The evolutionary links between fixed and variable traits

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This paper discusses the evolutionary relations between fixed and varying traits. The first puzzle is, how did the fixed traits become fixed? In one scenario, after speciation establishes the independence of the daughter gene-pools, selection canalizes different traits in different species. In each clade, some still-variable traits coevolve with the canalized trait. This embeds the canalized trait in a network of interactions with other traits so that continued successful function depends upon that trait remaining canalized. Clade-specific constraints result because the canalized trait cannot now be changed without incurring costs too high to be paid in the fitness contributions of the other traits, and because the canalized trait is clade-specific. In another scenario, colonization of a new habitat, or evolution of a new stage in the life cycle, produces some 'temporarily neutral traits' that had been useful in the old habitat or life cycle. Those traits are then free to evolve for other purposes; some of them become incorporated in structures serving other functions than their ancestral homologues. The process is irreversible, for they cannot evolve back to their previous structures and functions without an unacceptable fitness cost. The second major puzzle is, do the fixed traits affect the further evolution of the traits that remain genetically variable, thus producing clade-specific patterns of response to selection? The impact of discontinuous growth on the expression of genetic variation in size-related traits in arthropods suggests that the answer is yes. Comparative, phylogenetic analysis of the impact of prior fixations on patterns of variation may also yield insights; potential problems are discussed.

Key words: canalization, phylogenetic constraint.

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Libchaber believed that biological systems used their nonlinearity as a defense against noise.
(J. Gleick)

Introduction

The three major themes of biological evolution – natural selection, history, and chance (Williams 1992) – are connected. The patterns now called historical originated through natural selection and chance, and historical patterns in their turn now constrain the response to selection and the scope of chance events. However, microevolutionary theory does not yet explain how varying traits evolve into fixed traits, and how fixed traits become one source of constraint on the further evolution of the still-varying traits. These deficiencies define a challenge to be faced.

Theories of phenotypic evolution, like demographic life history theory and evolutionary quantitative genetics, are ahistorical, as is physics. To make successful predictions about phenotypes, they must assume, explicitly or implicitly, historical particulars which appear as boundary conditions or empirically fitted parameters in the models (e.g. tradeoff functions, genetic variance-covariance matrices). To make local predictions they must take much of the state of the system as an already highly-evolved given, *deus ex machina.* This is not satisfactory; it should be possible to connect the variable traits to the fixed ones; doing so would connect macro- to microevolution.

There are two major puzzles in the relation of the traits that are fixed within clades to the traits that still vary among individuals. The first is, how did the fixed traits become fixed? I assume that traits were primitively variable and that fixation is a derived state. One can start from models of the evolution of canalization, and from the assumption that different traits get fixed in different clades for historical reasons.

The second puzzle is, what are the effects of the fixed traits on the further evolution of the variable ones? Is the expression of the genetic variation of the variable traits affected by which other traits happen to be fixed in that clade? How do the fixed traits affect the further evolution of the traits that remain genetically variable, thus producing clade-specific patterns of response to selection? Here one tries to derive constraints on the varying traits from the properties of the fixed traits.

Clade-specific patterns of variation were documented by Vavilov (1922) for crop plant's. Important theoretical and experimental analyses of the causes of clade-specificity in patterns of variation have been made by Riedl (1975), by Alberch (1989) for amphibian limbs, by Nijhout (1991) for butterfly wings, and by Ebert (1991) for crustacean life histories.

Definitions

Clade. — A 'clade' is anything that can be fairly represented by a dendrogram in which properties of descendants are determined by properties of ancestors within isolated gene pools (Williams 1992). 'Clade-specificity', or autapomorphy, indicates a property of a clade that is characteristic of that clade and not of related clades.

Tradeoff. — By 'tradeoff I mean connections between traits that constrain their simultaneous evolution. Physiological tradeoffs result from allocation decisions between processes competing for limited resources within an organism. The simplest physiological tradeoff is a 'Y' tradeoff, where one resource must be divided between two competing functions. Microevolutionary tradeoffs occur within populations, not within individuals, when an evolutionary change in one trait that increases fitness causes a change in another trait that decreases fitness. Some authors describe macroevolutionary tradeoffs, but these are only patterns, relations between traits detected by comparisons of clades in which a change in one trait that would increase fitness if it occurred within a deme is associated with a change in another trait that would decrease fitness if it occurred within a deme. All three types of tradeoffs are discussed in the literature. However, the relations among them are not clear. To what degree are they the product of the same mechanisms, and to what degree do they differ in their causation? Might they all reflect a basic constraint shared by all individuals, demes, species, and higher taxa belonging to a given clade? We do not yet know.

Evolutionary constraint. — By 'evolutionary constraint' I mean the things that cannot be changed by selection, that are fixed within clades or made otherwise unavoidable by physical, chemical, or evolutionary law. Evolutionary constraints fall into two broad classes: external constraints (physical and chemical) and internal systems constraints (developmental, morphological, physiological).

Specialists emphasize their own fields in defining constraints. Geneticists talk about genetic constraints, morphologists about developmental and morphological constraints, physiologists and students of biomechanics about physical and chemical constraints, ontoecogenophylogeneticists about ontoecogenophylogenetic constraints (Antonovics & van Tienderen, 1991). Whatever one calls them, one needs to know how long one can expect a constraint to be in effect, and here there are some fundamental differences.

Physical and chemical constraints will last as long as this universe exists. Their existence is not controversial even if their consequences remain research topics in evolutionary and developmental biology.

Lack of genetic variation is one criterion for detecting all classes of biological constraints (developmental, morphological, physiological), but, in contrast to physical and chemical constraints, the number of durable constraints that are ultimately caused by lack of genetic variation is

probably small. Not many constraints can be helpfully called genetic constraints. A lack of genetic variation is not a reliable indicator of long-term constraint, for a trickle of mutations can still enter the population and lead to slow, steady phenotypic change while any known method of measuring heritabilities would not be able to estimate an additive genetic variance that was significantly different from zero.

Although all genes mutate, some traits are invariant within clades. On the other hand, some traits vary a great deal without any corresponding genetic variation (e.g. in clones). Plasticity and canalization decouple variation in traits from variation in genes. Reductions in genetic variation produced by canalization should last much longer than those produced by selection, for the restoration of variation would require not just mutations and gene flow but the further evolution of the develpmental system to de-canalize the phenotype.

For all these reasons, true genetic constraints, defined by a real lack of genetic variation itself, and not an apparent lack of genetic variation caused by canalization or systems constraints, can only constrain the dynamics of phenotypic evolution for a few generations. Asexuality, which prevents the recombination of information, and diploidy, which allows the accumulation of recessive mutations, are two of the few long-term genetic constraints on evolution.

Systems constraints fall into two general classes. First, there are evolutionary irreversibilities caused by the use of redundant structures elsewhere in the body for new, vital functions. One cannot go back to the old organization because the elements previously used to create vanished structures are now used somewhere else. This kind of constraint is exemplified by the plethodontid salamanders studied by Wake and his colleagues (e.g. Wake & Larson 1987).

These salamanders have lost their lungs and breathe through their skins, and the bones and muscles previously associated with lung breathing have moved forward in the thorax to enable the construction of a more efficient protrusible tongue used in capturing food. Some have also switched from larval to direct development, and the structures previously used in larval gills have found new application in the adult structures of the directly developing species. Thus plethodontid salamanders are constrained in the sense that it would be hard to select for reversal to lung breathing and larval development because the structures that ancestral salamanders use for those two processes have been employed elsewhere and could not be recovered without killing the developing animal. Recall that the bones of the mammalian inner ear were originally breathing aids as gill arches in fish, became feeding aids in the jaws of amphibians and reptiles, then were hearing aids when the mammalian jaw was simplified (Romer 1962). To select the mammalian jaw to incorporate the incus, malleus, and stapes again would imply drastic hearing loss.

A second class of systems constraints arises in one structure that has two or more essential functions. This functional superposition is the

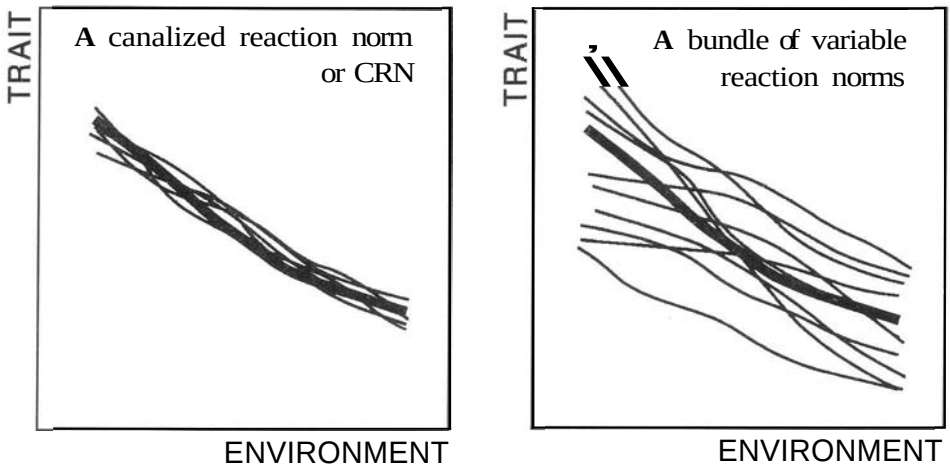

Fig. 1. Canalized reaction norms (CRNs) are environmentally variable but genetically invariant. Each line represents *a* genotype.

opposite of redundancy. Take as example the gills of mussels; they are involved both in feeding and in breathing. Gills could not retain their feeding function while being transformed into lungs, nor could they retain their breathing function while being transformed into jaws with teeth. This does not mean that an air-breathing bivalve is an evolutionary impossibility, but like a lung-breathing plethodontid, it would take a long time, for the same condition would have to be met: tissues freed up from .other functions would have to take over either breathing or feeding.

Some comments on canalization

Canalization of primitively variable traits is a central element in scenarios for the fixation of key traits and the origin of systems constraints Schmalhausen (1949).Canalization has two main benefits. The first pertains to traits under stabilizing selection whether they occur in sexual or asexual organisms. If a trait has an optimum value, or an optimum reaction norm, then it is under stabilizing selection. Organisms that deviate less from the optimum value will have higher fitness, and canalization should evolve to hold traits close to that optimum value. The second benefit of canalization is that it produces traits that can be relied upon for trait coevolution by reducing the problem of trait recombination, discussed below. For both reasons, the evolution of development created the necessary conditions for the evolution of clade-specific constraints.

What gets fixed: canalized reaction norms (CRNs). — The definition of fixed and variable traits is not straightforward, for a trait may be phenotypically plastic but genetically fixed. Consider a phenotypically plastic trait with little heritable variation. Its expression is environment-

dependent but perfectly predictable; every time an environment occurs, the trait takes a certain value, albeit a different value in each environment. For the other traits in the evolving population, it is a reliable and unchanging element of their developmental environment so long as they are also sensitive to the same environmental variation that it is. Such a trait is a tightly canalized reaction norm (Fig. 1).

The developmental problems posed by gene and trait recombination. — In asexual organisms, mutations add variation sequentially, only rarely in parallel; the selection pressure to canalize traits against genetic perturbations will be steady but small. In sexual organisms, progeny encounter the problem of genetic recombination, and in time this greatly increases the selection pressure for genetic canalization. Traits have to be produced in stably functioning combinations despite the shuffling of the genes. In a sexual population with no canalization and recombination among traits, every time a trait is expressed in a different individual it encounters a developmental environment in which many other traits take on different values. It must become adapted to a variable environment described by means and variances of the traits with which it interacts. This poses severe design problems.

There are at least four ways out of this difficulty.

The first is natural selection alone: the only survivors will be those with the correct match among traits. This can only be a sufficient solution in a population with high fecundity, for it implies low juvenile survival. It also generates strong selection for more efficient solutions.

The second is that the developing organism somehow automatically (without natural selection, solely as a result of the physical and chemical laws governing self-assembly of components) produces traits that are appropriately matched to each other. Allometry will take care of some of this, but only (according to the assumption) to the degree that allometric coefficients have not yet been adjusted by natural selection. This could only be part of the solution.

The third is that natural selection will favor uncanalized developmental systems that reply on phenotypic plasticity to adjust the varying traits to each other in all genetic combinations and in all environments. If there is no canalization of key traits, this would appear to be implausibly complex.

The fourth is genetic canalization of key traits through the evolution of redundancy in developmental mechanisms – fail-safes, back-ups, functional redundancy, over-engineering – and through nonlinearities that stabilize expression, evolved or not. In a sexual population with canalized reaction norms for key traits, the problem is reduced for the interactions of the variable traits with the canalized reaction norms, for they interact with traits with predictable values. This could accelerate their own approach to an optimum value in the given environment, no matter what the original reason for the evolution of the CRN with which they are interacting. Having CRNs for key traits makes the adaptation of the rest of the phenotype happen more rapidly.

Under this view, genetic canalization began to evolve in a serious way after the origin of sex as a method of stabilizing the development of organisms confronted with the new problem of trait recombination. There then followed refinements of the developmental system to adjust the still-varying traits to one another in combinations that maximized mean fitness while minimizing variation in fitness.

Scenarios for the origin of clade-specific constraints

To guide the search for the origin of clade-specific constraints, we need scenarios of how they might have arisen. Here some are proposed and their key features are noted. Common to all of them is the evolution of canalization. The first two are developments, in different language, of ideas in Riedl (1975).

Scenario 1: Canalization, embedding interactions, fixation

Step 1. — Speciation establishes the independence of the daughter gene-pools.

Step 2. — Selection now canalizes different traits in different species.

Step 3. — In each clade, some still-variable traits coevolve with the canalized trait. This step embeds the canalized trait in a network of interactions with other traits in such a way that the continued successful functioning of the organisms depends upon that trait remaining canalized.

Step 4. — Clade-specific constraints result because the canalized trait cannot now be changed without incurring costs too high to be paid in the fitness contributions of the other traits, and because the canalized trait is clade-specific.

If this process is repeated many times, one trait after another will be canalized and then fixed by a web of developmental, physiological, and biomechanical interactions.

Why does the process not continue until all traits are fixed? There are costs and benefits to canalization and to variation. The short-term, individual benefits of variation include the production of variable offspring that, as a set, can better exploit heterogeneous environments and can better resist diseases and parasites. The long-term, population benefits of variation include more rapid evolution, elimination of deleterious mutations, less chaotic population dynamics, and lower extinction probabilities.

In a steady-state environment, one would expect the canalization of key traits to proceed along a trajectory of decreasing returns until it reached a point where further canalization would not bring much and further reduction in variation would cost more. At that point, the canalization of the organism should stop and the ratio of the degree of canalization to the

degree of variation of each trait should reach an equilibrium value. That is why the process does not continue until all traits are fixed.

Scenario 2: Old structures used in new functions

Step 1. — Colonization of a new habitat, or evolution of a new stage in the life cycle, with the result that some traits that had been evolved for the old habitat or for the old life cycle are no longer necessary. Let us call them 'temporarily neutral traits', whatever the reason.

Step 2. — Those temporarily neutral traits are then free to evolve for other purposes; some of them become incorporated in structures serving other functions than their ancestral homologues. The process is adaptive throughout, but it is irreversible, for once the temporarily nearly-neutral traits have been incorporated in new structures with important functions, they cannot evolve back to their previous structures and functions without an unacceptable fitness cost. If the organism reinvaded its ancestral habitat, it would have to find other means of adapting to it than the ones that had been used before.

A good example of Scenario 2 are the plethodontid salamanders mentioned above.

In both scenarios, the reason that a focal trait cannot be changed by selection is that other traits can only make their maximal contribution to fitness if the focal trait remains fixed. Thus one cannot distinguish them with formal properties of the current state of the system. One must use other types of information to infer the history of the homologues of the focal trait. For Scenario 1 it is not logically necessary that any of the traits have changed their function relative to homologues found in inferred ancestral states. For Scenario 2, finding such a change in function from its homologues to the focal trait would strengthen the case; failing to find such a change would eliminate Scenario 2. The test can only be done comparatively and with reliable identification of homologues.

Scenario 3: Clade-specific biomechanical constraints

Oster and Alberch (1982), Meinhardt (1982), Alexander (1983), and Vogel (1988), following Thompson (1961) and Turing (1952), show that many constraints have their origin in the physical and chemical properties of biological materials. One can get clade-specificity out of biomechanics if the materials and structures that imply physical constraints are themselves clade-specific.

Such models are particularly convincing as mechanisms for the origin of major transitions of the sort involved in symmetry breaking, e.g. the Turing model of gastrulation. It may well be that, because of the tension holding the cells together, the first spherical hollow balls of cells gastrulated willy-nilly when they grew to a certain size and were forced to live with the consequences. Once tube-like organisms with layers had been

formed, however, the usual processes of natural selection came into play, genetic modifiers of the process were selected, and the present-day result may have little to do with the original mechanism. For example, organisms that today gastrulate by the movement of individual, independent cells could well have had ancestors that gastrulated by spontaneous symmetry-breaking.

Modelers of the biomechanics of early development often assume, following von Baer, that events occumng early in development are more constrained, harder to change in evolution, than events occurring late in development. This idea is currently under attack; good counter-examples are known; Wagner & Misof (1993) give a table of ten examples of developmental pathways that are more variable than the adult characters that they produce. Natural selection works on *all* stages of the life cycle, and with particular strength on the juvenile stages. If a change in the environment of the juvenile stages – e.g. larvae – is not mirrored by a parallel change in the environment of the adult stage, then one should expect more rapid evolution of the larvae than of the adult.

The counterargument runs, larval structures are necessary precursors of adult structures; larva and adult are bound together by systems constraints; the key traits upon which adult traits depend are in the larvae; if the larva changes very much, the adult will no longer function. The accumulating cases of larval change with adult stasis in anurans and echnoids shine suggest that larval-adult systems constraints are either not general or are not strong, or both.

Microevolutionary consequences

> *Therefore, we* **may** *conclude that, in general, closely allied Linnean species are characterized by similar and parallel series of varieties; and, as a rule, the nearer these [species] are genetically, the more precise is the similarity of morphological and physiological variability. Genetically nearly related [species] have consequently similar series of hereditary variation... Whole botanical families in general are characterized by a definite cycle [series] of variability which goes similarly through all genera of the family.*
>
> [N.I. *Vavilov*, 1922)

The impact of mode of growth

With every body plan there is associated a characteristic mode of growth. Hydroid colonies grow like plants with apical dominance, branching points being determined by distance along the stalk from an organising center that produces a hormone. When the concentration of the hormone drops below a certain threshold, a bud is produced and a new stalk forms. Arthropods are constrained by their chitinous exoskeleton to grow discontinuously, molting periodically and taking up water during the molt to increase body size. Mollusks, echinoderms, and chordates grow continu-

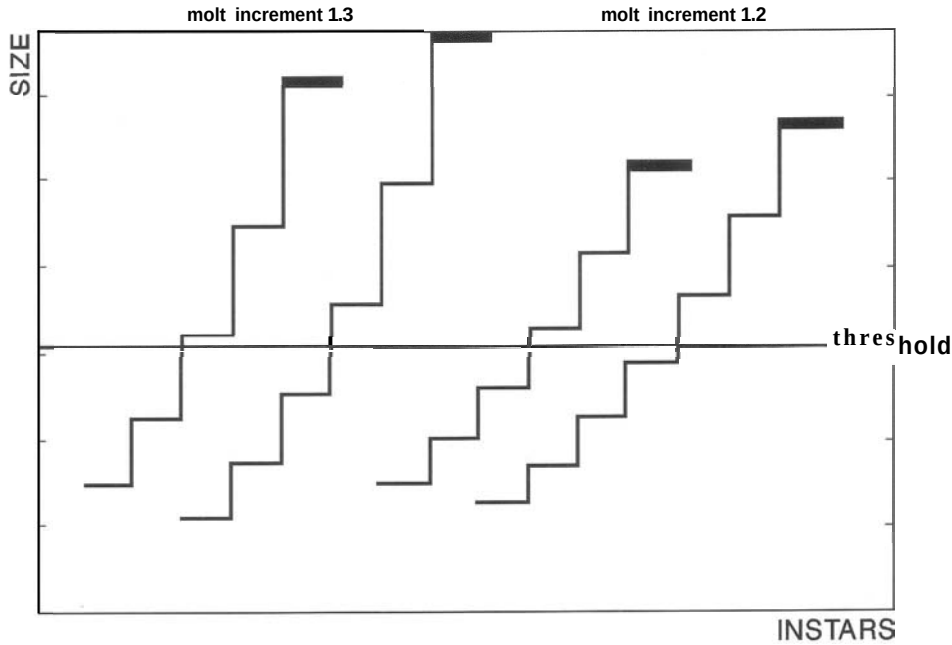

Fig. 2. In discontinuously growing organisms with a fixed maturation threshold, size at birth and growth rate interact to produce structured phenotypic variation in age and size at maturity (after Ebert, 1991b). In particular, organisms larger at birth can be smaller at maturity.

ously. That these different patterns of growth could have consequences for the expression of genetic variation for all size-related traits, and thus for the response to selection, is a possibility first explored in detail by Ebert (1991a, b; 1992).

The essence of the idea is this: if an organism grows discontinuously by molting but has a size threshold for maturation, then it will either overshoot or undershoot that size threshold on its nth molt. If it overshoots, it matures after n instars. If it undershoots, it matures after n+1, n+2, ... instars. This generates considerable discontinuous phenotypic variation in size at maturity, age at maturity, and all subsequent size-related traits, including fecundity and size of offspring (Fig. 2). The variation in size of offspring then feeds back into the growth pattern by generating size variation at the start of the growth trajectory.

This all sounds simple enough. However, Ebert has shown that it can have surprising consequences. For example, directional selection on increased body size of adults can, depending on the phenotypic distribution of sizes at birth, translate into directional selection for increased size at birth, decreased size at birth, stabilizing selection on size at birth, or disruptive selection on size at birth (Fig. 3). Because size at birth and size at maturity are related by discontinuous functions, this can set up a

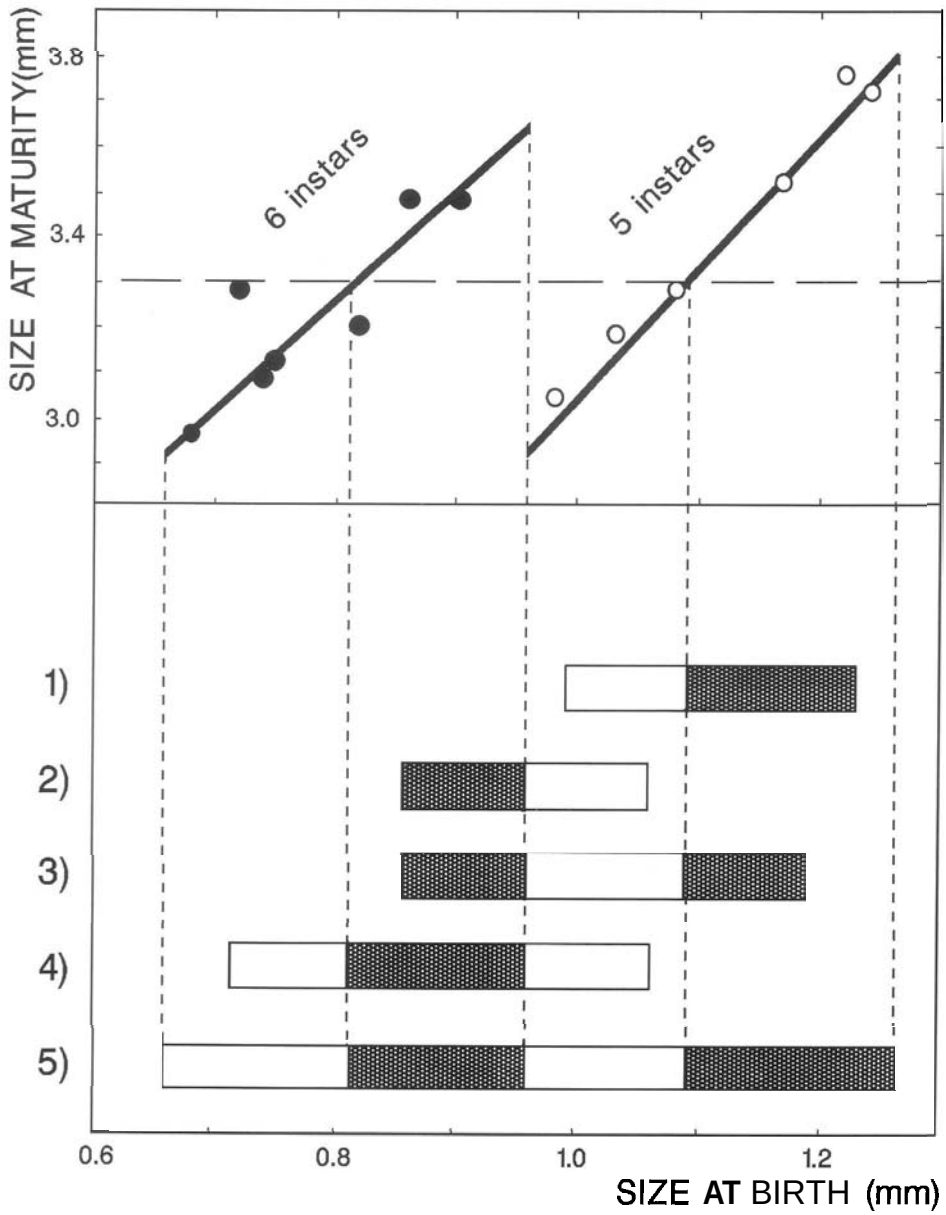

Fig. 3. The consequences of discontinuous growth can produce counterintuitive effects on the response to selection. (a) Size at maturity for ***Daphnia*** that matured in either 5 or 6 instars. (b) Patterns of selection on various ranges of size at birth if adults larger than 3.3 mm are selected: (1) directional upwards, (2) directional downwards, (3) disruptive, (4) stabilizing, (5) complex.

complex dynamic within which the expression of genetic variation for growth rate and size at birth is modulated by the discontinuous growth pattern.

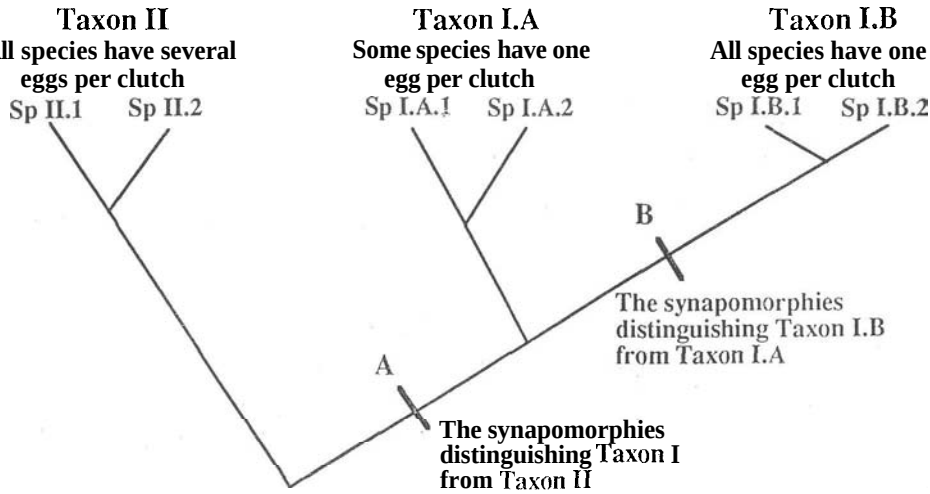

Fig. 4. An attempt to define clade-specific constraints with phyletic analysis.

The example makes one point clear: a body plan implies a mode of growth, and certain modes of growth strongly constrain the patterns of expression of genetic variation and covariation of size-related traits.

Comparative analysis of phylograrns

Specific to a clade are its body plan, development, and physiology. A glance into comparative studies suggests one problem in interpreting clade-specific patterns: explanations of clade-specific constraints based on physics, chemistry, and biomechanics. For example, there are no water-breathing true homeotherms because water has 90,000 times the heat capacity of air per unit of oxygen extracted; the heat lost could never be paid back by the oxygen gained (Steen 1971). Thus a constraint may be clade-specific not for reasons of internal design but because the entire clade encounters an external physical constraint associated with the habitat in which all members of the clade live.

Do clade-specific constraints exist? Phylogenetic analysis can suggest that they do but cannot, by itself, demonstrate their existence. Consider Fig. 4.

Let us assume that the phylogenetic hypothesis is soundly argued. We want to know why all the species in Taxon I.B (e.g. the tubenosed birds, order Procellariiformes) only lay one egg per clutch. The cladogram itself suggests some possibilities. Our first hypothesis would be that the synapomorphies labelled B, distinguishing Taxon I.B from Taxon I.A, imply clade-specific constraints on clutch size in Taxon **I.B.** This hypothesis suggests we search for physiological, developmental, and morphological changes at point B on the tree that can be plausibly connected to restrictions on clutch size. Our second, more complex hypothesis, is that

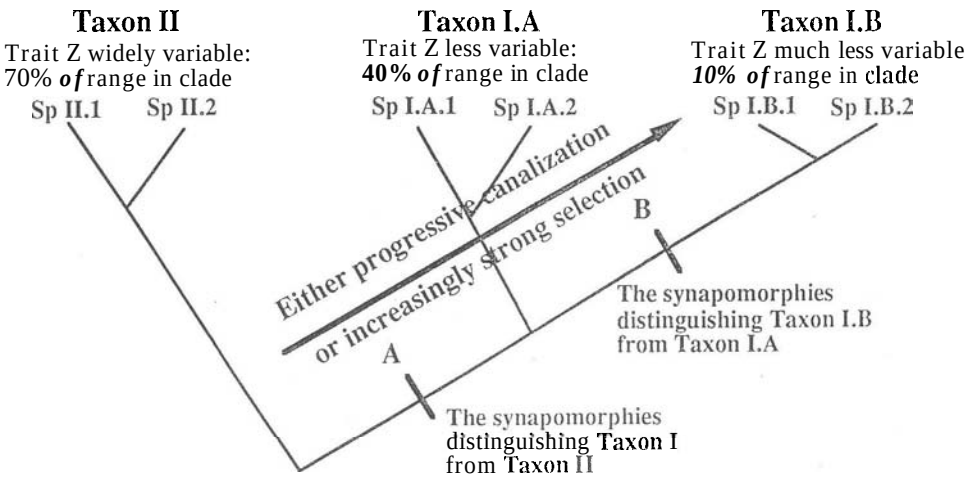

Fig. 5. Phylograms can indicate subtler clade-specific constraints.

the changes made at point B are necessary but not sufficient, for they only restrict clutch size when combined with the changes made at point A, defined as the synapomorphies distinguishing Taxon I from Taxon II and naturally inherited by Taxon I.B along with the rest of its traits. This hypothesis suggests we search for the origin of constraints on clutch size in the interactions between the new states of the traits at Point B and the trait states that changed at Point A.

A few problems need mention. First, anything read off the cladogram but not analyzed functionally can only indicate correlations, not causes. The cladogram analysis could be strengthened by demonstrating, in a series of independent evolutionary events, that every time trait collection Z (say a part of A+B) occurred, clutch size was restricted to 1 in all species beyond that trait collection. That would be strong evidence, certainly helpful in motivating the functional analysis, but not a substitute for the functional analysis in demonstrating the existence of a constraint. Secondly, the traits used in the analysis have been pre-screened by the phylogenetic paradigm, which means that they are very likely to be traits fixed within species. It is not clear that traits that still vary within species are incapable of being involved in clade specific constraints on other traits that are fixed within species. That assumption should be tested.

There may be more subtle clade specific constraints than those that stand out in a cladogram. Fixed traits, such as those contained in the synapomorphy sets A and B, may restrict the range of variation that the variable traits can express. Suppose we observe a pattern like that in Fig. 5: the range of variation of a trait Z becomes progressively restricted as we move from the clade as a whole into a specific taxon (e.g. I.B).

There could be two reasons for this. First, contained in the synapomorphy sets A and B, we might find the reasons for the progressive

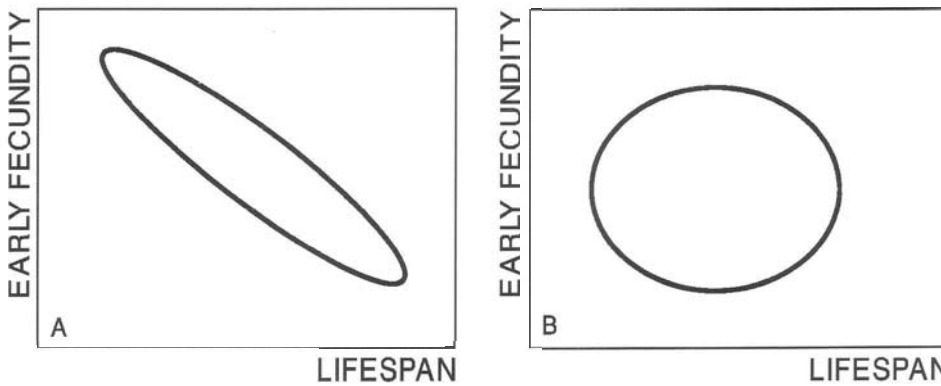

Fig. 6. A. A two-trait trait (a tradeoff). B. Lack of a tradeoff in a related species.

canalization of trait Z. Alternatively, trait Z might be coming under increasingly strong selection as we move into Taxon I.B, and its smaller range of variation there could simply be the result of the exhaustion of genetic variation. There is no way to distinguish these hypotheses by analyzing cladograms. However, the alternatives can be tested experimentally. One approach would be to test genetically equivalent samples of each of the four species (e.g. equal numbers of isofemale lines) for the responses in the variability of trait Z to different environments and to induced mutations. That would test for differences in the degree of genetic and environmental canalization of the trait as we progress through the clade. The second approach would be to relax selection – 'domesticate' each of the four species – and see if the genetic variation increases the most in the least variable taxon and the least in the most variable taxon.

If the pattern of variation in the clade remains the same when challenged by different environments, by mutations, and by relaxing selection, then the explanation is canalization, both environmental and genetical. If the differences among the species disappear when selection is relaxed, and if the variation in all species is equally responsive to different environments and to mutations, then the explanation is that selection pressure on Z increases as we move through the clade.

In such analyses, one must use measures of variation appropriate to the question asked. Measures like heritability are defined relative to the variation existing within a population, not relative to the variation existing within a clade. For proper comparison, we need a measure of phenotypic variation within each species expressed as a percentage of the phenotypic variation present in the clade.

If the same manipulations are made of a set of related species, those results can be placed on cladograms to stimulate questions about tradeoffs. For example, one could measure the tradeoff between early fecundity and lifespan by enhancing the expression of a single gene with effects on both traits. If the tradeoff existed in a given species, we would get a

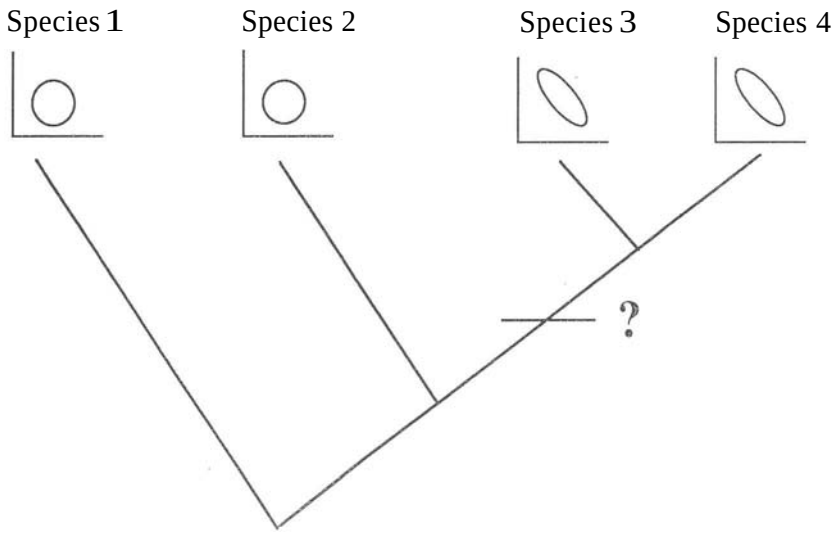

Fig. 7. Phyletic analysis of a tradeoff.

picture like Fig. 6A, and if it did not exist in a related species, we would get a picture that looked like Fig. 6B.

If we had such information for all the species in a clade, we could then make a picture like Fig. 7.

No matter how the tradeoffs had been measured, such a cladistic pattern would clearly provoke the question indicated in the figure: what traits changed in evolutionary history at the point indicated that might explain why the tradeoff occurs in some organisms but not in others? If the pattern had been measured as a genetic correlation in a selection experiment, we would be a something of a loss as to how to proceed, for we would not have any idea of the nature of the genes involved or the processes they affected. If the pattern had been measured by molecular perturbation of both traits, we would immediately have a hypothesis, if not a very sharp one: it might have something to do with the gene we had manipulated. However, in such cases there is always the alternative, and the alternative is less helpful: the tradeoff exists independent of the gene manipulated, and the changes induced in the expression of that gene are only a method of revealing a preexisting and independent pattern caused by other physiological processes. In short, the cladogram helps to pose a puzzle – why is there a difference among clades in presence or absence of certain tradeoffs – but it does not give us much insight into the solution. However, a well-posed question is often more than half the answer.

Caveats for comparative biologists. — The comparative onion is many layers deep. If we look for constraints on reproduction in a polygynous mammal, can we be sure that they have anything to do with being a mammal? The ideal life cycle, from a purely genetical point of view, might

well be cyclical parthenogenesis with sequential hermaphroditism in the sexual generation. However, recall Weismann's (1902) comment about sexual reproduction: he expected that we would only get reliable information from the most primitive forms, where the fewest alternative explanations were available and where the least subsequent evolutionary 'embedding' had occurred. The question is not well posed for vertebrates, for it offers no clues to the historical contingencies and systems constraints that are probably necessary to find the answer.

Adaptationist alternatives to constraint explanations and constraint alternatives to adaptationist explanations can help. For example, suppose one finds that a trait is fixed within a clade. Someone interested in constraints might think that an irreversible change had occurred in a key trait in the common ancestor, followed by changes in traits functionally associated with the new character state of the key trait, creating systems constraints internal to the organism. However, the trait might be fixed in the entire clade because the clade is only found in an environment in which that state of the trait is favored by natural selection, and if one could change the selection pressure, the trait would not be constrained internally and would change.

A strong null hypothesis for clade-specificity is simply that no clade-specific constraints exist at all, we see only the vestiges of history that remain because they are neutral. This seems plausible for vestigial organs (the human coccyx, limb remnants in snakes and whales). However, when can we be sure that the vestiges that remain are not equally well explained by the constraint hypothesis: they persist as byproducts of essential functions reduced to the minimum consistent with maintenance of those essential functions?

Conclusions

The impressive progress made possible by the simplifying assumptions of evolutionary genetics was matched by the inability of evolutionary genetics to explain phenotypic design. People were well aware of that deficiency by the 1970's and early 1980's, when it was widely suggested that the role of development in evolution and the new phenotypic theories – optimality theory, quantitative genetics, and game theory – would provide the solution. Some progress has been made, but the key questions still await solution: how did the fixed traits become fixed, and do the fixed traits constrain the pattern of expression of the variable ones? Answering those questions would go a long way towards satisfying the strictest logical criterion for causation, sufficiency, by making clear the connections between clade-specific patterns of fixation of traits and the patterns of expression of the traits that remain variable. To take a small step down that path has been the purpose of this paper.

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Streszczenie

W artykule tym przedyskutowane są stosunki ewolucyjne pomiędzy genetycznie utrwalonymi i zmiennymi cechami. Podstawowym zagadnieniem jest to, w jaki sposob następuje utrwalenie cech.

Zgodnie z jednym z ewolucyjnych scenariuszy specjacja otwiera możliwość niezaleznej ewolucji rozdzielonych pul genowych potomnych gatunkow i selekcja moie stopniowo kanalizowad roine cechy w każdym z powstałych odrębnych ciągów ewolucyjnych. W każdej gałęzi ewolucyjnej pewne wciąż zmienne cechy mogą koewoluowad z cechami skanalizowanymi. To wciąga cechy skanalizowane w sied współzależności z innymi cechami, co z kolei powoduje, ze skuteczność funkcjonowania calego organizmu wyrnaga ich trwalosci. W rezultacie powstajq cechy specyficzne dla gałęzi ewolucyjnych, bowiem zmiany w utrwalonych już cechach łączyłyby się ze zbyt dużymi kosztami – zmniniejszeniu bowiem moglaby ulec wartość przystosowawcza powiqzanych z nimi innych cech.

Zgodnie z alternatywnym scenariuszem kolonizowanie nowych siedlisk lub ewolucyjne wyodrębnienie nowego stadium rozwojowego prowadzid moze do uwolnienia pewnych cech jako czasowo neutralnych, chod uprzednio były cechami zdecydowanie użytecznymi. Umożliwia to ich swobodnq ewolucję w kierunkach związanych z nowymi przystosowaniami – niektore z nich mogą byd włączone w struktury służące innym funkcjom niz te wlasciwe dla wyjsciowych homologow. Proces jest. nieodwracalny, jako że powrót do pierwotnych struktur i funkcji tu również łączyłby się ze znacznymi kosztami.

Inne zagadnienie o fundamentalnym znaczeniu to czy istnieje wpływ utrwalenia jednych cech n a ewolucję innych, genetycznie zmiennych, który mógłby dawad specyficzne dla gałęzi ewolucyjnych sposoby reagowania n a selekcję. Że tak może byd, sugeruje związek nieciągłości wzrostu z ekspresją zmiennosci genetycznej cech zależnych od rozmiarów wśród stawonogow. Rozpoznanie tego rodzaju zjawisk wymaga przeprowadzenia szczegółowych analiz filogenetycznych wpływu uprzedniego utrwalenia cechy n a charakter zmiennosci.