

Critical Perspective, Origins & Design 18:2 Homology: A Concept in Crisis

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Abstract

Before Darwin, homology was defined morphologically and explained by reference to ideal archetypes -- that is, to intelligent design. Darwin reformulated biology in naturalistic* rather than teleological terms, and explained homology as the result of descent with modification from a common ancestor. Descent with modification, however, renders design unnecessary only if it is due entirely to naturalistic mechanisms. Two such mechanisms have been proposed, genetic programs and developmental pathways, but neither one fits the evidence. Without an empirically demonstrated naturalistic mechanism to account for homology, design remains a possibility which can only be excluded on the basis of questionable philosophical assumptions.

* In this article, "naturalism" and "naturalistic" refer to the philosophical doctrine that nature is the whole of reality, and that intelligent causation does not qualify as a scientific explanation.

Morphological and Phylogenetic Homology

From at least the time of Aristotle, people who study living organisms have noted some remarkable similarities among very diverse creatures. Bats and butterflies are quite different from each other, yet both have wings to fly; bats fly and whales swim, yet the bones in a bat's wing and a whale's flipper are strikingly alike. The first kind of similarity involves different structures which perform the same function, and in 1843 anatomist Richard Owen called this "analogy." In contrast, the second kind of similarity involves similar structures which perform different functions, and Owen called this "homology." Owen (and other pre-Darwinian biologists) attributed homology to the existence of archetypes: biological structures are similar because they conform more or less to pre-existing patterns. (Bowler, 1989; Panchen, 1994)

In 1859, Charles Darwin offered a different explanation for homology. According to Darwin, bats and whales possess similar bone structures because they inherited them from a common ancestor, not because they were constructed according to the same archetype. By replacing archetypes (which imply design and intelligent causation) with a

natural mechanism such as common descent, Darwin hoped to render idealistic explanations unnecessary and to place biology on a securely naturalistic basis.

Not all structural similarities, however, are inherited from a common ancestor (as Darwin and his followers recognized). For example, the eye of a mouse is structurally similar to the eye of an octopus, yet their supposed common ancestor did not possess such an eye. In 1870, Ray Lankester coined the term "homoplasy" to describe such features. Implicit in this distinction was a new definition of homology. As evolutionary biologist Ernst Mayr put it, after Darwin the "biologically most meaningful definition" of homology was: "A feature in two or more taxa is homologous when it is derived from the same (or a corresponding) feature of their common ancestor." (Mayr, 1982) In other words, what Darwin proposed as the explanation for homology became its definition. For many biologists, the post-Darwinian (or phylogenetic) definition of homology has replaced the structural (or morphological) definition. (Hall, 1992; Panchen, 1994)

Darwin's reform -- explaining homology by material descent with modification -- was incorporated into the neo-Darwinian synthesis of the mid-twentieth century with the discovery of the mechanisms of transmission genetics (i.e., inheritance), about which Darwin knew nothing. Figure 1 displays a flow diagram with the key elements of the neo-Darwinian explanation of homology. The cardinal "explainer" (so to speak), or cause, which Darwin advocated classically in chapter XIII of the *Origin of Species*, is material descent. Every organism in our experience has at least one parent. Thus, humans (for instance) possess two large bones, the radius and the ulna, in their forelimb because, by hypothesis, their distant non-human primate ancestors also possessed two such bones, albeit with slightly different shapes -- and so on, back to the primary progenitor which first evolved the radius-ulna pattern.

Neo-Darwinian biologists added to this the new causal dimension of the physical basis of heredity. In brief, at reproduction, each parent (in a sexually-reproducing species) passes half of its genetic material (DNA) to its offspring. What descends from generation to generation, therefore, are genes: DNA. These genes, in turn control the processes of development in the fertilized egg, as the phenotype (adult morphology) is being constructed. Evolution, or the adaptive modification of adult form, occurs because genes are subject to mutation. These mutations affect development; and differing phenotypes are constructed among the offspring, which are then selected by their ability to compete and reproduce.

This explanation has a beautiful plausibility. It is also in very serious trouble. Within the past decade or so, a flood of new data on the genetic constituents of development, as

well as the revisiting of older but still unsolved puzzles (see below), have battered the foundations of the neo-Darwinian explanation of homology. In a recent commentary on the troubled state of the concept, David Cannatella, of the Department of Zoology at the University of Texas wrote:

... Wake (1994) offered that homology is the central concept of all biology. If this is true, then a large group of comparative biologists lacks a guiding principle. One does not have to look far to see that homology (and therefore homoplasy) is not understood by many biologists. (Cannatella 1997, 369)

In this article we review in broad outline some of the major difficulties with the neo-Darwinian explanation of homology, in particular, the incongruent causal relationship between genes, development, and phenotypic form. Despite the standard textbook claims, homology has never been adequately explained by neo-Darwinism. The time is ripe, we contend, to reconsider biology's exclusion of intelligent design as a possible cause.

The Need for a Naturalistic Mechanism

Ask your neighborhood evolutionary biologist how he knows intelligent design is unnecessary to explain homology, and odds are he will say something like, "Well, we have a demonstrated natural mechanism which accounts for the phenomenon." In actuality, however, the mechanism has not been demonstrated; rather, homology is simply taken as *prima facie* evidence of descent, and design is excluded out of hand. The problem is unintentionally illustrated by biologist Tim Berra in his 1990 book, *Evolution and the Myth of Creationism* (Stanford University Press). According to Berra, "If you look at a 1953 Corvette and compare it to the latest model, only the most general resemblances are evident, but if you compare a 1953 and a 1954 Corvette, side by side, then a 1954 and a 1955 model, and so on, the descent with modification is overwhelmingly obvious. This is what paleontologists do with fossils, and the evidence is so solid and comprehensive that it cannot be denied by reasonable people." (p. 117)

As the title of his book indicates, Berra's primary purpose is to show that living organisms are the result of naturalistic evolution rather than intelligent design. Structural similarities among automobiles, however, even similarities between older and newer models (which Berra calls "descent with modification") are due to construction according to pre-existing patterns, i.e., to design. Ironically, therefore, Berra's analogy

shows that even striking similarities are not sufficient to exclude design-based explanations. In order to demonstrate naturalistic evolution, it is necessary to show that the mechanism by which organisms are constructed (unlike the mechanism by which automobiles are constructed) does not involve design.

One could simply postulate that the mechanism of biological evolution is naturalistic, arguing that the postulate is justified because science is limited to studying natural mechanisms. Although such a philosophical move may seem very reasonable, it gravely compromises the status of evolutionary biology as an objective science. Asserting that something is objectively true implies that it is based on empirical evidence, not merely assumed a priori on philosophical grounds. A methodological exclusion of design-based explanations constitutes a limitation on one's science, not a description of objective reality. If evolutionary biologists want to show that the actual mechanism of evolution does not involve intelligent design, they cannot merely exclude the possibility a priori, but must take the more difficult approach of proposing and corroborating a naturalistic alternative.

This alternative must account naturalistically for what evolutionary biologist Leigh Van Valen has called "continuity of information." (Van Valen, 1982) According to Van Valen, homologous features are produced during the development of each individual organism by information which has been inherited, with modification, from the organism's ancestors. Thus the first step toward understanding the mechanism of evolution would be to determine the nature of the information which controls the development of the embryo.

Homology and Genetics

One possibility is that this information is encoded in the organism's genes. In the 1930's, the synthesis of Darwin's theory and population genetics explained evolution as a change in gene frequencies, and several decades later the discovery of the structure and function of DNA extended this explanation to the molecular level.

According to the neo-Darwinian synthesis, a genetic program encoded in DNA directs embryonic development; the process of reproduction transmits this program to subsequent generations, but mutations in the DNA sometimes modify it ("descent with modification"); thus descendants of the original organism may possess structures which are similar but not identical ("homologies") (See Fig. 1). No design is required, so the explanation is thoroughly naturalistic. By 1970, molecular biologist Jacques Monod felt

justified in announcing that "the mechanism of Darwinism is at last securely founded," and that as a consequence "man has to understand that he is a mere accident." (quoted in Judson, 1980, p. 217)

Efforts to correlate major phenotypic evolution with changes in gene frequencies, however, have not been very successful. Detailed studies at the molecular level fail to demonstrate the expected correspondence between changes in gene products and the sorts of organismal changes which constitute the "stuff of evolution." (Lewontin, 1974, p. 160). According to Rudolf Raff and Thomas Kaufman, evolution by DNA mutations "is largely uncoupled from morphological evolution;" the "most spectacular" example of this is the morphological dissimilarity of humans and chimpanzees despite a 99% similarity in their DNA. (Raff and Kaufman, 1983, pp. 67, 78).

Some biologists have proposed that the remaining 1% consists of "regulatory genes" which have such profound effects on development that a few mutations in them could account for dramatic differences. For example, mutations in homeotic genes can transform a fly's antenna into a leg, or produce two pairs of wings where there would normally be only one, or cause eyes to develop on a fly's leg. Furthermore, genes similar to the homeotic genes of flies have been found in most other types of animals, including mammals. Based on the profound developmental effects and almost universal occurrence of such genes, biologist Eric Davidson and his colleagues recently wrote that "novel morphological forms in animal evolution result from changes in genetically encoded programs of developmental regulation." (Davidson et al., 1995, p. 1319)

According to this view, homologous features are programmed by similar genes. Assuming that genes with similar sequences are unlikely to originate independently through random mutations, sequence similarity would indicate common ancestry. Features produced by similar sequences could then be inferred to be phylogenetically homologous.

The very universality of homeotic genes, however, raises a serious problem for this view. Although mice have a gene very similar to the one that can transform a fly's antenna into a leg (*Antennapedia*), mice do not have antennae, and their corresponding gene affects the hindbrain; and although mice and flies share a similar gene which affects eye development (*eyeless*), the fly's multifaceted eye is profoundly different from a mouse's camera-like eye. In both cases (*Antennapedia* and *eyeless*), similar homeotic genes affect the development of structures which are non-homologous by either the classical morphological definition or the post-Darwinian phylogenetic definition. If similar genes can "determine" such radically different structures, then those genes aren't really

determining structure at all. Instead, they appear to be functioning as binary switches between alternate developmental fates, with the information for the resulting structures residing elsewhere. (Wells, 1996)

Not only are non-homologous structures produced by organisms with supposedly homologous genes, but organisms with different genes can also produce similar structures. The most famous examples involves the genes, mentioned above, which affect wing and eye development in flies. Fly embryos with a normal gene for wing development, when treated with ether, can be induced to grow a second pair, just as though they possessed the mutant form of the gene (For a review, see Hall, 1992). Flies with a mutant form of the eye gene fail to develop eyes; but if eyeless flies are bred for many generations, some of their descendants will develop eyes even though they still possess the mutant form of the gene. Such anomalies led embryologist Gavin de Beer to conclude that "homologous structures need not be controlled by identical genes," and that "the inheritance of homologous structures from a common ancestor ... cannot be ascribed to identity of genes." (de Beer, 1971, pp. 15-16)

The underlying assumption that a genetic program directs embryonic development has been seriously questioned by developmental biologists (For a review, see Wells, 1992). Sydney Brenner, who originally proposed genetic programs in 1970, repudiated the idea when he realized that the information required to specify the neural connections of even a simple worm far exceeds the information content of its DNA. (Brenner, 1973) A decade later, developmental biologist Brian Goodwin noted that "genes are responsible for determining which molecules an organism can produce," but "the molecular composition of organisms does not, in general, determine their form." (Goodwin, 1985, p. 32) And in a 1990 critique of the notion of genetic programs, H.F. Nijhout concluded that "the only strictly correct view of the function of genes is that they supply cells, and ultimately organisms, with chemical materials." (Nijhout, 1990, p. 444)

Clearly, the genetic explanation for homology is inadequate. As an alternative, some biologists have suggested that homology results from complex developmental mechanisms which are not reducible to a genetic program.

Homology and Developmental Pathways

Since homologies cannot be explained by equating developmental information with DNA sequences, some biologists have attempted to explain it by attributing it to similar developmental pathways. Although DNA determines the amino acid sequence of

proteins essential for development, such pathways also involve other factors, such as the localization of cytoplasmic constituents in the egg cell, physical constraints resulting from the size of the embryo, and so on. (Wells, 1992)

Efforts to correlate homology with developmental pathways, however, have been uniformly unsuccessful. First, similar developmental pathways may produce very dissimilar features. At the molecular level, it is well known that virtually identical inducers may participate in the development of non-homologous structures in different animals. (Gilbert, 1994) At the multicellular level, the pattern of embryonic cell movements which generates body form in birds also generates body form in a few species of frogs. (Elinson, 1987) And even at the organismal level, morphologically indistinguishable larvae may develop into completely different species. (de Beer, 1958) Clearly, similar developmental pathways may produce dissimilar results.

Second, and more dramatically, similar features are often produced by very different developmental pathways. No one doubts that the gut is homologous throughout the vertebrates, yet the gut forms from different embryonic cells in different vertebrates. The neural tube, embryonic precursor of the spinal cord, is regarded as homologous throughout the chordates, yet in some its formation depends on induction by the underlying notochord while in others it does not. (Gilbert, 1994) Evidently, "structures can owe their origin to different methods of induction without forfeiting their homology." (de Beer, 1958, p. 151) Indeed, as developmental biologist Pere Alberch noted in 1985, it is "the rule rather than the exception" that "homologous structures form from distinctly dissimilar initial states" (see Figure 2). (Alberch, 1985, p. 51)

Production of similar forms from dissimilar pathways is also common at later stages of development. Many types of animals pass through a larval stage on their way to adulthood, a phenomenon known as indirect development. For example, most frogs begin life as swimming tadpoles, and only later metamorphose into four-legged animals. There are many species of frogs, however, which bypass the larval stage and develop directly. Remarkably, the adults of some of these direct developers are almost indistinguishable from the adults of sister species which develop indirectly. In other words, very similar frogs can be produced by direct and indirect development, even though the pathways are obviously radically different. The same phenomenon is common among sea urchins and ascidians (see Figure 3). (Raff, 1996)

Even the classic example of vertebrate limbs shows that homology cannot be explained by similarities in developmental pathways. Skeletal patterns in vertebrate limbs are initially laid down in the form of cartilage condensations, which later ossify into bone.

The sequence of cartilage condensation is the developmental pathway which determines the future pattern of bones in the limb. Yet similar bone patterns in different species (i.e., homologies) arise from different sequences of cartilage condensation. (Shubin, 1991) In the words of biologist Richard Hinchliffe: "Embryology does not contribute to comparative morphology by providing evidence of limb homology in the form of an unchanging pattern of condensation common to all tetrapod limbs." (Hinchliffe, 1990, p. 121)

The constancy of final patterns despite varying pathways has prompted developmental biologist Günter Wagner to suggest that homology might be due to conserved developmental "constraints". (Wagner, 1989) Wagner's critics, however, object that this notion is too vague to be useful. Although developmental constraints emphasize the fact that embryos are capable of producing similar end-points by a variety of routes, they do not constitute a naturalistic mechanism accessible to empirical investigation.

So embryology has not solved the problem of homology. In 1958, Gavin de Beer observed that "correspondence between homologous structures cannot be pressed back to similarity of position of the cells in the embryo, or of the parts of the egg out of which the structures are ultimately composed, or of developmental mechanisms by which they are formed." (de Beer, 1958, p. 152) Subsequent research has overwhelmingly confirmed the correctness of de Beer's observation. Homology, whether defined morphologically or phylogenetically, cannot be attributed to similar developmental pathways any more than it can be attributed to similar genes. So far, the naturalistic mechanisms proposed to explain homology do not fit the evidence.

Conclusion

In 1802, William Paley wrote that someone crossing a heath and finding a stone could reasonably attribute its presence to purposeless natural causes. Upon finding a watch, however, and seeing that "its several parts are framed and put together for a purpose," one could conclude that the watch had been designed. By analogy, Paley argued, one could also conclude that living things are designed. (Paley, 1802, p. 2) In 1859, Charles Darwin argued that living things are more like Paley's stone than Paley's watch, and claimed that everything which Paley attributed to design could be accounted for naturalistically, by descent with modification.

As Berra's automobile analogy shows, however, descent with modification is not enough to exclude design. It is necessary, in addition, to show that the mechanism of descent with modification is thoroughly naturalistic. Darwin thought he had done this with his

theory of natural selection, but as the problem of homology demonstrates, he failed to accomplish his goal.

Diverse organisms possess homologous features. Homology in some cases may or may not be due to inheritance from a common ancestor, but it is definitely not due to similarity of genes or similarity of developmental pathways. In 1971, Gavin de Beer wrote: "What mechanism can it be that results in the production of homologous organs, the same 'patterns', in spite of their not being controlled by the same genes? I asked this question in 1938, and it has not been answered." (de Beer, 1971, p.16) Twenty-six years later, the question still has not been answered.

Without a naturalistic mechanism to account for homology, however, Darwinian evolution cannot claim to have demonstrated scientifically that living things are undesigned, and the possibility remains that homologies are patterned after non-material archetypes. Without a demonstrated mechanism, naturalistic biologists are left with only one alternative: exclude design a priori, on philosophical grounds.

This exclusion could be taken as a statement that intelligent design does not exist, or it could be taken as a statement that intelligent design is beyond the reach of empirical science. The first is a philosophical or theological statement, and warrants the same response. The second is a methodological limitation which cannot be logically extrapolated to a limitation on reality. In other words, a scientist who makes the first move is engaging in metaphysical disputation, while a scientist who makes the second is declining to investigate a possible aspect of reality.

Unfortunately, many biologists make both moves, but fail to distinguish logically between them. While justifying their exclusion of intelligent design on methodological grounds, they act as though science has disproved its existence by providing a naturalistic explanation for homology. When confronted with the fact that science has failed in this regard, they reaffirm their methodological commitment and express faith that a naturalistic mechanism will someday be discovered.

And perhaps it will. But what if living things really are designed? Someone who finds a watch on the ground, and wants to investigate its origin, would be mistaken to rule out design a priori. Having already jumped to the wrong conclusion, that person might go on to waste an entire lifetime dabbling in spurious explanations. If science is truth-seeking, then this is a strange way to do science.

According to an old joke, a passer-by walks up to a drunk stumbling around under a street light. The passer-by asks the drunk what he's doing, and the drunk replies, "Looking for my watch." "Oh, did you lose it here?" asks the passer-by. "No," the drunk replies, "I lost it across the street, but there's no light over there!" Letting naturalistic philosophical assumptions limit one's search for the cause of homology may not be the best way to study living things.

Figure 4: Cleavage Stages

References

Alberch, Pere (1985). "Problems with the Interpretation of Developmental Sequences," *Systematic Zoology* 34 (1): 46-58

Berra, Tim M. (1990). *Evolution and the Myth of Creationism*. Stanford, CA: Stanford University Press.

Bowler, Peter J. (1989). *Evolution: The History of an Idea*. Revised edition. Berkeley: University of California Press.

Brenner, Sydney (1973). "The Genetics of Behaviour," *British Medical Bulletin* 29: 269-271.

Cannatella, David (1997). Review of *Homology*, B.K. Hall, ed., and *Homoplasy*, M. Sanderson and L. Hufford, eds. *Systematic Biology* 46: 366-369.

Davidson, E. H., Peterson, K. J. and Cameron, R. A. (1995). "Origin of Bilaterian Body Plans: Evolution of Developmental Regulatory Mechanisms," *Science* 270: 1319-1325.

de Beer, Gavin (1958). *Embryos and Ancestors*, 3rd ed. Oxford: Clarendon Press.

de Beer, Gavin (1971). *Homology: An Unsolved Problem*. London: Oxford University Press.

Elinson, Richard P. (1987). "Change in Developmental Patterns: Embryos of Amphibians with Large Eggs." In Rudolf A. Raff and Elizabeth C. Raff, eds., *Development as an Evolutionary Process*, vol. 8, pp. 1-21. New York: Alan R. Liss.

Gilbert, Scott F. (1994). *Developmental Biology*, 4th ed. Sunderland, MA.: Sinauer Associates.

Goodwin, Brian C. (1985). "What Are the Causes of Morphogenesis?" *Bioessays* 3: 32-36.

Hall, Brian K. (1992). *Evolutionary Developmental Biology*. London: Chapman & Hall.

Hinchliffe, Richard (1990). "Towards a Homology of Process: Evolutionary Implications of Experimental Studies on the Generation of Skeletal Pattern in Avian Limb Development." In J. Maynard Smith and G. Vida, eds., *Organizational Constraints on the Dynamics of Evolution*, pp. 119-131. Manchester, UK: Manchester University Press.

Judson, Horace Freeland (1980). *The Eighth Day of Creation*. New York: Simon & Schuster.

Lewontin, R.C. (1974). *The Genetic Basis of Evolutionary Change*. New York: Columbia University Press.

Mayr, Ernst (1982). *The Growth of Biological Thought*. Cambridge, MA: Belknap Press.

Nijhout, H.F. (1990). "Metaphors and the Role of Genes in Development," *Bioessays* 12: 441-446.

Paley, William (1802). *Natural Theology*. Reprinted in 1972. Houston, TX: St. Thomas Press,.

Panchen, Alec L. (1994). "Richard Owen and the Concept of Homology." In Brian K. Hall, ed., *Homology: The Hierarchical Basis of Comparative Biology*. pp. 21-62. San Diego: Academic Press.

Raff, Rudolf A. (1996). *The Shape of Life: Genes, Development, and the Evolution of Animal Form*. Chicago: The University of Chicago Press.

Raff, Rudolf A. and Kaufman, Thomas C. (1983). *Embryos, Genes, and Evolution*. New York: Macmillan.

Shubin, Neil H. (1991). "The Implications of 'The Bauplan' for Development and Evolution of the Tetrapod Limb." In J.R. Hinchliffe, J.M. Hurle, and D. Summerbell, eds., *Developmental Patterning of the Vertebrate Limb*, pp. 411-421. New York: Plenum Press.

Van Valen, Leigh M. (1982). "Homology and Causes." *Journal of Morphology* 173: 305-312.

Wagner, Günter (1989). "The Biological Homology Concept," *Annual Review of Ecology and Systematics* 20: 51-69.

Wells, Jonathan (1992). "The History and Limits of Genetic Engineering," *International Journal on the Unity of the Sciences* 5: 137-150.

Wells, Jonathan (1996). "Unseating Naturalism: Recent Insights from Developmental Biology." Presented at a conference on *Mere Creation: Reclaiming the Book of Nature*, Biola University, Los Angeles.

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