5 Convergent Evolution as Natural Experiment

George Gaylord Simpson, one of the most influential evolutionists of the twentieth century and an architect of the modern biological synthesis, begins his skeptical paper on the search for extraterrestrial intelligence (SETI) by proclaiming that "we can learn more about life from terrestrial forms than we can from hypothetical extraterrestrial forms."[1] Simpson's argument would be elaborated on extensively several decades later by macroevolutionist Peter Ward and planetary scientist Donald Brownlee in their influential book *Rare Earth: Why Complex Life Is Uncommon in the Universe*.[2] Lamenting that we are unlikely to discover an extraterrestrial instance of life, Simpson concludes that SETI monies would be better spent on terrestrial concerns. Whatever one thinks of this triaging recommendation, Simpson's methodological point stands: in the absence of extraterrestrial data, studies of earthly evolution are our best bet for assessing the prospect of a cosmic biology.

We saw in the last chapter that the radical contingency thesis (RCT) is addressed to big-ticket philosophical questions about large-scale evolution that controlled experiments simply cannot answer. This leaves open the possibility that "natural experiments" in macroevolution might play this crucial evidentiary role. In what sense might convergence constitute "experimental" evidence that bears on the contingency debate? How should the validity of natural experiments in convergent evolution be assessed? In this chapter, we will explore the evidential logic of convergence in greater detail.

1. The Experimental Logic of Convergence

1.1 The Nature of Natural Experiments

We normally think of scientific experiments as involving the controlled manipulation of independent variables to assess their causal influence on dependent variables. But to conduct an experiment, broadly construed, is simply to put

oneself in an epistemic position from which to make observations that affect our confidence in hypotheses about the causal, nomological, or historical structure of the world. Given such a broad framing, it is not easy to distinguish between observational studies that make use of conditions already found in nature and experiments that involve the controlled manipulation of nature, be it in terms of their methods, epistemic goals, or ability to confirm (or refute) theories.

As philosopher of science Samir Okasha points out, many classic experiments in physics, such as the crucial tests of general relativity, are essentially observational and do not involve human intervention in nature.[3] The solar eclipse of 1919 made it possible to test Einstein's prediction that starlight would bend twice as much under the gravitational influence of the sun than is predicted by Newtonian physics because the light of stars traveling close to the sun could be measured at that time. In such cases, nature presents conditions that make it possible to test rival hypotheses, and it seems unproblematic to think of tests that take advantage of these natural conditions as "experiments." At the same time, studies in historical sciences, such as paleontology, are rarely purely observational—they often involve controlled, systematic searches for traces of the past that draw on a wealth of background theory, employing refined methods of data collection and setting out with the aim of testing hypotheses. There is thus no clear conceptual difference between observational and interventionist studies.

Nor is there an obvious epistemic difference between the two. One might be inclined to distinguish manipulation-based experiments from observational studies on the grounds that the former generate more reliable inferences about the causal structure of the world. Yet this is far from evident. In fact, in evolutionary biology, unlike in physics, controlled laboratory studies can create highly artificial conditions that are only weakly projectible to the natural living world. This is in part because the natural living world is complexly causally configured in ways that are not reflected by idealized laboratory conditions, and these differences in causal complexity can result in substantially different results under controlled and natural conditions, respectively.[4]

Nevertheless, "natural experiments" are in certain ways more like proper manipulationist experiments than they are like observational studies. This is because natural experiments are fortuitously structured in ways that resemble interventionist experiments that might be designed by rational agents were they capable of manipulating large-scale natural conditions. In natural experiments, researchers select and analyze samples that differ naturally in one or more independent variables but are similar with respect to other variables, with the aim of developing, corroborating, or refuting hypotheses. Although natural experiments in evolutionary history lack the control of classic experiments, and thus

risk internal validity, they have the advantage of allowing biologists to gather data across numerous taxa and habitats reflecting vast timespans of evolution.[5] This breadth and depth of study, impossible in the laboratory or field, permits inferences about the robustness of macroevolutionary patterns.

For instance, isolated islands are often described as "natural laboratories of evolution" that can reveal law-like evolutionary patterns and processes. "Dwarfing," for example, can occur when large mainland vertebrates (especially mammals and birds) invade or become trapped on isolated islands. Dwarfing has been observed for a wide range of animals, from dinosaurs to humans. Fossils of dwarf sauropods and ankylosaurs have been found on an ancient offshore island in what is now modern-day Romania[6]—may they have been hunted by similarly dwarfed carnosaurs? Likewise, dwarf populations of *Homo erectus* have been found on the island of Flores in Indonesia, where they apparently hunted dwarfed elephants, known as stegodons.

Studies of ecosystem recovery after the removal of human influence, such as in the demilitarized zone between North and South Korea, also have the structure of a natural experiment. Most recently, the Japanese tsunami of 2011, triggered by a magnitude 9.0 earthquake, carried a staggering diversity of sea creatures on tsunami debris from the coast of Japan to the West Coast of the United States in the greatest maritime migration ever recorded. Researchers have described this event, too, as a natural experiment that probes the stability of local ecosystems to large-scale invasion. These events and others like them amount to replays of the tape of life from which biologists can glean law-like generalizations about ecology and evolution.

1.2 Evaluating Natural Experiments in Convergent Evolution

Natural experiments in convergent evolution can probe deeper into the causal structure of the living world. But like laboratory experiments, they can be contaminated, poorly structured, and misleading. A crucial feature of any experiment, whether natural or artificial, is that it is isolated in ways that control for the relevant confounding variables. The variables that must be controlled in any given experiment are determined by the research questions being posed and the hypotheses being investigated. It is critical, therefore, when it comes to natural experiments in convergent evolution, that we are crystal clear about the claims that the experiments are designed to test, as this will determine how to assess their validity.

Our goal here is to establish the validity criteria for natural experiments in convergence for purposes of testing the RCT. Evaluating the setup of natural experiments in convergence for this specific evidentiary purpose requires, at

a minimum, that we assess the "independence" of observed replications by controlling for the influence of conserved developmental constraints. We saw in the previous section that certain types of evolutionary iteration are consistent with, and perhaps even corroborative of, the RCT. It is critical, therefore, that we distinguish the underlying causes of the repetitions observed. Failure to do so has resulted in flawed experimental setups, even in paradigmatic studies of convergence.

One such paradigmatic natural experiment in convergence is the series of pioneering studies conducted by Jonathan Losos and his collaborators documenting the iterated evolution of *Anolis* lizard "ecomorphs," or distinct forms adapted to specialized microhabitats on isolated islands in the Caribbean.[7] Arriving on floating debris, these beautiful lizards established new populations on numerous islands in the Greater Antilles. These populations then evolved in an iterated, predictable way on each "laboratory island," producing at least six distinct ecomorphs, each associated with a suite of anatomical and behavioral properties that are matched to a specialized ecological zone.[8]

For example, the "crown-giant" ecomorph exhibits a cluster of traits adapted to life in the highest tree canopies, whereas "trunk" ecomorphs are anatomically and behaviorally suited to life on tree trunks; "grass-bush" and "twig" ecomorphs, meanwhile, are adaptively matched to selective regimes with their own distinct modes of predation, predator-avoidance, terrain, climate, and so forth (see figure 5.1). Each ecomorph has a quantifiably distinct body plan, including limb and tail length, head and toe-pad size, and coloring, and each ecomorph is more closely related to the other ecomorphs on their own island than they are to the similarly adapted anoles on other islands. For instance, tokens of the twig ecomorph found on Cuba, Puerto Rico, Hispaniola, and Jamaica, despite their affinities, are more distantly related to one another than they are to the very different ecomorphs on their own respective islands.

These patterns of convergence appear to suggest that there is a limited set of "attractors" in *Anolis* morphospace, and that selection reliably overcomes any contrary forces or tendencies in driving the repeated differentiation of lizard populations toward these respective ecomorph attractors. This, in turn, may be taken to indicate not only that selection is a dominant force in macroevolution but also that selection *works in highly repeatable and circumscribed ways*—two key elements of the robust replicability thesis (RRT). *Which* particular ecomorph a subpopulation is driven toward may be highly contingent on numerous factors, such as the ecological zone in which it first becomes established, the ordering of its genetic mutations, the effects of drift and migration, and so on. But the fact that isolated island populations will

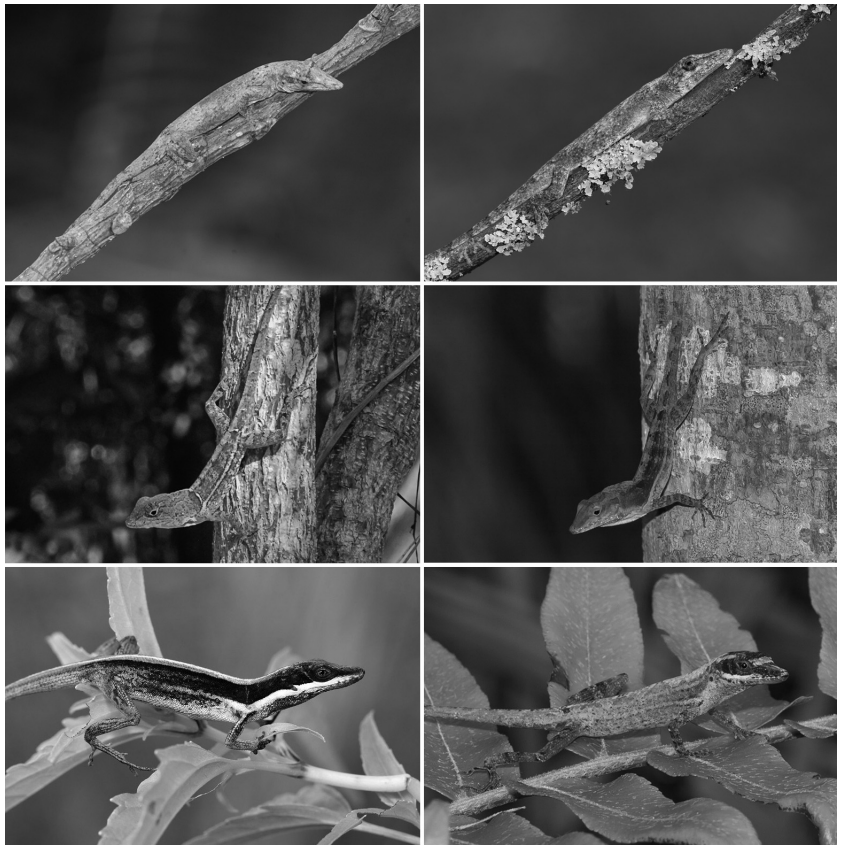

Figure 5.1
Recurrent evolution of Anole ecomorphs on isolated Caribbean islands. Photograph panel courtesy of the Luke Mahler laboratory at the University of Toronto. From left to right, the top row depicts twig specialists *A. garridoi* (Cuba) and *A. occultus* (Puerto Rico); the second row depicts trunk and ground specialists *A. cybotes* (Hispaniola; photo by B. Falk) and *A. lineatopus* (Jamaica); the third row depicts grass specialists *A. alumina* (Hispaniola; photo by M. Landestoy) and *A. alutaceus* (Cuba). Photographs not otherwise marked are by Luke Mahler.

differentiate into these respective *Anolis* equilibria appears to be robustly replicable across natural island experiments.

The Losos studies have been widely taken to be "valid" natural experiments in the sense that they employ the necessary controls of confounding variables in order to adjudicate the hypothesis being tested. Clearly, these studies have ruled out the hypothesis that ecomorphs are more closely related to one another than they are to other ecomorphs on their own island. However, this is not the hypothesis being tested; rather, it is the RCT that is under scrutiny. Both biologists

and philosophers of science have assumed that the geographic isolation of islands ensures the independence of the replications observed.[9] The problem, however, is that *geographical* isolation is not the only kind of isolation that is necessary for establishing the validity of natural experiments in convergent evolution. *Developmental* isolation is equally important. The *Anolis* ecomorph experiments demonstrate unequivocally that selection can optimize form within a given set of developmental parameters. This is an important finding, to be sure, but *as tests of the RCT*, the *Anolis* experiments are invalid. For despite their geographical separation, the observed evolutionary systems are not isolated in crucial developmental respects. Although the target systems end up in similar ecomorphological attractors, they begin from highly similar initial developmental conditions. As such, they fail to rule out the possibility that the observed attractors are caused by shared developmental constraints that facilitate the repetitions.

At this point, one might respond with a shrug of exasperation: "Of course selection is working in these cases with a common developmental plan! What else could possibly account for these outcomes?" Yet this is precisely the thrust of Gould's thesis, and it explains why observations such as these fail to speak to the RCT. Conceptually speaking, there may be no way of delineating ecomorphological attractors, such as the ones observed in the *Anolis* studies, absent a stipulated set of developmental parameters within which selection can optimize form.[10] Yet if these parameters are radically contingent, then the ecomorphological iterations that hinge on them will be radically contingent as well. That is to say, the replicability of ecomorphs will extend no farther back than the evolution of the specific developmental parameters on which they causally depend and with respect to which they are defined. If a pattern of iteration is due to selection acting on conserved developmental parameters, then the question of contingency falls back on the evolutionary robustness of the parameters themselves, and this can only be established by observing iterations *of* (rather than *within*) the developmental plans at issue.

A general problem with appeals to natural experiments in convergent evolution is that the sense of "independence" that is operative in dominant definitions of convergence does not support some of the evidential uses to which the phenomenon has been put in the contingency debate. To see this, it is not necessary to navigate the dizzying array of concepts that cluster around "convergence."[11] A few key distinctions will suffice. We can begin by distinguishing "homology" from "homoplasy." On the standard "taxic" account, *homology* refers to a character resemblance or similarity (S) between two lineages (L1 and L2) that is present in their last common ancestor (LCA), with the inference being that S arose once in LCA and was continuously transmitted in L1 and L2 since they diverged from LCA. *Homoplasy*, on the other hand, describes

(using the same placeholders) a case of S between L1 and L2 that was not present in LCA, and thus is inferred to have arisen at least twice.[12] Whereas some terms, such as "analogy," describe similarity that results from adaptation to a common selective regime, the term "homoplasy" is causally neutral and makes no claims about adaptive motivations. Whether a character resemblance is a homoplasy is determined solely in virtue of its *phylogenetic pattern*. For example, the rodent-like common ancestor of marsupial and placental saber-toothed lions (see chapter 4) did not have a sabertooth morphology, let alone one that was transmitted continuously for the hundred-odd-million years since the divergence of marsupial and placental mammals. Thus, the sabertooth suite is homoplasious, not homologous. End of story.

The taxic account, though dominant, is not the only account of homology/homoplasy on offer in the literature, nor does it exhaust the relevant terminology. Some authors treat homoplasy as synonymous with convergence, while others treat convergence as a causally distinct type of homoplasy. A minority view holds that for a homoplasy to count as a convergence, the developmental machinery causally responsible for the relevant character resemblance must not have been continuously inherited from a common ancestor.[13] On this view, "parallelisms" (see section 3) would count as homologies. Other authors sympathetic to the idea of distinguishing between iterations based on their underlying causes have chosen to retain standard definitions of homology and homoplasy, instead distinguishing between parallel and convergent homoplasies, where "parallelism" refers to homoplasy that is underwritten by conserved developmental generators.[14] The latter approach is the one that will be adopted here.

Our focus for the moment, though, is on the limitations of the taxic approach, because it is the account most often relied upon by proponents of the "critique from convergence" (CFC) discussed in chapter 4. On the taxic account, convergence/homoplasy can ensue from both highly similar and highly disparate initial developmental conditions. So long as S—delineated phenotypically—was not present in the LCA of L1 and L2, then S is deemed to be homoplasious. This pattern-based approach to homoplasy is not problematic in and of itself; but when convergence is called upon to serve as evidence in the contingency debate, the failure to parse the category of homoplasy can undermine the validity of convergence experiments.

As noted in this and the previous chapter, the contingency/convergence dispute turns on whether evolutionary repetitions have the kinds of causes that bespeak their evolutionary robustness. Because the taxic approach does not distinguish among homoplasies on the basis of their underlying causes, convergence data gathered in this way cannot adjudicate the RCT. The problem is not that convergence data fail to provide a "crucial" test that could decisively

confirm or refute the RCT; that is much too demanding a requirement, given the kinds of questions we are investigating. The problem, rather, is that because the CFC has operated exclusively with the taxic conception of homoplasy, it fails to provide any clear evidence against the RCT at all. Without a more targeted analysis of the causes underlying cases of convergence, the CFC is unable to distinguish natural experimental setups that fail to speak to the RCT from those that genuinely undercut it. In essence, studies of convergent evolution carried out with the intent of testing the RCT have been working with tainted experimental setups.

1.3 The Lumping Problem

The failure to parse convergence data in this way results in a heterogeneous class of convergent events with varying implications for evolutionary robustness. Because it does not control for the internal developmental determinants of iterated evolution, the CFC is unable to differentiate biological regularities that reflect deep truths about the living universe from those that are essentially accidental. Let us refer to this as the "lumping problem."

For instance, George McGhee's state of the art review of convergent evolution (discussed in chapter 4) is a significant improvement over previous efforts in part because it includes extensive phylogenetic information.[15] However, because McGhee is operating with a taxic notion of convergence, he does not use this information to control for the developmental facilitators of iterated evolution. To take but one example, consider McGhee's inclusion of more than twenty iterations of "cantharophilous" flowers, which have evolved specialized morphologies to facilitate pollination by beetles. This specialized "pollination syndrome" includes large dish-shaped flowers that are typically dull in color and heavily scented, have easily accessible pollen, and protect their ovaries from beetle mouthparts. Bees, flies, lepidopterans, beetles, and birds have acted as unwitting couriers of male angiosperm gametes since the Cretaceous. Thus, it is not surprising that similar observations have been made for other angiosperm–pollinator pairings.[16] Predictions such as "If there are beetles, then there will be cantharophilous flowers" may be law-like on weakened accounts of biological laws that presuppose radically contingent antecedents (as discussed in chapter 3). But if the clusters of traits that compose "beetle" and "angiosperm" ground plans are highly contingent accidents of Earthly evolution, then this adds little to our understanding of the deep structure of evolution.

The same goes for Simon Conway Morris's discussion of ant mimicry, which is so ubiquitous—evolving more than seventy times in insects and spiders (see figure 5.2)—that it even has its own name in the literature: "myrmecomorphy."[17]

Figure 5.2
Synemosyna formica, jumping spider ant-mimic. Note the two large, forward-facing camera eyes. Photo courtesy of Tom Murray.

If the "ant" and broader "arthropod" phenotype is radically contingent, then ant mimicry will not be stable across deep replays of the tape of life. In other words, ant mimicry is only replicable across shallow replays of the tape that begin with the developmental parameters in question—which in this case, includes the arthropod parameters of the mimic and the ant-specific parameters of the subject of mimicry. Regularities such as these may indicate the deep evolutionary robustness of very generic phenomena like "coevolution" and "mimicry," which can be found across the whole of complex multicellular life. But as noted earlier, and as will be discussed in more detail in the next chapter, this is not the level of detail at which the contingency debate takes place.

The take-home message is that the sheer number of iterations, without further analysis, tells us very little about the evolutionary robustness of the regularity described. Dozens of coevolutionary convergences on flower and ant morphologies offer far weaker evidence against the RCT than do convergences that are fewer in number but that can be shown to arise from broad physicochemical constraints on life. For the latter do not hinge on the peculiar—and singularly evolved—developmental parameters of particular lineages.

For instance, gills have evolved countless times in arthropods, vertebrates, mollusks, and annelids for the universal biochemical function of mediating gas exchange between an organism and its surrounding fluid medium. The same may be said of image-forming eyes, a topic that will be explored in some detail in the next chapter and again in part II. It is only once the "quality" of a convergence is established that the "quantity" can become very telling.

The upshot is that the high frequency of an evolutionary outcome in this history of life, without further analysis of its underlying causes, cannot be taken to imply a high frequency across alternative histories of life. The fact that certain structures or functions recur in evolution is not evidence that they are cosmic in scope or even robust across alternative histories of life on Earth. Lumping is harmful to the case against contingency because it causes the CFC to overplay its hand, while obscuring the strongest evidence against the RCT. In compiling a body of undifferentiated data on convergence, the whole is less than the sum of its parts. In short, the greatest asset of the CFC—its impressive inventory of convergent events in the history of life—is also its greatest flaw. The task before us is to begin the work necessary to sharpen this promising class of evidence.

2. Parsing the Reference Class

As we saw in the last chapter, the Gouldian disagreement with convergence proponents turns not on *whether* evolutionary iteration is theoretically important, but *why* it is important, and what it signifies about the causal—and in particular, modal—structure of the biological world. Understanding the theoretical import of convergence requires that iterations be distinguished in accordance with their underlying causes. What does it mean to be a cause of an iteration, and since iterations may be said to have multiple causes, what makes one cause or type of cause comparably more important than another? These are challenging questions, but they must be answered if convergence is to serve as evidence, one way or the other, in the contingency debate.

2.1 Gouldian Repetitions versus True Convergence

The challenge is to distinguish iterations that are indicative of deeper modal robustness from those that are evidence for shallower degrees of replicability. How can this be achieved in concept and operationalized in practice? This is the million-dollar question, and I do not pretend to offer a definitive answer to it here. My less ambitious aim is to sketch a preliminary picture of what such an account might look like, though this sketch will no doubt be incomplete.

If, when all is said and done, one remains unconvinced that this distinction can be drawn, then one must accordingly resign to the fact that the contingency debate is permanently underdetermined by convergence data—and that we must look elsewhere, perhaps beyond Earth, to make any headway on it. I hope, however, to show that this distinction is not incoherent, and that it can be drawn in an epistemically accessible way.

The first thing we need to do, if we are to make good on this distinction, is break down "iteration" or "homoplasy" into further, more finely tuned categories. Let us call it "Gouldian repetition" when evolutionary iteration results from selection acting on conserved developmental components or their sequelae. Let us refer to all other iterations as cases of "true convergence." Where we are epistemically neutral as between these two types of homoplasy, we shall refer to biological "iterations," "repetitions," or "homoplasies" to signify this neutrality. Although there are clear-cut cases of each type of homoplasy, there is also likely to be a gray zone in between.

The existence of gray areas should not deter us from drawing the distinction between Gouldian repetition and true convergence any more than it prevents us from drawing useful common language distinctions like "night and day." Gray areas should be expected in the living world, where fuzzy ontologies are the norm rather than the exception. The notion of "evolutionary individual," for instance, is important and meaningful for biological theory, even if there is a fuzzy borderland of pseudo-individuals (such as holobionts[18]) between clear-cut cases of individuals (such as organisms) and nonindividuals (such as populations).

Before proceeding, let us reiterate why this distinction is so important to the evidential value of homoplasy. Iterations that are properly characterized as Gouldian repetitions do not support the CFC because they do not lend credence to the three additional philosophical claims, over and above strong adaptationism, that underpin the RRT (see chapter 4). First, Gouldian repetitions fail to show that certain design problems are pervasive in any history of life, because the design problems that prompt this type of iteration may be shaped by the internal developmental parameters of the lineage in question (in a form of organism-niche codetermination). Second, Gouldian repetitions fail to show that the set of solutions to the design problems they solve is highly circumscribed in a way that would support their law-like projectibility, because in such cases accidental internal parameters are constraining (and facilitating) the solution space. Third, Gouldian repetitions fail to show that certain solutions are accessible to selection notwithstanding the internal constraints of phylogeny, because internal constraints are integral to explaining the morphospace that is repeatedly explored by Gouldian repetitions. This last point warrants further elaboration.

Researchers may in some cases take developmental parameters as a given in attempting to isolate the effects of selection. This is a legitimate mode of evolutionary inquiry, but it is orthogonal to Gould's project.[19] The optimization of form within a given set of contingent developmental constraints may be a useful way of assessing adaptationist theses,[20] but it does not speak to the RCT/RRT debate. Even if we were to achieve a "periodic table of forms" based on a thorough understanding of vertebrate development and how it interacts with natural selection—as first envisioned by the Soviet biologist Nikolai Vavilov and echoed in George McGhee's work (see chapter 4)—this would not show that the parameters of vertebrate development are themselves modally robust. And if they are not, then the periodic table analogy, with all its nomological undertones, is misleading.

Jonathan Losos, director of the *Anolis* lizard studies discussed earlier, acknowledges that iterations work on a common developmental platform, but he denies that this indicates a significant role for internal constraints in explaining *Anolis* evolution.[21] Losos maintains that because all the key features of *Anolis* ecomorphs are continuous, quantitative characters, they are subject to standard selection explanations without requiring any appeal to a lack of variation on which selection can act. Losos's remarks are correct, but they are talking at cross purposes with the RCT. If one takes the overarching developmental parameters of *Anolis* as fixed, then selection does figure most prominently into explanations of these iterated outcomes. Further, if one interprets "developmental constraint" in the exclusively negative sense, then a finding of repeated adaptive optimization within these stipulated developmental parameters undercuts an internal constraints-based explanation. But as discussed in the previous chapter, this overlooks the positive role of internal constraints in facilitating iterative adaptive evolution within the highly contingent parameters of existing body plans. Selection can explain why a particular range of morphospace has come to be populated within a restricted set of internal parameters, but this says nothing about the modal robustness of the internal parameters themselves.

How can we determine whether a given iteration is a case of Gouldian repetition or a case of true convergence? This launches us face first into a thicket of thorny conceptual and methodological problems. The first hurdle is to carve out the elements of underlying development that are causally relevant to a given iteration. This is not easy to do. The trouble is that because all known life on Earth is related to one degree or another (see chapter 1), there will always be crucial developmental mechanisms underlying cases of iteration. As we proceed down taxonomic ranks from eukaryotes to metazoans, from metazoans to bilaterians, from bilaterians to deuterostomes, from deu-

terostomes to vertebrates, from vertebrates to tetrapods, from tetrapods to mammals, and so on, more and more homologous genetic-developmental machinery will be shared. There is a sense, therefore, in which all iterations must be caused by shared development because all traits in any two lineages will be produced by a partially homologous developmental apparatus.

Does this make all repetitions necessarily Gouldian and thereby gut the value of the term? Does any amount of shared development undermine the independence of the evolutionary replications observed? If so, then natural experiments in convergence that aim to test the RCT would be incurably confounded. These questions become even more pressing in light of the growing body of work on "deep homology," or upstream regulatory networks that are conserved by animal groups that diverged more than 600 million years ago. Deep homologs are implicated in the development of some traits that are the subject of impressive repetitions across very distant groups, such as the evolution of eyes and various body plan modifications. Does deep homology undermine the independence of iterations in which they are involved?

Distinguishing Gouldian repetitions from true convergence will require that we identify shared developmental characters that bear the right sort of causal relation to the iterated outcome. In other words, if we are to distinguish repetitions that are consistent with the RCT from those that undermine it, we must parse cases of convergence in accordance with their underlying developmental causes—and to do this, we must parse the developmental causes themselves.

One workable approach would be to infer a degree of independence that is proportional to phylogenetic distance. Let's call this the "taxonomy heuristic." On this approach, the greater the evolutionary distance between converging lineages, the greater the independence of the iterations observed. Evolutionary distance is used here as a proxy for developmental independence, much as the number of higher-level taxa has been used as an indirect measure of morphological disparity. The evolutionary distance of convergence admits of degrees: for example, converging classes of mammals bridge a much smaller phylogenetic gap than do converging phyla of animals. Similarly, the independence of natural evolutionary replications would admit of degrees, with independence proportional to the phylogenetic distance of convergence.

The taxonomy heuristic explains why a small number of convergences across kingdoms, phyla, and classes (or clades that roughly map on to these Linnaean categories) tend to indicate deeper modal stability than numerous convergences confined to families, genera, and species. The trouble with this approach, however, is that phylogenetic distance is not always a good indicator of developmental difference when it comes to evolutionary iteration. As biologists Jeff Arendt and David Reznick point out, evolutionary iteration between

closely related groups—even populations within species—can be produced by different developmental mechanisms, while evolutionary iterations between distantly related groups can sometimes be produced by the same developmental mechanisms (the "same" developmental mechanisms here means homologous developmental mechanisms, or those that have been transmitted continuously from a common ancestor).[22]

For instance, evolutionary iterations of pigmentation in populations of mice within the same species have been shown to be produced by different developmental pathways. Convergence between higher taxa, meanwhile, can involve the repeated activation of deep homologs. *Pax6*, for instance, is involved in the development of eyes in numerous eye-bearing phyla (such as vertebrates, mollusks, and arthropods), and "homeobox" genes are implicated in the anatomical evolution of groups as distant as animals, plants, and fungi. Thus, we must develop ways of assessing independence that do not rely entirely on the blunt instrument of phylogenetic distance.

3. The Problem of Parallelism

One way of glossing the distinction between Gouldian repetitions and true convergence is by falling back on a conception of "parallelism," which does a reasonable (though far from perfect) job of tracking these phenomena. In his comprehensive analysis of convergent evolution, Conway Morris states in a footnote that he will ''avoid that old chestnut of whether it is convergent evolution as against parallel evolution,''[23] reasoning that the difference is merely one of degree rather than of kind. Conway Morris is in good company here, as evolutionists the likes of G. G. Simpson have held that parallelism and convergence "intergrade continuously and are often indistinguishable in practice."[24] Yet as we shall see, the conflation of parallelism and convergence causes proponents of the CFC to conclude that external constraints on design space are more pronounced than the evidence in fact warrants. If the distinction between parallelism and convergence offers a promising avenue for distinguishing Gouldian repetitions from true convergence, then crack this "old chestnut" we must try.

Parallelism could have important implications for the use of homoplasy as evidence in the contingency debate, depending on how the concept is cashed out. For example, parallelism could indicate that certain developmental parameters or "informational substrates" that lineages share due to their contingent history strongly increase the probability of certain evolutionary outcomes while dramatically reducing the probability of others. We saw this earlier with the example of the repeated evolution of flight in walking stick insects (see

chapter 4). Path dependency need not entail irreversibility, and the kind of path dependency contemplated by the RCT does not entail irreversibility at all phylogenetic grains of resolution—a point that is illustrated powerfully by the positive potential of parallelism.

3.1 Parallelism as Homoplasy in Closely Related Groups

The seminal treatment of the "homoplasy" family of concepts is found in a paper by Otto Haas and G. G. Simpson published in the 1940s. According to their study, the earliest definition of "parallelism" came from W. B. Scott in 1891, who used the term to describe "the independent acquisition of similar structure in forms which are themselves nearly related," in contrast to "convergence," which referred to the acquisition of similar structures "in forms which are not closely related."[25] Scott later added that "the more nearly related any two organisms are, the more likely are they to undergo similar modifications."[26] The idea that closely related lineages will tend to undergo similar modifications because of shared development and lifeways is also one expressed by Darwin in a later edition of *On the Origin of Species*, where he writes,

> Members of the same class, although only distantly allied, have inherited so much in common in their constitution, that they are apt to vary under similar exciting causes in a similar manner; and this would obviously aid in the acquirement through natural selection of parts or organs, strikingly like each other, independently of their direct inheritance from a common progenitor.[27]

Thus, parallelism has a long history of being associated with homoplasy in closely related lineages. In addition, the greater frequency of iterations in closely related lineages (as compared to distant lineages) has historically been *attributed to shared developmental plans*.

Although Simpson read parallelism this way, his coauthor, Haas, opted for a purely geometric definition, using the term "parallelism" to describe situations in which two clades evolve along parallel trajectories but do not converge (i.e., do not come to resemble one another more than they did before the homoplasy). For instance, horses and brontotheriids (a family of extinct perissodactyl mammals) both evolved small molar cusps, but this did not result in morphological convergence because these lineages simply maintained their current levels of affinity by virtue of the homoplasy; in other words, they evolved in geometric parallel and hence did not increase their resemblance to one another. Simpson's reading of parallelism has come to be the predominant one, however.

With the growth of "evo devo" over the last two decades, there has been increasing recognition that developmental homologies are important for explaining

certain homoplasies. Distinguishing between parallel and convergent homoplasy is a way of acknowledging the different causal frameworks that underlie iterative outcomes in evolution. As mentioned earlier, some accounts of homology break entirely with phylogenetic patterns of morphology to label parallelism as a type of *homology*.[28] Even if parallelism smacks of a certain "homologyness" (as Gould suggests in *The Structure of Evolutionary Theory*), in my view it is preferable to retain a pattern-based account of homology, if for no other reason than to document the distribution of iterations in the history of life and to examine causal explanations of these patterns.

This still leaves two unresolved questions. First, how can we delineate parallelism from convergence, given the universal gradations of developmental homology? Second, to what extent do parallelisms map onto Gouldian repetitions? To the first question, we could stipulate some coarse-grained relatedness metric to separate parallelisms from convergence. For instance, we could hold that iterations between taxa at or above the class level are convergences, whereas homoplasies at or below the family level are parallelisms. But this simply brings us back to the limitations of using a blunt relatedness heuristic to make inferences about developmental causes, as discussed earlier. What is needed is a more targeted causal analysis of the developmental mechanisms underlying iteration. The rest of this chapter is devoted to this philosophical task.

3.2 Causal Accounts of Parallelism

If this section had a motto, it would be, "It is not the *extent* of developmental homology, but rather the *causal type*, that counts." The last two decades have witnessed great strides in our understanding of the molecular basis of development—or what evolutionary developmental biologist Sean Carroll has called "genetic dark matter"[29]—and its role in adaptive evolution. Although organisms sharing a homoplasious trait will always share varying degrees of developmental homology merely by virtue of their relatedness, the decision to categorize a homoplasy as a parallelism need not be arbitrary or rely on coarse-grained phylogenetic affinities. The key is to identify homology in the generators that are *directly causally responsible* for the relevant iteration.

The big question, of course, is what we mean by "directly causally responsible." If by this one simply means "an actual difference-making cause" (a technical term that will be unpacked later), then iterations underwritten by deep homologs could indeed be considered parallelisms. This seems to be how Gould interpreted iterations that implicate deep homologs, which he argued are better thought of as internally constrained parallelisms than proper convergences. If this is right, then it would render many paradigmatic cases of convergence, such as the evolution of image-forming eyes (see chapter 6),

consistent with the RCT—cleverly pressing the strongest cases for robust replicability into the service of radical contingency.

Take for instance *Pax6*, a so-called master control gene that is involved in the development of camera-type eyes in vertebrates and cephalopods as well as in the compound eyes of arthropods. We know from gene "knockout" and substitution experiments that *Pax6* plays a causal role in the development of eyes in each of these distant phyla. The question before us is twofold. Does this deep homology convert the iterated evolution of eyes from convergence into parallelism, as Gould contends, and, regardless, does it undercut the experimental independence of the iterations observed? I will answer "no" to both counts.

There are several ways to gloss the proposition that some developmental mechanism is "directly causally responsible" for an evolutionary iteration. One way is to hold that a homologous developmental mechanism is directly causally responsible for an iteration when it is a *proximate* cause, rather than simply *a* cause, of the phenotypic similarity. We know that the misexpression of *Pax6* transcription factors can lead to the formation of ectopic eyes (e.g., on legs, wings, etc.) in both vertebrates and arthropods. We also know, thanks to gene knockout studies, that *Pax6* is crucial for the development of healthy eyes in both animal groups. Yet neither of these important facts tell us whether *Pax6* is a proximate developmental cause of eyes.

One approach is to use a "screening-off" test to determine whether some developmental factor is a proximate cause of a given phenotypic outcome. Two basic experimental manipulations can be carried out to test whether deep homologs like *Pax6* screen off other developmental factors with respect to the production of eyes (see figure 5.3).

The first experiment, which has already been carried out by Walter Gehring and his collaborators, is to insert the arthropod version of *Pax6* into, for example, the mollusk camera-type eye development cascade or vice versa and see what type of eye develops.[30] Low and behold, if we substitute the drosophila version of *Pax6* for its homologue in the octopod eye cascade, we get a normal octopod camera-type eye, not an insect compound eye. Likewise, in a series of ectopic eye studies, Gehring and his collaborators showed that the mouse version of *Pax6* can be used to induce compound eyes in various body parts of drosophila, such as on the tips of antennae. What this shows is that while *Pax6* is acting as an early trigger of eye morphogenesis, it is not causally responsible for the specific parameters of eye development in diverse eye-bearing groups. The macro-morphological arrangements of camera and compound eyes are directed not by *Pax6* but by thousands of nonhomologous genes that lie downstream from *Pax6* in the developmental cascades that lead to the various eye types.

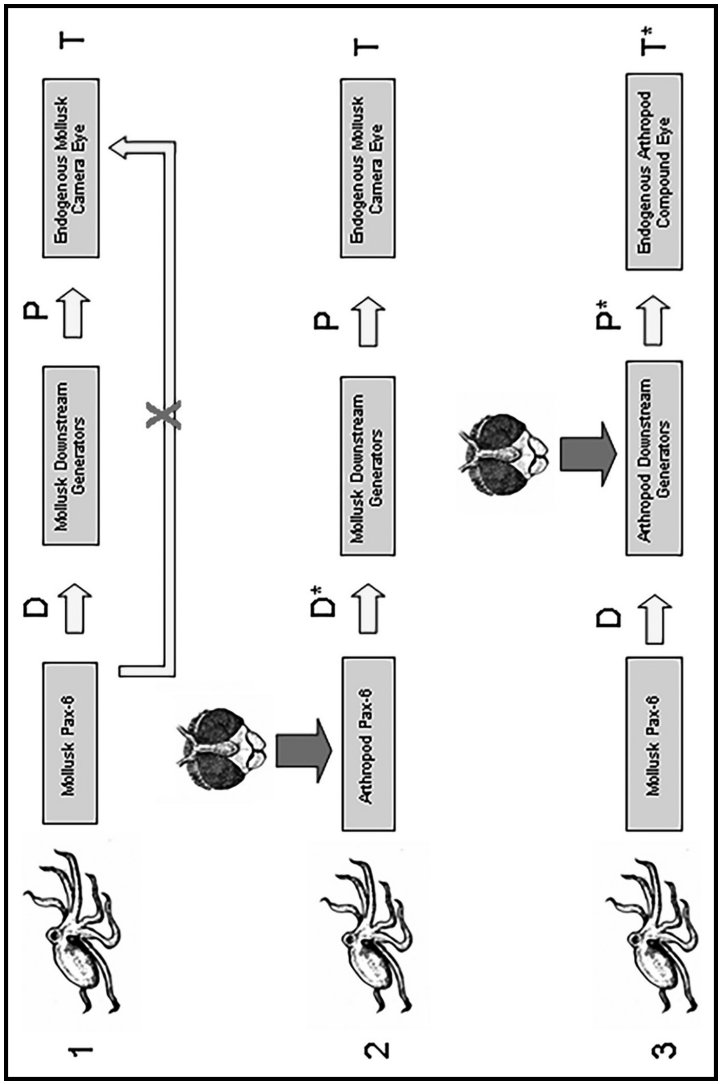

Figure 5.3
Three different manipulation scenarios show that *Pax6* is "screened off" by downstream developmental generators in the production of eyes. (1) In a normal cephalopod, *Pax6* (distal cause D) triggers downstream generators (proximate cause P), which produce a normal cephalopod camera-type eye (T). If either D or P is nonfunctional, T will not be produced, so both D and P are causes of T. (2) In the first (actualized) manipulation, an arthropod *Pax6* (D*) is inserted into the mollusk camera-type eye development cascade, resulting in the development of a normal cephalopod camera-type eye. (3) In the second (hypothetical) manipulation, normal cephalopod *Pax6* is left intact but arthropod downstream generators (P*) are substituted for their mollusk counterparts in the cephalopod developmental cascade. If the result is an arthropod compound eye (T*) rather than T, this would show that the probability of T given P is equal to the probability of T given D and P, and different from the probability of T given D; therefore, P may be said to screen off D with respect to the production of T. See text for details. From R. Powell, "Is Convergence More than an Analogy? Homoplasy and Its Implications for Macroevolutionary Predictability," *Biology and Philosophy* 22 (2007): 565–578.

This conclusion would be even more decisively confirmed by a second manipulation, which has not yet been performed and likely cannot be performed given the complex causal structure of gene-regulatory networks. This would be to replace the downstream, nonhomologous generators of the octopod camera-type eye with those of the arthropod compound eye (or vice versa), while leaving the mollusk *Pax6* intact. If in this scenario an arthropod eye develops (even if it is poorly integrated into the phenotype), then it will have demonstrated that the macroscopic arrangements of the eye are causally determined by their proximate downstream generators, which screen off *Pax6* and other upstream homologues. This second, fanciful manipulation need not be carried out, however, because ectopic eye studies in drosophila, in which the mouse *Pax6* is substituted for the insect *Pax6*, are sufficient to establish this asymmetrical causal relation, even if the downstream generators remain largely a black box. A similar causal analysis would extend to other *Pax* family genes implicated in skeletal-muscle differentiation, as well as to *Hox* genes involved in iterated appendage modifications.

Philosopher Treavor Pearce criticizes this screening-off approach to parallelism on the grounds that it only works in the case of topographically linear developmental pathways, when in fact most complex traits that are subject to evolutionary iteration will be produced by messy, nonlinear gene networks, not tidy causal chains. This seems right, although as we have seen there is an important temporal asymmetry between upstream components of the developmental cascade and their downstream sequelae. Gene networks may not behave in a simplistic linear fashion, but it is still plausible to say that certain upstream components in the cascade are screened off by certain downstream components with respect to some phenotypic outcome (e.g., the macromorphological arrangements of the eye), even if the downstream battery of causes is interactive and poorly understood.

Pearce also points out, again quite rightly, that we should not assume that the proximate cause of any given outcome is the most important cause or explanation of that outcome. Philosopher Christopher Hitchcock argues that pragmatics are an unavoidable aspect of explanation, and consequently that explanation need not track proximate causes. Hitchcock offers the following example to illustrate the point: consider the case where an individual's unprotected sexual activity leads to an infection with human immunodeficiency virus (HIV), which in turn leads to acquired immunodeficiency syndrome (AIDS).[31] Does the HIV infection (the proximate cause) provide a better explanation of why that individual has AIDS than does that individual's earlier unprotected sexual behavior (the distal cause)? The microbiologist and the public health worker might very well disagree on which explanation is best,

given their differing interests. So which explanation is best will be relative to a given set of pragmatic interests and goals.

Elliott Sober is likewise skeptical that proximate causes are better explanations than distal causes, as this would imply that *synchronic* (morphological/physiological) explanations are categorically better than *diachronic* (evolutionary) explanations.[32] Yet, as Sober correctly points out, these two explanatory programs are largely orthogonal to one another, and so neither has a claim to explanatory primacy over the other. If the causal-explanatory import is relative to the research questions being posed and the pragmatic goals being pursued, then the question becomes whether the current investigation justifies prioritizing proximate developmental causes over distal ones in explaining evolutionary iterations. Perhaps the best way to appreciate the role of *Pax6* in eye development and evolution, for purposes of assessing the independence of eye iteration, is to shift the discussion away from screening-off relations and toward the different causal roles that developmental factors play in iterated evolutionary outcomes.

3.3 The Specific Causes of Parallelism

Another way of glossing the notion of a direct developmental cause is in terms of "specific causation." Recall that the problem we are confronted with in attempting to delineate parallelism is double-edged. First, all iterations will be underwritten by a certain degree of developmental homology, given the broad relatedness of life; conversely, rarely will there be homology in all of the developmental components that are necessary and sufficient to produce a given homoplasy. If there is no nonarbitrary way of privileging some developmental causes over others, then the causal conception of parallelism may be unworkable. If so, then we cannot use parallelism to parse convergence data in ways that allow it to play the evidential role that many want it to play.

However, when it comes to the pragmatic goal of explaining evolutionary iterations, not all developmental causes are of equal relevance. Building on Jim Woodward's counterfactual account of causation, philosopher Ken Waters offered a typology of causes that can be usefully pressed into service for thinking about causal explanations of development and evolution.[33] On Woodward's interventionist theory of causation, a causal relation is defined as a statistical relationship between variables as revealed by manipulations of their respective values.[34] More specifically, *A* is the cause of *B* only if some intervention with respect to *A*—that is, a change in *A*'s value while holding other variables constant—would result in some change in the value (or probability distribution) of *B*.

Waters gives the following example to illustrate how the interventionist theory of causation works. We can say that Mary's striking a match is a cause

of it lighting, because if we hold all other variables constant, had Mary not struck the match, the match would not have lit. By the same token, the presence of oxygen in the room is also a cause of the match lighting, because had oxygen not been present—again, *ceteris paribus*—the match also would not have lit. To the contrary, the position of the planet Saturn is not a cause of the match lighting, because variation in Saturn's position does not affect whether the match lights, whereas Mary's striking of the match and the presence of oxygen in the room do.

It follows from this view that developmental factors are causes so long as their manipulation would have a statistical bearing on the outcome. On this view, *Pax6* is clearly a cause of eye development and therefore of eye evolution, providing the molecular substrate of the phenotypic structures on which selection has acted to shape the visual sense in disparate groups. However, not all causes of an outcome are of equal explanatory value. We can further distinguish the "actual difference-makers" from the vast set of "potential difference-makers" in explaining the variation observed across a population of outcomes. This enables us to say that Mary's striking the match is a distinct cause of it lighting in one case, and it not lighting in another. Other causes, such as the presence of oxygen, do not vary across scenarios in which there is an actual empirical difference in the outcome (though of course they could). Waters extends Woodward's counterfactual theory of causation into the realm of developmental biology, allowing us to say that a DNA sequence is the actual cause of RNA structure in a bacterium, even though RNA polymerase and other accessory proteins are necessary causes as well. This is because actual differences in DNA explain the actual variations in RNA sequence, whereas the accessory proteins do not vary. In situations where DNA and accessory proteins vary, both will be actual causes of RNA structure because both are causes that make a difference.

The distinction between potential and actual causes solves the broad developmental overlap problem for parallelism because it enables us to distinguish the actual developmental causes of the iteration from all potential developmental causes in the background, such as the structure of the DNA code, mechanisms of transcription and translation, cellular signaling machinery, patterns of embryogenesis, and so on. These causes of eye development and evolution do not vary such that they figure in explanations as to why some lineages have eyes and others do not. In contrast, *Pax6* is an actual (not merely potential) cause of eye development and evolution because it has been recruited for similar developmental functions in numerous eye-bearing animal groups. *Pax6* is certainly not sufficient for eye development, as it is present in various non–eye-bearing animal groups such as echinoderms, which suggests that it had a more primitive, nonvisual function (or, alternatively and less probably,

that it lost its ancestral function in the vast majority of animal phyla—see chapter 9 for a detailed discussion). Nevertheless, even if *Pax6* is not *the* actual cause of bilaterian eyes, it is *an* actual cause of eye development and evolution, as it provides some of the shared molecular substrate on which disparate eye systems have been built.

Our causal ontology is not exhausted, however. Again following Waters, we can further distinguish between specific and general actual causes. "Specific causes" are those factors that, if subjected to a battery of interventions, tend to change the outcome in *detailed ways*. "General causes," on the other hand, merely determine *whether* or *when* an outcome will occur; they have no influence on precisely how it will do so, where "how" relates to the particular parameters of the outcome. Let us consider RNA synthesis again. On Woodward's causal regime, there is no basis by which to assign causal priority to DNA over and above accessory proteins with respect to the construction of RNA so long as both causes are necessary and actually vary. However, Waters's specific-general distinction enables us to say that DNA is a specific actual difference-maker with respect to RNA structure, because alterations in DNA engender particular changes in RNA sequence whereas interventions with respect to accessory proteins are limited to halting the synthesis process entirely or merely altering the rate at which it occurs. (The same cannot be said of RNA splicing agents, which do have specific effects on RNA sequence.) In the context of morphogenesis, alterations in the rate and timing of developmental events (heterochrony) can have profound morphological consequences and thus are not limited to Waters's "whether/when" criteria for nonspecific actual causation. The point, however, is that we can prioritize some token biological causes over others, even if we cannot a priori privilege certain classes of biological entities (e.g., genes) over others (e.g., proteins).

This philosophical machinery can also help resolve the second prong of the parallelism problem—namely, the fact that many or most cases of iteration implicate some proportion of nonhomologous developmental mechanisms in addition to homologous ones. Pressing this subtler causal typology into service, we can hold that a homoplasy is a parallelism when some of its developmental machinery is both homologous and causally specific. On this definition, an iteration is not precluded from being a parallelism merely because it is bound up with nonhomologous gene products or pathways that affect "whether/when" the iterated phenotype develops. For the same reasons, homology in regulatory regions of the genome, such as promoters, enhancers, silencers, and other factors affecting gene expression, will often (but not always) be insufficient grounds for parallelism. The more specific-causal homology that underlies an iteration, the more clearly that iteration will be a case of parallelism.

The notion of specific causation not only resolves the problems that arise from degrees of homology in the developmental machinery underlying iterations, but it helps to clarify some key cases. For instance, it explains why upstream homologues like *Pax6* fail the screening-off manipulation test: namely, they are general rather than specific causes of the iterated outcome. Deep homologs essentially act as triggers that merely allow traits like eyes or limbs to develop or not, but they do not specify *how* these traits should develop in three-dimensional space. Thus, *contra* Gould, the repeated activation of deep homologs like *Pax6* does not convert these cases of convergence into parallelisms.

Conversely, emerging examples of parallelism appear to satisfy the definition given above. Take, for instance, the iterated evolution of elongated and shortened pelvic spines in stickleback fish over the last 10,000 years in isolated North American glacial lakes—a pattern that has been interpreted by Beatty and others as in tension with the Gouldian view of life.[35] Stickleback populations have repeatedly assumed two ecomorphs in response to common selective pressures: a benthic short-spined form and a pelagic long-spined form. The former configuration reduces the chances of the fish being snagged by predatory dragonfly larvae in the shallows, while the latter increases the diameter of the fish so as to exceed the gape of many open-water predators. This adaptive feat has been accomplished independently numerous times by recurrent genetic modifications of hind-limb development—specifically, in regulatory regions of the pituitary homeobox transcription factor 1 (*Pitx1*) gene,[36] which plays a crucial role in pelvic fin development in sticklebacks and determines the specific length of the pelvic spine and girdle. The iterative evolution of *Anolis* ecomorphs (section 1.2) is also likely to implicate causally specific developmental homologs, given the close relatedness of the lineages.

There are also examples of what we might think of as "potential parallelism"—conserved genetic potential which, if activated under suitable selection regimes, could be the basis of future parallelism. Extant birds, for instance, have retained the ability to develop archosaurian teeth, which have been experimentally induced in chicken embryos.[37] Birds lost their dinosaurian teeth about 75 million years ago; similar losses of dentition occurred several times in theropod dinosaur evolution in favor of keratinized beaks. We can speculate that if in the future history of life mammals were to suffer a major setback and thereby surrender their predatory niches to birds, then dormant avian developmental programs (if they have not irreversibly deteriorated into pseudogenes) could be reactivated by natural selection and avian dinosaurs could once again express teeth. The same may be said for re-evolving claws in the place of wings, as occurs in juvenile forms of some living birds, such as the hoatzin. Given the conserved

potential to produce archosaurian teeth and claws, birds could then "re-evolve" a guild of Mesozoic-grade "theropodomorphs"—not identical to theropods proper, of course, but close enough to deserve the moniker. Such iterated outcomes, as striking as they might be, would only be possible due to a shared set of specific genetic potentials and body plan constraints.

Birds may contain other dormant dinosaurian potentials, such as the ability to modify feathers into scales and vice versa. The standard view has it that feathers evolved from modified scales in dromaeosaurid dinosaurs (informally, "raptors") and were retained by birds; birds, in turn, lost their scales entirely but then re-evolved scales around their feet. Very recent findings from China, however, indicate the presence of feather-like structures in pterosaurs—archosaurs that evolved powered flight well before the dinosaurs.[38] Pterosaurs have long been thought to have a fuzz-like covering made up of simple "pycnofibers." However, the presence of bilaterally branched filaments of varying functionalities and colors in pterosaurs suggests that either feathers originated even earlier in archosaurian evolution, before the emergence of dinosaurs, or else functional feathers evolved multiple times in archosaurs (the group that includes dinosaurs, pterosaurs, and crocodylomorphs), perhaps from an ancestral pycnofiber fuzz. Either way, the developmental potential for scale-to-fuzz-to-feather transitions appears to be confined to archosaurs, with no similar feather-like structures having evolved in any other animal group. Unlike fuzzy coverings and gossamer skin wings, feathers may be a peculiar evolutionary innovation of a single clade that is only replicable, if at all, within that group.

On the other hand, some functionally constrained morphologies, broadly defined, can be achieved through multiple developmental pathways. For instance, teeth have in a sense revolved in birds: Pelagornithids are a fascinating group of large Cenozoic seabirds whose relatively fragile "teeth" lacked any mineralized dental tissue. Instead, they were bony outgrowths of the jaw covered in a hardened keratinized beak, designed more for grasping prey than for forcibly dismantling them.[39] In short, some repetitions may be due to loaded developmental dice that belie their independence and robustness. Just what proportion of repetitions can be characterized in this way is unclear.

3.4 Parallelism and Modal Robustness

Why is it that the same genes often act as specific difference-makers in the production of homoplasies, even in distant lineages? Francois Jacob famously likened natural selection to a tinkerer, working with the developmental odds and ends at hand to repurpose existing machinery for novel and unforeseen tasks.[40] But this only partly explains the repeated evolutionary cooptation of dormant developmental potential. A fuller explanation has to do with evolu-

tionary constraints imposed by the causal topography of developmental systems (discussed in detail in chapter 2). There may be only a handful of genes that can produce major phenotypic effects, and thereby drive substantive adaptive evolution, without having deleterious consequences for other crucial features of the organism. Genes that are expressed "downstream" in the developmental cascade, or those that only affect a particular cell or tissue type, may be more amenable to selective modification. In contrast, "upstream" genes with ramifying pleiotropic effects that are not confined to a single tissue type may be limited to regulatory (rather than structural) modification, because any fundamental alteration in their function is likely to result in a nonviable organism. If this is so, then the vast majority of morphological innovations will be achieved through the regulatory activation of upstream networks and the structural modification of downstream components.

Given these topographic constraints, it is not surprising that two lineages working within a shared developmental plan will tend to arrive at similar solutions to common ecological design problems by drawing upon homologous generators. Although such cases of parallelism are evidence for a degree of modal robustness—namely, modal robustness within a given set of developmental parameters—they do not preclude the possibility that the set of developmental parameters within which selection acts to optimize form is itself radically contingent.

The final question is the extent to which parallelism, conceived roughly along the lines suggested above, maps on to the category of Gouldian repetitions. I have suggested that the more overlap there is in the causally specific developmental homology that underlies an iteration, the more that iteration resembles a clear-cut case of parallelism. And the more an iteration resembles a clear-cut case of parallelism, the less independent that evolutionary replication may be said to be, and thus the weaker its evidentiary force against the RCT. That said, parallelism is neither necessary nor sufficient to undermine the deep modal robustness of an iteration. It is not necessary because some Gouldian repetitions will not be parallelisms. Take, for instance, the evolution of ant mimicry, discussed earlier; even if this outcome were produced by different developmental pathways in different mimics (spiders, beetles, etc.), it would still be radically contingent so long as the ant phenotype itself is radically contingent.

Consider another case where some radically contingent set of developmental parameters makes a certain outcome a "Good Trick" in Dan Dennett's terminology (see chapter 4). An example might be the iterated evolution of retractable necks in turtles, which offers one of the best-known illustrations of competitive replacement.[41] The retractable neck has been achieved through

at least two distinct specialized methods: flexing the neck sideways (*Pleurodira*) and flexing the neck into an S-curve (*Cryptodira*). Even if these iterations were produced by disparate developmental mechanisms, the reason why the retractable neck is an attractor in turtle morphospace is because of the shared (and quite possibly, radially contingent) parameters of the turtle body plan, combined with a common selective regime. If this is right, then such replications are Gouldian repetitions whether or not they are properly considered parallelisms. The same may be true of countless other iterations, such as the evolution of barracuda-like forms in both marine (sphyraenids) and freshwater (characins) lineages of ray-finned fish.

By the same token, parallelism is not sufficient for an iteration to be a Gouldian repetition because we can imagine an iteration that is at once a parallelism and a case of true convergence. For instance, if a particular developmental substrate is highly externally constrained and thus robustly replicable—such as the proteins involved in photoreception or light refraction—then the repeated specific cooptation of those conserved mechanisms in distant lineages would not undermine the evolutionary robustness of the iterations they underlie. This is why, in the final analysis, it is best to avoid tying our notion of Gouldian repetition too closely to the concept of parallelism, even though their theoretical motivations are closely allied.

The take-home lesson of this chapter is that many evolutionary iterations start from similar developmental conditions that bias the set of solutions that are accessible to selection. This in, turn, undermines the causal independence of the natural experimental replications observed and thus invalidates them *qua* tests of the RCT. Some of these internally constrained/facilitated iterations may constitute proper parallelisms, while others, even though they are produced by nonhomologous developmental mechanisms, are the product of broader body plan parameters that could easily have been otherwise and thus amount to Gouldian repetitions. Yet there is still much work to be done if we are to situate convergence within a broadly Gouldian view of life. The next chapter will delve deeper into this problem.

Contingency and Convergence

Toward a Cosmic Biology of Body and Mind

By: Russell Powell

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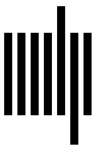

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