

The Role of Historical Factors in the Evolution of Complex Organismal Functions

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Abstract. Two major classes of historical factors have been proposed as regulating the diversity of organismal design. First, key innovations may be causally related to diversification of structure and function in a clade. Second, developmental constraints may limit the diversity of phenotypes. A major difficulty with these proposals is the lack of a methodology for testing proposed constraints or innovations. Hypotheses of developmental constraints, in particular, have rarely been tested using phylogenetic methods and are usually based on descriptive and correlative analyses.

We present a comparative phylogenetic methodology for testing the role of historical factors in the evolution of complex organismal functions. This six-step procedure incorporates phylogenetic and outgroup analysis as well as an a priori causal model and statistical tests. A specific historical factor is discussed in detail: duplication of structural elements is proposed to be causally related to structural and functional diversity in descendant clades. As the number of independent elements rises in a design, the number of possible interconnections and novel functions rises.

This hypothesis is illustrated with examples from gene evolution and the biomechanics of vertebrate skulls. In both cases, the duplication of structural elements appears to be causally related to subsequent historical diversity of structure and function. Innovations in the lower vertebrate skull have often been introduced by the acquisition of duplicate biomechanical pathways (primitively regulating a single function) that subsequently undergo diversification.

We emphasize the importance of formulating and testing hypotheses of causal historical factors, and of addressing a key set of potential constraints on the evolution of design: the interactions among different levels of biological design. The organization of neuronal circuits and the design of the musculoskeletal system, for example, may play an important role in regulating the origin and transformation of behavioral novelties.

INTRODUCTION

How do we explain the diversity of organismal form and function? To what extent is this diversity an effect of the production of morphological and functional novelties and their consequences, and to what extent may it be explained by constraints and limitations imposed by either the environment or by intrinsic properties of biological designs? These are questions to which no general satisfactory answers are currently available. Yet, they represent a major area of interest in evolutionary biology.

The general issue underlying these questions is the extent to which historical factors have regulated the process of morphological and functional diversification. In order to understand fully the design of organisms, we need to understand the history of the design and to integrate historical analyses with equilibrium analyses of the relationship between organisms and their present-day environment (Gould 1986; Huey 1987; Lauder 1981; Liem 1987, 1988; Ridley 1983). For analysis of the evolution of structural diversity in a group of species, a historical factor is any general feature or aspect of a clade that (a) arose at a level plesiomorphic to the phylogenetic level at which the pattern to be explained exists, and (b) is used to explain in a causal sense some aspect of that pattern. Historical factors have been viewed as both possible constraints on the diversity of organismal design and as morphological, functional, behavioral, and/or ecological novelties that permit diversification (e.g., Gould 1980; Liem 1973).

In this paper we consider the two main classes of historical factors that have been proposed as regulating morphological and functional diversification: key innovations and structural constraints. There are two general themes that we wish to emphasize. First, it is our contention that while considerable effort has been devoted to propose and describe historical factors, few attempts have been made to test historical hypotheses. Most analyses in the literature are descriptive and correlative rather than causal, and do not attempt to establish a causal link between a proposed innovation or constraint, and morphological or functional diversity. Second, we suggest that a general class of constraints and innovations has been largely ignored: the relationship among different levels of biological organization.

KEY INNOVATIONS AS HISTORICAL FACTORS

The idea that a clade may possess a "key innovation" or "evolutionary novelty" that confers an advantage to the species possessing it, thus giving species in this clade a competitive enhancement, is one historical factor that has been proposed to regulate the evolution of organismal design (see Larson et al. 1981). A key innovation was originally considered to be any novel feature (e.g., morphological, physiological, or behavioral) that

characterizes a clade and is proposed to be correlated with the adaptive radiation of that clade (Liem 1973; Mayr 1960). As the concept was initially described, the possession of a novelty was proposed to be both necessary and sufficient to explain the extent of diversification of species within a clade (Fig. 1A). A classic example was provided by Liem (1973), who proposed that the possession of a morphological novelty (a lower pharyngeal jaw suspended in a muscular sling) was related to the extensive diversification of cichlid fishes of the great lakes of Africa.

As discussed by Lauder (1981; see also Stiasny and Jensen 1987 and Liem and Wake 1985), the concept of a key innovation as originally applied has several major difficulties. First, there is no a priori basis for assigning any particular feature to be the novelty. As illustrated in Fig. 1B, many synapomorphies characterize each level of a cladogram, and we have no reason to prefer one over another as a putative causal agent in regulating

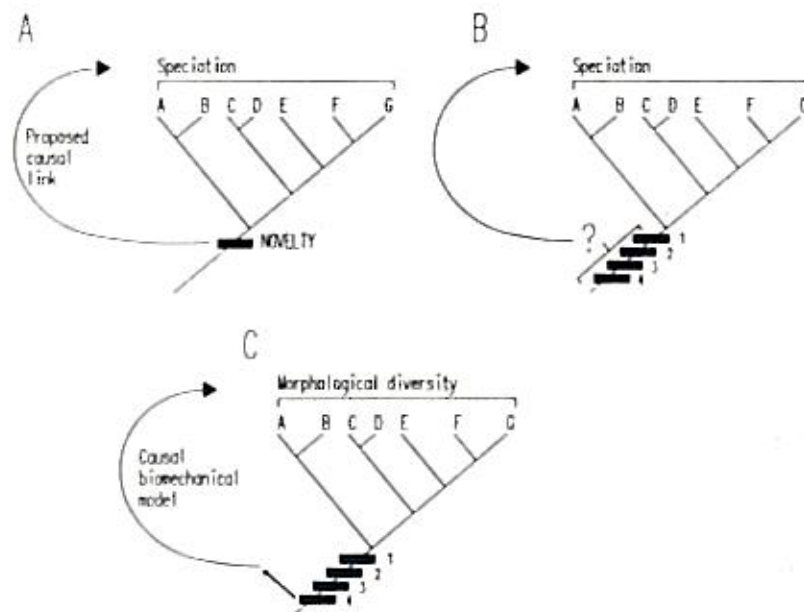


Fig. 1.—Diagram to illustrate in a phylogenetic context the classical concept of a key innovation (A), one major difficulty with this concept (B), and an aspect of a proposed revision to the key innovation concept (C). Note that many synapomorphies usually characterize any given lineage (B), and that in the absence of a causal model we have no reason to prefer any one of the synapomorphies as being causally related to diversification. In addition, the link between a morphological or physiological novelty and speciation rate is usually tenuous.

diversification: each is uniquely associated with the origin of the clade. Second, the causal connection often suggested between possession of some specific morphological feature and increased rates of speciation (or decreased extinction rates) is often tenuous. Exactly how does a change in a physiological pathway or musculoskeletal anatomy increase speciation rate in a clade? What are the causal links between possession of a new muscle, for example, and evolutionary "success"? Third, key innovations by definition are unique features (synapomorphies) of a clade. How do we test a hypothesized key innovation when it has only evolved once?

In order to avoid these difficulties, several modifications can be made to the key innovation concept that enhance our ability to test the historical consequences of novel features (Fig. 1C). First, in order to avoid the assumption of a link between a novelty and speciation rate, key innovations as hypothesized historical factors may be most testable when they are limited to explicit *structural* predictions about morphological or functional diversity. Rather than attempting to explain how possession of some morphological trait leads to increased speciation, our ability to test the historical consequences of a novelty will be enhanced if predictions are limited to structure per se (Fig. 1C).

Second, the proposed innovation is testable in the broad sense to the extent that it is a general (emergent) feature or property of a clade. It is easier to test the historical consequences of possessing general structural properties, such as decoupled biomechanical pathways, serial arrangement of parts, or complexity of design (Lauder 1981), than to test the effect of a specific novelty, such as the development of a new articulation between the parietal bone and the otic capsule. If a proposed historical factor has only evolved once, then testing by comparison is impossible. To some extent, then, convergent acquisition of structural specializations will enhance our ability to test for historical consequences of a novelty.

Third, an explicit causal model is needed (see Zweers 1979, 1985) that predicts what the relationship should be between the possession of a morphological novelty and the pattern of structural diversification in a clade. Without such a general causal model, it is easy to rationalize any correlation that is found between a derived novelty and the structural diversity that exists among terminal taxa in a clade, and to interpret that correlation as reflective of a causal historical relationship. A causal model allows us to assign one particular synapomorphy (e.g., Fig. 2, character 4) as causally related to structural and functional transformations.

Testing Historical Innovations: A Procedure

A general phylogenetic procedure for testing the historical consequences of novel features is illustrated in Fig. 2. The protocol advocated here for

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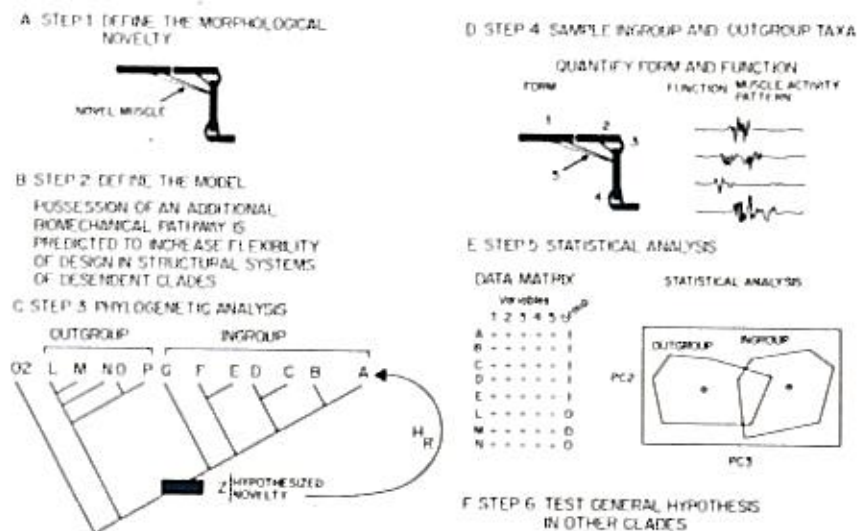


Fig. 2—Schematic diagram illustrating a six-step phylogenetic procedure (discussed in the text) by which historical hypotheses of structural innovation and constraint may be tested. A: A hypothetical musculoskeletal system is illustrated that resembles a vertebrate limb, and a novel muscle has been discovered connecting two of the bones (black bars). B: A general causal model is defined. C: The phylogenetic distribution of the novel muscle (Z) is shown, and this novelty is hypothesized to be causally related to structural and functional diversity in the ingroup clades (a relational hypothesis, H_R [Lauder 1981]). D: Form and function of the musculoskeletal system are quantified in both ingroup and outgroup taxa. For example, five variables (1 to 5) are measured from the limb. These morphological variables could be muscle cross-sectional areas, lever arms, or shape of bones. Function could also be quantified by measuring muscle activity and quantifying the motor pattern (e.g., Lauder and Shaffer 1988). E: A variable by taxon data matrix is constructed for both ingroup (I) and outgroup (O) taxa. This matrix may be subjected to statistical analyses such as principal components (PC) and the group centroids (circles) or variances compared. F: If the novelty (Z) is a general one, then this same procedure could be repeated on other clades.

examining the historical consequences of a key innovation or novelty involves six steps. This description builds on the earlier proposals of Lauder (1981, 1982a) and the subsequent modifications of Stiasny and Jensen (1987). As an aid to understanding this procedure, a hypothetical example involving the evolution of the musculoskeletal system in vertebrates will be used.

First, define the key innovation or novelty (Fig. 2, step 1). Typically, a novelty might come to the attention of an investigator through the examination of a morphological system or through the study of the behavior or ecology of species in a clade. For example, examination of the

musculoskeletal system in a clade of vertebrates may result in the observation that a novel muscle exists in some species between two bones in the limbs (Fig. 2A).

Second, propose a priori the historical effect of the novelty and elaborate a causal model that predicts (even in general terms) the consequences of possessing the novelty (Fig. 2, step 2). In the hypothetical example, it might be predicted that the addition of a novel muscle to the limb, through its demonstrated ability to permit a wider range of function, should result in increased functional and morphological diversity in the musculoskeletal system of the clade possessing this novelty. Experimental functional morphology (with electromyography, cinematography, and experimental manipulation) in a few ingroup species might reveal that the activity of this muscle is related to increased kinematic versatility, allowing the limb to be moved in novel ways. In general terms, the causal model predicts that the possession of an additional biomechanical pathway will increase flexibility of design and function in a structural system in descendant clades.

Third, obtain a phylogeny of the clade under consideration and several outgroup taxa (based on characters other than those related to the novelty). An independent estimate of genealogical relationships for the taxa under analysis is critical for testing historical hypotheses. Then, map the proposed innovation onto the cladogram (Fig. 2, step 3). This is perhaps best accomplished by coding each of the terminal taxa in the in- and outgroups for the presence or absence of the novelty, entering the topology of the cladogram into a quantitative phylogenetics program such as PAUP (Swofford 1984), and testing for congruence between the distribution of the novelty and input topology. Typically, within the clade, the proposed novelty might only have evolved once. The historical hypothesis to be tested is a relational one (Lauder 1981): possession of a morphological novelty (*Z*) is related in a causal historical sense to structural and functional diversity in descendant species (Fig. 2). In the hypothetical example, the novelty in the limb maps onto the cladogram once (Fig. 2, step 3; *Z*), and we wish to test the hypothesis that structural diversity in the ingroup (the clade for which the novelty is a shared derived feature) is greater than in the outgroup.

In our example, we have predicted in advance that the ingroup clade should exhibit increased structural diversity based on a model of musculoskeletal function and the expected consequences of adding structural elements to a complex design.

This prediction does not take into account any extrinsic factors such as differing ecological conditions in ingroup and outgroup taxa, nor does it address the origin of the novel muscle (which may perhaps have originated as a new subdivision of ancestral myogenic tissues). Rather, this is a second-level prediction about pattern that has a causal component.

Fourth, quantify the relevant structural features of the ingroup and

outgroup (for example, through a morphometric analysis [Fig. 2, step 4]). This involves several procedures. If the ingroup and outgroup clades are large, one should randomly sample taxa in both groups. For structural comparisons, it is best if the species diversity of the ingroup approximates that of the outgroup so that relative species diversity does not bias the estimate of structural diversity. Then, measure a suite of morphological and/or functional variables in each species. This data matrix will serve as the data set to test the historical hypothesis of ingroup versus outgroup differentiation in morphology. In the hypothetical example of the musculoskeletal system, a suite of morphological and/or functional features could be measured in each of the in- and outgroup species (such as muscle activity patterns, cross-sectional areas of the bones, truss measurements of limb bone shape, and muscle mass) and these measurements assembled into a data matrix of variables by species (Fig. 2, steps 4 and 5).

Fifth, conduct a statistical test comparing the outgroup to ingroup pattern of differentiation in form and/or function. For example, this might take the form of a principal components analysis of the variables, removal of size effects (Bookstein et al. 1985), and a multivariate analysis of variance to test for significant differences between the centroids of the ingroup and outgroup polygons (Fig. 2, step 5). Also, depending on the nature of the predicted effect of the novelty, one might wish to compare the variance about the multivariate mean for ingroups and outgroups. In general, one would predict that if the novelty has the predicted historical effect, then differentiation will be seen between ingroup and outgroup species.

Sixth, proceed to other clades to test the general hypothesis by comparison. While the procedure outlined above allows us to test the historical effect of a morphological novelty in one clade, in order for a relationship of general interest to emerge we must corroborate a historical effect of the addition of biomechanical linkages in other clades (see also Emerson 1988). The extent to which a general congruent pattern of historical consequences is found will determine the extent to which intrinsic historical factors may be viewed as having played an important role in the evolution of complex organismal functions.

It is important to note that this procedure only addresses one source of variation in structure in a clade. The methodology does *not* address questions relating to speciation or extinction rate and it does *not* address the causal basis of the production of novelties or their spread in a population. There are multiple levels of causal explanation for any historical phenomenon, and this procedure only allows a test of one level of causal explanation. In addition, we wish to emphasize that the methodology proposed above only tests the explicit structural hypothesis of historical effect of an aspect of design. Ingroup and outgroup clades may have such differing ecologies and have undergone such different patterns of contingent historical events that

no relationship can be discerned between possession of a novel feature and structural/functional diversification. Also, possession of a novelty by a clade may be a necessary but not sufficient event for a change in structural variation within the ingroup clade. However, only by utilizing an explicit methodology for testing for historical effects of aspects of organismal design will we be able to decide to what extent the possession of novelties in the body plan determines subsequent evolution in design.

An Example: Duplication of Genes and Biomechanical Linkages

An increase in the number of independent elements in a complex functional system has been implicated in the evolution of morphological and functional diversity in several systems (Lauder 1981, 1982a; Liem 1987, 1988; Schaefer and Lauder 1986; Vermeij 1973a, b). While few explicit tests of this idea have been conducted (see Emerson 1988 for the best test to date), several cases have been adduced that seem to support the concept. Stated more explicitly, the hypothesis is that clades possessing, as a novelty, a greater number of independent (decoupled) structural or functional components of design will exhibit an increase in morphological and functional diversity when compared to sister clades with fewer independent design elements. The underlying model for this historical hypothesis is that diversity of structure and function is positively related to the number of independent elements because of the increased possibility for change and new interconnections as the number of independent components in a complex system increases. The presence of several independent elements as a derived condition, where primitively only one element is present, also releases constraints on the duplicated elements that may have limited structural and functional diversification on the single element previously.

The predicted relationship between decoupled structural components and historical flexibility of design by no means rules out the possibility that primitively decoupled elements may subsequently become highly coupled. Indeed, as structural design is modified within a clade and the diversity of body plans increases, primitively distinct elements of design may acquire tight functional associations that constrain, at another phylogenetic level, directions of morphological change.

A particularly good example of the role of independent elements of body plans in governing historical diversification occurs in the evolution of diversity in gene function. Well-studied cases include the evolution of hemoglobins, tubulins, and lactate dehydrogenase (see Markert et al. 1975; Ohno 1970; Raff et al. 1987; Romero-Herrera et al. 1978; Zuckerkandl 1976). The phenomenon of gene duplication is well documented and results in a second copy of a gene for which there was primitively only one copy (Fig. 3), thus forming a duplicate structural element in the genome. When

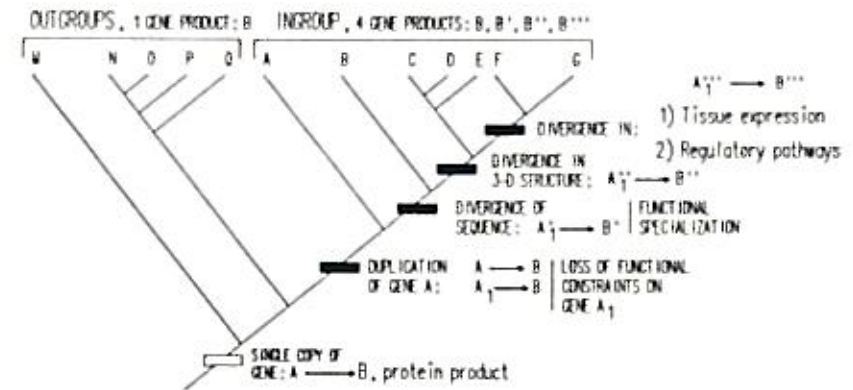


Fig. 3—Diagram of a typical pattern of phylogenetic diversification in gene families following gene duplication. Primitively, gene A is present in only one copy and makes a protein product B. The ingroup for this analysis is characterized primitively by a gene duplication event which results in two copies of gene A (A and A_1) which both primitively make protein B. Duplication of gene A releases functional constraints on the protein product of gene A_1 and is causally related to subsequent structural, regulatory, and functional diversification in the protein products of gene A_1 (B' , B'' , and B''').

there is only one copy of a gene whose product is of considerable importance to the maintenance of normal regulatory function (e.g., hemoglobin) there is little divergence in structure and function of the gene through time: functional constraints on the gene product are high and the consequences of modifying it are severe. However, duplication of genes frees the duplicate from these constraints as the other gene copy can continue to produce the essential product.

As illustrated in Fig. 3, a pattern of gene and gene-product divergence typically follows the duplication event. Primitively, a clade may possess one copy of a gene (Fig. 3, A) that makes one protein product (Fig. 3, B). Following the initial duplication of gene A, divergence in both gene sequence (and thus amino acid sequence and the three-dimensional structure of the gene product) may occur. In addition, divergence in regulatory pathways governing the timing of gene expression and the tissue location of gene expression may occur. This pattern of divergent specialization of structure, function, and regulation in duplicated gene products is repeated in the gene families whose evolutionary history has been studied to date.

The net result of this process of duplicate gene divergence and specialization is that the ingroup (Fig. 3), which primitively possesses the duplicate genes, has considerably increased structural and functional diversity as compared to outgroup clades (Fig. 3). While clades in the ingroup retain the ability

to make the primitive gene product (Fig. 3, A→B), they also have acquired the ability to make three new gene products (B', B'', and B''') as well as a differential pattern of regulation of these products (Markert et al. 1975). This provides an excellent example of the role of an intrinsic historical factor (duplicate structural components) in governing (in a causal sense) morphological and functional diversity in a clade. It is clear that a substantial component of the variance in gene product diversity is attributable to the historical factor of structural duplication. Without duplication of genes primitively in the ingroup (Fig. 3), very little structural, regulatory, or functional divergence would have occurred in the protein product of the primitive gene A.

A second example of duplication of design elements is provided by the musculoskeletal system in the jaw of ray-finned fishes. Here, duplication of biomechanical linkages has resulted in divergent specialization and an increase in structural and functional diversity in a manner directly equivalent to the gene duplication example presented above. We suggest that the causal model underlying the diversification of jaw structure in lower vertebrates and the diversity of structure and function in gene families is identical: an increase in the number of pathways to achieve a function.

As illustrated in Fig. 4, ray-finned fishes primitively possess one biomechanical pathway to depress the mandible during the mouth opening phase of feeding (Lauder 1980, 1982b; Lauder and Shaffer 1989). This biomechanical system involves ventral body musculature and connections between the hyoid and mandible (schematically illustrated in Fig. 4). The primitive presence of only one mechanism for opening the mouth is equivalent to the primitive occurrence of only one copy of a gene (Fig. 3): functional constraints on modification of this biomechanical pathway are great (since this pathway is the only way the fishes have of depressing the mandible) and limit the extent to which functional and structural divergence can occur.

Within ray-finned fishes, a second (duplicate) biomechanical system for depressing the lower jaw evolved at the halecostome level (Fig. 4); this provided two independent biomechanical pathways for mediating mouth opening in this clade and released the second system from the functional constraints imposed on the primitive system. Just as possessing two copies of a gene making one protein product frees one copy for subsequent modification, so the possession of duplicate biomechanical pathways permits the development of specialization and divergence in structure and function of the second biomechanical pathway. In ray-finned fishes, the effect of this release from functional constraint in the mouth opening pathway was particularly dramatic, as a primitive clade of teleost fishes possessing the dual biomechanical pathways, the Osteoglossomorpha, has greatly modified the primitive hyoid system for chewing and has developed a novel bite

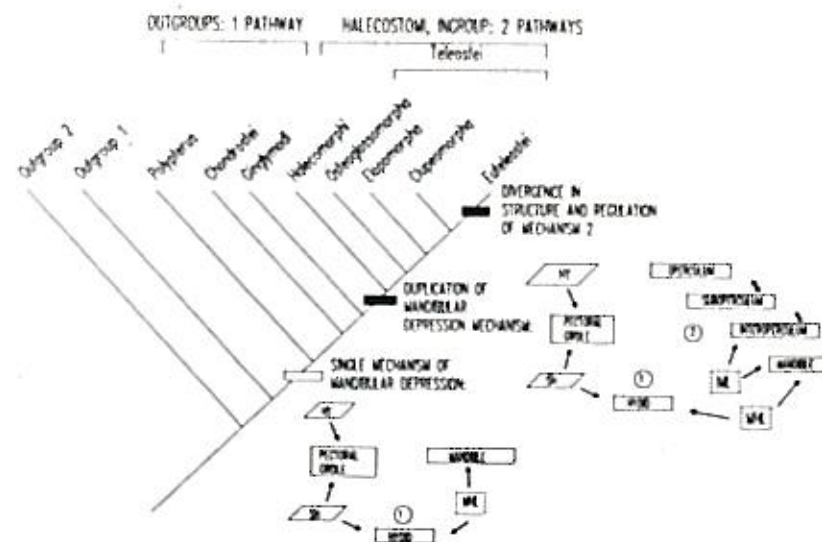


Fig. 4—Diagram of the duplication in biomechanical pathways in the feeding mechanism of ray-finned fishes. We contend that the diversity of jaw structure and function in the skull of lower vertebrates is causally related to the primitive duplication of biomechanical pathways in a manner directly analogous to the effects of gene duplication. Primitively in ray-finned fishes, there is one biomechanical pathway for depressing the mandible when the mouth is opened. This mechanism involves co-contraction of the hypaxial muscles (HY), the sternohyoideus muscle (SH), and the application of a force to the mandibulohyoid ligament (MHL). The halecostome clade (the ingroup) is characterized primitively by two biomechanical pathways, both of which act to open the mouth. One of these (1) is identical to the primitive system, while the second (2) is a novel functional linkage between the interoperculum and the mandible via the interoperculomandibular ligament (IML). In the diagrams of the biomechanical pathways, muscles are shown in parallelograms, bones in solid rectangles, and ligaments in dashed rectangles. Note that much subsequent divergence of function and structure in the hyoid linkage system occurs in teleost fishes. We suggest that this diversification is causally related to the duplication of biomechanical pathways that allowed specialization to occur.

between the base of the skull and hyoid. The novel morphological changes in the hyoid and the novelties in the muscle activity pattern associated with use of the hyoid biomechanical system during chewing are unlikely to have evolved when the hyoid was functionally required for mouth opening. Decoupling of the hyoid biomechanical system from its primitive mouth opening function is predicted to have permitted the evolution of a morphologically and functionally specialized chewing system in osteoglossomorph fishes.

While neither of these examples relating duplication of structural elements to historical diversification has been rigorously tested using the protocol for

key innovations outlined above, the available data strongly support the interpretation that duplication of a major structural feature is causally related to subsequent historical diversification. This provides an indication of the importance of historical factors in the evolution of protein structure and jaw function in vertebrates, and provides a concrete basis for a broader investigation of the role of historical factors.

HISTORICAL CONSTRAINTS

In contrast to the discussion above of factors that might result in an increase in structural and/or functional diversity in a lineage, many workers have emphasized constraints or factors that might limit the expression of diversity in design (e.g., Alberch 1982; Gould 1980; Gould and Lewontin 1979; Maynard Smith et al. 1985; Wake 1982).

Perhaps the major proposal for limitations on design diversity is the concept that ontogenetic pathways constrain the diversity of adult phenotypes (Alberch 1980; Maynard Smith et al. 1985). If developmental pathways are such that only a limited array of phenotypes may be produced, then the expression of phylogenetic diversity may be limited by the narrow range of permissible morphologies allowed by conservative developmental programs and interactions (Wake and Roth, this volume).

Those concerned that developmental constraints may limit the diversity of phenotypes seen in a clade should address several important issues, including (a) phylogenetic patterns to developmental pathways (see Fink 1982), (b) the phylogenetic distribution of the phenotypic output of the developmental pathways, and (c) the production of a causal model that explains why particular developmental interactions should be causally related to limitations on phylogenetic diversity in design. While a solid attempt has been made to relate variation in ontogenetic trajectories to patterns of morphological differentiation (Alberch et al. 1979), few specific causal models are available to explain how interactions among developmental pathways limit phylogenetic diversity in design. Even fewer cases are available that test proposed developmental constraints.

Most proposals for developmental constraints are highly descriptive and lack explicit causal hypotheses that can be tested. Merely describing the range of actual variation in a structure and finding that it is less than the range of theoretically possible variants does not by itself test hypotheses of constraints. If the mode of development of the aorta was proposed to be a causal factor underlying the limited diversity of aortic branching patterns in amniotes (Alberch 1980), then the appropriate methodology for testing this hypothesis is the six-step procedure illustrated in Fig. 2. Mechanistic developmental research on individual species or the description of ontogenetic

trajectories will not test what is fundamentally a historical and comparative hypothesis (Fink 1982; Haynes 1988).

Despite the wide discussion of constraints in the evolution of organismal design, we feel that one class of constraints has not received the recognition it deserves. Relatively little attention has been paid to the possible constraints imposed by interrelationships among different levels of biological organization. For example, to examine the diversity of behavior exhibited by species in a clade and to understand the mechanistic bases for differences among species, one might want to consider other levels of analysis that might explain how differences in behavior arise (Lauder 1986, 1989; Lauder and Shaffer 1988). As illustrated in Table 1, these other levels might include an analysis of peripheral morphology, muscle activity patterns controlling behavior, physiological properties of the musculature, and the morphology and function of central nervous system circuits.

An example of these constraints is provided by anabantoid fishes in which air ventilation is accomplished by either a water-dependent quadruphasic or a water-independent triphasic motor pattern (Liem 1987). These contrasting motor patterns have profound effects on the behavior of these fishes. Only those taxa with the water-independent motor pattern can emerge from the water, while taxa with water-dependent quadruphasic motor patterns are constrained in their behavior to remain in the water even during environmental catastrophes.

How does the design of central circuitry constrain the range of behaviors exhibited by a clade? To what extent is peripheral morphology linked to central nervous programs? How tightly coupled are interspecific differences in behavior to alterations in musculoskeletal design? These are fundamental

TABLE 1 Levels of analysis in complex morphological systems.

Level of analysis	Structures/functions to be studied
1. Behavioral	Movements and movement sequences
2. Peripheral morphology	Anatomy of the musculoskeletal system
3. Muscle physiology	Physiological properties of the peripheral musculature (time-to-peak twitch tension, contraction time, etc.)
4. Motor pattern	Pattern (sequence and timing) of central nervous output to the peripheral musculature
5. Central nervous system structure	Anatomy of brain and spinal cord nuclei and their interconnections
6. Central nervous system circuits	Neural networks and the pattern of neuronal excitation

issues in our understanding of the diversity of design in organisms that have not yet been addressed in a quantitative way.

CONCLUSIONS

We wish to emphasize our view that the importance of historical factors in regulating the evolution of diversity in organismal design needs to be rigorously assessed. If we aim to explain the diversity of organismal design, then we must have a research program that allows us to examine quantitatively the role of historical factors and to answer the following question: How much of the variance in design within clades is predictable on the basis of intrinsic design features of those clades?

We have outlined one procedure for testing the role of historical factors in the evolution of complex organismal functions. While this is not the only procedure that could be used to examine the evolutionary significance of a novelty or key innovation, it has the advantage of using an explicit phylogenetic framework, an explicit a priori prediction of patterns of morphological diversification, and a causal functional model. While there are several levels of causal explanation for any historical phenomenon, this procedure addresses one important class of explanations. Another important avenue of research on historical factors has been initiated by Cheverud and his colleagues (Cheverud 1984; Cheverud et al. 1985). Relatively few studies involve a priori predictions of patterns of morphometric variation; most attempt a posteriori rationalizations. It is especially important to emphasize the role of a comparative phylogenetic analysis in testing the role of developmental constraints.

In underscoring the importance of historical factors in explaining organismal design, we do not deny major explanatory roles for the processes of selection and adaptation, or the contributions of population biologists to understanding the origin of novelties within populations. Indeed, these are critical components of any general understanding of the process of structural diversification. Rather, we wish to highlight the contribution that a comparative and phylogenetically based research program can have in enhancing our ability to test hypotheses of design versatility and constraint.

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