

# ADAPTATIONISM AND OPTIMALITY

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## *Chapter 10*

### **Adaptation, Development, and the Quest for Common Ground**

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*Science* magazine recently surveyed over 100 developmental biologists on two questions. The first asked for opinions on the greatest unanswered questions in developmental biology. The second asked which areas would see the most progress in the next five years. Among the unanswered questions "What is the relation between development and evolution?" scored second, just behind "What are the molecular mechanisms of morphogenesis?" But when asked to name the areas in which progress was expected, the role of development in evolution fell to eleventh out of twelve. No other question showed so much disparity between importance and expectation of progress (Barinaga 1994).

The study of evolutionary adaptation is at the core of modern evolutionary biology. Natural selection, the explanation of adaptation, is also the primary force for evolutionary change. Developmental biology is only at the periphery of this study, even with the recent explosion of knowledge in molecular and developmental genetics. The *Science* questionnaire showed that developmental biologists are very interested in the relevance of development to evolution, but pessimistic about discovering its nature.

Developmental biology is not integrated into mainstream evolutionary biology. I will argue that this situation has resulted from clashes in methodology between the primarily adaptationist practices, methodologies, and theories of mainstream evolution theory and the contrasting practices, methodologies, and theories of developmental biology.

Contemporary evolution theory has its origins in the Modern Synthesis of the 1920s and 1930s (Mayr and Provine 1980). The Synthesis was centered on population genetics.

Population genetics

contained the central insights of Darwinian selection theory and Mendelian genetics, two traditions which had up until that time been perceived to be inconsistent with one another. The Synthesis placed Darwinian adaptation at the center of evolutionary biology, and was a virtual death knell for the competitors to Darwinian evolution theory. Most of the specialized studies within biology (e.g. paleontology, systematics) found a place within the Synthesis. It has been widely noted that developmental biology was not included among these Synthesized specialties (Waddington 1953, Hamburger 1980, Horder 1989, Gottlieb 1992, Newman 1992, Depew and Weber 1995, Gilbert et al. 1996, Raff 1996, Smokovitis 1996). The problematic position of developmental biology with respect to evolution theory has continued to the present day.

The current gulf between developmental biology and mainstream evolutionary theory is an intriguing scientific, historical, and philosophical issue. It has consequences not only for the practice of biology but also for broader topics such as the alleged unity of science. Since the late 1970s a number of developmental biologists have been arguing for the inclusion of developmental biology in evolutionary theory (Bonner 1982, Goodwin et al. 1983, Raff and Kauffman 1983, Thomson 1988, Hall 1992a, Gottlieb 1992, Raff 1996, Gilbert et al. 1996, Wake 1996, Gerhart and Kirschner 1997). Carving a place for development within evolutionary theory often brings these workers into conflict with adaptationist theorists, practitioners of an evolutionary research program, which has gotten along without significant developmental input for the 60 years of the Synthesis. Many of the debates seem peculiarly inconclusive. The two sides are working from such distinct sets of presuppositions that a mutually satisfactory resolution is hard to conceive. There often seems to be no common ground on which the debates can be satisfactorily adjudicated (Arnold 1992, Mitchell 1992).

I would like to be able to clear a patch of common ground for the resolution of these debates, but that task is well beyond my abilities. Instead I will attempt to chart some of the fissures and crevices which fractionate this logical terrain. In contrasting the two modern traditions I will use the terms *developmentalist* and *adaptationist*. Developmentalists are developmental biologists like those cited above who are attempting to introduce a developmental evolutionary biology and articulate its place within general evolutionary theory. My use of "adaptationist" will be more broadly drawn. It is my contention that many

aspects of Synthesis evolutionary theory show a methodological clash with developmental biology. These include population thinking and population genetics, the special significance of intraspecific variation, and the core importance of selection. I will illustrate the clash by use of quotations from a range of Synthesis biologists. For the most part, my "adaptationists" are not chosen because of a specific theoretical agreement among them. Rather, they are mainstream Synthesis biologists chosen to illustrate the contrast I wish to highlight – the contrast between developmental methodologies and those of Synthesis biology. The contrast is seen somewhat more vividly among biologists with certain theoretical interests. Dobzhansky distinguished between historical studies of evolution and experimental approaches (Dobzhansky 1951, p. 11). Reeve and Sherman (1993) similarly distinguish between studies of "evolutionary history" and of "phenotype existence." While both kinds of studies are often focused on adaptation, practitioners of the experimental, phenotype-existence kind show more vividly the contrast with developmental approaches. Reeve and Sherman's persuasive and influential paper will be especially useful in this regard.

Like any scientific tradition, distinct fields of biology are equipped with distinct sets of conceptual tools. Acquiring skill in the use of these tools is a part of the professional education of a biologist. Problems arise from the fact that the sets of tools of related scientific disciplines can be very different. In the present case it often seems that each side has tools highly suited for demonstrating the irrelevance of the alternative approach. An examination of the uses (scientific and rhetorical) of these tools, and even their historical origins, may allow each side to gain a broader perspective.

Sometimes distinct theories can seem to be in conflict when they actually are not. One analysis of such conflicts is based on the pragmatics of explanation (Dretske 1972, Garfinkel 1981, Sober 1986, Mitchell 1992). Any scientific explanation operates against a background of presuppositions. One of the presuppositions is the *contrast class* of alternative explanations. An explanation of some phenomenon *P* involves a presupposition about the proper or relevant description of *P*, and the contrast class against which the phenomenon is explained. A given explanation of *P* might successfully explain the occurrence of *P* *rather than B, C, or D*; a different one might explain the occurrence of *P* *rather than X, Y, or Z*. If a person who desires an explanation of *P* (rather than *X, Y, or Z*) hears the explanation of *P*

(Rather than B, C, or D) she might well conclude that no explanation has been given at all. This point is customarily illustrated with an anecdote. A priest supposedly asked the great bank robber Willie Sutton why he robbed banks. Sutton's answer was "Because that's where the money is." This joke gets its humor from the fact that we can all recognize the two distinct contrast classes: the one assumed by the priest (robbing banks rather than obeying the law) and the different one assumed by Sutton (robbing banks rather than robbing, say, libraries). But when one is working within a research tradition, and is skilled in conceptualizing the subject matter in that tradition's manner, an alternate contrast set is not so easily conceived, or understood as relevant. This can be called the principle of *explanatory relativity*. Explanations can only be properly understood in relation to the presuppositions and the contrast class with which they were offered. It is not unusual in the history of science to come across theoretical debates in which one scientist asserts that an adversary's account of a given phenomenon *is not even an explanation*. This usually indicates that the writer and the adversary have different presuppositions and contrast classes. (See Amundson 1985 for an example from the history of experimental psychology.) Debates of this sort are peculiarly inconclusive, but are fertile grounds to search for clashes of presuppositions.

Some of the conflicts between developmental and adaptationist thinking may stem not from genuine contradictions between the traditions, but from explanatory relativity. We will see that the two traditions differ even in the meanings they attach to certain key concepts. The mismatch in presuppositions is considerable, widened by decades of isolation. It would be naïve to expect too much from the mere recognition of the methodological contrasts; recognizing them does not make them go away. Nevertheless, recognition is a good place to begin.

### **Functional and Structural Biology**

The present anomalous position of developmental biology with respect to evolution can be understood as arising from contrasts between the explanatory styles and presuppositions of developmentalist biology and the adaptationist theoretical commitments that comprise the mainstream of modern evolutionary thought. The modern situation can be seen as an example of a particular theoretical contrast that has

been well studied by historians of biology. In his classic 1913 book *Form and Function*, Russell showed how the contrast between functionalism and structuralism had permeated biological debates up to his day. Ospovat (1981) showed that chief biological controversy of the early 19<sup>th</sup> century was not evolution versus special creationism, but function versus structure. The function/structure debates of the 19<sup>th</sup> century show intriguing similarities to the present situation.

I will present aspects of the modern debates as instances of the age-old function/structure clash. Broadly adaptationist biology will be interpreted as functionalist, and developmental biology as structuralist. This will give a perspective from which certain explanatory relativities will become visible.

Functionalism is the view that function is in some sense prior to form; objects have their form in virtue of the function they serve. Historical functionalists have had various theories of the origin of functional adaptation; divine design, use-inheritance, and natural selection are examples. Structuralism is the view that form or structure is prior to function. Form, to a structuralist, is not to be explained in terms of function, but rather in terms of autonomous formal/structural properties and processes. Like functionalists, structuralists have differed widely on what kinds of causes *do* appropriately explain form. Abstract ideals, geometry, and material embryological processes have all been candidates. The generation of form is the common target of explanation. Even though specific explanations of form varied widely, the point of agreement is that form is not explained by function. Function, to a structuralist, is the secondary putting-to-use of an already existing structure.

A brief review of the 19<sup>th</sup> century debates will help set the stage. Functionalists included Cuvier and the British Natural Theologians. Structuralists included Geoffroy and other continental morphologists, and a number of British followers eventually including Owen (Appel 1987, Desmond 1989, Amundson 1996). The debates centered on the reality of Types and the nature of homologies. Structuralists were committed to Unity of Type as the basic fact of biology, and to homologies as revealing of Type. Adaptations were secondary overlays. Functionalists focused attention on the distinct adaptations of different species to their environments. Functionalists were willing to recognize some Types, such as Cuvier's four great *embranchements*. To Cuvier, the *embranchements* reflected distinct

adaptive styles and had no significance independent of adaptation. Cuvier was willing to recognize the homologies proposed by structuralists like Geoffroy only when the proposed homologs had the same function in different species. For functionally similar homologs, no similarity beyond adaptation needed to be assumed. But structuralists believed that homologies and Types reflected a reality that lay deeper than adaptation, and so were most interested in functionally distinct homologies – mammal lungs and fish swim bladders, ear bones and jaw bones– the very homologies functionalists refused to acknowledge. These functionally distinct homologs were taken by functionalists to imply *maladaptation*, because the asserted similarity could not be explained by similar function. The British functionalist Bell rejected the proposed homologies between inner ear bones of mammals with jawbones of lower animals on the grounds that "the organ of sense is not imperfect" in the animals lacking the inner ear bones (Bell 1833, p. 139). Since maladaptation had not been proven (and in Bell's opinion it never would be) the ear/jaw homology was meaningless to him. The structuralists had arrived at the homologies by analyses of morphology and embryology, with no attention to the function of those body parts. To them neither adaptation nor maladaptation was under investigation; the structural, morphological facts stood on their own. The functionalists and structuralists were working with different standards of explanatory success.

The functionalist Natural Theologians were special creationists, anti-evolutionists, and proponents of the Argument from Design. The structuralists of this period have sometimes been described as similarly anti-evolutionary. Some were. But some were evolutionists, and many simply didn't care about "ultimate" origins. Structuralists were united not by a common theory about the "ultimate" cause of biological phenomena, but rather a common goal – that of *explaining form* in a non-functional way (Nyhart 1995). Most had a strong methodological caution towards hypothetical or theoretical reasoning. This allowed a consensus on structuralism as a biological orientation while keeping metaphysical (and evolutionary) disagreements in the background. But the structuralist program was crucial to evolution in one way. Structuralism asserted *real* relationships among certain groups of species, relationships reflected in their homologies and Type. So functionalists were individualistic about species in a way that structuralists were not. For

functionalists species are *really* related only to their adaptive niche; "Types" were just coincidences of those niches. For structuralists, species were *really* related to other species, via their common membership in Types. The real unities documented by structuralists were a necessary premise in Darwin's argument for evolution (Amundson 1998).

One of the striking facts about Darwin's *Origin* is that it was the first genuine scientific reconciliation of the form/function dichotomy. More than any previous work, the *Origin* provided a unified account of the biological phenomena that were central to both the functional and the structural schools of thought. But although form and function were reconciled in the *Origin* they were not on equal footing. Unity of Type (the evidence from embryology and homology) provided Darwin with what he considered his strongest evidence for common ancestry. Natural selection was the cause both of adaptation and of evolutionary change. So adaptation (the "law" of Conditions of Existence) was said to be "the higher law; as it includes, through the inheritance of former adaptations, that of Unity of Type" (Darwin 1859, p. 206). This "higher law" argument reaffirms, somewhat ironically, the Natural Theological intuition that adaptation rather than common structure was of primary biological importance.

It is now widely recognized that the early success of the *Origin* was not that it convinced the scientific community of the importance of natural selection, but rather of the fact of evolution itself. Darwinism did not become the dominant view of evolution until the Modern Synthesis (Mayr and Provine 1980). Bowler (1988) has documented the strength of non-Darwinian evolutionary schools between 1860 and 1930. Bowler describes most of these schools as "developmental," meaning that phylogenetic evolution was to be understood on the model of ontogenetic development, and that evolutionary change was in this way directed. In the present paper I use the term "developmental" only in its ontogenetic sense, to refer to the biological study of individual development, with no implications that phylogeny somehow reflects ontogeny. The relevance of embryological development to phylogeny was taken as obvious by most biologists between the time of Darwin and the Modern Synthesis. Many of these "developmental" schools of evolution were based on linkages between ontogeny and phylogeny that are no longer tenable – Lamarckian inheritance, orthogenesis, and recapitulation, for example.

Developments of the early 20<sup>th</sup> century revealed how unsatisfactory these theories are, and simultaneously rendered problematic *any* relevance of development to evolution. I will argue that the result has been a reinstatement of the function/structure theoretical contrast in a form highly reminiscent of the days of Cuvier and Geoffroy.

### **The Sequestration of Embryology:**

Between 1859 and the Modern Synthesis it was assumed by most biologists that developmental biology would play a crucial role in understanding evolution. The Synthesis changed all that. In the course of unifying the Darwinian and Mendelian traditions in evolutionary biology, the Synthesis drove a wedge between developmental biology and the population genetic understanding of adaptation and evolution. Underlying the events of the 1930s was the articulation and reinterpretation of a set of distinctions which now almost seem to be matters of logic, but which actually embody important methodological policy. These include Weismann's distinction between germ plasm and soma, Johannsen's genotype/phenotype distinction, Morgan's distinction between transmission genetics and developmental genetics, and Mayr's explication of the distinction between proximate and ultimate causation.

During the 19<sup>th</sup> century everyone including Darwin realized that the *Origin* lacked an account of heredity, and knew that a full understanding of evolution would require one. This fact is common knowledge today. It is less well understood that the 19<sup>th</sup> century concept of heredity was very different from our modern concept. Current ideas about "Darwin's need for a theory of heredity" are quite different from those of Darwin and others of his century. In those days the term *heredity* referred not only to the transmission of traits from parent to offspring, but also to the development of those traits in the ontogeny of the offspring. To *explain heredity* was to account for the kinds of similarities observed between generations. This was seen to require not merely a summary of patterns of those similarities, but also an account of the causal properties of gametes that are carried to the offspring, and an account of how gametic properties give rise during development to traits in the offspring similar to those of the parent. I will refer to this notion as "broad heredity," a causal chain beginning with the parent, continuing through then-unknown properties of the gamete, and ending after ontogeny

with the manifestation of parental traits in the offspring. Theoretical devices such as Darwin's pangenesis and Weismann's germ plasm traced this causal chain, schematically describing the material mode of transmission as well as the origins in the embryo of the transmitted traits. Many embryologists opposed particulate theories of inheritance on the grounds that they could not account for the hierarchical processes of development. For this reason they could not account for the ontogenetic origin of Form, and so could not *really* account for heredity. All agreed that the ontogenetic origin of inherited similarities was a part of heredity. As a part of heredity, its importance to evolution was unquestioned.

How did the modern concept of narrow heredity come about? In hindsight it might seem that Weismann's 1883 doctrine of the continuity of the germ plasm and its separation from the soma was a big step towards it. But Weismann himself worked with the concept of broad heredity. He studied development for the specific purpose of understanding evolution, attempting to "trace heredity back to growth" (Weismann quoted in Webster and Goodwin 1982, p. 27). Parental gametes give rise to a sequestered germ line within the developing embryo, but also exert their influence on the developing soma. Explaining (broad) inheritance required explaining not only the passage from gamete to germ line, but also from gamete to somatic trait. Development was a part of inheritance, and so was necessarily implicated in understanding evolution.

Weismann's developmentalism contrasts sharply with certain modern understandings of his doctrine. Assuming the modern concept of heredity, the sequestration of the germ line is seen by some as blocking the relevance of development to evolution. Wallace states that embryologists could only contribute to an understanding of evolutionary mechanisms if they can answer the question "How does a developing organism alter the genetic program carried by its originating germ cells?" But this question can have no answer because of the sequestration of the germ line, and so embryologists have nothing to say about evolutionary mechanisms (Wallace 1986, p. 160). Maynard Smith states that "One consequence of Weismann's concept of the separation of the germ line and soma was to make it possible to understand genetics and hence evolution, without understanding development" (Maynard Smith 1982, p.6). It is true that the Lamarckian route from development to evolution is blocked by sequestration. But other routes may still be open.

Weismann's germ/soma distinction today seems to parallel the distinction between genotype and phenotype. This terminology was introduced in 1909 as part of Johanssen's pre-Synthesis Mendelian opposition both to Weismann's speculative developmental theories and (ironically) the Darwinian belief in the importance of continuous variation. The distinction originally "represented an implicit acknowledgment of the beginning and end of a production – a developmental process" of embryogenesis, but it soon came to name the two completely distinct aspects of an organism which are relevant to the modern understanding of evolution (Sapp 1983, p. 329). Quite at odds with Johanssen's intent, genotype and phenotype came to designate the functional aspects of germ and soma in an individual organism. The genotype/phenotype distinction served to bracket the internal and scientifically intractable processes that were causally responsible for individual characters. As the processes of embryogenesis were bracketed, there seemed to be no loss to evolutionary explanations.

The narrow concept of heredity was first explicitly stated in 1926 by Morgan (Morgan 1926, p. 27; Gilbert 1978). On Morgan's schema the study called *genetics* took over the entire workload of broad heredity, but the task was divided. Transmission genetics characterized certain aspects of the particles that carried traits between generations, with scientific understanding based primarily on distributions of phenotypic traits among generations. Developmental genetics had the task of explaining the ontogenetic processes by which genes give rise to phenotypic traits. Ownership of the term *heredity* was eventually won by transmission geneticists, although embryologists continued for a while using it in the old, broad sense (Sapp 1983). Today the unmodified term genetics almost always refers to transmission genetics, as it does in the quotation from Maynard Smith above.

The final distinction to be noted is between proximate and ultimate causation, best articulated by Ernst Mayr (1961). Functional or physiological biology deals with proximate causation, while evolutionary biology studies ultimate causation. The distinction can itself be seen as an explication of a certain sort of explanatory relativity, showing that physiological explanations do not necessarily contrast with evolutionary ones even though they sometimes seem to. Mayr has often used the distinction to defend evolutionary studies against threats from reductionist fields, for example the fascination with molecular approaches in the 50s and 60s (Beatty 1994, p. 350).

Creating a logical space for evolutionary studies in a world full of microreductionist science is not the only use of the distinction, however. It can also be used to block entry into that logical space. In 1994 Mayr stated he had recently read five or six works on development and evolution, and all of them violated the proximate/ultimate distinction. This is because embryogenesis, called by Mayr the "decoding of a genetic program," is a proximate process (Mayr 1994, p. 357; 1984, p. 1261). The authors of the works in question had *intended* to discuss ultimate (evolutionary) processes. If embryology and developmental genetics are by definition proximate processes, it is hard to imagine how they could have succeeded. The proximate/ultimate distinction is undeniably useful for exposing certain explanatory relativities. But its application can be problematic, and other relativities overlooked. I will later show how the perceived 'ultimacy' of a biological process depends on one's explanatory presuppositions.

The modern understanding of these four dichotomies has contributed to the present anomalous position of developmental biology with respect to evolution. The black-boxing of development follows naturally. The narrow concept of heredity allows Mendelism to be seen as *the* solution to Darwin's need for a theory of heredity. After Morgan, a theory of heredity no longer requires a solution to the problem of Form, or an explanation of how the material carrier of inheritance gives rise to phenotypic traits. The notion that an evolutionarily satisfactory theory of heredity would leave ontogenetic development untouched would have surprised Darwin.

### **The Causal Completeness Argument**

Advocates of the relevance of development to evolution reject the black-boxing of development that is made to seem so natural by the dichotomies of the previous section. This section will discuss one kind of argument in favor of the relevance of development. While it is ultimately inconclusive, it helps to reveal differences between developmentalist and adaptationist explanatory presuppositions.

In 1937 Dobzhansky stated the population genetic definition of evolution: "Since evolution is a change in the genetic composition of populations, the mechanisms of evolution constitute problems of population genetics" (Dobzhansky 1982, p. 11). The statement is a mirror image of the scientific developments that led to its adoption.

The successes of population genetics were what inspired the consensus that evolution was indeed changes in gene pools. As with any science, the subject matter is conceived in terms of the most successful methods of studying it. One might expect that scientists working in other traditions than population genetics might have other definitions of evolution, and so they do. Developmentalists may favor Van Valen's definition of evolution as "the control of development by ecology" (Thompson 1988, p. 78; Van Valen 1974, p. 115). Dobzhansky's population genetic definition of evolution places development in a black box, Van Valen's definition black-boxes (transmission) genetics.

Developmentalists have expressed a specific kind of argument against the black-boxing of development, which I will call the *Causal Completeness* argument. Changes in genotypes can have an effect on phenotypes only by modifying developmental processes; therefore the understanding of developmental processes is necessary to the understanding of evolution. "In order to achieve a modification in adult form, evolution must modify the embryological processes responsible for that form" (Horder 1989, p. 340; for the history of the argument see Raff 1996, p. 23, and Johnston & Gottlieb 1990, p. 472).

The point of the Causal Completeness argument can be seen in developmentalists' reactions to the traditional dichotomies discussed above. Waddington was unwilling to accept the genotype/phenotype dichotomy, and in 1939 interposed an "epigenotype," the hierarchical processes of embryogenesis which generate a phenome from a genome. The phenotypic effect of a genotypic change is constructed by the epigenotype (Hall 1992b, p.118). The dichotomy between proximate and ultimate causation has recently received similar treatment. Mayr's classification of developmental biology as a study of proximate causation (genetic program decoding) places conceptual limits on its relevance to evolutionary biology. That dichotomy must be expanded for development to be relevant to evolution. One recent expansion places developmental biology as an intermediary between Mayr's functional (proximate-causal) and evolutionary (ultimate-causal) categories.

Functional biology = anatomy, physiology, cell biology, gene expression

Developmental biology =  $\delta$  | functional biology | /  $\delta t$

Evolutionary biology =  $\delta$  | developmental biology | /  $\delta t$

(Gilbert, et al., 1996, p. 362)

Changes in functional biology through (ontogenetic) time constitute developmental biology, and changes in developmental biology through (phylogenetic) time constitute evolutionary biology. Evolution is defined in Van Valen's style, as changes not in a gene pool but in the developmental processes that give rise to phenotypes. This arrangement "suggests that to go from functional biology to evolutionary biology without considering developmental biology is like going from displacement to acceleration without considering velocity" (ibid.).

The interposition of developmental processes between proximate and evolutionary processes, or between genotype and phenotype, shows that a fuller causal story can be told about how evolutionary change occurs. A different version of the Causal Completeness argument brings us closer to conflict with the study of adaptation. This argument draws attention to the irrelevance of transmission genetics for the explanation of organic form. Transmission genetics identifies genes only by the phenotypic variations that they cause. A selective explanation might explain why one variant *rather than another* exists in a population, but it does not explain how *any* of the variants are ontogenetically generated. If it happens that a developmental gene (or complex) is accurately identified in a transmission-genetic study, a structural explanation of how that gene gives rise to "its" trait would require reference to many other genes and embryological processes, most of which may be invariant in the population. Since they are fixed in the population, they cannot be identified by transmission genetic methods, and so are not of independent interest to students of adaptation. In a case like this, adaptationists are interested in the effects of only a single gene out of the many embryological components (genetic and epigenetic) which causally explain the presence of the trait in the organism, and that gene plays a very small (even though interesting) role in ontogenesis. This kind of critique can be seen in embryologists' early reaction to Morgan's genetics: "Genes could determine the number of bristles on a fly's back, but they could not determine how a fly constructed its back in the first place" (Gilbert, et al., 1996, p. 361). A genetic explanation of polydactyly explains (at best) only the difference in digit number, and assumes without explanation the organism's ability to develop digits in the first place. Adaptationists pick out traits and genes on transmission genetics principles. So from a developmental perspective, adaptive explanations do not really explain why a trait *exists* in a population

(or in an individual or taxon) – they only explain why *it rather than some other trait* exists. They explain why Sutton robs the bank rather than the library, but not why he robs the bank rather than making an honest living.

The Causal Completeness argument actually does very little to prove the evolutionary importance of developmental biology to a skeptic (e.g. an adaptationist). Its chief use is in preaching to the converted. It draws attention to the activities going on within the black box of development, activities from which our attention is distracted by the dichotomies of proximate/ultimate and genotype/phenotype. But the mere existence of these processes does not prove their relevance to evolution. Some black boxes need never be opened because their insides really *are* uninteresting. It would be futile to argue, for example, that statistical thermodynamics suffers from insufficient attention to the actual paths of the individual molecules of a gas as they impact the walls of a container. Such a detailed causal account would be uninteresting even if it were achievable. Perhaps the facts of development are similar, and the gap between genotype and phenotype is filled with masses of physiological trivia that cannot affect our understanding of evolution in any way. Some adaptationist evolutionary explanations are similar to statistical thermodynamics in their aloofness from grubby causal details. Like thermodynamics, adaptationist biology can be seen as an *equilibrium* study, dealing with systems in which a very large number of different causal histories could be expected to converge on a stable state. This often relieves adaptationist biology from the need to reconstruct detailed phylogenetic or populational histories (Sober 1983, Reeve and Sherman 1993). Perhaps it also relieves adaptationist biology from a concern with the causal details of the black box of development. The Causal Completeness argument only shows that causal factors are *present* which are being ignored by mainstream biology. It does not show that those causal factors are *important* to the understanding of evolution. They may be no more crucial than the paths of individual gas molecules are to the understanding of gas pressure.

Advocates of developmental evolutionary biology must do more than prove that details of development are causally involved in evolutionary change. They must show how an understanding of development contributes to an understanding of evolution in significant ways. This requires either that developmentalist explanations address phenomena not addressed by mainstream evolution theory,

or that they provide better or more complete explanations of phenomena that are already addressed. At this point explanatory relativity becomes a problem. Whether a phenomenon is worthy of explanation depends on one's methodological commitments. (An explanation of the origin of Bauplans will carry no fascination for someone who doesn't believe that Bauplans exist.) Even when the target of explanation is agreed upon, the standards by which explanations are judged differ between research traditions. An explanation proposed from within one tradition can too easily be judged to be irrelevant, "not an explanation," from the perspective of the other tradition.

### **Consequences of Attending to Unity Rather than Diversity**

*Biological functionalists study biological diversity and structuralists study biological commonality.* This generalization applies clearly enough to the 19<sup>th</sup> century structuralists and functionalists, with their disagreements on Unity of Type. When applied to modern approaches, it glosses over complexities in both traditions. Nevertheless, it highlights an important contrast. The theoretical commitments and explanatory techniques of each approach are so arranged that successful explanations will involve the *assumption* of different positions on the significance of biological diversity or commonality. As in Darwin's hands, modern adaptive explanations more often explain differences than similarities between species. Homoplasy is the kind of similarity that is most often given adaptive explanation. An adaptationist explanation of homoplastic traits would hypothesize convergent selection of originally diverse species under similar environmental influences. The resulting similarity is attributed to the similar selective forces rather than pre-existing similarities between the species. A developmentalist explanation might refer to common developmental mechanisms of the two converged species, and attribute the homoplastic trait to these internal commonalities. This kind of explanation will be discussed below.

First, consider the role of diversity and variation in adaptationist studies. Unlike the 19<sup>th</sup> century adaptationists, modern explanations of variation between species are based on heritable variation within populations. The techniques of transmission genetics can identify a trait as heritable only if it varies within a pop-

ulation. Natural selection only operates on heritable, varying traits. Mendelian genetics synthesizes so nicely with Darwinism because heritable variants, the output of Mendelian genetic analysis, are the input of Darwinian natural selection. Transmission genetics identifies precisely the traits that are relevant to natural selection and so to evolutionary adaptation. One might naively assume that the non-varying traits within a taxon are also heritable, since they are shared among organisms that are related by descent. But transmission genetics is unable either to "atomize" non-varying traits (identify which character groupings are inherited together) or to apply the operational definition of "heritable" to them; one cannot do Mendelian crosses between members of reproductively isolated populations. Heritably varying traits are those associated with genetic differences (by definition). What about non-varying traits? Non-varying traits are simply irrelevant to the population dynamics of natural selection. The causes of non-varying traits play no role in forward-looking equilibrium styles of adaptive explanation (the "phenotype existence" questions of Reeve and Sherman 1993). In historical, phylogenetic studies of adaptation, traits which vary between but not within species are assumed to have shown heritable variation in ancestral taxa (although non-varying traits in *that* generation were *then* irrelevant to evolutionary change). For varying traits, one fact about ontogenetic causation is critical, namely whether or not the trait's causal origin is the kind that makes it heritable. A population of flies with heritable variation in bristle number is open to selection for bristle number. But the causation of non-varying traits is of no theoretical consequence. Heritability is undefined for these traits, and the lack of definition is inconsequential. Non-varying traits may have varied in the past, and they may vary in the future. But for contemporary population dynamics they are simply background conditions. A fly must have a back before it can have bristles on it, but selection is "blind" to non-varying aspects of its back. Non-varying traits are the canvas on which the adaptive evolutionary picture is painted. Admittedly this canvas changes as traits become fixed, or begin to vary. But the explanatory action always takes place in the zone of variation.

The situation is very different in developmental biology. Many processes studied are fixed not only in a species, but in very high taxa. The processes shared within each stem and branch of the hierarchical tree of life are of interest, from those most widespread (e.g. early cleavage patterns) to those which differ between closely related

species. Much research is devoted to anatomical items that characterize high taxonomic ranks, such as the neural crest or the vertebrate limb. Traits that vary within a population have an embryogenesis as well, of course, as the Causal Completeness argument implies. But there has been little traditional interest in these traits. The theoretical interest is in understanding how an organism develops out of a zygote *at all*, how it gets its segments, and its gut, and its legs, and its back. There is less interest in such developmental details as the number of bristles on one particular organism's back. Intraspecific variation is not entirely ignored among embryologists. The oldest continuing embryological interest in intraspecific variation is in teratology, one of Geoffroy's specialties (Alberch 1989). Monsters, of course, are notoriously out of step with Darwinian evolution. Some recent developmental studies have begun to focus on more intrapopulational variation. But, as we will see, they too have commonality rather than diversity as their theme.

The traditional structuralist research goal of explaining form can be seen in modern developmental biology; how does adult form emerge during embryogenesis and later development? It is *possible* to interpret this project as the study of the proximate mechanisms involved in each ontogenetic event, each individual "decoding of the genetic program." On this interpretation, developmental biology explains how this chick developed out of its zygote, and how this fly did it, and this snail, and this sponge, and so on. But one can only interpret developmental biology as a proximate study by ignoring the emphasis that developmentalists place on the commonality of developmental processes. Developmental theorists study *the vertebrate limb*, not this particular chick's wing. The vertebrate limb has been a subject of structuralist study for over 150 years. Although nothing approaching a complete theory has been proposed, sketches have been made, and it is possible to imagine what a full theory would be like. A structural theory of the vertebrate limb (if a complete theory were available) would apply to *all* vertebrate limbs, with more specialized theories addressing the limbs of vertebrate taxa. Sketches of such a theory distinguish between "permitted" and "prohibited" morphologies, and infer these morphologies from what is known about mechanisms of limb development across the vertebrate lineage, as well as from observed interspecies variation (Holder 1983, Hinchcliffe 1989, Shubin and Alberch 1986). This theory would not be a proximate theory about the building of any single limb, or

about the building of the limbs of a particular species. It would be a theory about the processes of limb embryogenesis, and how common and divergent elements of these processes range across a large chunk of the evolutionary nexus. It would reveal how the nested sets of homologies of limb morphology reflect the interplay of conserved and divergent form-generating processes in the embryos of tetrapods.

Students of limb development would consider such a theory to be relevant to evolutionary biology. Here are the reasons. When two related species evolve different limb morphologies, this happens as a result of specifiable modifications in the particular processes that they had shared. Understanding of the morphogenetic processes will allow prediction and explanation of certain evolutionarily interesting phenomena (examples to be examined below). The processes of development are not just causally implicated in evolution, as the Causal Completeness argument asserts. They also play a role in explanations of evolutionary phenomena. This is the Causal Completeness argument cashed in.

Given the developmental mechanisms by which limbs are constructed, some evolutionary modifications are developmentally likely, some unlikely, and some impossible. If the structuralist limb theory is a good one, it would help us to see which are which. An example can be seen in recent studies of intraspecific limb variation among newts and salamanders. One such study examined 452 newts from a single population for variation in the configuration of cartilage and bone elements in the digits, carpus, and tarsus (Shubin, et al. 1995). Possible variations include the loss, addition, or amalgamation of the seven carpal or nine tarsal elements. Of the many possible variations, only a few were observed in the population. Forty percent of the variants represented typical configurations in other species. The most interesting were bilaterally symmetrical patterns. Because they are present in both left and right limbs, these indicate a global developmental influence. Of the five bilateral variants observed, two represented atavisms, reconstituting inferred ancestral morphologies. The other three represent derived conditions in nested clades of other urodeles. The important point is that this restricted pattern of intra- and interpopulational variation can be seen to follow from quite specific, empirically discovered mechanisms of limb development. "Underlying developmental influences on anatomical variation may exert their effect on cladistic topology

because of the structural hierarchy of *the urodele limb*" (ibid., p.882, emphasis added).

This talk of *the urodele limb* is not just a way of referring to the limbs of urodeles. *The urodele limb* is an abstract theoretical construct, like the Bauplan, that expresses shared patterns of development. Its nature is inferred from comparative morphology and experimental embryology. This kind of explanation may make no reference whatever to selective forces. Even in studies of intrapopulational variation there is often no interest shown in either the heritability or the fitness effects of the variants. From a study similar to the above: "...[T]he observed pattern of variation from a variety of clades is consistent with Shubin and Alberch's model of chondrogenic pattern formation. A functional explanation is at least not required to explain the bias in the variation pattern" (Rienesl and Wagner 1992, p. 318; Shubin and Alberch 1986).

Recall Mayr's comment that development was a matter of proximate causation. Notice that theoretical concepts like *the urodele limb* are seen as embodying the hierarchically structured developmental mechanisms available within a taxon. This is why development is not seen as merely proximate. The theoretical *urodele limb* pre-existed even the selective processes that produced the modified limb of a particular urodele species. From this perspective, development (or its set of possibilities, as expressed in *the limb*) is more ultimate even than natural selection, because selection can act only on the variation 'allowed' by *the limb*!

Following Gould and Lewontin (1979), advocates of development have sometimes criticized adaptationists for failing to acknowledge that the facts of embryology place limits on adaptive change (Dawkins 1986, p. 311; Sober 1996). This is an inaccurate criticism; modern adaptationists recognize that evolution can only proceed in directions allowed by the developmental system of an organism. But the criticism can be understood as following from the explanatory differences between the two camps. The difference is that adaptationists do not care *why* a particular variation is or is not available. The most complex and fascinating epigenetic explanation of a prohibited variant means the same, to an adaptationist, as the simple absence of a gene in the population. This adaptationist *disinterest* in embryological details can be misconstrued by developmentalists as a *disbelief* in the causal efficacy of developmental processes. Adaptationists can accept the fact of restrictions on heritable variability caused

by embryogenesis. They simply do not see the *relevance* of the underlying causes of that restriction for the understanding of evolution. Heritable variability is where the action is, because selection is blind to non-varying traits.

The reason for this contrast may be that adaptationists consider facts about restricted variation to be facts about particular populations. Developmentalists view embryological processes (that restrict variation) as distributed throughout taxa. Thinking of "the vertebrate limb" as a theoretical concept involved in causal explanations of evolutionary phenomena runs contrary to the adaptationist intuition that the variation actually present in a population is what causally matters. The phylogenetically distributed nature of a developmental type like the vertebrate limb is sometimes expressed historically, as features of ancestral developmental systems that (under modification) continue to operate in embryogenesis within current species. Reeve and Sherman vividly express their adaptationist disdain for this notion. "[W]hatever is important about phylogenetic history will be recorded in the species' current environmental and biological attributes. Ancestral species do not otherwise mysteriously reach from the past to clutch the throats of their descendants" (Reeve and Sherman 1993, p. 19). Shared developmental processes are here depicted as superstitious bogeymen, since they do not "really" exist in a current population. It is surely true that the *sharing* does not exist in any current population, since it is a fact about phylogenetic distribution. Developmentalists have their eyes on the distribution of the developmental processes making up *the vertebrate limb*, and regard these processes as exemplified or expressed in individual species. From this perspective, nothing mysterious or throat-clutching seems to follow from the persistence, under modification, of developmental processes in descendent species. Reeve and Sherman can accept the *fact* of restricted variation, but they reject the *relevance* of developmentalist explanations of that restriction. Some developmentalists misread them as rejecting both.

There is a great deal of skepticism among adaptationists about structuralist concepts like the theory of the vertebrate limb, or the Bauplan. They are often seen as theories about epiphenomena, about the mere artifacts of a history of evolutionary change that was not influenced in any meaningful way by conserved ontogenetic processes (Williams 1992, p. 87). A.J. Cain has asserted that even the deepest homologies of the vertebrate archetype are adaptations for

existing vertebrate species. "[T]heir remarkable constancy of plan combined with plasticity in pretty well every detail of that plan over hundreds of millions of years almost forces us to the conclusion that they are as they are because that is what, in competition with all the other great groups, they need to be" (Cain 1964, p. 37). This claim is astonishing to a developmentalist, especially because Cain bases it only on "what we know about natural selection" (ibid.), which includes no information at all from developmental biology. It is difficult to see how to resolve this conflict given the current divergence in presuppositions.

### **Characterizing the Contrast**

I will offer a sketch of the contrast between these two kinds of evolutionary explanation. Adaptationist explanations depict evolution as changes to what I will call the *current genetic state* of a population, with selective forces fully accounted for but the current genetic state itself assumed as an unexplained background condition. The current genetic state includes both non-varying traits and the existing gene frequencies of the varying traits. (For historical adaptationist studies an ancestral state is the "current state.") Structuralist explanations depict evolution as changes to the *processes that give rise to phenotypes*, with those developmental processes fully accounted for at each step but selective forces assumed as unexplained background conditions. There are shortcomings to each approach. The structuralist approach ignores selection, and so contains no account of the motor of evolutionary change. Selection is an assumed background condition in a structuralist account of evolution, just as the current genetic state is a background condition for adaptationists. On the other hand, by treating the current genetic state as mere background the adaptationist account ignores the biases in available variation that are created by the existing developmental system. For selectionist purposes, those biases can only be expressed as the existence or non-existence of heritable variation. With theoretical interest only in the *existing* variation, the adaptationist feels no direct need to explain the absence of a different range of variance. Adaptationists speak of the "opportunism" of evolution, meaning that selection "takes advantage" of whatever variation is available. These progressive metaphors direct attention towards the consequences of selection, but away from the causes that shape the availability of variation.

Variation that does *not* exist is out of sight of selection, and so out of the minds of selectionists.

Neither approach denies the existence of the phenomena explained by the other. Those phenomena are merely treated as the background against which the real action is projected. Developmentalists might study the developmental consequences of a change in size of a particular bone (the morphological modifications that might cascade from it) without any interest in the ecological circumstances that comprise the selective force that changed the bone. Adaptationists might consider a phenotype that shows heritable variation for vertebra length but not number of vertebra, with no interest at all in the embryological facts that determine why one but not the other variant is expressed. In this way adaptationists can *claim* to take account of developmental constraints, and structuralists can *claim* to take account of selection, even though adaptationists don't care about the embryological causes of developmental constraints, and structuralists don't care about the ecological causes of selective forces. "Taking account" here only means acknowledging the existence of a phenomenon (selection or developmental bias). It does not mean directing attention to the constituent causes of the phenomenon – causes like ecological relationships, or embryological mechanisms. This minimal account-taking does not require attention to the explanatory contrast classes of the other research tradition. Adaptationists are deeply interested in *why* there was selection for bone size (e.g. whether the change improved flight from predators, or attracted mates, etc.). Developmentalists only care *that* such selection existed, but don't care why. Developmentalists are interested in *why* some morphologies are developmentally prohibited, and what accounts for the permitted range. Adaptationists acknowledge *that* the existing variation is limited, but don't care why.

There are also advantages to each approach. When an evolutionary change occurs, an adaptationist can explain *why the change occurs* (rather than not occurring) and also why the change that does occur *affects fitness in the way it does*. The structuralist can explain neither. However, the structuralist may be able to explain why that change *rather than a different but functionally equivalent change* occurred (i.e. why it was available to selection when others were not). In addition the structuralist can predict other changes that will occur in the phenotype as correlated effects of the change that was selected for.

The structuralist lacks a way to pick out the motor of evolutionary change. But this handicap is perhaps compensated by the potential ability to explain *all* aspects of correlated evolutionary change, not just those aspects that were the targets of selection. It is an empirical question how much morphological change is directly selected for and how much comes about through developmental correlations. Many adaptationists think that pleiotropies are relatively unimportant. Richard Dawkins very explicitly acknowledges the importance of constraints created by embryogenesis, and complains that the critics of adaptationism do not accept this acknowledgment (Dawkins 1986, p. 311). Nevertheless, he considers pleiotropies relatively unimportant because "there is no reason why selection should not favour modifier genes that detach the two phenotypic effects" (Dawkins 1982, p. 35). If there *is* a reason why selection does not favor such modifier genes, that reason might well be found in the developmental system that does not produce such variation. An embryologist might know how to answer questions about it. Dawkins's acknowledgment of embryological complexity does not lead him to look to embryology for facts about developmental correlations. In contrast, developmentalists have their attention fixed on the complex embryological *causes* of correlations rather than to selective forces. It is not surprising that they consider developmentally correlated change to be the norm in evolution. Perhaps it is not needless to remark here that the biases of research programs will not by themselves answer this question.

### **Two Meanings of 'Constraint'**

In divergent research traditions the same word is often applied to very different concepts. Ernst Mayr has shown that geneticists and naturalists had different concepts attached to the word "mutation" in the period before the Modern Synthesis, and that this exacerbated the theoretical conflicts between those disciplines (Mayr 1980, p. 20 ff.; 1982, p. 742 ff.). Today, with attention focused on distinct central phenomena, developmentalists and adaptationists can use common vocabulary to express different concepts. An example is the notion of *constraint* itself. When adaptationists speak of a constraint, they refer to a factor that restricts adaptive optimality. An optimality model in foraging theory might specify external constraints such as food distribution, and internal constraints such as the organism's perceptual

acuity (Stephens and Krebs 1986, p. 6). Constraints are defined by their effect on adaptation. When developmentalists speak of constraint, they refer to a developmental factor that restricts the range of possible phenotypic forms generated. Nothing is implied about the relative fitnesses of the constrained forms. I have elsewhere elaborated on this explanatory contrast, distinguishing between the "constraints on adaptation" discussed by adaptationists and the "constraints on form" of structuralists (Amundson 1994). The contrast can lead to significant miscommunication. Adaptationists often discuss constraints (on adaptation), and find it puzzling that developmentalist critics do not recognize this (e.g. Stephens and Krebs 1986, p. 212). The reason developmentalists fail to recognize many adaptationists' discussions of constraint is that they do not address the embryological processes by which form is generated, and so no constraints (on form) are acknowledged.

Let us consider a case in which theoretical discussion is affected by this contrast. Wake (1991) proposed a developmental explanation of observed homoplasy among taxa of plethodontid salamanders. One example was the distribution of digit loss among taxa. The mechanisms of amphibian limb morphogenesis are fairly well understood. Experimental manipulations have determined that the development of digits is influenced by cell number in limb buds. Cell size is influenced by genome size, which is extremely large in some amphibian taxa. Wake explains the loss of digits in certain taxa as a combination of two effects. One is small limb bud sizes, due to overall miniaturization. The other is large cell size, caused by extremely large genome sizes that allow fewer cells in a limb bud of a given size. Wake assumes some unspecified selective force for miniaturization, and derives digit reduction from known factors of limb morphogenesis. Reeve and Sherman criticize this account, calling attention to Wake's failure to measure the relative fitnesses of four- and five-toed salamanders. They offer the possibility, not considered by Wake, that there was direct selection for digit reduction in addition to selection for overall miniaturization. "Since Wake offers no evidence of the relative fitnesses of small four-toed and five-toed individuals within plethodontid taxa, design constraints offer at best a description, not an explanation, of the occurrence of four-toedness" (Reeve and Sherman 1993, p. 22).

The aspect I wish to highlight is not the question of whether digit reduction was adaptive. It is rather the assessment "not an explanation,

just a description" that Reeve and Sherman offer.

Like the examples of Cuvier and Bell mentioned above, these adaptationist authors consider structuralist accounts explanatorily irrelevant unless adaptation has been refuted. Wake had asserted a constraint *on form* (Wake 1996). Reeve and Sherman showed that he had not proven a constraint *on adaptation*, and concluded that he had not offered an explanation. They are certainly correct that Wake had not tested the fitness of the variants. Their adaptationist alternative might, after all, be correct. But this is an empirical matter, not a methodological one. It does not warrant the methodological condemnation of "not an explanation," at least from a structuralist perspective. If a structuralist account must exhaust all adaptive alternatives before being considered even a potential explanation, similar requirements might in fairness be placed on adaptationist accounts. Selectionist accounts might be required to prove that a particular adaptive evolutionary change could *not* have been caused pleiotropically by selection on a developmental correlate. I am afraid that this would require a knowledge of developmental possibilities and impossibilities well beyond that presently available.

Reeve and Sherman offer a new definition of developmental constraint that would (unlike Wake's) count for them as an explanatory alternative to direct selection for digit number. "Phenotypes are developmentally constrained when the alternative phenotypes have lower fitnesses because they depend upon developmental processes that would seriously disrupt the original developmental program, thereby reducing viability" (ibid.). Wake's hypothesis is reinterpreted into "the production of four toes minimally disrupts the development of small individuals." The contest is now between two adaptationist scenarios; was it embryonic or ecological selection that caused digit loss? Wake's original structuralist alternative is dismissed as methodologically flawed. Other adaptationists have offered similar "corrections" of developmental explanations, in which developmental disruption of the embryos is the selective force that restricts adult variation (Dawkins 1982, p. 39; Williams 1992, p. 80). I will call this the *adaptationist version of developmental constraint*.

This reinterpretation of developmental constraint changes a structuralist assertion into an adaptationist claim about the existence of particular selective forces. The result is a very different hypothesis from the structuralist original. Reeve and Sherman's adaptationist version of constraint requires historical episodes of variation and

selection. A developmentally disruptive five-toed miniaturized plethodontid must actually have existed together with a less disruptive four-toed form, at least as embryos, during the history of the lineage. This is an empirical assertion about the existence of heritable variation in the taxon. Wake's structuralist hypothesis has the opposite historical implication. The developmental knowledge about limb morphogenesis that underpins Wake's hypothesis leads him to believe that such variation never existed. There is a certain symmetry in these debates. Just as Wake's account lacked measurements of fitness, Reeve and Sherman's lacked evidence of heritable variation in digit number. What are the facts of this case? Did miniature four-toed plethodontids outcompete (or outsurvive) their five-toed siblings? Or was \*\*\*the fifth toe automatically lost in the developmental changes that led to miniaturization, leaving a population of four-toed salamanders with no digit variation to be selected upon? At this point, explanatory relativity can be seen to influence the questions that are asked or not asked. Heritable variation plays a role in every adaptationist explanation; Reeve and Sherman assume that it must have played a role in salamander digit loss. Such variation plays little or no role in developmental explanations, and Wake doesn't bother to look for it. But the existence of such variation is an empirical question, not a matter to be decided by methodology alone.

### **Conclusions**

The contrast in explanatory assumptions between adaptationists and developmentalists is so striking that speculation about the possibility of a synthesis seems hazardous. But I will conclude by noting a few of the possibilities.

First, consider the two proposed interpretations of developmental constraint. The adaptationist interpretation of developmental constraint and the structuralist interpretation of constraint differ in assumptions about embryonic variation. Here is a point in the methodological debate at which we may be able to find a toehold in empirical facts. The developmentalist version of constraint asserts that certain anatomical variants never occur (or never occur heritably) because of morphogenetic mechanisms within the lineage. The adaptationist interpretation of developmental constraint assumes that heritable variants did occur, and were selected against during

early development. The existence of such early variants is a difficult question, but an empirical one. It is certainly not a logical or methodological truth that the variants *must have* existed. Each version of constraint is probably true of *some* 'developmentally constrained' traits in the world. Knowledge of the relative frequencies of these two very different kinds of "constraint" would go some ways towards adjudicating this particular conflict. Practical difficulties are tremendous, since the hypothesized developmental disruption might be lethal so early in development that the embryo has no digits to count. But progress might be made if we could even reach agreement on what would count as evidence for the existence or the absence of this not-yet-observed variation.

Second, consider the problematic ontological status of typological theoretical concepts like the archetype, the Bauplan, or the urodele limb. Many modern philosophers and biologists have considered typology the very antithesis of evolution. I have elsewhere argued that this is a misunderstanding of typology and of history (Amundson 1998). Nevertheless typological concepts contrast strongly with neo-Darwinian population thinking. Everyone agrees that development itself evolves. Typological concepts like the urodele limb are not mere descriptions, but refer in some way to the causal properties of shared developmental systems. Does this mean that the *urodele limb* evolves? A non-evolving urodele limb would seem to create tension with the concept of development itself evolving. But an evolving urodele limb would be applicable to extant species in virtue of something other than their common ancestry, presumably in virtue of a set of modifications in the developmental system that are shared by modern urodeles but not shared with their common ancestor. But what could keep reproductively isolated species on the same evolutionary track? If typological concepts like these are to be a part of an expanded developmental evolutionary theory consistent with neo-Darwinism, significant interpretive work has still to be done.

Under one proposed scenario, the contrasts between adaptationist and developmentalist explanatory styles might be finessed. It has been argued that adequate quantitative genetic studies of trait covariance might provide common ground between students of adaptation and those of development (Arnold 1992, Kirkpatrick and Lofsvold 1992). These constraints, called "genetic constraints" by Arnold, would be discovered by phenotypic rather than developmental

analysis. The constraints would be defined in terms of transmission genetics rather than developmental genetics, and so would fit into the population dynamic models of adaptationist biology. Constraints thus uncovered would then be potential targets of developmental explanation, since embryogenesis is presumably the cause of many of the patterns of covariance. (If embryogenesis is not the cause of many such patterns, then the adaptationist skeptics are correct and development really is irrelevant to evolution.) Such an analysis might provide a stepping stone between the research programs of developmental and adaptationist biology. Developmental biologists would study the causes of Arnold's genetic constraints, while adaptationists study their consequences. Neither side would be required to adjust its methodology to the other. The results would be disconcerting from the point of view of theoretical unity, however. A quantitative-genetic screen between developmentalists and the rest of evolution theory would merely conceal the methodological disunity of biology. If Bauplans or other typological notions are necessary to explain the genetic correlations of traits, we may as well admit it.

The conflicts between developmental and adaptationist approaches to evolution are not caused by simple methodological errors committed by one side or the other. Neither the proximate/ultimate distinction nor the Causal Completeness argument can chop through this methodological knot. On some issues, such as the existence of a population of unnoticed embryonic variants, empirical studies might narrow the gap. On others, such as typological versus population thinking, conciliation is hard to imagine at present. It is possible that an evolutionary developmental biology of the kind projected by Raff (1996) and by Gilbert et al. (1996) might proceed without accommodating its methods to mainstream adaptationism. On the other hand, the rapid advances in developmental genetics might modify the conceptual landscape so much that today's conflicts are transformed. Can the unities of the Hox genes replace the unities of Bauplans for purposes of evolutionary developmental biology? If so, can they synthesize more readily with population thinking? I do not know.

We might take hope from the fact that the 19<sup>th</sup> century structure/function debates eventually arrived at a sort of synthesis in which structural and functional biology both participated. Perhaps the same will happen in the modern debate. It is sobering to recall, however, that the 19<sup>th</sup> century synthesis between structure

and function required no less an achievement than Charles Darwin's *Origin of Species*.

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