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INTRODUCTION

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Homology and homoplasy are terms that travel together; homoplasy being close to, but not quite, the inverse of homology. If homology is "the same thing" (persistence of traits in their various transformed states), homoplasy is the *appearance* of "sameness" that results from independent evolution. Homoplasy is *derived similarity* that is *not* synapomorphy (Archie, in his chapter, usefully differentiates between evolutionary and phylogenetic homoplasy). To my knowledge, this is the first book devoted to the subject, and the authors have assumed the challenge to define the field of investigation and to lay the foundation for future research.

Similarity, and the reasons for it, are central issues in studies of homoplasy, as in controversies over homology. Virtually all of the problems associated with the study of homology (well displayed in the diverse contributions in Hall 1994; see also Wake 1994) also relate to homoplasy. However, some issues relate more importantly to homoplasy than to homology. I believe that a full exposition of these issues has long been needed, and

this volume explores homoplasy in broad perspective, while at the same time giving attention to many detailed aspects of homoplasy.

There are two general categories of issues that relate to homoplasy. One is the means of detection and the other is the reason that apparently similar features evolve independently. Detection was made objective, but not simplified, by the development of a well-formulated, logical philosophy of phylogenetics and of associated analytical methods. The seminal work of Hennig (1966) accelerated progress in this area, and thanks to contributors such as Farris (Hennig-86), Felsenstein (PHYLIP), Swofford (PAUP), and Maddison and Maddison (MacClade), to mention only the most frequently cited, a wide variety of tools is now available for phylogenetic analyses. Every contributor to the present volume explicitly (and appropriately) discusses this area of research on homoplasy.

The second category concerns the biological basis of homoplasy, that is, the biological processes and mechanisms responsible for the production of homoplastic traits. This is a venerable area of inquiry that was largely shunted aside during the past 25 years as interest focused on phylogenetics *per se*. But there has always been an interest in the underlying causes of homoplasy, and our vastly improved means of detection has made the study of causes more sophisticated. Recent efforts exploring the biological basis of homology (Hall 1995; Wagner 1989, 1995) have relevance to this topic, and I have examined the causes of homoplasy in some recent papers (Wake 1991; Shubin et al. 1995). Most of the authors in this book discuss at least some of the proximal biological reasons for homoplasy and some (e.g., McShea, Armbruster, and Hufford) emphasize it.

For one focused on phylogenetic analysis it matters little whether a trait subject to homoplasy is a convergence, a parallelism, or a reversal, but instead what is critical is that the phylogenetic signal (based on "true" synapomorphies in classical and mainly morphological cladistics) be stronger than the homoplastic noise. As data bases grow, with concomitant analytical challenges, there is less interest in analysis of individual traits. In molecular systematics it is not unusual to encounter hundreds of equally parsimonious trees for a large data set (Sanderson and Donoghue, this volume, find morphological data sets to be no better in this respect). In such circumstances homoplasy is rampant and one does not worry about determining synapomorphies (but if one is using a coding sequence there will probably be more interest in specific second-position transversions than in third-position transitions). For those focused on trait evolution and evolutionary mechanisms, however, the phylogenetic analysis is usually a precursor to a study of the reasons for particular homoplasies.

At no hierarchical level is homoplasy more common, and arguably better understood, than at the most reduced—the nucleotide level. Possibilities are severely constrained by the availability of only four bases, and in parts of the genome the rate of substitution is relatively high. At the level of detection of a "G" in a particular nucleotide position, homoplasy may be common, although homology (in the sense of chemically being "the same thing") may be precise. By reducing the homoplasy problem to the lowest hierarchical level that is practical, all statements of homology can be seen to be provisional and cladogram dependent. Homoplasy may increase as the number of taxa studied increases, but it also increases when there is low trait persistence or when there are few alternatives and transitions between them are possible.

Problems with homoplasy parallel those related to homology, and they are unlikely to be resolved any more readily. Homologous traits provide insight into the connectedness of life, its genealogy. For some researchers, mainly phylogeneticists (or "phylogenists"), history and homology are essentially congruent, while for others, mainly morphologists and developmentalists, history and structural identity need to be teased apart. Wagner (1989, 1995) argued that homologous characters are parts of the genotype that become individualized because of the local regulation of development, which acts as a constraint on further change; consequently, the individuated parts tend to persist. Wagner's (1995) "building block" hypothesis is a recent and I believe heuristic attempt to explain how characters become individuated (possibly as a result of selection "taming" spontaneously produced morphological variants) and then play important roles in adaptive processes by virtue of their replaceability and combinability. This conceptualization is relevant for homoplasy of characters that on face value appear too complicated or novel to evolve repeatedly. Perhaps characters reappear because small changes in regulation of local developmental systems trigger latent building blocks. This connects to a long recognized area of discussion in character homology. Latent homology is the situation in which the developmental precursor of a structure persists and is triggered in different lineages, sometimes by different means (de Beer 1971; Hall 1995). There is a long history of interest in the phenomenon, expressed variously in such concepts as homologous series (Vavilov, 1922), canalized evolutionary potential (Saether 1983), and apomorphic tendencies (Rasmussen 1983) (reviewed by Sanderson 1991). This is a "levels problem," so that the precursor itself is the appropriate level for homology but the phenotypic outcome of phylogenetically independent triggerings is the level at which homoplasy occurs. One interested in trait evolution might then classify the different triggers, thus sorting different reasons for parallel evolution which could lead to improved character coding or additional characters to be coded.

It is not possible to state a hard and fast distinction between parallel and convergent evolution, but categorization can help sort morphological homoplasies. In general, parallelism is the production of apparently identical traits by the same generative system and convergence involves the production of similar traits by different generative systems. Elongation is a common homoplasy in salamanders: parallelism is the independent increase in numbers of segments, and ultimately vertebrae, during early embryogenesis in

different taxa; convergence is the much rarer increase in vertebral length in a few taxa. This example illustrates the importance of levels of analysis in homology and homoplasy research. "Elongation" can be explained by "added segments" and "increased segment length," each of which in turn generates homoplasy.

Organisms are integrated systems showing complicated couplings that limit and bias the kinds and direction of trait evolution (Roth and Wake 1989; Schwenk 1995). The homoplastic evolution of freely projectile tongues in salamanders has been explored in some detail with respect to these ideas (Lombard and Wake 1977, 1986; Wake 1982, 1991; Roth and Wake 1985). The hyolingual system is used for both filling lungs and propelling the tongue. Lung reduction and loss is a common homoplasy in salamanders and appears to be a necessary, but not sufficient, condition for the evolution of extreme tongue projection by releasing the constraint of lung filling and permitting specialization. Specialization takes specific, alternative forms. For example, the tongue skeleton must fold for long-distance projection, and only two geometrical arrangements are possible, both of which have been used by different clades to accomplish the same end, which produces the same homoplasy at the level of tongue projection, but is revealed to be convergence at the level of biomechanics.

Paedomorphic homoplasy may be the easiest to understand and one of the most common kinds. The pattern of some taxa having "backed down" plesiomorphic ontogenetic trajectories in relation to others can produce confusingly similar morphologies in separate lineages within a clade. Perennibranchiate species of salamanders, forms in which sexual maturity is attained by gilled larvae and metamorphosis is eliminated, come to resemble each other in most morphological details. This is because differences among taxa are most commonly terminal ontogenetic additions and the larval stage is relatively undifferentiated. The perennibranchiate forms represent evolutionary reversals to more simple, and hence less variable, early ontogenetic stages. Less obvious kinds of ontogenetic transformations are probably associated with many homoplastic trait transformations.

Certain homoplasies appear commonly in some clades but not others, and hierarchical approaches may be heuristic. Parthenogenesis has evolved repeatedly in lizards, and Moritz et al. (1992) hypothesized that a general ("phylogenetic") constraint in vertebrates had been overcome in some manner in squamate vertebrates, representing a necessary condition for the homoplasy. Schwenk (1995) offered what he called an internalist (or structuralist) perspective as an alternative, suggesting that there is no prior constraint, but rather that squamates have evolved novel genetic or developmental conditions which bias them toward the homoplastic evolution of parthenogenesis. In this case the parallelism is taken as evidence of a synapomorphic evolutionary constraint. I offer this example to show that homoplasy may give insight into underlying traits that are of both phylogenetic

and evolutionary significance. Despite widespread interest in homoplastic tendencies such as parthenogenesis in lizards, Sanderson's (1991) statistical analysis failed to find compelling evidence for their existence. However, it may be that homoplastic characters of this sort are routinely excluded from phylogenetic analyses by systematists (the "file-drawer" problem identified by Sanderson and Donoghue, this volume).

Since I began biological research I have been fascinated with morphological homoplasies, especially the biological basis for their independent generation. Perhaps this fascination developed because I chose to pursue evolutionary morphological and systematic studies of a difficult group, relatively featureless salamanders. The most featureless were the most difficult—clades that contained miniaturized species, clades that displayed general uniformity despite being speciose and in which the few derived traits were distributed in bewildering arrays, and clades that contained species displaying varying degrees of paedomorphosis. It was my studies of salamanders that first made clear to me that the study of the causes of homoplasy requires a hierarchical approach.

That homoplasy detection is accomplished through a genealogical hierarchical (phylogenetic) analysis is now widely understood. Less attention has been given to hierarchically based, biological explanations for homoplasy; I am pleased that several authors in this book (e.g., Armbruster, Bateman, Brooks, Hufford, and McShea) emphasize such perspectives, because I believe they are key to our understanding of the phenomena involved in the production of biological similarity. Hierarchical approaches have been used with respect to homology by several researchers (e.g., Roth 1991) with excellent results.

Important issues in evolutionary biology involve the reasons that morphological change takes specific forms (e.g., Wagner 1989; Wake 1991), and hierarchical investigations of the biological bases of homoplasy have provided insight. Homoplasy alerts the researcher to the possibility of limits on character production and spurs inquiry into the mechanistic foundation for change. If we demand an explanation for specific homoplasies without taking into account the full extent of homoplasy and the degree of intercorrelation, a hierarchical error can result. For example, researchers might devise a research program that demands a selective explanation for a specific homoplastic trait, when this is an inappropriate explanatory level. The trait in question might be part of a more general phenomenon. An example which illustrates many points related to homoplasy is miniaturization.

Miniaturization results from dynamics at the level of populations and communities, but often has evolutionary consequences quite independent of factors that led to size decrease (Hanken and Wake 1993). A frequent outcome is ontogenetic truncation, or progenesis (Gould 1977), and the homoplasy thus generated is failure of plesiomorphic characters to appear, recorded as a loss. One of the best understood in vertebrates is the repeated

loss of a toe in miniaturized amphibians (Alberch and Gale 1985). In frogs it is the first and in salamanders it is the fifth toe that invariably is lost. This can be understood, and even predicted, from the different, but standard, patterns of morphogenesis in the two taxa. Frogs display postaxial-to-preaxial digital morphogenesis, a pattern shared with amniotes, while salamanders display a preaxial-to-postaxial pattern. The first toe of frogs and the fifth of salamanders fail to appear in four-digited taxa. However, the last formed digit is often larger than expected from out-group comparisons. Occasional individuals of five-toed species of salamanders are found in which only four external digits are present but in which there are five skeletons; the full skeleton of the last digit may be imperfectly duplicated, so that there are two skeletal but only one integumentary digit (Wake 1991). The last digit of four-toed salamanders then is not number four but some undifferentiated combination of four and five. Here another hierarchical issue becomes relevant. Amphibians have large to very large genomes, relative to other tetrapods, and genome size is a relatively persistent trait, less likely to change during phylogenesis than body size, for example (Sessions and Larson 1987). Genome size translates directly to cell size, so miniaturized amphibians are caught in a hierarchical crunch, organismal form being simultaneously affected by downward causation from factors at the population level leading to small body size and by upward causation from persistent genome size. Limb buds of amphibians have large cells, so miniaturized taxa with smaller limb buds will have fewer cells. There are important allometric considerations. Reduction in limb bud size in a large celled species will have greater consequences than in a small celled species, because cell number is critically important in morphogenetic processes such as condensation, segmentation, and bifurcation (Shubin and Alberch 1986). The large-celled species may encounter developmental thresholds that they fail to cross because they have too few cells and digital reduction may result. The combination of few cells and reduced cell division rate (a consequence of large genome size; Sessions and Larson 1987) causes truncation of developmental pathways and digital loss occurs. In contrast, miniaturized lizards, derived from small-genomed ancestors, are capable of producing five small digits. Some miniaturized lizards are nearly as small as tiny frogs and salamanders, but the amphibians are effectively (i.e., with respect to developmental mechanics and some other organismal-level phenomena) much smaller because of their much larger cells (Hanken and Wake 1993).

One might expect the hierarchical factors outlined above to have general effects beyond digit loss and such effects do occur. Homoplasy at the level of tissue histogenesis in the brains of large-genomed lungfish, frogs, and salamanders has been documented (Roth et al. 1994); the proximal reasons for such homoplasy are relatively well understood (e.g., Roth et al. 1995). It would be a mistake to try to explain on a point-by-point basis the reasons

for detailed neuroanatomical similarity between lungfishes and plethodontid salamanders when there is a more general explanation available at a different hierarchical level (this of course assumes that evidence is good that the two taxa do not form a monophyletic group). There are many examples of paedomorphic homoplasy in amphibians, and unless one uses an hierarchical approach, traits that are correlated are treated as separate. A large problem with ontogenetically based homoplasy is that there may be different thresholds for different characters. Thus, some four-toed salamanders also lack prefrontal bones and have large cranial foramina (e.g., *Batrachoseps*), but others (e.g., *Hemidactylium*) do not (Wake 1966).

Genome size, patterns of limb morphogenesis, and tissue histogenesis in the brain can all be viewed as factors that produce bias in the production of variation and make some outcomes far more likely than others (see also discussions in this book by Brooks, Hufford, and McShea). Traits characteristically are not "free" to vary in any direction; they vary predictably (Alberch 1989; Wake 1991). Variant traits within a population frequently duplicate conditions fixed in other taxa, illustrating why study of factors responsible for production of homoplastic traits is likely to be a fruitful area of inquiry (Shubin et al. 1995).

Once one initiates a hierarchical approach to homoplasy, unanticipated insights emerge. Surprisingly, retinotectal projections of paedomorphic bolitoglossine salamanders with large genomes share a homoplastic relationship with primates and "megabats." This is an example of ontogenetic repatterning in the salamanders (Wake and Roth 1989), in which paedomorphosis has led to a derived morphology that in turn serves as a point of departure for a derived ontogenetic trajectory. In this case, one level of homoplasy that has predictable results (in essence, backing down a persistent ontogenetic trajectory, using the formalism of Alberch et al. 1979; see also Hufford 1996) leads to another, unanticipated and less-predictable level of homoplasy. In all three cases of homoplasy stereoscopic vision is enhanced, but whether there is a common ontogenetic phenomenon is unclear. Ontogenetic repatterning of the kind found in the salamanders is a far more profound event than a simpler, phase- or stage-specific ontogenetic transformation, which, however, might also have major consequences (again, see Hufford 1996).

The study of homoplasy offers a rich and diverse array of opportunities. The chapters in this book represent the collective focus on homoplasy by a wide array of biologists, mainly systematists, but also organismal and developmental biologists. The rigor and discipline represented in these contributions establish a solid foundation on which to build future studies of homoplasy. I look forward to a unified approach that incorporates functionalist (selectionist or externalist), structuralist (mechanistic or internalist), and phylogenetic perspectives. One can view homoplasy as the major problem in phylogenetic inference; alternatively, once we have some assurance that ho-

moplasy exists it becomes a challenge to explain its origins. The pursuit of such questions will contribute greatly to our understanding of how diversity evolves.

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