineage must be explained independently. It amounts to a denial of the possibility of general explanations in evolution. Each lineage has a unique history, and that history must be explained in terms of the particular evolutionary forces acting on that lineage. Gene flow is the only causal force that binds lineages together; once it is cut off, they go their separate ways.

Such an interpretation certainly excludes evo-devo's Cuvieran functionalist explanation of deep conservation, but it excludes much else besides. For example, why do many bird and mammal lineages tend to be larger in cooler climates (Bergmann's rule; Meiri and Dayan 2003)? And why does this trend fail in other lineages, such as amphibians and insects (Adams and Church 2008; Shelomi 2012)? If explanations that transcend single populations are forbidden, then each instance of this trend must be explained independently. It's reasonable to ask, however, for a more general explanation, one in terms of shared features of the groups that experience the trend leading those groups to experience similar selection pressures in similar environments. In the case of Bergmann's rule, the standard explanation is in terms of the need for endotherms (such as birds and

mammals) to minimize heat loss in colder environments (Walters and Hassall 2006). This explains why some (but not all) independent lineages face similar selection pressures and so show similar evolutionary trajectories. Though the lineages experience these causes independently, we can nonetheless offer a unified explanation of their evolutionary trajectory because the same causal factor is repeated in multiple lineages.

The strong interpretation of exclusive population thinking is therefore untenable. Exclusive population thinking should require only that causes cannot transcend populations – that is, that independently evolving lineages should experience and respond to evolutionary causes independently. But it should not require that explanations cannot transcend populations, because similar causes can affect independent populations in similar ways. Once we see this, however, we can see that Cuvieran functionalism underwrites appeals to body plans in a way that is compatible with this more defensible understanding of exclusive population thinking. According to Davidson and Erwin, what underlies conserved body plan elements are conserved GRNs. These are conserved because changes to their structure are rarely if ever viable. Their functional integration gives rise, in every population in which they are present, to the same, strong selection pressures. Thus, though they can change, they do not (Section 3.7). So far, no cause that unifies separated populations is required. All that's needed is that similar selection pressures arise independently in multiple lineages. There is no mystery in that.

Once it is allowed that key portions of the regulatory networks underlying the development of conserved body plan elements are themselves conserved, the puzzle about how conserved body plan elements can unify the evolution of distinct populations

vanishes. If changes that affect certain regions of the GRN are routinely selected against, then evolutionary divergence in the relevant characters must occur via changes at other regions of the GRN. The conservation of some GRN regions can thus impose constraints on the evolution of those characters. Since these conserved regions of the GRN are present in each descendant lineage,²⁷ they can impose the same constraints on each population (cf. Arthur 2001, 276). This is no more mysterious than the explanation of trends in body size evolution: in both cases, similar causes affect independent lineages, allowing for a unified explanation of their evolutionary trajectories.²⁸

In this manner, developmental types are able to unify separate populations in a manner compatible with exclusive population thinking, once that is itself interpreted in a manner compatible with the practice of neo-Darwinian evolutionary theorizing. No “spooky” causal influence of the ancestor on its descendants is needed. All that is required is the repeated presence, in diverged lineages, of the same underlying GRNs. The ancestor doesn’t clutch its descendants by the throat. It simply bequeaths to them, with severe selection pressures as its faithful executor, the regulatory architecture that constrains their evolution.

²⁷ This is only strictly true when there is perfect conservation, as appears to be the case with GRN kernels. Other characters may, during rare evolutionary events, change, as in the transition from the teleost fin to the tetrapod limb (for further discussion of such issues, see Wagner 2014, chaps. 1–6).

²⁸ David Stern (2011, chap. 8) explicitly integrates Cuvierian functionalist reasoning with population genetics when he argues that network-disrupting mutations are more likely to be fixed in small populations than in large populations.

3.9 UNIVERSALITY AND DIVERSITY REVISITED

In this chapter, I have attempted to elucidate the role of anti-gradualist arguments in contemporary developmental genetic explanations of deep conservation, and to show how these arguments can be integrated into evolutionary theorizing without falling afoul of classic objections to “typological” thinking. In doing so, I have defended the following claims:

- There exist at least two distinct paths to anti-gradualism – Cuvieran functionalism and natural state thinking – and that these paths cannot be unified under any more general heading, such as Mayr’s notion of “typological thinking.” (Section 3.4)
- Contemporary developmental genetics draws on localized applications of Cuvieran functionalist reasoning to explain deep conservation (Section 3.3, Subsection 3.4.2, Section 3.5). It does not draw on natural state thinking (Subsection 3.4.1).
- By relying on neo-Darwinian interpretations of the nature of variation, selection, and continuity, Cuvieran functionalist reasoning in evo-devo complements neo-Darwinian theorizing while expanding its explanatory power (Section 3.6).
- Cuvieran functionalist reasoning in evo-devo does not fall afoul of the errors with which typological thinking has been charged. It does not treat conserved

characters as definitional of taxonomic groups; it treats them merely as diagnostic (Section 3.7). Moreover, it is compatible with any defensible understanding of exclusive population thinking (Section 3.8).

In light of this, it is worth revisiting Raff's (1996, chap. 1) claim that evo-devo differs from synthesis-derived evolutionary theorizing in emphasizing universality rather than diversity. This is often taken to support the view that evo-devo is a structuralist program in biology, in contrast to the functionalism of neo-Darwinian theorizing. This view structures Amundson's (2005) book-length treatment of the issue, and it has been endorsed by various evo-devo biologists (e.g., Alberch 1989; Wagner 2014). This is not the place for a full treatment of structure-function disputes (see chapter five), but, roughly, structuralists see internal and/or ahistorical causal factors as the primary drivers shaping organismal form, while functionalists see external, historical causal factors (e.g., selection) as the primary drivers.

There is truth to this narrative—evo-devo has, from its inception up to the present day, had a strong structuralist strain (e.g., Alberch 1989; Kauffman 1993; Müller 2003; Newman 2014b)—but, in light of what I've argued here, it's overly simplistic. If functionalism is conceived as including only Darwinian functionalism, with its emphasis on continuous variability and environment-driven selection pressures, and if structuralism is understood as the only alternative to functionalism, then evo-devo's emphasis on deep conservation can only seem structuralist.²⁹

²⁹ Alberch (1989, 25), for instance, seems to endorse something like these assumptions when he argues that functionalist approaches inherently treat nature as continuous.

But there's a third option: Cuvieran functionalism. Like Darwinian functionalism, Cuvieran functionalism (in its post-Darwinian forms) emphasizes the role of selection in driving evolutionary change (or evolutionary stasis). Like structuralism, Cuvieran functionalism emphasizes what is universal (evolutionarily conserved) over what is diverse (evolutionarily labile), and does so by focusing on the internal organization of the organism, not its relation to any particular environment. While it's true that evo-devo has always had a sizable structuralist contingent, evo-devo, especially those regions of evo-devo centered on developmental genetics, also has a strong Cuvieran functionalist contingent (e.g. Carroll 2008; Wagner 2014; Peter and Davidson 2015), and structure-function disputes arise within evo-devo.³⁰

In order, therefore, to understand both the internal structure of evo-devo and the place of evo-devo within the broader landscape of evolutionary theorizing, we must recognize Cuvieran functionalism as an alternative to both structuralism and Darwinian functionalism.

³⁰ For instance, Stuart Newman and Ramray Bhat (2008, 2), representing the structuralist approach to evo-devo, refer to Sean Carroll's approach as "Neo-Neo-Darwinian."

4.0 THE FINE STRUCTURE OF 'HOMOLOGY'

4.1 INTRODUCTION

The pectoral fin of a dugong, the forelimb of a mole, and the wing of a bat, though they do not appear especially similar and though they serve distinct functions (swimming, digging, and flying, respectively), are nonetheless the “same” part: they are all variations on the vertebrate limb (Owen 2007). They are all, as biologists put it, homologous.

Homology is among the most important and most controversial phenomena in biology: important because it is the “basis of comparative biology” (Hall 1994), controversial because biologists rely on multiple, possibly incompatible accounts of homology. In particular, there is a longstanding conflict between accounts of homology based on Darwinian evolutionary theory (e.g. Lankester 1870; Hennig 1966) and accounts of homology based on morphology and development (e.g. Owen 2007; Wagner 1989).

The relationship between these two types of account remains contentious. Genealogical accounts, which today are generally framed in terms of phylogenetic systematics (Wiley and Lieberman 2011), are dominant. The status of developmental accounts of homology is less clear. Some take the two types of account to stand in an antagonistic

relationship (Amundson 2005, pp. 238–44; Ramsey and Peterson 2012; Currie 2014), while others see them as merely different, useful for different purposes (Brigandt 2002; Jamniczky 2005; Griffiths 2007). If we accept the latter suggestion, then we need some picture of how the accounts relate. Not much has been written on this score (but see Brigandt 2007; Laublichler 2014).

This chapter presents a general framework showing how genealogical and developmental accounts of homology fit together. In this framework, both types of account capture aspects of homology that the other type cannot. Both types of account therefore work together to contribute to a full understanding of homology.

In rough outline, I argue that the two types of account relate as follows (Figure 2).³¹ Genealogical accounts rely on notions of ‘character’ and ‘descent’ that abstract away from the particular mechanisms by which characters are inherited. Thanks to this abstraction, phylogenetic systematics can incorporate data drawn from all kinds of biological characters (genes, body parts, behaviors, etc.). However, the genealogical account presupposes the existence of inherited units. These units are inherited in different ways. Considering these differences leads to the development of enriched accounts of homology that, though tied to the genealogical account, apply to more limited domains (e.g. just to genes, or just to body parts). Even as their domain is restricted, however, these accounts expand the reference of ‘homology’, for they include phenomena (e.g. paralogy, serial homology)

³¹ In Figure 2, large boxes represented accounts of homology, while small boxes represent sub-concepts.

that are excluded by a strict genealogical account. Developmental accounts of homology are best understood as enriched accounts that apply specifically to body part homology.

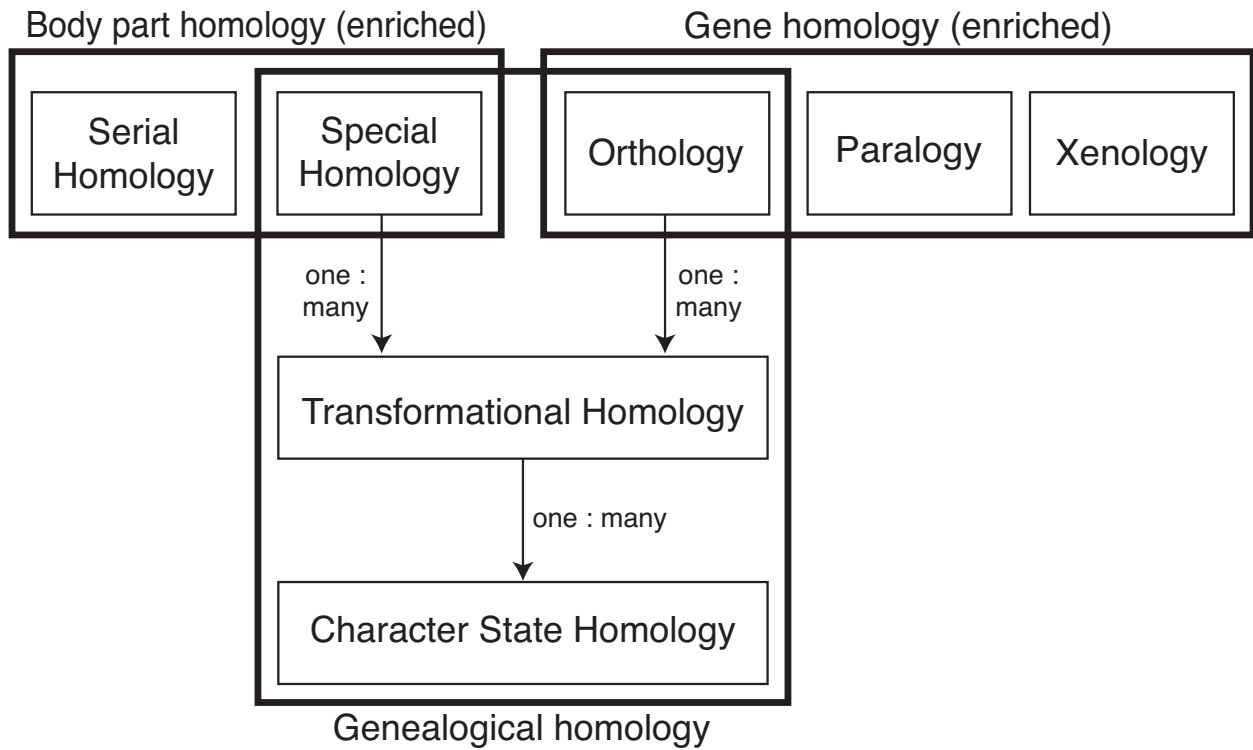


Figure 2. The fine structure of 'homology'.

There is thus a complex conceptual structure underlying the various uses of the term 'homology'. This structure consists of (1) an abstract, genealogical account that applies to all kinds of biological characters and (2) a set of locally enriched accounts that complete the genealogical account within a limited domain (e.g. body parts) while also including

additional phenomena (e.g. serial homology) not covered by the genealogical account. I aim to show that, once this fine structure is appreciated, the longstanding tensions between the two types of account vanish.³²

4.2 HOMOLOGY: THE PROBLEM

Any adequate account of homology must explain how the parts of organisms can be the same part, despite potentially great dissimilarity. This problem is captured by Owen's (1843, p. 379) classic definition of a homolog as "the same organ under every variety of form and function." This definition does not say in what sense homologs are "the same." The problem of homology might thus be put as follows: Owen's definition is correct, but what does it mean?

Here the disputes begin. Accounts of homology can be grouped into two main classes—genealogical and developmental—that provide different types of answers to this question. There are four key sources of tension between these two accounts.

Tension 1: what is the nature of homological sameness? Genealogical accounts explicate sameness in terms of shared descent: two parts are homologous if they derive from the same part in a common ancestor. Developmental accounts, by contrast, explicate

³² The view that 'homology' has a complex conceptual structure resonates with Mark Wilson's (1982, 1994, 2006, 2018) studies of conceptual behavior in classical mechanics and materials science.

sameness in terms of shared features of development. Günter Wagner's (1989, p. 1163) account of homology, for instance, treats two parts as homologous if they share "historically acquired and genetically regulated developmental constraints." This basic difference over the nature of homological sameness gives rise to three further sources of tension.

Tension 2: how much dissimilarity is permitted? Genealogical and developmental accounts of homology differ over the extent to which homologs can be dissimilar. Genealogical accounts explain homological sameness in terms of a shared origin. Because the origin is a fixed historical event, no amount of subsequent divergence can destroy the homology between two parts. Genealogical accounts thus allow for indefinite divergence. Developmental accounts do not. They explain sameness in terms of extant, causally active factors operative in development, and the requirement that these factors be conserved constrains the degree to which homologous parts can diverge.

Tension 3: how many types of homology? Genealogical and developmental accounts of homology disagree about whether serial homology (the same part repeated within a single individual) is genuine homology. Strictly speaking, genealogical accounts exclusively concern special homology (the same part in different species). Phylogenetic systematics ties homology to the topology of phylogenetic trees (see below, Section 4.3), and no account of serial homology emerges from this (Cracraft 2005; cf. Lankester 1870; De Beer 1971). Developmental accounts, by contrast, explicitly include serial homology (Wagner 2014, p. 418).

Tension 4: what is the proper target of homology assessments? Genealogical and developmental accounts of homology appear to disagree over what is properly homologized. Whereas developmental accounts homologize *characters* (e.g. the vertebrate forelimb, whatever form it takes), genealogical accounts homologize *character-states* (e.g. having a forelimb shaped like a fin) (Wagner 1989; Brigandt 2007). In this way, they disagree concerning the proper targets of homology assessment.

On account of these four sources of tension, a number of philosophers have taken genealogical and developmental accounts of homology to be in competition (Amundson 2005, pp. 238–44; Ramsey and Peterson 2012; Currie 2014). Others, however, have defended the compatibility of the two accounts, on the grounds that they are useful for different purposes (Brigandt 2002; Jamniczky 2005; Griffiths 2007). Roughly, the genealogical account is useful for reconstructing phylogenies, while developmental accounts are important for understanding the evolution of novel structures. Once the different purposes of these accounts are recognized, they can be seen as no longer fighting over the same ground. Among biologists, advocates of both compatibility (Panchen 1999, discussion; Wagner 2014) and conflict (Cracraft 2005) can be found.

This chapter sides with the compatibilist camp. It is not enough, however, to show that genealogical and developmental accounts serve different purposes, for that does not explain in what sense they are both accounts *of homology*. A viable compatibilist analysis of homology must both (a) make clear how the two types of account are related and (b) do so in a way that shows how to resolve the four sources of tension described above. In

what follows, I defend a framework for understanding homology that satisfies both desiderata.

One feature of this framework is that the scope of different accounts of homology varies not only in terms of the range of characters covered, but in terms of the range of taxa covered. Accordingly, the sections of this chapter vary in scope. The discussion of the genealogical account of homology (Section 4.3) covers all taxa, reflecting the unifying role of the genealogical account. The discussion of gene homology (Section 4.5) likewise applies to all taxa. By contrast, the discussion of body part homology (Section 4.6) covers only animal taxa, since that is the scope of the enriched account under discussion. The scope of the chapter as a whole, however, is general: it provides a framework for making sense of applications of ‘homology’ to all taxa and for all characters.

The easiest way to introduce this framework is by presenting a genealogical account of homology (Section 4.3), as the limitations of such an account reveal the need for enriched accounts of homology. After explaining the general features of enriched accounts (Section 4.4), I provide examples of two such enriched accounts: one for gene homology (Section 4.5) and one for body part homology (Section 4.6). I then return to a more abstract discussion of the nature of enriched accounts of homology (Section 4.7). With the framework in place, I show how it resolves the four sources of tension just described (Section 4.8), then compare my account to two other recent proposals (Section 4.9).

4.3 A GENEALOGICAL ACCOUNT OF HOMOMOLOGY

In this section, I present a genealogical account of homology, based on the methods and conceptual framework of phylogenetic systematics. This account unifies applications of 'homology' to all kinds of biological entities. It does so by relying on abstract notions of 'descent' and 'character'. Subsequent sections will show how making these abstract notions concrete by incorporating mechanistic details give rise to locally enriched accounts of homology.

According to the genealogical account of homology, homologs are characters or character states (the distinction is explained below) that descend from the same character (state) in a common ancestor. Character state homology is tied to the topology of phylogenetic trees, while homology of characters ("transformational homology") is importantly presupposed in the methodology of phylogenetic systematics. I take up these two types of genealogical homology in turn.

4.3.1 Homology of Character States

Phylogenetic systematics aims at reconstructing the phylogenetic relationships between taxa. Phylogenetic relationships are distinct from tokogenetic relationships (Figure 3; see Hennig 1966, pp. 29–32). Tokogenetic relationships hold between parents and offspring within an interbreeding population. When a single interbreeding population splits into two, this yields phylogenetic relationships between the ancestral and descendant

populations. Phylogenetic relationships produce a strict hierarchy representable in a phylogenetic tree; tokogenetic relationships do not.

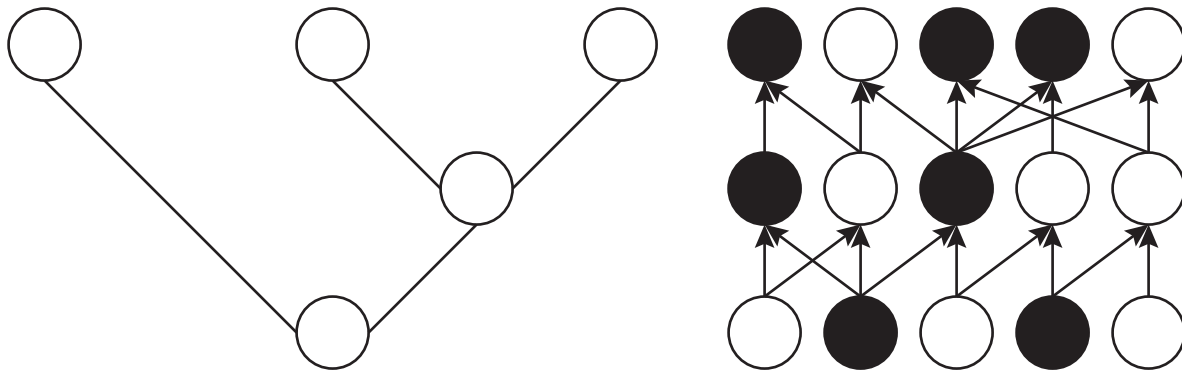


Figure 3. Phylogenetic relations (left) and tokogenetic relationships (right).

To reconstruct phylogenetic relationships systematists record the similarities and differences between a given set of taxa (Wiley and Lieberman 2011). Figure 4 shows a data matrix recording the similarities and differences of nine characters among four taxa. In the figure, the columns are taxa, while the rows are characters. Each character can occur in at least two character states. For example, “number of digits” might be a character, while “one” and “five” are possible states of that character. For simplicity, the characters in Figure 4 come in only two character states, coded as ‘0’ or ‘1’.

	Taxon A	Taxon B	Taxon C	Outgroup
Character 1	0	1	1	0
Character 2	1	0	0	0
Character 3	1	1	0	0
Character 4	1	1	0	0
Character 5	0	0	1	0
Character 6	0	0	0	1
Character 7	1	1	0	0
Character 8	1	1	1	0
Character 9	1	1	1	0

Figure 4. Hypothetical data matrix. Rows are characters; columns are taxa.

Next, the systematist searches for the phylogenetic tree that is best supported by the data (Figure 5). Inferring a phylogenetic tree from the data requires adopting a model of evolution, capturing the possible and probable transitions between states of a given character. For instance, for molecular data, the Jukes-Cantor model of DNA evolution assumes that all four bases of DNA occur with equal frequencies, and that the rate of transition is the same for all pairs of bases (Huson, Rupp, and Scornavacca 2010, pp. 29–31). For morphological data, a model might assume that loss of a complex character is more likely than gain. In generating Figure 5, I assumed that 0 is the ancestral state for each

character, that $0 \rightarrow 1$ is the only possible transformation for each character, and that otherwise all transitions are equally likely.

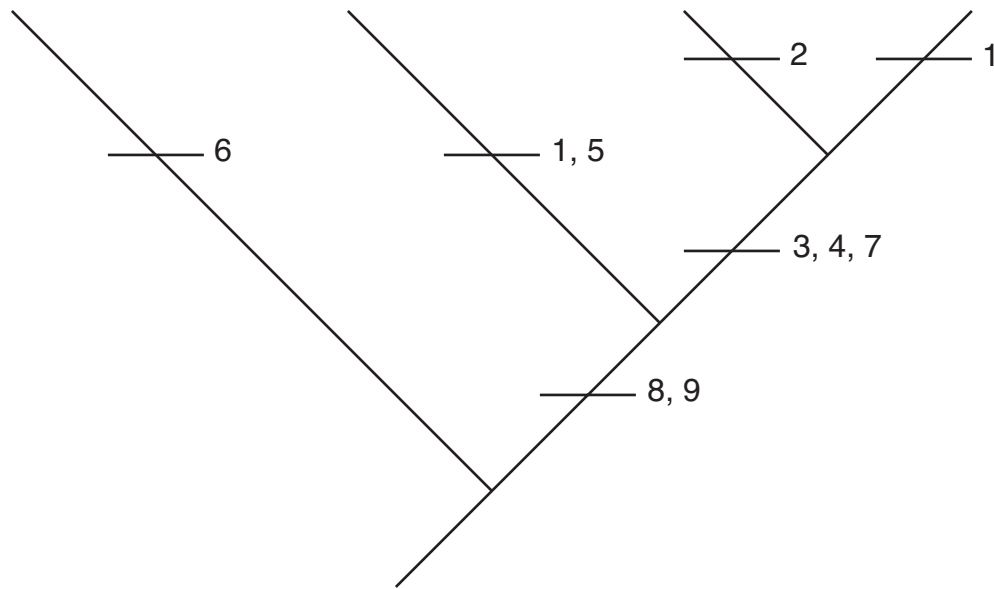


Figure 5. Phylogenetic tree produced from the data matrix in Figure 4.

For understanding character state homology, however, what matters is not the model used to infer the phylogenetic tree from the data, but rather the topology of the tree produced. Once a tree is inferred, homology relationships can be read off the tree directly. There are two ways in which character states can be homologous. Sympleisomorphies are shared ancestral character states, while synapomorphies are shared derived character

states. As both are shared due to common descent, both fall under a genealogical account of homology.

4.3.2 Transformational Homology

Character state homology does not exhaust the genealogical account of homology. To construct a data matrix, one must recognize a second kind of homology: transformational homology. Even to ask whether two shared character states are homologous or independently derived, they must be treated as states of a single character. Characters must be able to transform from one state to another (e.g. reduction in digit number during the evolution of horses). In this sense, characters form “transformation series” (Hennig 1966). Just as a data matrix encodes hypotheses of character state homology by giving two taxa the same value for some character, so it encodes hypotheses of transformational homology by placing the features of distinct taxa in the same row.

This point is independent of the sort of data one considers in phylogenetic analysis. In the morphological case, for instance, it would be incorrect to compare the coloration of bird wings to the coloration of butterfly wings, since the two groups evolved wings independently. Wing coloration thus does not form a transformation series in the two groups. In the molecular case, it is essential to compare base identity at homologous loci. This is the purpose of sequence alignment, a necessary stage in the phylogenetic analysis of molecular data (Huson, Rupp, and Scornavacca 2010, chap. 2).

Though the process of inferring a phylogenetic tree from the data evaluates the hypothesis that shared character states are shared due to common ancestry, it presupposes hypotheses of transformational homology. Hypotheses of transformational homology are thus “logically prior” to hypotheses of character state homology (Brower and Schawaroch 1996, p. 269).

Both transformational homology and character state homology are intelligible within and indeed necessary to a phylogenetic framework (Assis and Brigandt 2009, p. 251). The genealogical account of homology thus includes both.

4.3.3 ‘Descent’ and ‘Character’

The genealogical account of homology just described unifies applications of the term ‘homology’ to biological entities of all kinds. It is able to serve this unifying role because it relies on an abstract, purely formal understanding of both ‘descent’ and ‘character’. Anything can be a “character” (i.e. used in phylogenetic systematics), provided that it yields transformation series. Likewise, a phylogenetic descent relationship is simply any relationship that gives rise to phylogenetic patterns recoverable by phylogenetic analysis. Such patterns hold among biological taxa, but can also be found outside of biology, as in the relationships between languages: Darwin quite properly spoke of linguistic homologies (Darwin 1981, p. 59). Genealogical accounts thus abstract away from the details of particular kinds of character, focusing only their formal features (in terms of the topology of phylogenetic trees).

By relying on formal notions of ‘descent’ and ‘character’, the genealogical account achieves broad applicability. Any character that is informative of phylogenetic relationships between taxa can be homologized. This is so even though the processes by which different kinds of parts are inherited can be quite different. DNA is replicated by copying from a template. Body parts are not. Nonetheless, both can form transformation series. Later, we will see how these differences give rise to locally enriched accounts of homology. For now, however, what matters is that the genealogical account of homology is able to apply to all kinds of characters, despite these differences.

The formal nature of the genealogical account also makes room for the fact that homology “dissociates” across different kinds of biological entity. For instance, homologous body parts may develop via non-homologous developmental pathways and involve the expression of non-homologous genes (De Beer 1971; Wray and Abouheif 1998). In the other direction, homologous genes and developmental precursors may be involved in the development of non-homologous adult structures (Havstad, Assis, and Rieppel 2015). The genealogical account of homology permits (but does not require) such dissociations, because the formal notions of ‘descent’ and ‘character’ impose no *a priori* requirements on the relations between different homologs. Once the means by which different kinds of homologs are inherited are considered, constraints on dissociation will become important, but no such constraints are required by the genealogical account considered in isolation.

As represented in Figure 2, the genealogical account of homology, in addition to covering transformational and character state homology, also includes special homology and orthology. Special homologs are body parts shared due to descent from a common

ancestor (e.g. the vertebrate forelimb); orthologs are the same for genes. These stand in a one-to-many relationship to transformational homologs: each special homolog/ortholog is the basis for multiple transformation series. For instance, orthologous genes contain multiple loci, each of which forms a transformation series. Likewise, the vertebrate forelimb is the basis of many transformation series, such as digit number and length. They thus stand in the background of phylogenetic analysis without featuring in it directly. To see how they enter the picture, we need to look at the reasons why enriched accounts of homology are required.

4.4 ENRICHED ACCOUNTS OF HOMOLOGY

The methodology of phylogenetic systematics requires the identification of characters whose character states are informative of phylogenetic relationships among taxa. Only once such characters are recognized can phylogenies be reconstructed. How are they to be recognized? Answering this question will reveal the need for locally enriched accounts of homology. I first explain why such accounts are needed (Section 4.4.1), then summarize their key features (Section 4.4.2).

4.4.1 Why Enriched Accounts of Homology are Needed

Phylogenetic relationships are produced by the evolutionary process of descent with modification. When an ancestral species splits into two descendant species, the descendants resemble the ancestor (and each other) in some respects, but differ in others. Thus, shared descent can explain shared similarities. A phylogenetic relationship, however, is simply one that answers to a particular formal structure (Hennig 1966, pp. 18–21). There is no principled reason that ancestors and descendants, *qua* ancestors and descendants, cannot be radically different. Phylogeny alone places no limits on the extent of possible divergence between them.

To understand why real-world phylogenetic relationships show these similarity relationships, we must therefore look beyond phylogeny itself. Because offspring resemble their parents, descendant populations resemble their ancestors. If we want to know why common ancestry can explain similarities between taxa, we therefore need to consider the processes that produce parent-offspring similarities.

This is especially important for understanding homology, because, as we saw above, applying the methods of phylogenetic systematics requires that we first recognize comparable features of distinct taxa. The data used to infer phylogenetic relationships are laden with assumptions about which characters can be and are shared by descent. The grounds for such assumptions, however, lie in our understanding of how such characters are inherited. In this sense, *the genealogical account of homology is internally incomplete*: it assumes that certain characters are shared by descent without explaining how this is

possible. It requires completion by consideration of the details of inheritance, which will furnish an explanation of what it means for parts to be shared by descent.³³

At this point, a complication arises: what it means for two parts in different taxa to be shared by descent depends on the nature of the part in question. The reason is that different processes underlie the inheritance of different kinds of biological entities. The mechanisms of DNA replication, which involve copying from a template, are responsible for genomic parent-offspring resemblances. Body parts, by contrast, are reproduced each generation without the benefit of a template, relying instead on complex networks of regulatory interactions between genes (among other causes). Still other kinds of characters (e.g. behaviors) are inherited differently than either genes or body parts.

For this reason, the manner in which the genealogical account is to be completed will depend on the type of character in question – thence the need for local, not global enrichment. These local differences in the nature of inheritance matter for two reasons. First, from the standpoint of phylogenetic systematics, comparisons between character states that do not belong to the same transformation series introduce error into the process of phylogeny reconstruction (Fitch 1970). As accurate identification of transformational homologs is essential, it is necessary to understand how different kinds of characters are inherited.

³³ This argument resembles others in the literature on homology (Wagner 1989, p. 1158; Müller and Newman 1999, p. 65; Laublichler 2014, p. 73). These authors defend the need for a developmental account of homology, but do not draw the broader conclusion about the need for enriched accounts.

Second, though phylogenetic systematics is the basis for the genealogical account of homology, homology matters beyond systematics (Jamniczky 2005). For instance, understanding how a particular kind of character evolves requires establishing comparability for that kind of character. For example, during the modern synthesis, the attempt to uncover the genetic basis of species differences required identifying homologous genes across species (Spencer 1963). Likewise, contemporary evolutionary-developmental biology's attempt to explain the origin of morphological novelty requires contrasting the evolution of genuine novelties from the (often extreme) modifications of pre-existing parts (Müller and Wagner 1991).³⁴

We should therefore expect that consideration of the local processes of descent for different kinds of characters will inflect our understanding of homology both within and beyond systematics. Consideration of these processes leads to accounts of homology that apply to only a limited subset of biological entities. These I refer to as enriched accounts of homology.

4.4.2 Three Features of Enriched Accounts of Homology

Enriched accounts of homology have three key features. Here, I state them dogmatically; the examples of the next two sections will justify my claims.

First feature. Enriched accounts of homology apply to a more limited domain than the genealogical account of homology; enrichment is therefore local. For instance, the

³⁴ This distinction is controversial (Minelli 2016). The issue is treated below (Section 4.6).

enriched accounts considered below apply, respectively to genes (Section 4.5) and to (animal) body parts and cell types (Section 4.6). These domains must be determined empirically. It happens to be the case that Günter Wagner's account of homology applies to both body parts and cell types. It may turn out that Wagner's account is wrong, and that these actually require separate enriched accounts. Further, it is an open question whether Wagner's account can be extended to the parts of plants. He suggests it can in at least some cases, but there are reasons to worry (Wagner 2014, chap. 12; Kendig 2016). There is no way to intuit what types of characters can be subsumed under a single enriched account.

Second feature. Enriched accounts of homology are connected to the genealogical account of homology. This connection has two aspects. *First*, enriched accounts must pick out a type of homolog that is shared due to common ancestry, and that forms the basis for recognizing transformation series. Wagner's enriched account of body part homology includes special homology, while the enriched account of gene homology includes orthology (Figure 2). As mentioned above (Section 4.3), special homologs and orthologs stand in a one-to-many relationship to transformational homologs. Enriched accounts thus constrain but do not determine the choice of transformational homologs, and so play an important background role in the methodology of phylogenetic systematics.

Second, enriched accounts must, for the relevant type of genealogical homolog, explain what it means for such homologs to be shared by descent. That is, enriched accounts must elucidate the particular processes that enable these parts to be related by descent. As we saw above, the genealogical account, because it presupposes the ability of parts to be so related, is internally incomplete. In explaining how such relationships are possible for a

particular kind of character, *enriched accounts of homology complete the genealogical account within a limited domain.*

Third feature. Enriched accounts of homology include types of homology that do not fall under the genealogical account. This is because enriched accounts of homology explain homological sameness not in terms of a purely formal notion of 'descent', but rather in terms of the particular processes that make shared descent possible. It turns out (empirically) that these enriched accounts of homological sameness can be applied in cases where the genealogical account cannot, yielding phenomena such as serial homology, paralogy, and xenology (Figure 2). While the genealogical account recognizes no connection between, e.g., special and serial homology (Cracraft 2005), there is such a connection, and this is captured by enriched accounts.

In combination, these three features show that enriched accounts of homology overlap with the genealogical account but are not nested within it. Neither is complete without the other. The genealogical account unifies the application of 'homology' to many different kinds of character, but only by ignoring the details that explain how these characters can be related by descent at all. In ignoring these details, it overlooks the real connections between genealogical homology (e.g. special homology and orthology) and non-genealogical homology (e.g. serial homology, paralogy, and xenology). Enriched accounts, by focusing on these details, are able to (a) explain the connections between genealogical and non-genealogical homology (third feature) and (b) explain how characters can be related by descent (second feature). Because enriched accounts apply only within limited

domains (first feature), however, the sense in which they are all accounts of *homology* is lost without the genealogical account, to which each is connected (second feature).

That is why a compatibilist picture that recognizes the need for multiple co-existing accounts is correct. As noted above (Section 4.2), any viable compatibilist view must explain in what sense developmental and genealogical accounts of homology are accounts of the same thing. The framework just described satisfies this demand. In their regions of overlap, the genealogical account and a given enriched account provide different perspectives on the same phenomenon. The next two sections justify this framework by looking in detail at two particular enriched accounts, one for gene homology and one for body part homology.

4.5 THE ENRICHED ACCOUNT OF GENE HOMOLOGY

Enriched accounts of homology complete the genealogical account within a limited domain. They do so by considering the processes that allow entities within that domain to be related by descent. In the case of DNA, these processes are well understood, with the result that homologizing genes is conceptually, though not always practically, a simple process. DNA consists of two anti-parallel strands with complementary nucleotide sequences. During replication, the strands are separated, with each strand serving as a template for its complement, such that two new double-stranded DNA molecules are created,

each consisting of one old and one new strand. Though errors may occur in this process, it is generally quite faithful, with the result that the new molecules are nearly identical to each other and to the original.

On this basis, an enriched account of gene homology emerges: genes (in this context, any stretch of DNA of interest) are homologous just in case they descended, via this replication mechanism, from the same stretch of DNA in a common ancestor. Thus far, this is just a genealogical account of homology that makes reference to the specific mechanism by which DNA is inherited. It possesses the first two features of enriched accounts, but not the third. However, the behavior of DNA during replication forces us to complicate the account. Occasionally, the molecular machinery required for replication “slips” and copies a particular stretch of DNA twice, resulting in a new DNA molecule with two genes that both descended from a single gene in the ancestor. Other mechanisms (e.g. unequal crossing over and mobile genetic elements) can also give rise to duplicated genes, or even to a duplication of the entire genome.

Biologists thus recognize two sameness relations that can obtain between genes, both of which involve being descended from the same stretch of ancestral DNA. Two copies of the same gene that are the result of duplication and so coexist within a single organism are *paralogs*, while two copies of the same gene that are the result of speciation are *orthologs*. Only orthologs fall under the genealogical account of homology. Character states of paralogous genes cannot be treated as part of the same transformation series: “phylogenies require orthologous, not paralogous genes” (Fitch 1970, p. 113). Paralogy thus falls under the enriched account of gene homology, but not under the genealogical

account of homology, illustrating how enriched accounts expand the reference of ‘homology’ beyond the domain of the genealogical account.

Other features of how DNA is inherited complicate the account still further. Lateral gene transfer, in which stretches of DNA (not necessarily functional) are transferred between different species, is rampant among prokaryotes and known, though rare, in eukaryotes (Eme and Doolittle 2016). It allows for the same gene to be present in different species. Like orthologs, laterally transferred genes are the same gene in different species, and biologists perfectly readily speak of laterally transferred genes as “homologous” (e.g. Mohanraju et al, 2016). Unlike orthologs, however, laterally transferred genes produce tokogenetic rather than phylogenetic relationships between species, and so require the addition of a third category of gene homology: xenology (Gray and Fitch 1983).³⁵

Thus we can see that consideration of the mechanisms by which DNA is inherited is the basis for an enriched account of gene homology that possesses all three features of a locally enriched account. It applies to a limited class of biological entities, namely stretches of DNA. It can also apply to proteins (Fitch 1970), but not, for instance, to body parts or behaviors (first feature). It is connected to the genealogical account, explaining how it is possible for genes to stand in descent relationships, i.e. be orthologous (second feature). Lastly, the enriched account of gene homology expands the reference of

³⁵ Unlike paralogy, xenology is not simply a problem to be avoided in systematics. It is arguably a phenomenon to be included. Where xenology is prevalent, systematists cannot simply assume that relationships between taxa can be captured by a strict tree, and must instead infer from the data to a phylogenetic network (Huson, Rupp, and Scornavacca 2010). However, whether one infers a tree or a network, one must still undertake a sequence alignment step that furnishes the relevant transformation series, and this is presupposed, but not tested, by the data-to-tree/network inference.

'homology' by including paralogy and xenology, even though these are excluded by the genealogical account (third feature).

4.6 WAGNER'S ENRICHED ACCOUNT OF BODY PART HOMOLOGY

Understanding body part homology requires understanding development, and knowledge of developmental processes has played a role in determining body part homologies since at least the late 18th century (Goethe 2009; Owen 2007). The fertilized embryo contains a nucleus (including DNA) and surrounding cytoplasm. In the strictest sense, that is all that an offspring inherits from its parents.³⁶ Adult morphology must be developed epigenetically each generation. In contrast to DNA replication, a parent's limbs do not serve as templates for its offspring's limbs. Thus, when body parts are homologized, they are homologized in accordance with an enriched account distinct from the enriched account that applies when genes are homologized. The crucial consideration is the nature of the continuity between ancestral and descendant body parts.

Because homology dissociates between genes and body parts (De Beer 1971; Wray and Abouheif 1998), body part homology cannot be easily reduced to gene homology

³⁶ The discussion of development in this section primarily applies to animal development, and it focuses exclusively on the role of gene regulation in development, ignoring the role of non-genetic resources that shape development. I exclude such considerations because they do not feature in Wagner's account of body part homology.

(Brigandt 2002). Furthermore, homologous body parts may develop via different developmental pathways (De Beer 1971), and different body parts may develop from the same developmental precursors (Havstad, Assis, and Rieppel 2015). An enriched account of body part homology must be compatible with these phenomena. Specifically, any attempt to explain body part homology in terms of shared developmental processes must (a) identify what developmental features are conserved between homologs and (b) explain why dissociation in other features is possible.³⁷

No consensus account of body part homology currently exists. I here present Günter Wagner's (1989, 1994, 1999, 2014) account, not out of commitment to its correctness, but because it illustrates in detail what an enriched account of body part homology might look like.³⁸ Wagner (1989, p. 1163) sets himself the task of accounting for three core explananda. First, homologous body parts share conserved features. Despite variation in form and function, there is deep evolutionary conservation of animal body plans, and an account of body part homology should explain why body plans are conserved. Second, homologous body parts are individualized, i.e. they possess "a certain minimal degree of complexity, differentiation, and genetic/epigenetic autonomy" (Wagner 1989, p. 1160). Wagner emphasizes variational individuality: the ability for genetic mutations to affect

³⁷ Some authors (Ramsey and Peterson 2012, 261; Currie 2014, 703) have used the mere fact of dissociation as an objection to developmental accounts of homology. This is a mistake. Dissociation in some respects does not entail dissociation in all. Dissociation is properly understood as an explanandum for accounts of body part homology, not as an objection to such accounts (cf. Wagner 2014, 90–93).

³⁸ Gerd Müller (2003) offers a distinct enriched account of body part homology. Alessandro Minelli (2016) raises serious challenges to Wagner's approach to homology. I defer discussion of these challenges to the end of this section.

one part but not another. Third, homologous body parts possess a single evolutionary origin and thus characterize a monophyletic taxon.

The latest incarnation of Wagner's account explains the conserved similarity, individualization, and phylogenetic uniqueness of homologs in terms of shared character identity networks, or ChINs (Wagner 2014, chap. 3). ChINs are gene regulatory network (GRN) subcircuits, usually wired in a positive feedback loop, that "form the interface between developmental signals and those genes that actually engender the morphological character during morphogenesis and differentiation" (Wagner 2014, p. 97).

Animal development involves the precise control of gene expression in space and time (Peter and Davidson 2015, chap. 1).³⁹ As the embryo develops, it is progressively subdivided into more and more domains, each characterized by a distinct regulatory state. In this process, transient chemical signals furnish positional information that blocks out a domain. These signals activate a positive feedback loop that stabilizes the regulatory state of that region (Peter and Davidson 2015, chap. 6). Downstream of this positive feedback loop, "realizer" genes are expressed that are responsible for the formation of a particular body part within that domain (morphogenesis). This feedback loop (Wagner's ChIN) stabilizes the identity of the region, fating it to express a particular set of realizer genes. As lineages evolve and diverge, the realizer genes downstream of the ChIN can change,

³⁹ GRN theory in its current form is primarily applicable to animal development. However, developmental information is relevant for resolving homology assessments in plants as well (cf. Sajo, Longhi-Wagner, and Rudall 2008; Vrijdaghs et al. 2009; Golding and Ponder 2010; De-Paula et al. 2011)

leading to variation in form and function. So long as the ChIN is conserved, however, these body parts share what Wagner calls a “character identity” – they remain homologous.

Just as the complications of DNA replication and lateral transfer forced the recognition of different types of gene homology, so the complications of development force the recognition of different types of body part homology. Wagner ties homology to character identity. Character identity can be shared across species (special homology), as in the case of the dugong’s fin and the bat’s wing. However, it can also be shared within an individual (serial homology). The dugong, after all, has two pectoral fins, and the bat two wings. If Wagner’s account is correct, this is because the same ChIN is activated in two regions of the embryo.

Wagner’s account can explain all three explananda described above. Because ChINs are recursively wired, and because they are responsible for ensuring the expression of an entire suite of genes essential for body part development, they are likely to be refractory to evolutionary change (Davidson and Erwin 2006). They thus tend to be conserved, even as the downstream genes they regulate are gradually changed. Wagner can thus explain the sense in which two body parts can retain the same identity despite substantial modifications of form and function.

Wagner can also explain the individualization of body parts. A given gene can come to be regulated by a given ChIN without necessarily being regulated by any other ChIN. Body parts whose development is controlled by distinct ChINs can therefore vary independently. At the same time, Wagner can explain why individualization is often

incomplete. Serial homologs, in which the same ChIN is activated in multiple regions of the embryo, tend to vary in tandem because a downstream gene activated in one serial homolog is likely to be activated in every other.

Lastly, any individual ChIN emerges in a particular lineage at a particular time and, unless it is modified or lost, can be found in all members of that lineage. In this way, Wagner captures the phylogenetic uniqueness of homologs.

Wagner's account of body part homology has all three features of enriched accounts of homology. It applies only to a limited domain of biological entities, namely animal body parts. Wagner (2014, chap. 8) also extends his account to cell types, on the grounds that cell type identity is also determined by ChINs (first feature). Wagner's account is tied to the genealogical account, because character identities are phylogenetically unique. By tying character identity to the expression of conserved ChINs, Wagner explains how body parts can be shared due to descent from a common ancestor, even as they are modified. Wagner's special homologs are thus the proper basis for recognizing transformation series (second feature). Lastly, Wagner's account expands the reference of 'homology' to include serial homology, which is not covered by the genealogical account (third feature; Figure 2).⁴⁰

⁴⁰ In this discussion, I have simplified things for ease of exposition. In fact, the appropriate bearer of character states is not the entire organism (or part) over the entire course of its life, but a suitably thick time-slice of the organism (part), called a *semaphoront*. In an excellent paper, Havstad, Assis, and Rieppel (2015) show that ontogenetic identity (identity of a part across the different semaphoronts of a single individual) and phylogenetic identity (identity of a part across evolutionary transformations) can come apart. For example, in *Drosophila melanogaster*, female genitalia develop from the embryonic segment A8. In males, however, A8 develops into a tergite-like structure (Keisman, Christiansen, and Baker 2001). A8 in females is phylogenetically identical to A8 in males, and it is

While Wagner's account of homology illustrates what an enriched account of animal body part homology might look like, it is controversial. In a recent paper, Alessandro Minelli (2016) argues that Wagner is too sanguine about the manner in which body parts remain "the same" over time, even as their features change. On Wagner's account, body parts possess an underlying identity that persists even as the features of those parts change. Wagner thus has a two-layer ontology, in which characters possess both an identity (determined by their underlying ChIN) and a particular realized state (determined by the operation of downstream realizer genes). Minelli rejects this approach in favor of a single-layer ontology. On Minelli's view, traits are to be understood as "complex and ever-changing intersections of an indeterminate number of features." This disagreement has further consequences. Wagner's approach lends itself to the traditional assumption that homology is an all-or-nothing relation (cf. Fitch 2000), while Minelli favors a combinatorial approach to homology that allows for parts to be partially homologous. Relatedly, Wagner's account permits a sharp distinction between the origin of novel features and their respective diversification, while Minelli's account denies the possibility of drawing such a distinction.

The crucial question, for the purposes of this chapter, concerns the basis for Minelli's objections. Here Minelli is explicit that his criticisms are founded on an understanding of

ontogenetically identical to the adult female genitalia. Likewise, A8 in males is ontogenetically identical to the tergite-like structure. Yet the female genitalia and the male tergite-like structure are not homologous. Over the course of development, a homologous precursor develops into non-homologous structures. One task of an enriched account is to explain why this is so. Wagner's account would analyze such cases as involving initially homologous precursors that come to express non-homologous ChINs.

how body parts are inherited, which includes their manner of development. He accepts that there exist conserved developmental modules, but argues that these modules are related to body parts in a many-to-many fashion: many such modules go into the building of any single part, and each individual module is used in the building of distinct parts. Body parts are thus the product of a “peculiar intersection (both spatial and temporal) of developmental modules” (Minelli 2016, p. 49). This has two implications: first, that there are no grounds for a Wagnerian two-layer ontology, since all developmental modules are on a par; and, second, that these intersections, due simply to the number of modules they involve, are unlikely to be deeply conserved.

Though Minelli’s account challenges Wagner’s at crucial points, it serves the same basic function: it attempts to explain how it is that body parts can be related by descent. That is, Minelli challenges Wagner not by denying the need for an enriched account of body part homology, but by offering a competing enriched account. Whichever view of body part homology should prove correct, my central contention—that some enriched account is needed—stands.

4.7 ENRICHED ACCOUNTS OF HOMOMOLOGY ARE LOCAL

I have shown how consideration of the processes by which characters are inherited leads to the development of enriched accounts of gene and body part homology. The

genealogical account is unifying in the sense that it furnishes abstract requirements that all (genealogically) homologous characters must meet: they must be shared due to common descent. The ability of particular kinds characters to satisfy these requirements depends on concrete processes by which those kinds of characters are inherited. These processes are distinct for different kinds of characters. Genes are homologous in case they descend via replication from the same ancestral sequence. Body parts are homologous (if Wagner is right) in case they develop via the activation of shared ChINs. Enriched accounts thus show how it is possible to satisfy the requirements of the genealogical account within particular domains.

A central feature of the framework I have presented and defended is that these enriched accounts are local, in the sense that they apply to different domains. According to this framework, biologists work with more than two accounts of homology (one genealogical and one enriched). They work with one genealogical account and multiple enriched accounts, each with its particular, limited domain. My aim in this section is to defend this claim against an objection.

The objection I have in mind claims that enriched accounts are more unified than I have let on. Granting that, at the level of details, enriched accounts are clearly distinct, the objection claims that the resulting pictures, conceived more abstractly, share certain structural similarities. In this regard, only a single enriched account is required. Such a view might be motivated by considering an apparent structural similarity between the two enriched accounts provided above. For both gene and body part homology, we can distinguish the same part/gene in different species (special homology, orthology) from

the same part/gene in a single organism (serial homology, paralogy). It is true that the enriched account of gene homology recognizes xenology, whereas Wagner's enriched account of body part homology involves nothing of the sort. However, there is no conceptual difficulty in imagining a laterally transferred ChIN, merely a host of practical difficulties.

On this basis, one might suggest that there is a basic template for constructing enriched accounts. No matter what processes are responsible for the inheritance of a particular type of character, there are three ways in which two tokens of that character type might be related. They might be related in different species due to shared descent (special homology, orthology), or in different species due to lateral transfer (xenology), or in a single individual due to duplication (serial homology, paralogy). In some cases, one or more of these conceptual possibilities may be unrealized (as there is no analog of xenology for animal body parts), but these three conceptual possibilities are exhaustive, no matter the type of character.

I do not deny that enriched accounts will involve some subset of these three possibilities. However, I contend that, despite these similarities, the differences between distinct enriched accounts are more important. In the remainder of this section, I consider special homology and orthology, in order to illustrate the nature of these differences and the reasons they matter.⁴¹

⁴¹ For reasons of space, I do not discuss serial homology and paralogy, but similar considerations apply.

On Wagner's (2014, pp. 58–65) account of body part homology, two types of special homologs are recognized: strict special homologs and variational modalities. Variational modalities capture cases where special homologs come in two or more distinct forms. The tetrapod limb and teleost fin are special homologs, even though they are structurally quite distinct. The array of actual fin forms occupies a distinct region of morphospace than the array of actual limb forms. They are thus two variational modalities of the same special homolog. Note that this is a structural distinction. In the case of gene homology, however, no similar structural distinction can be drawn.

Among genetic phenomena, the closest corresponding distinction is between orthologs that share a molecular function and orthologs that serve distinct molecular functions (Wagner 2014, p. 80). The defining difference is in terms of molecular function, in contrast to the case of body parts, where the difference is in terms of variational properties. It is true that orthologs with different molecular functions are likely to occupy discrete regions of sequence space, and so generate a pattern similar to variational modalities. But it is shared function that creates this pattern. By contrast, a tetrapod limb is a tetrapod limb even if it serves, as in the case of a whale's flipper, the same function as a fish fin.

In short, it is possible to draw a structural division between types of special homolog, but not between types of ortholog. This matters for understanding how special homologs and orthologs evolve. Special homologs are subject to both developmental and functional constraints, whereas orthologs are subject only to functional constraints. As Amundson (1994) has argued, developmental and functional constraints have distinct evolutionary

implications and should not be conflated. The reason for this difference lies in the nature of the homological sameness relation. Body parts are homologous if they share the same character identity. This requires sharing an underlying ChIN, which in turn constrains their patterns of variability. The mechanisms of DNA replication do not furnish any comparable constraints on variability.

These differences between special homology and orthology point to a deeper difference in how these two enriched accounts explain sameness “under every modification of form and function.” The genealogical account explains this in terms of shared descent (with modification). Because the genealogical account refers homology to historical origin, it permits potentially unlimited divergence after that origin. No amount of subsequent divergence can change the fact that two parts share a common origin. The genealogical account *by itself* thus places no constraints on subsequent divergence.

But, as we saw above (Section 4.4), the genealogical account is incomplete. It furnishes abstract requirements that homologous characters must satisfy, but does not consider the specific processes of inheritance that make the satisfaction of these requirements possible. Once the details are considered, limits to divergence may be discovered. In Wagner’s theory of body part homology, what is homologized are character identities, as fixed by ChINs. Preserving body part homology thus requires the evolutionary conservation of ChINs. Homology-preserving divergence is limited to divergence that occurs via the modification of the genes downstream of the relevant ChIN. Wagner’s account thus makes the conservation of particular similarities essential to the conservation of homological sameness.

By contrast, the mechanisms of DNA replication set no limits on divergence. Two stretches of DNA in different species may be homologous (descended via replication from the same stretch in a common ancestor) even if, over evolutionary time, every single nucleotide has diverged between the two sequences. Unlike the case of body part homology, no particular similarities are essential to the preservation of homology. It is true that the recognition of orthologous DNA sequences requires the preservation of sufficient similarity to distinguish similarity due to common descent from similarity due to chance (Strimmer, von Haeseler, and Salemi 2009, pp. 137–40). However, the difficulty of recognizing that two highly dissimilar sequences are related by descent does not change the fact that they are so related. Even leaving that point aside, recognizing orthology requires only a sufficient degree of overall similarity, not similarity in any particular subset of bases. Wagner’s account of body part homology, by contrast, sets no limits on overall dissimilarity, so long as the essential similarity (the ChIN) is preserved.

The enriched accounts of body part homology and gene homology thus lead to importantly divergent pictures of how these different kinds of characters evolve. These differences matter both within systematics (recall that inferring phylogenies from morphological or molecular data requires adopting a model of how the characters used evolve) and in the study of evolutionary change more generally. Thus, the surface similarities between orthology and special homology are just that: superficial. Enrichment truly is local, ineliminably dependent on the particular processes involved in the inheritance of particular kinds of characters.

4.8 RESOLVING THE PROBLEM OF HOMOLOGY

According to the framework I am defending, developmental accounts of homology should be understood as enriched accounts of body part homology. Other enriched accounts arise when other kinds of characters (genes, behaviors, etc.) are homologized. These enriched accounts are not in competition with the genealogical account of homology. Rather, they complete (and extend) that account within particular domains.

This compatibilist view of the relationship between enriched accounts and the genealogical account is tenable only if the four sources of tension between genealogical and developmental accounts (Section 4.2) can be resolved. My aim in this section is to show that the framework I have offered eliminates these tensions.

The first tension concerns the nature of homological sameness. Genealogical accounts say that homologous parts are the same in virtue of their shared descent. 'Descent' here is understood purely formally, in the context of the methodology of phylogenetic systematics. There are multiple mechanisms that can produce such descent relationships between parts: replication in the case of genes, development controlled by a ChIN in the case of body parts. In saying that body parts are homologous because they share the same character identity, Wagner is not denying the genealogical account. He is explaining how it is possible that body parts can answer to that account's formal requirements. This point is not limited to Wagner's account. Other enriched accounts are compatible with the

genealogical account for the same reason: one of their key features is that they explain how characters can be shared due to descent. Thus the first tension disappears.

The second tension concerns the amount of dissimilarity between homologs that each account permits. Developmental accounts limit the amount of allowable dissimilarity between homologs. Genealogical accounts do not. This generates no inconsistency, however. The key consideration here is that the genealogical account imposes purely formal requirements on homology, in terms of the topology of phylogenetic trees. Because the genealogical account says nothing about what it is to be the same character, it is silent about the degree to which homologs can diverge. For this, we must look to enriched accounts. Wagner's developmental account says that body part homology requires the conservation of the underlying ChIN. By contrast, in the case of genes, the appropriate enriched account does not require the conservation of any essential similarity. The genealogical account is consistent with both. It permits but does not require the possibility of complete dissimilarity. Similar considerations apply to the issue of dissociation. The genealogical account is consistent with all kinds of dissociation, while developmental accounts reveal limits to the dissociation that is actually possible.⁴² The second tension disappears along with the first.

⁴² This point is very important, and not always recognized. For instance, Jenner (2006) accuses Wagner's account of homology of failing to respect that homologs, like taxa, are individuals, not classes (cf. Ghiselin 2005b). The problem, Jenner argues, is that Wagner does not allow for homologs to diverge indefinitely. Jenner's error is to attempt to draw a physical conclusion from a conceptual point. It is true that no amount of divergence can, *simply in virtue of being divergence*, erase common ancestry. But one cannot conclude from this, as Jenner does, that the physical mechanisms underlying inheritance cannot be such that, in reality, they forbid indefinite divergence.

The third tension concerns the role of serial homology: the genealogical account excludes it, while developmental accounts include it. Here it is important to see that the term ‘homology’ covers multiple distinct (though related and overlapping) phenomena. Genealogical sameness (shared descent) and developmental sameness (shared ChIN) are distinct types of sameness. There is no special homology of body parts without the overlap of both of them. It is in virtue of sharing a ChIN that body parts are able to stand in descent relationships. Thus, in this region of overlap, developmental sameness is part of the account of what genealogical sameness is.

But neither sameness relation is limited to this region of overlap. Genealogical sameness is shared by other kinds of characters that lack developmental sameness altogether (e.g. genes) or that lack the specific kind of developmental sameness (conserved ChINs) shared by body parts (e.g. behaviors). Equally, character identity is shared not just among special homologs, but also among serial homologs. The third tension, too, has vanished.

The fourth tension concerns the proper target of homology assessments: characters or character states. We are now in position to see that this is misleading. “Character” is ambiguous here: it can refer both to transformation series (e.g. number of digits) and to the parts that underlie these transformation series (e.g. the vertebrate forelimb). Since we already have the term ‘transformation series’ for the former, I will use ‘character’ for the latter. Then we can distinguish three targets of homology assessment: (1) characters (special homologs, orthologs, etc.), (2) transformation series, and (3) character states. As we have seen, the genealogical account covers all three (Figure 2). Genealogical and developmental accounts overlap in the first category and, insofar as developmental accounts

constrain (without determining) the identification of transformation series, also the second. Homologizing character states is indeed peculiar to the genealogical account, but there is no incompatibility here. Both accounts agree that characters and transformation series can be homologized. About homologizing character states, developmental accounts are simply silent. And so the fourth tension disappears along with the rest.

In the end, we can see that there is truth in both of the traditional views about the relationship between genealogical and developmental accounts of homology. Those who have treated them as antagonistic have correctly recognized that they cover, at least in part, the same phenomena. The genealogical sameness of body part homologs is not a wholly distinct phenomenon from their developmental sameness. Rather, their developmental sameness is precisely what allows them to be genealogically the same. Thus, the two accounts had better be consistent in what they say about special homology. Nonetheless, the compatibilists are correct that they cannot be unified into a single account. The developmental sameness relationship (and enriched sameness relationships more generally) can be instantiated in cases where genealogical sameness is not (e.g. serial homology), and vice versa (e.g. orthology). Genealogical and enriched accounts overlap but are not identical. Where they overlap, they are compatible. Where they do not overlap, the issue fails to arise.

4.9 OTHER ACCOUNTS OF HOMOLOGY

I am not the first to attempt to bridge the gap between genealogical and developmental accounts of homology. Recently, Brigandt (2007) and Ramsey and Peterson (2012) have defended accounts with similar aims. Ramsey and Peterson defend a genealogical account of homology that incorporates input from developmental biology, while Brigandt defends a developmental account of homology that fits with systematic practice. I contend that the framework I have defended marks an improvement over both. It is compatible with Brigandt's account but has greater generality, and it preserves the advantages of Ramsey and Peterson's account without sharing that account's central flaw (discussed below). I discuss them in turn.

Brigandt is concerned, as I am, to make sense of the relations between genealogical and developmental accounts of homology. But whereas I begin from genealogical accounts and expand outward to enriched accounts (including developmental accounts), Brigandt starts with development. He characterizes homologs as units of heritable phenotypic variability – i.e. as characters that can take different character states. Understanding the developmental sameness of body part homologs offers an explanation for why it is possible to perform phylogenetic analyses that take for granted the existence of characters that come in different character states.

According to the framework I have defended, Brigandt can be understood as showing how a developmental account of homology fulfills the function of an enriched account. Brigandt does not, however, draw the conclusion that such accounts are required for

every kind of character used in phylogenetic analysis. Philosophical work on homology has focused on understanding the legitimate roles (if any) of a developmental account of body part homology, largely ignoring the fact that there has long existed an enriched account of gene homology. In fact, however, the situation is exactly comparable in the two cases. My aim has been to draw attention to this more general state of affairs, while Brigandt's aim, expressed in my terminology, was to look at the proper role of a particular enriched account. Our views are compatible, and complementary.

I turn now to Ramsey and Peterson's genealogical account of homology. They argue that a single definition of 'homology' can be offered that (a) incorporates the role of information about the mechanisms of descent and (b) subsumes all legitimate uses of the term (including serial homology and paralogy). They present an abstract schema for how to include information about descent mechanisms. As this schema applies in all cases, they can be seen as offering a fully general template for constructing enriched accounts. I aim to show that this template imposes arbitrary restrictions on the nature of the relations between different types of character.

Ramsey and Peterson begin with a straightforward genealogical account of homology. Traits T and T^* belonging, respectively, to organisms O and O^* are homologous if they are present in every organism along the shortest path (on a phylogenetic tree) connecting O and O^* . But there is a problem: there can be failures of continuity that do not undermine homology. For instance, sexually dimorphic traits need not be continuously present. The posterior lobe is a male-specific genital structure found in certain *Drosophila* species. Suppose a male has a female offspring, who in turn has a male offspring. In this case, the

female lacks the structure (violating continuity), and yet clearly the lobes in the males are homologous.

What is required here is some account of how such characters are inherited, an account that explains how a part can remain the same through descent despite such violations of continuity – an enriched account, in other words. Moreover, though the example I chose involved a morphological structure, such violations of continuity can affect other kinds of character as well, for instance behaviors. Ramsey and Peterson provide a general-purpose solution to this problem.

Ramsey and Peterson's solution rests on the assertion that there exist distinct biological levels that stand in a strict ordering, such that for a given level L_{N-1} (e.g., development), one can distinguish a higher level L_N (morphology) and a lower level L_{N-2} (genes). They then allow for violations of continuity at L_N provided that there is continuity at L_{N-1} , but not if continuity is only preserved at L_{N-2} . For instance, a violation of continuity at the morphological level might be bridged by continuity at the developmental level, but not by continuity at the genetic level. Though they make the point using the morphology, development, and genes as example levels, their claim is general. Whatever levels biologists identify, violations of continuity can be bridged by continuity at the next level down, but no lower.

This account assumes the existence of an objective (i.e. research agenda-independent) hierarchy of levels in biology, with a clear ordering, such that it is unambiguous, given a particular L_N , what is L_{N-1} and what is L_{N-2} . At a minimum, this assumption requires

further elaboration.⁴³ Levels are not simply size scales, given that developmental processes involve entities at both genetic and morphological size scales. Moreover, whereas genes and morphological parts are entities, development is a process involving those entities. Thus, it is unclear what it means to say that development is a level intermediate between the genetic and the morphological levels.

Even granting the assumption, however, their account runs into serious trouble. In trying to solve the problem of failures of continuity, Ramsey and Peterson recognize the need for understanding how characters are inherited – the need, that is, for an enriched account. The role of this account is to show how a character can be continuously inherited even if the character itself, for whatever reason, fails to appear in particular individuals. What Ramsey and Peterson’s account does is to impose a restriction on what enriched accounts can include: they can involve continuity at L_{N-1} , but not at L_{N-2} . I contend, however, that this restriction is arbitrary, and that it runs the risk of ruling out successful enriched accounts of homology on illegitimate grounds.

For example, consider Wagner’s account of body part homology, taking body parts as L_N . If we take ChINs as L_{N-1} , his account fits their schema. But ChINs are almost certainly not L_{N-1} . Suppose we accept Ramsey and Peterson’s distinction of three levels: morphological, developmental, and genetic. On this division of levels, ChINs arguably fit best at the genetic level. Gene regulatory networks are based on regulatory information encoded in *cis*-regulatory DNA sequences. True, ChIN activation depends on processes of gene regulation that can reasonably be treated as developmental. In the case of our lobeless

⁴³ For critique of this assumption, see Currie (2014).

female *Drosophila*, however, the (hypothetical) ChIN is never activated, so the violation of continuity extends to these processes. All the female inherits is the underlying genetic information— L_{N-2} .

Nonetheless, we may wish to treat gene networks, which involve a great deal of genetic information distributed throughout the genome, as a level distinct from that of single genes. Salazar-Ciudad and Jernvall (2013) have made a recent proposal along these lines. They, however, distinguish three levels below morphology: genes, gene networks, and epigenetic networks. ChINs, once again, are L_{N-2} . Finally, even if we set aside these issues and allow that gene networks are one level below morphology, it is questionable whether all morphological characters belong to a single level. Morphology includes quite distinct kinds of characters besides morphological parts, such as tissues, cell types, and organelles. Many analyses treat them separately, including, pertinently, those based on GRN theory (Wagner 1989, 2014; Peter and Davidson 2015).

No matter how Ramsey and Peterson choose to flesh out ‘level’, then, we will almost certainly be forced to recognize at least one level intervening between ChINs and body parts. Thus, Wagner’s account is illegitimate, according to Ramsey and Peterson’s schema. This is exactly the wrong result. Wagner’s account, if correct, solves the very problem that motivated their schema. It explains how body parts can be continuously inherited despite violations of continuity at the morphological level. In insisting that continuity be preserved at L_{N-1} , Ramsey and Peterson arbitrarily limit the search space for solutions to the problem they identify. There is no reason to expect that the world will respect these limitations, as Wagner’s theory shows.

Nonetheless, Ramsey and Peterson do have a reason for imposing this limitation: without it, they worry, cases of spurious continuity will be admitted. Here they are concerned about cases of deep homology, in which independently evolved structures make use of homologous genetic resources. For example, homologous transcription factors (Eyeless in *Drosophila*, PAX6 in mice) play important roles in eye development in their respective taxa (Shubin, Tabin, and Carroll 2009). Our best evidence, however, suggests that eyes evolved independently in these lineages. In this sense, deeply “homologous” structures are not really homologous at all. Ramsey and Peterson’s account can account for this: deep homology involves genetic continuity (L_{N-2}), but developmental discontinuity (L_{N-1}).

But, while they get the right result, they get it for the wrong reason. The trouble with deep homology isn’t that it involves violations of continuity at both L_N and L_{N-1} . The reason why deep homology is not genuine homology is that it does not solve the problem posed by such violations. Eyeless and PAX6 are implicated in a conserved pathway involved in opsin production. But opsin production is not eye production, and the eyes that rely on this conserved pathway evolved independently. That is why the continuous presence (the homology) of the underlying pathway does not ensure the homology of the structure. It has nothing to do with the level at which the continuity occurs. If Wagner (2014, pp. 102–5) is right, there is a conserved ChIN underlying insect eyes, and this is at the same level as the opsin pathway (indeed, it includes the gene that codes for Eyeless). Unlike the opsin pathway, however, this ChIN is crucial for establishing eye identity. It

is this difference that explains why one, but not the other, can account for the preservation of homology across violations of continuity.

The difficulty with Ramsey and Peterson's account stems from their attempt to impose an arbitrary restriction on what an enriched account of homology can look like. Beyond setting out a problem that such accounts must solve (the problem of failures of continuity), they claim that any adequate solution must involve only a very particular sort of lower-level continuity, regardless of the sort of character under discussion. But it is a matter for empirical determination, not stipulation, what sort of continuity can and cannot solve the problem.

I conclude that, instead of trying to find a single schema that can simultaneously perform the unifying work of the genealogical account and the detailed work of locally enriched accounts, we should accept that 'homology' has a fine structure of the sort illustrated in Figure 2.

4.10 CONCLUDING REMARKS

In this chapter, I have presented a general framework for understanding how distinct accounts of homology are related. The core claim I have defended is that there is a fine structure underlying uses of 'homology', consisting of a single genealogical account and multiple locally enriched accounts (Figure 2). I have argued that recognizing the existence

of this fine structure dissolves the several alleged sources of tension between genealogical and developmental accounts of homology.

The genealogical account unifies the application of 'homology' to all kinds of biological characters by showing they can all play the same formal role in phylogenetic systematics. It does so by relying on notions of 'descent' and 'character' that abstract away from the processes by which characters are inherited. All that is required is that characters stand in phylogenetic relationships.

Because of this, however, the genealogical account takes for granted the existence of transformational homologs: stable characters that can come in multiple distinct states. Recognizing transformational homologs requires consideration of the manner in which characters are inherited. Accounts of homology that consider such information are enriched accounts of homology.

Enriched accounts of homology have three key features. First, they apply to a limited domain, i.e. they are locally, not globally enriched. Second, they are connected to the genealogical account. This connection has two aspects: (a) enriched accounts overlap with the genealogical account concerning certain phenomena (e.g. special homology and orthology), and (b) within the region of overlap, enriched accounts complete the genealogical account by explaining how it is possible for a particular kind of character to stand in descent relationships. Third, enriched accounts expand the reference of 'homology' beyond what is covered by the genealogical account, including such phenomena as serial homology, paralogy, and xenology.

According to this framework, genealogical and developmental accounts of homology are compatible and inextricably intertwined. Specifically, developmental accounts of homology are to be understood as one type of enriched account of homology, applying within a particular domain. Once this is recognized, developmental accounts are seen to be consistent with and indeed complementary to the genealogical account in the regions where they overlap.

I have defended a compatibilist view of the relationship between different accounts of homology. I have tried, not merely to register that distinct accounts serve distinct functions, but to make clear in what sense the accounts, despite their differences, concern the same thing. The key lies in recognizing the fine structure that binds together the many different uses of the term.

5.0 STRUCTURE AND FUNCTION

“The forms of the spicules are the result of adaptation to the requirements of the sponge as a whole, produced by the action of natural selection upon variation in every direction.” (Minchin 1898, 569)

“It would scarcely be possible to illustrate more briefly and more cogently than by these few words [...] the fundamental difference between the Darwinian conception of the causation and determination of Form, and that which is based on, and characteristic of, the physical sciences.” (Thompson 1992, 693, commenting on Minchin)

5.1 STRUCTURE AND FUNCTION

The history of biology is mottled with conflicts between structuralists and functionalist (Russell 1982; Asma 1996; Amundson 2005). The fiery dispute between Georges Cuvier and Etienne Geoffroy Saint-Hilaire before the French Academy of Sciences, during which the two grand principles of Conditions of Existence and Unity of Type struggled for dominance, is perhaps most famous (Appel 1987), but that is only the beginning. The debate

also played out in England in the divergent approaches of Richard Owen (2007) and Charles Darwin (1964). In the 20th century, Richard Goldschmidt (1982) and Otto Schindewolf (1993) offered structuralist critiques of the modern synthesis (Dobzhansky 1982; Mayr 1982; Simpson 1982). The rise of evolutionary-developmental biology around the 1980s generated (and continues to generate) new structure/function disputes (Webster and Goodwin 1982; Hughes and Lambert 1984; Alberch 1989; Ho and Saunders 1993; Kauffman 1993; Webster and Goodwin 1996; Newman and Bhat 2008). And, of course, there is the conflict between Thompson and Minchin highlighted in the epigraph. The list could be extended *ad nauseam*.

I have begun with a list of examples rather than with a characterization of the two competing positions, because the examples make perspicuous the major difficulty facing any attempt to develop such a characterization. What the examples show is that structuralism and functionalism have survived 200+ years of often drastic theoretical change in biology. Structuralism must be broad enough to capture Owen, Thompson, and Alberch; functionalism must subsume Lamarck, Minchin, and Mayr. At the same time, the positions must avoid being so broad as to be vacuous. My aim in this chapter is to develop an account of the nature of structuralism and functionalism that satisfies these desiderata.

Structuralism and functionalism are generally characterized in terms of the relative explanatory priority of structure and function: structuralists say that structure is explanatorily prior to function, while functionalists say the opposite (Russell 1982; Amundson 2005; Boucher 2015). Local structure/function disputes (over, say, the form of the spicules in sponges) then arise when structuralists and functionalists provide competing answers

to particular questions. On this approach, structuralism and functionalism, as positions or stances endorsed by individual scientists, are primary. Structure/function disputes are then characterized in terms of localized conflicts between these background positions or stances.

In this chapter, I take the opposite approach. I argue that local structure/function disputes are primary, and that structuralism and functionalism as background positions are secondary. More specifically, evolutionary biologists ask why-questions about the evolution of organismal form. These questions ask why X evolved rather than Y. Such questions can have either structuralist or functionalist answers, where the requisite notions of structuralist and functionalist answers can be precisely specified. On this basis, we can characterize structuralists as those who tend to give structuralist answers to the contentious why-questions of their day (and similarly for functionalists). Nonetheless, structuralists will happily give functionalist answers, and functionalists will happily give structuralist answers, to many why-questions. Moreover, the background theories that drive structuralists may be quite different, and so for functionalists.

The crucial piece of the analysis, in which the notion of structuralist and functionalist answers to why-questions is developed, comes late (Section 5.6), as substantial prep work is needed to motivate the view. I begin by arguing that standard presentations of structuralism and functionalism in terms of explanatory priority misdiagnose the issue. The disagreement concerns the proper role of function in explaining form (Section 5.2). I then discuss Darwin's functionalism and the key wrinkles his innovations introduced into structure/function disputes, as well as what they left unchanged (Section 5.3). With

evolutionary theory in the picture, I introduce the crucial distinction between the generation and the spread of variation (Section 5.4). I show that, in an evolutionary context, structure/function disputes stem from disagreements over issues concerning the generation of variation (Section 5.5). This then motivates my analysis of structuralist and functionalist answers to why-questions: structuralist answers involve the generation of variation, while functionalist answers involve the spread of variation (Section 5.6). Finally, I consider the status of structuralism and functionalism in light of my analysis of structure/function disputes (Section 5.7).

5.2 EXPLANATORY PRIORITY IS NOT THE ISSUE

Standard presentations of structuralism and functionalism treat them as disagreeing over whether form is explanatorily prior to function (structuralism) or vice-versa (functionalism). Here, I argue that, while there is a clear sense in which functionalists treat function as prior to form, there is no corresponding sense in which structuralists treat form as prior to function.

If structuralists and functionalists are to be understood as disagreeing over a matter of explanatory priority, then we need a characterization of explanatory priority. Here is a clear way to understand the issue: the explanans is prior to the explanandum. On this view, functionalists take function as explanans and structure as explanandum, and so

treat function as explanatorily prior to structure. Structuralists do the opposite. This appears to be how the issue is understood in much of the secondary literature. E. S. Russell (1982, ix), for instance, characterizes the debate as a dispute over the question, “Is function the mechanical result of form, or is form merely the manifestation of function or activity?” Stephen Asma (1996, 12) likewise writes, “the question was whether specific organic structure was the result of specific function or vice versa.”

This picture is true enough in the case of the functionalists, but it gets structuralists entirely wrong. This is best appreciated in the light of some examples. For functionalists, consider Lamarck and Cuvier. Lamarck offered a transmutationist theory according to which novel structures arise as a result of the origin of new organismic needs.⁴⁴ As organisms changed their behavior so as to better meet these needs, their behavior affected their physiology, leading to structural modifications inherited by their descendants. In this way, structural modifications arose in order to fulfill particular functions. Structure is explanandum; function is explanans. The same holds for Cuvier, who explained the features of organisms in terms of their conditions of existence (Coleman 1964; Burkhardt 1977; Russell 1982). Organisms, for Cuvier, were tightly integrated, structured so as to be suited for a very particular form of life. That form of life determined its structure. Once again, structure is explanandum, while function is explanans.

But now consider the manner in which Richard Owen, arch-structuralist, attempted to refute these functionalist views. Owen did not proceed by arguing that structure

⁴⁴ Lamarck’s theory also involved an environment-independent force that progressively drove organisms to greater complexity. This force is irrelevant to his functionalism, so I ignore it here.

explains function. Indeed, his arguments precluded that possibility altogether. In *On the Nature of Limbs*, Owen (2007) offered the example of the bat's wing, the dugong's fin, and the mole's forelimb. Each of these is adapted to a different function: the bat's wing for flight, the dugong's fin for swimming, and the mole's forelimb for digging. Nonetheless, each is structurally very similar. Each consists of the same bones in the same arrangement. The same structure thus serves many functions. Because of this one-to-many relationship between structure and function, Owen argued that function was impotent to explain these structural correspondences, or Unity of Type.⁴⁵

Note, however, that Owen's argument equally forecloses the possibility of explaining function in terms of structure. If the structure is the same in each case, then structure is insufficient to explain the differences in function. Owen's argument is a double-edged sword. In showing that function is not explanatorily prior to structure, it equally shows that structure is not explanatorily prior to function. But Owen was not sabotaging his own project, because Owen was not attempting to explain function at all. Owen was attempting to explain structure, just as Lamarck and Cuvier were attempting to explain structure. He argued that function could not explain structure. True, the bat's wing is admirably suited to flight, but the features that allow it to serve that function are minor modifications superimposed on a stable underlying structure. Function can explain those modifications, but not the archetypal structure that remains unchanged across multiple functions.

⁴⁵ Geoffroy made similar arguments (Appel 1987, 85; Asma 1996, 16).

Thus, for Owen, structure is independent of function, but it is not explanatorily prior to function. There is a sense in which structure is prior to function: functional modifications are superimposed on top of stable underlying structures. But this is not explanatory priority, at least not in the same sense. The claim that functionalists treat function as prior to structure, while structuralists treat structure as prior to function thus hides an equivocation: the sense of “prior” is different in the two cases. Moreover, the claim does not point directly to the really important difference between Lamarck and Cuvier on one side and Owen on the other.

The key issue at stake in the above structure/function dispute was the question: does function explain structure? The functionalists said yes. The structuralists said no. They shared exactly the same explanandum: both sought to explain structure. The disagreement entirely centered on the explanans. Note that this characterization makes functionalism a much more constrained position than structuralism. The functionalist must explain structure in terms of function. But the structuralist is only committed to denying that function explains structure.

This characterization imposes no requirements on what a structuralist believes does explain structure. That’s appropriate, since structuralists have offered quite diverse explanations of structure. Drawing on the list of structure/function disputes discussed earlier, we can get a sense of this variety. Richard Owen (2007) explained structure in terms of a quasi-Platonic archetype that lay behind the observed variation (but see Camardi 2001). Richard Goldschmidt (1982) and Pere Alberch and Emily Gale (1985) explain structure in terms of the potentialities of the developmental system to generate variants.

D'Arcy Wentworth Thompson (1992) and Stuart Newman and Ramray Bhat (2008) explain structure in terms of the action of physical forces. Despite these quite different (though not necessarily incompatible) explanations of structure, all minimize the explanatory role of function and are recognizable structuralists.

Thus far, then, we can understand functionalists as those who explain structure in terms of function and structuralists as those who claim that structure is largely independent of function. We will later see that this characterization is incomplete (Section 5.6). Especially within an evolutionary context, there are substantive constraints on how one can explain structure and still be a structuralist. In consequence, one can deny that function explains structure without being a structuralist. But, for now, this characterization will suffice.

5.3 DARWIN AND THE COMPLICATION OF FUNCTIONALISM

Darwin took Owen's challenge to functionalism seriously, and he used his evolutionary theory to develop more sophisticated resources for explaining structure in terms of function. In doing so, he reconfigured the nature of structure/function disputes in key ways. At the same time, key features of these disputes remain constant across the Darwinian revolution, and these constancies are equally important.

Owen, recall, had shown the impotence of function to explain structure on the grounds that the same structure in different species was used for many different functions. Darwin recognized the force of this argument and, in his genial fashion, accommodated it in his evolutionary theory. At the end of the sixth chapter of the *Origin*, Darwin (1964, 206) wrote:

It is generally acknowledged that all organic beings have been formed on two great laws—Unity of Type, and the Conditions of Existence. By unity of type is meant that fundamental agreement in structure, which we see in organic beings of the same class, and which is quite independent of their habits of life. On my theory, unity of type is explained by unity of descent. The expression of conditions of existence, so often insisted on by the illustrious Cuvier, is fully embraced by the principle of natural selection. For natural selection acts by either now adapting the varying parts of each being to its organic and inorganic conditions of life; or by having adapted them during long-past periods of time: the adaptations being aided in some cases by use and disuse, being slightly affected by the direct action of the external conditions of life, and being in all cases subjected to the several laws of growth. Hence, in fact, the law of the Conditions of Existence is the higher law; as it includes, through the inheritance of former adaptations, that of Unity of Type.

Darwin made two crucial moves in this passage. First, he explained Unity of Type in terms of unity of descent. Dugongs, bats, and moles share structurally similar forelimbs because they all descended from the same ancestor, and that ancestor likewise had a forelimb with the same structural arrangement. Natural selection then caused this structure to diverge in different lineages to serve different functions (thus explaining the Conditions of Existence). In this way, Darwin acknowledge and explained the central phenomenon motivating Owen's structuralism.

Darwin's view, based just on the above, is not yet a functionalist view. So far, he seems to have granted Owen's point entirely. Selection superimposes minor functional

modifications atop inherited structures. These structures are prior (in the literal, temporal sense) to their eventual functions. The role for functional considerations is, thus far, severely limited.

This is where the second move comes in. At the end of the passage, Darwin argued that Conditions of Existence is the “higher law” on the grounds that what an organism inherits are “former adaptations.” In this way, Darwin reduced Unity of Type to Conditions of Existence. Yes, selection superimposes modifications on top of inherited structure, but that inherited structure is itself the product of past selection. Unity of Type is just a product of the fact that Conditions of Existence exert their influence over time, rather than statically, at a single moment of creation.

By introducing history in this way, Darwin freed functionalist explanations from assumptions of optimality. Any explanation of current structure requires a two-part explanation: current function and past function. As Darwin put it in the B species notebook: “The condition of every animal is partly due to direct adaptation & partly to hereditary [*sic*] taint” (Barrett et al. 2008, 182). Past function explains ancestral structure, while current function explains modifications to suit current needs. Explanations are thus functionalist all the way down. But, because history is involved, form is now explained, not in terms of a single function, but in terms of the entire sequence of different functions that all left their mark on the structure. Darwin thus granted to Owen the impotence of current function to explain the shared structure of vertebrate forelimbs. But all vertebrate forelimbs have a substantially shared history, i.e., they were, for a good portion of their history, subject to the same functional constraints. For a Darwinian functionalist, able to cite

past as well as current function, the existence of a one-to-many relationship between structure and current function is not a problem.

The introduction of Darwin's evolutionary theory thus substantially changed the terrain of structure/function disputes. Darwin introduced a novel explanatory resource into the arsenal of functionalists (namely, past function), and in doing so he deprived the structuralists of one of their most powerful arguments.

At the same time, Darwin left much unchanged. The central issue in the disputes was still the role of function in explaining structure. It is true that Darwin tied the notion of function to the action of natural selection, and so gave it a new theoretical basis.⁴⁶ However, this does not make Darwin's functionalism radically different from that of his forebears. The adaptedness of organisms to their environments and the use of particular parts to serve particular functions is a phenomenon that is recognizable independently of our understanding of how it comes about. As has long been recognized in philosophy of science, phenomena (and characterizations thereof) have a life independent of the theories proposed to explain them and can remain stable across theoretical change (Bogen and Woodward 1988; Woodward 1989; Hacking 1992). The notion of function has, in just this manner, persisted across the Darwinian revolution (Asma 1996, chap. 4). Pre-Darwinian creationists offered one mechanism connecting function to structure (God's consideration of organisms' needs in the creation). Lamarck (1914, chap. VII) offered another (needs →

⁴⁶ The fact that structuralism and functionalism have survived radical theory change of this sort is a major motivator for Boucher's (2015) view that they are best understood as stances *sensu* van Fraassen, rather than as substantive theses (van Fraassen 2002; cf. Boucher 2014). More on this below (Section 2.7).

behavioral changes → physiological changes → inheritance of said changes). And Darwin offered a third (the struggle for life → selection → evolutionary change). All three invoke function as an explanans of structure, though they fill out the intervening links differently.

Considered from the perspective of structure/function disputes, Darwin's major innovation is therefore not his notion of natural selection. Natural selection offered a way of understanding how function shapes structure that was scientifically viable in a way that creationist and Lamarckian ideas were not. It did not, however, fundamentally reshape the explanatory task of functionalists. Far more important was his sophistication of the resources of functionalism, giving functionalists the ability to invoke past as well as present functions in explaining structure.

5.4 THE GENERATION AND SPREAD OF VARIATION

In a contemporary evolutionary context, a complete explanation of evolutionary change must have two components. It must state how variants were generated, and it must state why these variants spread or failed to spread within a population (Endler 1986). With the benefit of hindsight, we can see that older transmutationist views also provided answers to these questions, even though they did not always clearly distinguish them. In the next two sections, we will see that the distinction between these two questions is essential for understanding the structure/function disputes that arise in an evolutionary context. Here,

my main aim is simply to present the distinction clearly, and to draw a few preliminary implications for our understanding of structure/function disputes.

The distinction emerged most clearly in the context of population genetic modeling in the first half of the 20th century. These models include two kinds of evolutionary causes: those that introduce new genetic variants into a population (mutation, migration) and those that affect which mutations spread through a population (selection, drift). Mutation and migration are causes that affect the *generation* of variation; selection and drift are causes that affect the *spread* of variation.⁴⁷ In this way, the two processes (generation and spread) are separable. Evolutionary change involves both processes, and so a full explanation of any given evolutionary change will cover both.⁴⁸

This division of the problem space was an essential element of the modern synthesis. Here, for instance is Theodosius Dobzhansky in his important *Genetics and the Origin of Species* (Dobzhansky 1982, 13):

Mutations and chromosomal changes arise in every sufficiently studied organism with a certain finite frequency, and thus constantly and unremittingly supply the raw materials for evolution. But evolution involves something more than origin of mutations. Mutations and chromosomal changes are only the first stage, or level, of the evolutionary process, governed

⁴⁷ I am setting aside the many recent attempts to expand evolutionary theory to include substantial non-genetic components (cf. Pigliucci and Müller 2010). The same issue arises there: non-genetic variants must be generated, and they must spread through the population.

⁴⁸ This is not to say that an adequate evolutionary explanation always has to cover both—far from it (this will be important below). For instance, when a population encounters an environmental change, selection often operates on standing genetic variation. Simply looking at the population's response to the environmental change, no new variants are needed and so an explanation can involve only the spread of variation, ignoring how the variants were generated. Nonetheless, if we ask about the origin of the standing genetic variation, in the background were processes of both generation (to produce the variation) and spread (to maintain it).

entirely by the laws of the physiology of individuals. Once produced, mutations are injected in the genetic composition of the population, where their further fate is determined by the dynamic regularities of the physiology of populations. A mutation may be lost or increased in frequency in generations immediately following its origin, and this (in the case of recessive mutations) without regard to the beneficial or deleterious effects of the mutation. The influences of selection, migration, and geographical isolation then mold the genetic structure of populations into new shapes, in conformity with the secular environment and the ecology, especially the breeding habits, of the species. This is the second level of the evolutionary process, on which the impact of the environment produces historical changes in the living population.

Dobzhansky's two levels are, quite clearly, the generation of variation (first level) and the spread of variation (second level).⁴⁹ This distinction between problems of generation and problems of spread is maintained in contemporary evolutionary theory (Endler 1986).

One might reasonably worry that this distinction only applies to those biologists who accepted it explicitly, and that it distorts the views of others to force this distinction upon them. In fact, however, the distinction is illuminating when applied to other transmutationist thinkers. In applying this distinction to biologists as diverse as Lamarck, Thompson, and Goldschmidt, we can notice an interesting feature of their views. All three presented mechanisms for the generation of variation that also accounted for their spread. That is, their views entailed that both problems could be solved in a single step. For Lamarck, because organisms shared the same environment, each individual would generate and pass on the same sort of variant to their offspring. Thus, no additional factors need to be cited to explain the spread of variation. Meanwhile, for Thompson: the same physical forces act on all individuals of the same species, so any aspects of form that are

⁴⁹ Dobzhansky goes on to discuss a third level, concerning the mechanisms that create distinct populations, but this is irrelevant to my concerns here.

determined by these forces will be the same in all individuals. Again, what accounts for generation also accounts for spread. Goldschmidt's view also solves both problems in one step, though in a different way. For Goldschmidt, the developmental system has the potential to generate large novelties via a single mutation. The "hopeful monsters" so generated are so different from other individuals that—if they are viable—they found a new population in one step.⁵⁰ The novel features of the hopeful monster are thus immediately at fixation in the new population, so neither selection nor drift are required to aid their spread.

The distinction thus usefully illuminates each of their views, both individually and in virtue of showing interesting commonalities between them. Note that these commonalities exist across the divide between structuralists and functionalists: both functionalists (Lamarck) and structuralists (Thompson, Goldschmidt) can offer theories that solve both problems together. The inverse is true as well: both functionalists and structuralists can keep the two problems separate. This is obviously true for functionalists such as Dobzhansky, but it is equally true for the structuralist arguments of Pere Alberch and Emily Gale (1985; discussed further below, Section 5.6). Thus, in making this distinction, I am not prejudging the issue in favor of either camp.

The distinction is also useful for clarifying the structure/function disputes that arise within the context of Darwinian evolutionary theory. In Lamarck's transmutationist theory, functional concerns directly affect the generation of variation. Though Darwin was

⁵⁰ This generates its own problems: in sexual lineages, it would seem that at least two of the same hopeful monster must be generated at one time to make this possible. But this issue does not affect the points I am making.

sympathetic to Lamarckian ideas about the generation of variation, post-Mendelian Darwinians have strongly rejected them. Variants are generated without regard for the organism's needs. Functional considerations arise only when it comes to the problem of spread. Thus, for a Darwinian, *functionalist explanations must be expressed in terms of the spread of variants through a population*. In this regard, Darwin stands with the structuralists against Lamarck's functionalism. Both Darwin and his structuralist critics agree that the generation of variation cannot be explained functionally.⁵¹

At this point, if I have been successful, the prospects for the integration of structuralist and functionalist perspectives should be seeming pretty good. Structuralists and (Darwinian) functionalists both recognize the need to explain both the generation and the spread of variation. Both agree that functional explanations are restricted to the spread of variation. This leaves (or so it seems) a lot of room for structuralists to make important contributions concerning the generation of variation. Since any complete explanation of form must involve an account of both generation and spread, it would seem that such an explanation must integrate structuralist and functionalist concerns. So how do structure/function disputes arise?

⁵¹ On the flip side, insofar as proponents of the extended evolutionary synthesis (Pigliucci and Müller 2010) argue that at least some variation can be explained functionally, there is a limited sense in which they are Lamarckian.

5.5 THE ORIGINS OF STRUCTURE/FUNCTION DISPUTES IN AN EVOLUTIONARY CONTEXT

In this section, I defend two claims about the nature of structure/function disputes in an evolutionary context. I argue, first, that they concern the sources of direction in evolutionary theory. Second, I show that the driving force of these disputes are disagreements about how variation is generated. There may be downstream disagreements about how variation spreads in a population, but these are consequences of the more basic disagreement about how variation is generated.

The previous section painted an optimistic picture about the prospects of reconciling structuralism and functionalism. This optimistic picture might be further motivated by considering a biologist who managed to integrate the two approaches to at least some degree. I have in mind Charles Darwin. Darwin (1964, chap. 5) devoted an entire chapter of the *Origin* to the issue of the laws of growth. He was especially interested in correlated variation, in which two parts (call them S and T) that seem independent vary together. When variation in S is correlated with variation in T, then selection for changes in S will inevitably result in changes in T. To explain why T evolved as it did, we need to invoke both selection (acting on variation in S) and facts about the potentialities of the developmental system, namely that it produces correlated variation.

In this explanation, the direction of evolutionary change is explained in part by selection, in part by potentialities of the developmental system. Though this type of explanation is a happy case of integration, in other cases the question of the sources of direction

in evolutionary change is precisely what gives rise to structure/function disputes. One of the central claims of the (functionalist) modern synthesis was that selection is the only direction-giving factor in evolution (Mayr 1969a; Mayr and Provine 1981). In making this claim, the proponents of the synthetic theory intended to exclude both neo-Lamarckian theories that let functional needs influence the generation of variation and structuralist theories that dispensed altogether with the need for selection to explain directional evolutionary change. In this latter camp, besides Goldschmidt, were orthogeneticists who allowed internal features of the organism to drive evolutionary change (e.g. Schindewolf 1993).

The claim that natural selection is the only direction-giving factor in evolution requires careful exegesis. If it is taken to mean that the generation of variation plays no role whatsoever in determining the direction of evolutionary change, it is a remarkably strong claim. It is a strong claim because it is committed to the view that variation is *isotropic* (or nearly so): that variation is generated equally in all directions (cf. Minchin in the epigraph).⁵² The processes that generate variation furnish selection with varieties of all sorts, in sufficient frequencies that all the work of determining which variants survive into future generations is done by selection (and by drift, which is random and *ipso facto* not direction-giving). Variation by itself does not bias (let alone determine) the direction of evolutionary change.

There are two problems with this assumption: it is in many cases arguably incoherent, and where it is coherent it tends to be false. It is arguably incoherent whenever the

⁵² Pere Alberch (1980, 653–54) accuses neo-Darwinians of just this assumption.

directions of variation are considered qualitatively.⁵³ At some point in evolutionary history, scales evolved into feathers (Wagner 2014, chap. 9). But scales might have evolved into any number of other things: skin, hair, an outer shell, or any of the kinds of outer covering of animals that exist only in possibility space. Could there be variation in the direction of all of these infinite possibilities? Clearly not in any actual (hence finite) population. The notion can be made coherent in cases where a property is quantitative and variation can be characterized in terms of increases or decreases (e.g., height), since in such cases there are limited directions in which variations might arise. Even in such cases, however, the assumption that variation is isotropic is usually false (Arthur 2001).

But this strong form of the assumption cannot possibly be what the architects of the synthesis actually meant, for the simple reason that they were aware of correlated variation. In the case considered above, Darwin granted both structural and functional contributions to evolutionary direction. Without selection favoring changes in S, there would be no change in either S or T. But selection alone cannot explain the directional change in T. The features of the developmental system that produce correlated variation between the two parts are necessary to explain this. If, for instance, S and T were correlated in the inverse direction, then the same selection pressure on S would have driven T to evolve in the opposite direction. So features of development are direction-giving factors.

Since the synthesists accepted such cases as this, they could not have meant to exclude any role for the generation of variation in explaining evolutionary direction. A more charitable interpretation of their claim is two-fold: that variation is present in many (not all)

⁵³ I thank Jim Woodward for pressing me on this point.

directions, and that selection is essentially involved in any directional evolutionary change. That is certainly true of this case, where selection is required to for the directional evolution of both S and T. This view also rules out the views they rejected: orthogenetic invocations of “mutation pressure” explain directional change without selection, as do Lamarckian views which render selection redundant by having the variation generated in each individual. Goldschmidt is a trickier case, as he allowed selection a minor role in eliminating non-viable, “hopeless” monsters. But even in his case, the primary direction of evolution is determined by what mutants are generated, not by a process of selection accumulating small changes over time.

We are now in position to see that structure/function disputes, in an evolutionary context, invariably involve disagreements concerning the generation of variation. As we saw, on the strong reading of the synthesists’ claim that natural selection is the only direction-giving factor in evolution, this claim rests on a very restrictive assumption about how variation is generated, namely that it is generated isotropically. If variation is generated in an anisotropic manner, then developmental bias has a role to play in shaping the direction of evolutionary change (Arthur 2001). On a more plausible interpretation of the claim, the assumption of isotropy is relaxed, but certain pictures of how variation is generated are ruled out. Goldschmidt’s emphasis on large mutations that accomplish speciation in a single step, for instance, gives too great a directional role to the processes that generate variation. While selection is given a role in filtering out the nonviable of these large variants, selection’s role in accumulating small variants is restricted within species boundaries. Its direction-giving role is thus minimized.

The same is true for the dispute between Minchin and Thompson. Minchin explains the evolution of spicules in terms of selection acting on “variation in every direction.” Selection (and thus function) is of especial importance because variation by itself is non-directional. Thompson, in just the opposite manner, allows variation to entirely determine form, because all variation is in one direction (that determined by physical forces). No role remains for selection. While they do conflict over the role of selection, they do so in virtue of their views about the nature of variation. Their conflict is first and foremost about the generation of variation.

In both cases, then, the heart of the dispute concerns the generation of variation. There may be downstream conflicts over the role of selection in determining organismal form, but these conflicts are the consequences of the deeper disagreement over the problems of generation. Functionalists, as much as structuralists, are committed to some answer to the question of how variation is generated.

We may therefore say that structure/function disputes arise when disagreements over the way in which variation is generated lead to further disagreements about the sources of directional evolutionary change.

5.6 WHY-QUESTIONS AND LOCALIZED STRUCTURE-FUNCTION DISPUTES

In the foregoing, I have attempted to lay out some of the major issues that characterize conflicts between structuralists and functionalists. Here, I tie these threads together into a precise specification of what constitutes a structure/function dispute in the context of evolutionary theory. Specifically, I give an account of what it means to give a structuralist or a functionalist answer to a specific contrastive why-question. I argue that the twin notions of structuralist and functionalist answers to particular contrastive why-questions are prior to any characterization of structuralism and functionalism more generally.

Evolutionary biologists seek both (i) to develop a general picture of the evolutionary process and (ii) to explain the evolution of particular structures. The majority of the philosophical discussion of structuralism and functionalism has concerned general pictures of evolutionary theory. Goldschmidt's structuralist theory, for instance, conflicted with the functionalist theory established during the modern synthesis. It is tempting, on this basis, to seek to characterize structuralism and functionalism in terms of the type of overarching theory one endorses.

I propose to take the opposite approach. In understanding structuralism and functionalism, it is better to begin with structure/function disputes as they arise concerning the evolution of particular structures. One of the most profound lessons of evolutionary theory is that evolutionary processes are themselves evolved. Different processes dominate in different lineages. As a result, biologists trade in restricted generalizations, not universal laws (Beatty 1995; Mitchell 2000). It is theoretically possible that much

evolution occurs in accordance with the synthetic picture on which selection accumulates mutations with small effect sizes, but that in some cases Goldschmidtian hopeful monsters have arisen and founded a new species in a single step. Thus, while a background picture of how evolution works may generate expectations about a particular case, ultimately each case must be taken on its own terms.

When biologists seek to explain the evolution of particular structures, local structure-function disputes arise. These disputes enjoy at least semi-independence from any background commitment to a structuralist or functionalist general picture, however such might be characterized. In these local structure/function disputes, explanations of the evolution of any particular structure can be broken down into answers to distinct why-questions (on why-questions, cf. van Fraassen 1980, chap. 4). In answering these why-questions, structuralist answers (in terms of the generation of variation) or functionalist answers (in terms of the spread of variation) can dominate to the exclusion of the other.

Consider the case of digit reduction in amphibians (Alberch and Gale 1985). Both anurans (frogs) and urodeles (salamanders) contain lineages that have lost all or part of certain digits. These cases of digit reduction show two interesting patterns. First, there are distinct trends within both the anuran and urodele lineages. For instance, anurans that have lost only one phalange always lose a phalange from the first digit, while urodeles that have lost only one phalange always lose a phalange from the fourth digit. Second, as the example shows, these trends are different within the two lineages. Anurans and urodeles lose distinct phalanges first.

How are we to explain these patterns of digit reduction? Alberch and Gale contrast two strategies. The first, which they prefer (and experimentally substantiate) is that both urodele and anuran developmental systems are highly constrained in their potential for digit reduction, such that, if a mutation causes loss of a single phalange, it always causes loss of the same one. That is, selection can favor digit reduction over digit increase, but it cannot favor (in anurans) loss of a phalange from the first digit over loss of a phalange from the third digit, because only the first type of variant is produced. The trends within urodeles and anurans are thus explained by shared potentialities of their developmental systems, while the differences are explained by the fact that the anuran and urodele developmental systems have different potentialities.

On the functionalist approach, by contrast, any developmental constraints are weak and local: weak in the sense that many different phalanges might be the first to be lost, local in the sense that different urodeles would have different constraints (and same for anurans). If so, shared constraints can obviously not explain these evolutionary trends, in which case selection is the most likely explanation. Specifically, trends seen in multiple lineages are likely due to similar selection pressures operating in each lineage.

So far, this fits the basic pattern we have considered. The functionalist makes assumptions about how variation is generated that leave most of the work of determining evolutionary direction to selection. Meanwhile, the structuralist makes different assumptions according to which the generation of variation substantially limits the course of evolution (in this case, in the same way across multiple lineages).

In moving to a particular case, however, we are able to ask more precise questions. Now the task is not to explain organismal form in general, but to explain the evolution of particular forms, as well as to explain common features of multiple cases of such evolution. Explanations answer why-questions, which have the structure “why X rather than Y?” In the context of evolutionary theory, this amounts to: why did X evolve rather than Y? For example: why did those anuran lineages that lost digits lose digit one first, rather than any other digit? In asking these specific questions, we can precisely localize the structure/function dispute at issue. The key is to distinguish structuralist answers to these questions from functionalist answers. I define these as follows:

- **Structuralist answers** exclusively invoke biases in the generation of variation to explain why X evolved rather than Y.
- **Functionalist answers** exclusively invoke selection influencing the spread of variation to explain why X evolved rather than Y.

The emphasis on exclusivity is important. As we will see shortly, the structuralist and functionalist positions that Alberch and Gale consider both give answers to the why-questions at issue that satisfy the exclusivity requirement. But it is possible also to give a **hybrid answer** to such questions. Such an answer allows both selection and biases affecting the generation of variation to influence the direction of evolution (Arthur 2001). Finally, **neutral answers** are also possible – neutral in sense that they invoke neither structuralist nor functionalist factors. An explanation in terms of drift operating on isotropic variation would be neutral in this way.

Let us return to the case of amphibian digit reduction. In this case, there are many why-questions we might ask. Here is a sample:

- (W1) Why did these anuran lineages lose a phalange from its first digit, while those other anuran lineages did not?
- (W2) Why did these anuran lineages lose a phalange from its first digit, rather than gain (a) phalange(s) or not change at all?
- (W3) Given that they all lost a phalange, why did these anuran lineages all lose a phalange from their first digit, rather than some other phalange?
- (W4) Given that they all lost a phalange, why do anuran and urodele lineages lose a different phalange first, rather than the same phalange?
- (W5) Why did this particular anuran lineage lose a phalange from its first digit, rather than from some other digit.

On both the structuralist and the functionalist views, W1 and W2 receive answers in terms of selection (or potentially drift). That is, Alberch and Gale accept that there was selection pressure for digit reduction rather than increase (answering W2) that operated only in certain lineages (answering W1). In other words, both the “structuralist” and the “functionalist” offer a functionalist answer to these questions. Note that structuralist answers to both W1 and W2 are possible. For W2, for instance, Alberch and Gale might have argued that the potentialities of the amphibian developmental system are such that variants are only generated in the direction of reduction, never increase.

Moving to the opposite extreme (W5), in allowing that the functionalist could accept local developmental constraints (i.e. allowing the functionalist to reject that variation is

isotropic), Alberch and Gale suggest that the functionalist could say, of a particular lineage, that developmental constraint at least partially explains why that lineage lost a particular phalange first. In that case, once again the “structuralist” and the “functionalist” offer the same answer to a why-question, only this time they both accept a structuralist answer, on which the generation of variation is the key explanatory factor. Here, again, the functionalist could offer a selective explanation rather than an explanation in terms of developmental constraint.

The structuralist and functionalist disagree only in their answers to W3 and W4. W3 asks, over a range of cases, why evolution went in X direction rather than Y direction, while W4 asks why it went in X direction (rather than Y) in one set of cases and in Y direction (rather than X) in another. The structuralist, in both cases, gives an answer in terms of what variation was generated: variation in the X direction was generated, while variation in the Y direction was not. The functionalist, by contrast, assumes that variation was generated in both the X and Y directions, and lets selection do the work of determining which direction is followed.

In this way, the structure/function dispute is localized to specific questions. So localized, structuralist and functionalist answers can be precisely defined, in a way that is difficult when asking about “structuralism” and “functionalism” more generally. Note, also, that these local structure/function disputes have all the key features of such disputes discussed in earlier sections. Both parties share an explanandum, namely structure (Section 5.2), though now the explanandum is contrastive. These explanations involve both inheritance (the normal amphibian digit pattern) and subsequent modification (digit

reduction) (Section 5.3). The disagreement is the product of disagreement over the nature of the variation that is generated (Section 5.5). This leads to disagreement about the causes of directional change (Section 5.5), where direction, too, is understood contrastively. This analysis of structuralism and functionalism in terms of structuralist and functionalist answers to why-questions thus does justice to the conclusions of the foregoing sections.

While this analysis makes clear what it means to give a structuralist or a functionalist answer to a why-question, it makes fuzzy what it means to be a structuralist or a functionalist. Both the “structuralist” and the “functionalist” agree that functionalist answers are needed for some why-questions (W1, W2) and that structuralist answers are needed for others (W5). Both, that is, agree that both the generation of variation and the spread of variation contribute to determining the direction of evolutionary change. Both contribute to an explanation of digit reduction in amphibians. It is true that Alberch and Gale give structuralist answers to more why-questions than their hypothetical functionalist interlocutor, and in that sense are the “structuralists” in this particular structure/function dispute. But, in a dispute with someone who gave structuralist answers to W1 and W2, they would be on the functionalist side of a structure/function dispute.

And this is only looking at one case. When we consider the full range of cases – that is, when we attempt to present a general evolutionary picture – we have to keep in mind Beatty’s (1995) point about evolutionary contingency. The developmental systems whose potentialities create biases in the generation of variation are themselves evolved. Different lineages (and different traits within a given lineage) may experience more or less anisotropic variation. And even though physical forces are in a sense universal, what forces

are relevant to a particular lineage depend on that organism's size and its environment (Thompson 1992). Microscopic aquatic bacteria, water surface-dwelling insects, large mammals, and birds all experience remarkably different physical forces.

For these reasons, it is somewhat artificial to speak of "structuralists" and "functionalists" generally. Anyone who accepts evolutionary theory is *ipso facto* committed to explaining evolutionary change in terms of both the generation and spread of variation. Anyone who rejects that variation is isotropic (that is, everyone) is committed to allowing that some why-questions will receive structuralist answers. The differences concern the particular why-questions to which one gives structuralist or functionalist answers. Some biologists will give structuralist answers more often. Some will give functionalist answers more often. But there is no specific set of why-questions to which one must give a structuralist (functionalist) answer to count as a structuralist (functionalist). It is thus the local disputes that are primary. The rest is a relative significance dispute (Beatty 1995). High-level structure/function disputes, that is, disputes that are not about the answers to particular why-questions, but instead concern the relative frequency with which structuralist or functionalist answers will be appropriate over some range of cases, are best understood as inductions from known cases (potentially with the aid of general theoretical models) to unknown cases.

There is also another, more interesting way to understand structuralism and functionalism beyond the confines of particular why questions. I am thinking of views that might be called methodological structuralism and methodological functionalism. Much has been written about the latter under the heading "methodological adaptationism" (e.g.

Godfrey-Smith 2001). The basic premise is that, by seeing how selection has failed to optimize traits, we learn where to look for constraints on selection. In other words, one assumes that most why-questions will receive functionalist answers in order to discover those cases that do not.

Equally possible, though less discussed, is methodological structuralism. As we have seen, there is a trivial sense in which structure is prior to function: selection can only act on the variation provided to it. The generation of variation is thus prior to the spread of variation. Thus, by studying what variants a developmental system produces, we can better discover which why-questions will plausibly require functionalist answers. This approach is advocated by Alberch and Gale (1985, 18–19):

However, there are some differences between expectations from ontogeny and the morphologies observed in nature that do imply functional (selective) constraints on design. [...] This highlighting of dissociations from the expected patterns is one of the important insights of the developmentalist approach. The problem for a functionally oriented evolutionist is, now, not to explain the repeated reduction of the first toe in anurans but rather the persistence of the fourth phalange in the fourth toe.

Alberch and Gale found that experimental perturbations of anuran development frequently caused the loss of a phalange in the fourth toe. Digit reduction in actual anuran lineages, however, always proceeded by loss of a phalange in the first toe. This suggests that there was a role for selection in favoring one pattern of digit reduction over the other. That is, by beginning with an understanding of the nature of variation generated by the anuran developmental system, Alberch and Gale were able to determine that a particular why-question likely required a functionalist answer.

I take it as a virtue of my account that it makes clear what, exactly, methodological structuralism recommends, and how it contrasts with the recommendations of methodological adaptationism. This does not address, however, the value of methodological structuralism, which remains relatively unexplored in the philosophical literature, especially compared to the attention devoted to methodological functionalism. It is an area worthy of further study.

5.7 STRUCTURALISM AND FUNCTIONALISM REVISITED

I have argued that the key to understanding structuralism and functionalism lies in looking at local structure/function disputes. These disputes are empirical disagreements about the answers to particular why-questions. Once this is understood, the need to characterize structuralism and functionalism as high-level positions is lessened. The substance of my view is now on the table. It only remains to contrast my approach with another recent attempt to analyze structuralism and functionalism.

Sandy Boucher (2015) has recently defended the view that structuralism and functionalism should be understood as stances *sensu* van Fraassen (2002). It is not incompatible with my view that there should exist structuralist and functionalist stances of the sort Boucher describes. As such, our accounts are not in direct conflict. However, we disagree on what is most central to understanding structure/function disputes in biology: I take

local structure/function disputes to be most important and to be explicable independently of stances, while Boucher sees structuralist and functionalist stances as driving the disputes. Moreover, Boucher's defense of the stance view raises a serious objection to the account I have developed. As such, it is worth considering.

A stance, according to Boucher (2015, 388), has five key properties. First, it is a cluster of attitudes and not a set of beliefs (nor is it reducible to a set of beliefs). Second, as a cluster of attitudes, a stance can be adopted but not believed. Third, because a stance is a cluster of attitudes, it is not propositional and therefore not truth-apt. Fourth, adoption of a stance is driven by one's values (epistemic and non-epistemic). Fifth, adoption of a stance is justified pragmatically rather than epistemically.

The appeal of thinking of structuralism and functionalism as stances stems from their persistence across radical theory change. It seems we can meaningfully identify both structuralists (Geoffroy, Owen, Thompson, Goldschmidt, Alberch, Newman) and functionalists (Lamarck, Cuvier, Darwin, Minchin, Dobzhansky, Dennett, Carroll) across the last 200+ years of biological science. But our understanding of both structure and function has changed dramatically over this time. The worry is that any belief about the relation between structure and function that could survive the last 200 years of theory change in biology must be vague to the point of vacuity. If there is anything linking those biologists we call functionalists and those we call structuralists, then it must be something other than shared beliefs. And the best candidate seems to be a stance (Boucher 2015, 393).

This argument against the possibility of finding any contentful beliefs that all structuralists or all functionalists share casts doubt on my attempt in the foregoing to give a

general characterization of structure/function disputes. The fact that my analysis of local structure/function disputes involved taking on board aspects of contemporary evolutionary theory only furthers this worry. It is clearly not applicable in the form developed above to the various structuralists and functionalists who were not transmutationists (i.e. who rejected the possibility of evolutionary change).

These worries can be defused by separating out two projects I have been engaged in this chapter. One project is to clarify the nature of contemporary structure/function disputes. Given that all serious biologists today accept evolutionary theory in some form, it is useful to understand the specific nature of the structure/function disputes in which they are engaged, even at the expense of losing some historical generality.

At the same time (second project), I have attempted to capture what did remain constant in structure/function disputes across the Darwinian revolution. This is the analysis given in Sections 5.2 and 5.3, where I showed that such disputes were not over the explanatory priority of structure or function, but over the role of function in explaining structure. I already argued there that this question is substantive even in the absence of a shared background theory. Understanding structuralism and functionalism in terms of the distinction between structuralist and functionalist explanations eliminates the need to think of them in terms of stances.

This leaves open the question of whether my claims about the priority of local structure/function disputes apply to pre-Darwinian structuralists and functionalists. Such biologists obviously did not have arguments over the relative roles of the generation of variation and the spread of variation in determining evolutionary direction. Nonetheless,

they did engage in local structure/function disputes. Consider the following two why-questions:

(W6) Why do the dugong's fin, bat's wing, and mole's forelimb share the same underlying structure, rather than have different underlying structures?

(W7) Why is the dugong's forelimb a fin rather than a wing?

Richard Owen would answer W6 by invoking the vertebrate archetype that all three organisms share, and he would answer W7 in terms of the dugong's aquatic mode of life. Georges Cuvier, by contrast, would answer both questions in terms of the organisms' modes of life (conditions of existence). While these answers are not in terms of the generation or spread of variation, they are nonetheless recognizably structuralist (Owen's answer to W6) or functionalist (Owen's answer to W7; Cuvier's answers to W6 and W7) in a broader sense.

In Section 5.2, the issue was raised that structuralists have, historically, offered quite different explanations of structure. These explanations were primarily unified in virtue of not being functional. In an evolutionary context, however, we can recognize a greater unity to structuralist explanations: they invoke properties of the generation of variation to explain evolutionary direction. More work is required to determine whether a similar unity can be found for pre-Darwinian structuralism – perhaps in terms of non-functional restrictions on possible form. I think the prospects are promising, but it is possible that it was only with the Darwinian revolution (and subsequent developments) that structuralist approaches came to be unified in this way. Deciding between these two possibilities would require deep historical analysis that is beyond the scope of this chapter.

5.8 RECAPITULATION

In this paper, I have attempted to provide an analysis of structure/function disputes in biology that (a) accounts for the persistence of these disputes across 200+ years without rendering structuralism and functionalism vacuous and (b) explains the nature of contemporary structure/function disputes, which occur within the context of evolutionary theory. The key claims I have defended are:

- (1) Structuralism and functionalism are best characterized in terms of the role they ascribe of function in explaining structure. That is, they share the same explanandum, but differ over the explanans (Section 5.2).
- (2) As a consequence of (1), structuralism and functionalism should not be understood as disagreeing over the explanatory priority of structure or function (Section 5.2).
- (3) Darwin, though he substantially sophisticated the resources available to the functionalist and in doing so deprived structuralists of one of their strongest arguments, did not change the central issue identified in (1) (Section 5.3).
- (4) As a consequence of (3), structure/function disputes can be understood as persisting across the Darwinian revolution (Section 5.3). This does not require treating them as stances *sensu* van Fraassen (Section 5.7).

- (5) In the context of evolutionary theory, structure/function disputes are generated when disagreements about the generation of variation lead to disagreements about the sources of directional evolutionary change (Section 5.5).
- (6) In the context of evolutionary theory, local structure/function disputes take the form of divergent answers to contrastive why-questions. Functionalist answers to these why-questions invoke the spread of variation, while structuralist answers invoke the generation of variation (Section 5.6).
- (7) These notions of functionalist and structuralist answers to particular contrastive why-questions are prior to understandings of structuralism and functionalism that transcend such local contexts, in the sense that the latter should be characterized in terms of the former (Section 5.6). It is an open question whether this point applies to pre-Darwinian structuralists and functionalists (Section 5.7).
- (8) One particularly interesting higher-level form of structuralism is methodological structuralism, which is a counterpart to methodological functionalism (adaptationism). Methodological structuralism is worthy of further philosophical investigation (Section 5.6).

6.0 CONCLUDING REMARKS

In the foregoing, I have made the case that, when the conceptual foundations of evo-devo are properly understood, the prospects for integrating evo-devo with the rest of evolutionary theorizing look quite good. Viewed in light of my analysis, what appeared to be deep, intractable, non-empirical conflicts between evo-devo and synthesis-derived evolutionary theorizing turn out to be a mix of merely empirical conflicts and complementary approaches to understanding evolutionary phenomena. I've made that case above; it does not need to be summarized again here. Instead, I'd like to end by drawing out a more general moral for understanding conflict and theory change in science.

Baldly stated, the moral is that cases of theoretical conflict are best analyzed, not in terms of large, complex packages of views, but by trying to localize the conflict as much as possible. My approach, in other words, proceeds in the opposite direction from classic accounts of theory change in science, couched in terms of complex, multi-faceted paradigms (Kuhn 1962; Hoyningen-Huene 1993) or research programmes (Lakatos 1970). Where those approaches sought to explain local conflicts in terms of large-scale background structures that give rise to them—in short, to see local conflicts as the product of higher-level conflicts—I prefer to see large-scale conflicts as the byproduct of local

conflicts. While paradigms or research programmes may be fruitful units of analysis for many purposes, my contention is that conflicts are best analyzed at smaller scales.

Why favor the local approach? In each chapter, I've tried to show the merits of the local approach for the specific conflict at issue, but there are also two general reasons, which I'll discuss here. One of these is ontological, the other pragmatic. The ontological reason for favoring the local approach stems from the complexity of paradigms and research programs. Kuhnian paradigms contain exemplary explanatory successes, a set of theoretical resources for solving puzzles, shared epistemic standards, active intra-paradigm disputes, and more. Lakatosian research programmes contain a hard core and an auxiliary belt, with positive and negative heuristics to guide the revision of the auxiliary belt. At the same time, paradigms and research programmes are supposed to confront (and possibly replace) their rivals as units. This is difficult to maintain, however: precisely because these large-scale constructs contain independently movable parts, those parts can and will interact with the parts of other paradigms or research programmes in a variety of ways. Even if there is conflict at the higher level, there may be productive cross-talk at the lower level, and this cross-talk gets washed out if the analysis only focuses on the large-scale conflict (cf. Sterner and Lidgard 2018).

Relatedly, the internal diversity of paradigms or research programmes can also be washed out by excessive emphasis on the larger scale. As I noted in the introduction, identifying what is shared among all proponents of the modern synthesis (let alone what is shared among both them and all of their descendents) is an impossible task, and the same is true for evo-devo. Nearly every aspect of evolutionary theorizing important to

the synthesis (a) was disputed during the synthesis period and (b) has been revised in the half-century since. Evo-devo is younger, but it too has undergone near-constant revision (for instance, from an emphasis on master genes to an emphasis on networks and their topologies). As they have changed, their relations to one another have also changed, and they have changed in ways difficult to capture if one focuses only on what unites them as paradigms or research programmes.

Moreover, continuing the ontological theme, because these large-scale constructs contain multiple moving parts, there is the possibility that different ways of grouping these parts will lead to cross-cutting classifications of scientific conflict. Chapters three and five above furnish a clear example of this. The conflict between evo-devo and synthesis-derived theorizing have been identified as the latest in a long line of conflicts between structuralist and functionalist approaches to biology (Amundson 2005). In fact, however, functionalism lumps together parts of evo-devo with synthesis-derived theorizing and sets both off against other parts of evo-devo. Such cross-cutting classifications can lead to alliances that seem strange (insofar as they appear to jumble separate paradigms), as when Stuart Newman and Ramray Bhat (2008) criticize the functionalist wing of evo-devo for being neo-neo-Darwinian.

The pragmatic reason to favor a local approach to understanding theory change and theoretical conflict is that it makes it easier to see how theoretical conflicts can be empirically tractable. For example, understanding structuralism and functionalism as stances (Boucher 2015) leaves us in a position where the merits of each stance are difficult to evaluate. Good work has been done on both sides – is biology then caught in an eternal,

irresolvable conflict between them? By contrast, understanding structuralism and functionalism as explanatory strategies (chapter five) makes clear (a) why both strategies have proven and will continue to prove fruitful, (b) how the strategies can be integrated in unified explanations, (c) why local structure/function disputes may nonetheless be expected to regularly arise, and (d) how such local disputes can be rendered empirically tractable. This approach also allows us to recognize how distinct functionalist approaches—such as Darwinian and Cuvieran functionalism—may complement one another, the one useful for explaining evolutionary change, the other for explaining evolutionary stasis.

The conflicts between evo-devo and synthesis-derived evolutionary theorizing are real. They show up in the biological literature with some regularity (a sampling: Alberch 1989; Gilbert, Opitz, and Raff 1996; Gilbert 2003a; Cracraft 2005; Jenner 2006; Davidson and Erwin 2006; Coyne 2006; Erwin and Davidson 2006; Hoekstra and Coyne 2007; Carroll 2008; Davidson 2011; Laland et al. 2014, 2015). But if we understand them as conflicts between evo-devo and synthesis-derived evolutionary theorizing, they come to seem greater than they are, and Amundson's (2005) conclusion that the two research programs are incommensurable is difficult to avoid. On the other hand, if we break these programs down into their parts and localize their conflicts to the particular parts that give rise to them, we can mitigate the tensions and uncover the many fruitful possibilities for integrating evolutionary theory's diverse explanatory resources.

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