Hox Genes—Evolution's Hoax

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Abstract

Since the advent of molecular science, Darwinism has leaned heavily on genetic mutations to augment weaknesses observed in natural selection. Today, much of the evolutionists' attention has focused on a set of genes referred to as homeobox (Hox) genes, which are pieces of DNA that either promote or inhibit other genes that play a role in the development of a particular organism. Using this information, scientists have been able to produce organisms with superfluous ectopic appendages, wings, or eyes on various regions of their bodies. This has caused evolutionists to proclaim that they have uncovered an adequate mechanism supporting the concept of evolution by mutations. However, Hox genes are far from the "magic bullet" that many have described them as being. While a mis-expressed Hox gene can alter phenotypic expression, it does not do so in a "biological vacuum." Numerous other genes and proteins are needed downstream in order to produce phenotypic characteristics. Additionally, Hox genes are unable to produce new material—something that is essential for macroevolution. The data generated from researchers investigating Hox genes provide many insights into cellular physiology and development; however, the data do not support or confirm evolutionary theory.

From "Hopeful Monsters" to Genetic Mutations

For most of the past century, Neo-Darwinism has prevailed. It is "neo" (new), in that it has made the addition of genetic mutations a necessity for biological evolution. But this raises the question: Why must an addendum be made, except to serve as an inadvertent admission that the old theory of natural selection, or "survival of the fittest," is not sufficient? As the late eminent professor at Harvard, Stephen Jay Gould, observed: "The essence of Darwinism lies in a single phrase: natural selection is the creative force of evolutionary change. No one denies that selection will play a negative role in eliminating the unfit. Darwinian theories require that it create the fit as well" (Gould, 1977, p. 28). Gould was correct when he said that no one denies the conceptual process where the "unfit" are eliminated. This truism—which suggests that the "survivors survive"—is an obvious truth that creationists do not deny. An animal that is adapted poorly to its environment will be disadvantaged when it comes to survival. Eliminating weaker animals ensures that genetic defects cannot be spread throughout a species. However, Darwin's General Theory of Evolution requires natural selection to go beyond simply sifting out the "unfit" and, as Gould commented, "create the fit as well"—something that it is unable to do. Colin Patterson, the late senior paleontologist at the British Museum of Natural History, appropriately noted in a radio interview:

> No one has ever produced a species by mechanisms of natural selection. No one has ever gotten near it and most

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> of the current argument in neo-Darwinism is about this question: how a species originates. And it is there that natural selection seems to be fading out, and chance mechanisms of one sort or another are being invoked (Patterson, 1982).

The chance mechanisms discussed in the above quote are the premise for the Neo-Darwinist movement. Seeing that the efficacy of natural selection was waning, yet unable to properly support it with any factual evidence, evolutionists turned to the geneticists for help. In so doing, they resurrected an old theory about systemic mutations—a theory that had been ridiculed and rejected for decades due to its lack of scientific support. The original theory, by the late evolutionary geneticist Richard Goldschmidt, suggested that evolution could produce "hopeful monsters." It was a concept opposed to the general dogma of Darwin's slow and progressive evolution, and one that instead proposed that speciation occurred in one giant macroevolutionary step. This theory was formulated due to the absence of any intermediary fossils, which are required to facilitate the enumerable transition phases of the gradual evolutionary theories. Goldschmidt commented on his initial proposal of "hopeful monsters" in his book, *The Material Basis of Evolution*, in which he wrote:

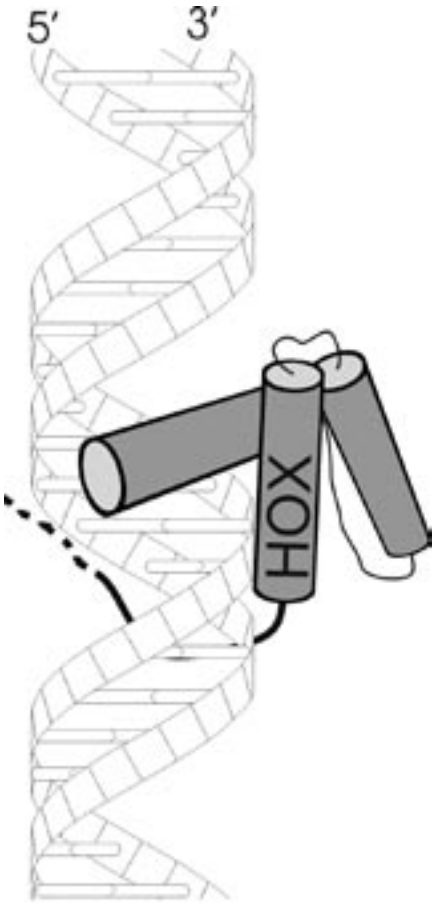

Figure 1. Three dimensional representation of homeodomain of Hox protein.

> I used the term "hopeful monster" to express the idea that mutants producing monstrosities may have played a considerable role in macroevolution. A monstrosity appearing in a single genetic step might permit the occupation of a new environmental niche and thus produce a new type in one step (Goldschmidt, 1940, p. 390).

However, the scientific community never embraced Goldschmidt's views, which deviated from the most commonly held evolutionary theory and invoked an even greater random occurrence than previous models. The "hopeful monsters" were, in actuality, "*hopeless* imaginations." Even Goldschmidt's evolutionary colleague, Stephen Jay Gould, berated such a notion by saying that Goldschmidt had "made a grand, not a paltry error," and that such a concept was a "manifestation of this deeply fallacious genetic theory," which, according to Gould (2002), should be dismissed as a "colorful term" for a "historical curiosity" (p. 457). Gould's stunning criticism did not stop there, but also was present in the introduction he was asked to write for a reprinting of Goldschmidt's book, in which he declared: "*The Material Basis of Evolution* is the major work of his full-fledged heresy" (Goldschmidt, 1940). The suggestions of a "hopeful monster," and the principle of reasoning behind it, are wholly based on evolution, but Gould's demeaning comments also must be taken with the proverbial "grain of salt." He himself repeatedly admitted consternations with his own theories:

> The fossil record with its abrupt transitions offers no support for gradual change. All paleontologists know that the fossil record contains precious little in the way of intermediate forms; transitions between major groups are characteristically abrupt (Gould, 1977, pp 22, 24).

He reiterated this thought three years later in the journal *Paleobiology* in an article titled "Is a New and General Theory of Evolution Emerging?"

> The absence of fossil evidence for intermediary stages between major transitions in organic design, indeed our inability, even in our imagination, to construct functional intermediates in many cases, has been a persistent and nagging problem for gradualistic accounts of evolution (Gould, 1980, p. 127).

Somehow it seems to elude evolutionists that the "absence of fossil evidence" for transitional organisms simply demonstrates there never were any such transitional forms.

Structural vs. Regulatory Genes

In 1865, Austrian monk Gregor Mendel introduced to the world his theory of heredity, through his study of garden pea plants (*Piscum sativum*). In 1906, William Bateson introduced the branch of biology known as genetics, which is founded on many of the precepts established by Mendel's research. The term "gene" was coined three years later by Wilhelm Johannsen (Keller, 2000). At first, of course, the term lacked a definition, and biologists' understanding of it was marginal. However, through the advancements in the new and blossoming field, much of the basis of mod-

ern genetics was fashioned. Today, the gene is defined as a "self-replicating unit of heredity; a portion of DNA (i.e., a sequence of nucleotide units) that encodes a protein" (Schwartz, 1999, p. 406). As the definition states, genes are portions of the deoxyribonucleic acid (DNA), which holds the genetic coding—a sort of blueprint for the design of the body. On a single strand of DNA, there can be numerous portions (genes), each specifying the design of a unique aspect of the body plan.

At one point it was thought, even by the eminent geneticist Thomas Hunt Morgan, "that all genes are acting all the time in the same way." However, he even noted that "this would leave unexplained why some cells of the embryo develop in one way, some in another, if the genes are the only agents in the results. An alternative view would be to assume that different batteries of genes come into action as development proceeds" (as quoted in Keller, 2000, p. 56). The concept set forth by Morgan concerning the problem of the one-gene—one-enzyme view was left unanswered for three decades. Finally, in 1959, Francois Jacob and Jacques Monod made what has been dubbed as the "most original discovery" in the field of genetics (Morange, 2001, p. 95). From their bacterial studies, they surmised that there must be at least two classes of genes: structural and regulatory. Their theory sought to answer the problem: "What caused gene expression in an organism's structure?" Evelyn Keller, in her book, *The Century of the Gene*, acknowledged this problem as the "Achilles' heel not just of the one-gene—one-enzyme hypothesis but of the very notion of gene action" (Keller, 2000, p. 56). Until this point, the process by which an organism's anatomy was constructed had remained a mystery. However, by 1961, Jacob and Monod had identified the "regulator gene," which they said "controls the rate of transcription of certain specific structural genes without itself contributing any structural information to the proteins" (as quoted in Keller, 2000, p. 56). Simply stated, the "regulator genes" are responsible for "directing" the information, and do not participate in the physical formation of any body structures. This laid down a clear separation between the two classes of genes—those known to make up an organism's structure (structural genes) and those responsible for gene expression (regulatory genes).

The regulatory genes (also known as developmental or control genes) are responsible for the major developmental decisions in the body—as opposed to specific details of engineering (Tedeschi, 1997). These genes are responsible for a variety of spatial and time-dependent functions. They have been found to be crucial contributors in setting up the directional gradient of developing embryos, whether anterior-posterior, dorsal-ventral, or radial. Later in the organism's development, regulatory genes help to supervise the formation of the body's diverse tissues, such as muscles and organs. Along with the basic symmetry of the organism, these genes also control the "position-specific adult structures" such as body segments, limbs, wings, sensory organs, etc. (see Martindale and Kourakis, 1999). Finally, regulatory genes do not always have a direct impact, but can pass their instructions indirectly by controlling numerous other regulating genes. French scientist Michel Morange, in his book *The Misunderstood Gene*, summarized the role of regulatory genes when he wrote: "Developmental genes do not directly construct the organism; rather, they provide cells with the relevant properties that enable them to interact in order to construct the organism" (Morange, 2001, p. 98).

Homeobox (Hox) History

The breakthrough by Francois Jacob and Jacques Monod fueled the hopes of many scientists who were engaged in a variety of genetic experiments. One of the experiments, built upon the work of Jacob and Monod, focused on the common fruit fly (*Drosophila melanogaster)*. Nobel laureate Thomas H. Morgan popularized *Drosophila* as an excellent test subject in the early part of the twentieth century, due to its brief life cycle, ease of culturing, high fecundity, and economic feasibility (Lewis, 1998). In the 1980s, researchers began to probe the molecular structure of the DNA in genes. They developed a technique where by a probe could be attached to the specific section of DNA for which they were searching. To their surprise, the probe located about a dozen sections, which they referred to as homeotic genes (Spice, 1999). It was through this research that the now-famous homeobox genes were first discovered and classified. Although initially found in the *Drosophila* experiments, similar Hox genes also have been found in every species subsequently investigated, including (but not limited to), worms, fish, crustaceans, mice, and humans.

Homeobox genes (most popularly known by their abbreviated name, "Hox" genes) are a "class of highly conserved regulatory, or control, genes" (Schwartz, 1999, p. 406). As regulatory genes, they are part of a hierarchical network that controls the expression of the body's structural genes (and thereby, the development of an organism's body). Hox genes are vital for any developing organism, as they serve in an "advisory position" for the other genes. Evolutionists have come to depend heavily upon regulatory genes, especially the Hox cluster, as the main mechanism of mutation. Evolutionists are using mutations in Hox genes as a new mechanism for evolution, thus, bolstering punctuated equilibrium, which was the pet theory of Stephen Jay Gould.

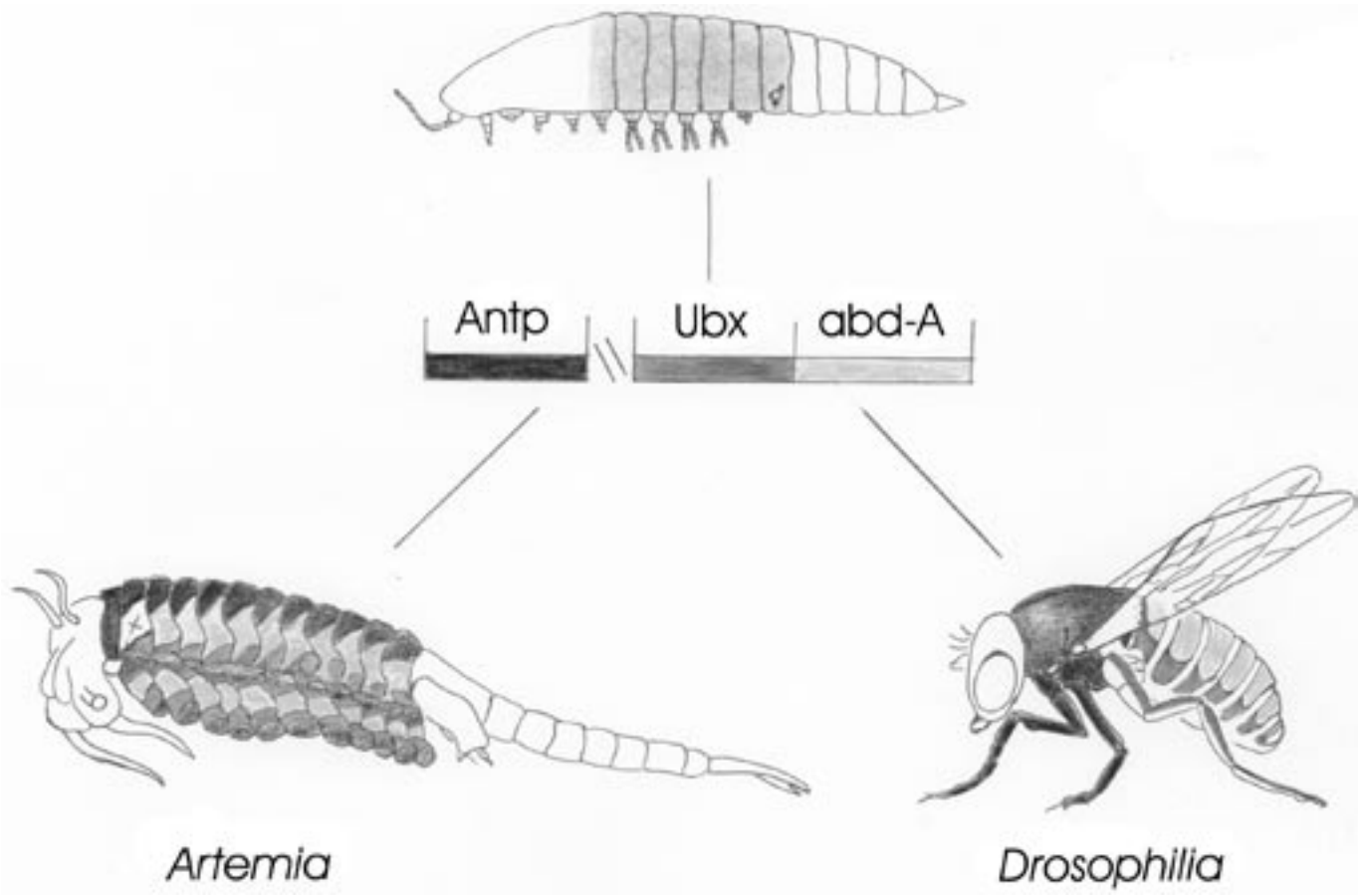

Figure 2. Body plans of *Artemia* and *Drosophilia* showing conservation of abd-P, Antp, and Ubx genes.

This theory is the recent adaptation of Goldschmidt's "hopeful monsters," invoking small mutations in Hox genes to produce the profound effects needed for macroevolution to occur (Batten, 2002).

Walter Gehring's research group initially started working with this homeobox "cluster," seeking to find the individual genes' functions and the mechanism by which these functions were carried out. Little by little, regulating functionality of the Hox genes became apparent. As Gould (2002) remarked: "Presumably, Hox genes 'read' positional information to set the location of differentiating structures, thereby triggering the cascade of downsteam [sic] architects, but not building the varied structures themselves" (p. 1099). This fit the mold, set forth by Jacob and Monod, for how a regulatory gene should operate.

By genetically altering Hox genes, the researchers were able to create mutations within the developing organisms. Some of the more radical mutations that were achieved involved the substitution of an appendage or an exterior organ at another location on the insect's body. An example of this is the alteration of the gene *Antennapedia* (first performed in 1987), which enabled scientists to grow a jointed leg in place of an antenna (Barinaga, 1995). They also were able to control the characteristics of body segmentation, allowing them to make two identical thoracic regions. In a series of three separate mutations, known as *bithorax*, *postbithorax* and *anterobithorax*, a four-winged variety of *Drosophila* resulted. In this mutation, the second set of full-sized wings replaced the halteres (balancing appendages), which normally are located in the third thoracic region behind the wings (Wells, 2000). Much of Gehring's later work focused on the genes involved in the organization of the eye. Gehring made headlines when his group published their work on the newly discovered gene, "eyeless." By altering this particular gene, the group produced eyes in various other tissues such as wings, legs, and antennae (Barinaga, 1995).

In more recent experiments, scientists expanded their research to include the crustacean *Artemia* (brine shrimp). William McGinnis, Nadine McGinnis, and Matthew Ronshaugen, all of the University of California, San Diego, have been conducting research on the Hox gene, *Ultrabithorax* (Ubx), which has been found in both *Artemia* and *Drosophila*. The team, whose findings were published in

Nature, has found that the Ubx gene is associated with limb development (see Ronshaugen, McGinnis, and McGinnis, 2002). By manipulating and transplanting the Hox gene from *Artemia* into *Drosophila*, they have been able to affect the development of the *Drosophila* subjects. The replacement of this gene affected the growth of the fly's legs. This was one of the first experiments to cross the Hox genes of separate organisms to see their correlating effects (Luskin, 2002).

According to Amanda Onion, "Past research has shown that Hox genes act as master switches that turn on and off other genes during embryonic development" (Onion, 2002). Matthew Ronshaugen, a graduate student working in McGinnis' laboratory, commented that "[t]his kind of gene is one that turns on and off lots of other genes in order to make complex structures" (as quoted in McDonald, 2002). Much of the prevailing thought concerning the premise of the Hox gene has been that it is a "master switch." However, this view also has engendered some controversy. As Nelson (2000) commented: "These genes are not master switches for making wings or legs, but they specify position in the fly's body." In an article, "Where Do Toes Come From," in *Time* magazine, J. Madeleine Nash wrote:

> Researchers are finding evidence that the Hox genes and the non-Hox homeobox genes are not independent agents but members of vast genetic networks that connect hundreds, perhaps thousands, of other genes. Change one component, and myriad others will change as well—and not necessarily for the better (Nash, 1995, vol. 146).

Master Genes—or Just Part of the Overall Machinery?

While convenience stores do not carry Hox genes, this does not stop evolutionists from portraying them as a common "one-stop-shop" solution for changing one species into another. Hox genes have been fashioned as self-reliant, relatively simple entities that can cause major mutations. Thus, these "master genes" are used to explain the vast differences in species we see today. The scientific literature on homeobox genes spans the spectrum from reporting experimental results, to pure speculation. Much of this speculation can be attributed to the fact that although most vertebrates and arthropods "have strikingly different body architectures, many of the regulatory genes they use to establish their body plan are conserved" (Kmita-Cunisse, et al., 1998, p. 3030). That is, the "same" homeobox genes are believed to be responsible for very different attributes in vastly different species. However, scientists have drawn conclusions regarding a common origin—with little regard for the empirical evidence. These inferences have led to a belief that animals experience a developmental cascade common to all animals—using these "conserved genes." However, these genes are not simply passive providers of encoded instructions responsible for development.

The cellular environment in which they reside is infinitely complex. In describing eukaryotic cells, Rose stated that the "ribosomal machinery itself consists of a giant assemblage of sub-units together containing more than 80 different proteins, and RNA sequences containing more than 6,700 nucleotide bases. Without it, without the complex biochemical environment the cell provides, 'gene'…simply can't function" (1997, p. 128). In this complex cellular environment, segments of DNA interact with proteins, metabolites, nutrients, and other segments of DNA. Thus Hox genes are "reactive complexes that are in constant and dynamic interaction with their carriers" (Plotkin, 1994, p. 39). In reality these "master control genes" are reliant on a vast network of cellular machinery in order to operate properly.

Gehring (1998) claimed that homeobox genes are the first active genes that lead to a particular outcome. He asserted that these "master genes" activate a series of other genes, leading eventually, in the case of the gene *Antennapedia* for example, to leg morphogenesis. However, this claim breaks down on several different points. *First, homeobox genes are not the first active genes in the embryo*. As Robert (2001) pointed out, mesoderm [the middle layer of cells of an embryo, from which the skeletal, reproductive, muscular, vascular, connective, etc. tissues develop] is genetically induced at the cleavage stage, long before homeobox genes are activated. Additionally, a large number of interacting agents [e.g., antennal disc only form in regions of the body not expressing *Hom-C* (homeotic), and agents responsible for specifying antennae in the larval imaginal disc are *extradenticle* (*exd*) and *homothorax* (*htd*).] and processes must be in place for *Antennapedia* to function at all, for *Antennapedia* does not arise out of thin air and then operate in a precursorless void.

William Bateson, who coined the term "homeosis" in 1894, "felt that he could further strengthen Darwin's case by exhaustively compiling the discontinuous variations that occur naturally within a species" (Lewis, 1994, p. 341). As such, evolutionists were quick to point out that homeobox genes are responsible (at least in part) for the evolutionary origin of body plans (Lewis, 1994; Raff, 1996; Gellon and McGinnis, 1998). Many evolutionists have taken this a step farther and have suggested that Hox genes are the agents responsible for saltation (saltation is the origin of a new species or a higher taxon in essentially a single evolutionary step that in some especially former theories is held to be due to a major mutation or to unknown causes).

Paleoanthropologist Jeffery Schwartz contends that the role of homeobox genes in individual development better explains the origin of new species. He even has suggested that in the production of structures such as the feet, brains, and "completely useful and fully formed eyes,…all that is necessary is that homeobox genes are either turned on or they are not" (Schwartz, 1999, pp. 362,368–369).

Alex Rosenberg, the co-director of the Duke University Center for the Philosophy of Biology, influenced by Gehring's work (Gehring, et al., 1995), as well as that of Lewis Wolpert (1994), contends that genes can "compute" the eye from nucleic acids and proteins alone. According to Rosenberg (1997), there is no need to explain the eye from a structural point of view, because upper-level structures are themselves computable from DNA. Rosenberg contends that the only thing that remains is to fill in the downstream blanks at the genetic level, and we will have a satisfactory explanation for eye morphogenesis. However, Schwartz and Rosenberg are grossly mistaken. As Robert (2001) noted:

> The development of an organism is not fully prescribed in its inherited zygotic or maternal DNA. Rather, development is hierarchical, characterized by the emergence of structures and processes not entirely predictable from lower-level (e.g., genetic) properties of the embryo. For instance, how cells behave collectively during morphogenesis cannot be predicted by examining the behaviour of individual cells prior to cell division, differentiation, or condensation, let alone by examining gene sequences (Hall, 2000, p. 177; see also Hall, 1999). *The very presence of the downstream targets of homeobox genes is due to the synergy of genetic, epigenetic, and environmental factors—not to genetic predetermination* (p. 293, emphasis added).

Even though scientists are able to detect a gene that is necessary for a given developmental event to occur (i.e., wing production), it is a grievous mistake to think that this is all there is to forming that end-product—i.e., that the causal pathway ends (or begins) there. As Nijhout (1990) correctly pointed out: "The causal pathway is endless and involves not only genetic, but manifold structural, chemical, and physicochemical events, a defect in any of which can derail the normal process" (p. 442). Thus, Hox genes cannot serve as so-called "master genes"—because the Hox genes themselves are controlled by external elements. Nijhout (1990) went on to comment: "In a system in which every component, and past history, all have come together at the right time and in the right proportions, it is difficult to assign control to any one variable, even though one may have a disproportionate effect" (p. 442). Are we to believe that all of these components evolved together at precisely the same time so that they could signal development of a leg?

So what of the evolutionists' claim that homeobox genes are "master genes" that can lead to a different species? While a mis-expressed Hox gene can lead to different phenotypic expressions of certain traits, such traits can occur only if the appropriate downstream targets are present at the new site. Akam (1998) noted: "When it comes to the downstream targets of the Hox genes, context is everything, in particular, which other transcription factors are present in the same cell will be a key factor determining the outcome of Hox gene action" (p. R678). That is, Hox genes are reliant on other postcursors (other genes that already are present) including in some cases, other Hox genes.

Mutations and Raw Material—Escape Route, or Dead End?

Hox genes are now the escape route of choice for researchers when they are asked how species evolve into another species. Evolutionary theory holds that mutations in Hox genes produce large-scale changes providing the needed macroevolutionary jumps. For instance, a press release from the University of California, San Diego, said in part:

> Biologists at the University of California, San Diego have uncovered the first genetic evidence that explains how large-scale alterations to body plans were accomplished during the early evolution of animals…. The achievement is a landmark in evolutionary biology, not only because it shows how new animal body plans could arise from a simple genetic mutation, but because it effectively answers a major criticism creationists had long leveled against evolution—the absence of a genetic mechanism that could permit animals to introduce radical new body designs (McDonald, 2002).

According to proponents of this ideology, such as physical anthropologist Jeffrey Schwartz, "[n]ew species would need not be the result of gradual changes [microevolution—BM/BT/BH] that accumulate over many generations as suggested by conventional evolutionary theory. Instead, new species could appear suddenly, as they do in fossil records" (as quoted in Spice, 1999). He went on to elaborate about the specifics of how this might occur: "Mutations could spread silently through a population over many generations, until animals suddenly begin producing mutated offspring. That would be the birth of a species" (as quoted in Spice, 1999). Schwartz (1999) further argued: "When particular genes are turned on for certain lengths of time and in certain regions, a worm may emerge. If the same or other genes are expressed for different lengths of time and in different regions, a more complex organism may

develop" (p. 342). And yet, there is no evidence for such a (false!) claim. Worms do not produce lizards or frogs, no matter how many experimental permutations there may be in which Hox genes are involved. While we can use species-X Hox genes to induce variations in positioning in species-Y specific structures, this is still an extremely long way from Xs birthing Ys based on manipulations of the Hox gene. *Scientific American* editor, John Rennie, erroneously concluded:

> On the contrary, biology has catalogued many traits produced by point mutations (changes at precise positions in an organism's DNA)—bacterial resistance to antibiotics, for example. Mutations that arise in the homeobox (*Hox*) family of development-regulating genes in animals can also have complex effects. *Hox* genes direct where legs, wings, antennae and body segments should grow. In fruit flies, for instance, the mutation called *Antennapedia* causes legs to sprout where antennae should grow. These abnormal limbs are not functional, but their existence demonstrates that genetic mistakes can produce complex structures, which natural selection can then test for possible uses (Rennie, 2002, p. 82).

In trying to go from single-celled "primitive" organisms to *Homo sapiens*, evolutionists commonly focus on mutations as the catalyst for transforming one species into another. As George Gaylord Simpson and William Beck noted: "Mutations are the ultimate raw materials for evolution" (Simpson and Beck, 1965, p. 430). Evolutionist Luigi Cavalli-Sforza, head of the international human genome diversity project, remarked in his book, *Genes, Peoples, and Languages*:

> Evolution also results from the accumulation of new information. *In the case of a biological mutation, new information is provided by an error of genetic transmission* (i.e., a change in the DNA during its transmission from parent to child). *Genetic mutations are spontaneous, chance changes, which are rarely beneficial, and more often have no effect, or a deleterious one.* Natural selection makes it possible to accept the good ones and eliminate the bad ones (Cavalli-Sforza, 2000, p. 176, emphasis added).

Cavalli-Sforza is correct on one of his points, and incorrect on another. It is true that genetic mutations "more often have no effect." Neutral mutations, as they are known, are of little use to evolutionists (see Hitching, 1982, pp. 62–63), as such mutations are dependent on still further mutations in order to be fully expressed and "useful" (in an evolutionary sense). But Cavalli-Sforza was *incorrect* when he stated, "new information is provided by an error of genetic transmission." It most certainly is not! As Sarfati (2002) commented:

> The issue is not *new traits*, but new genetic information. In no known case is antibiotic resistance the result of new information. There are several ways where an information *loss* can confer resistance. We have pointed out in various ways how new traits, even helpful, adaptive traits, can arise through loss of genetic information (which is to be expected from mutations).

Mutations do *not* result in *new* information! Hayward (1985) correctly noted:

> ...mutations do not appear to bring progressive changes. Genes seem to be built so as to allow changes to occur within certain narrow limits, and to prevent those limits from being crossed. To oversimplify a little: mutations very easily produce new varieties within a species, and might occasionally produce a new (though similar) species, but—despite enormous efforts by experimenters and breeders—*mutations seem unable to produce entirely new forms of life* (p. 55, emphasis added).

In the end, after mutations have occurred, no macroevolution has taken place. *None*! Let us not lose sight of the forest for the trees. Producing a four-winged fly, or adding a pair of legs to its head, is a far cry from explaining how plants, animals, and bacteria all descended from a nonliving source. Additionally, we need to keep in mind that the second pair of wings is nonfunctional, and provides more of a hindrance, rather than any benefit.

Hox genes themselves do not produce the information that results in such complex structures as legs, wings, antennae, or body segments (to use Mr. Rennie's examples). Hox genes do not act in a "biological vacuum." They rely on many other genes and proteins as valuable pieces of the overall puzzle. For instance, a light switch is great for turning on a light—but only if you have the necessary wires and bulb "downstream" from that switch. Without those, the switch is nothing more than, well, a switch. Keep in mind there is a well-balanced feedback mechanism in place inside every living cell. If more proteins are needed, genes are "turned on" so that those proteins can be produced. When genes mutate, this delicate balance of proteins is affected adversely, causing the production of either too much or too little of these much-needed proteins.

In trying to go from amoeba to fish to reptile to land dwellers, evolutionists clamor about mutations being the catalyst for transforming one species into another. But the question really is: How often do *good* mutations occur? Hermann J. Muller, Nobel laureate in genetics, said: "Accordingly, the great majority of mutations, certainly well over 99%, are harmful in some way, as is to be expected of the effects of accidental occurrences" (Muller, 1950, p. 35). Evolutionist Theodosius Dobzhansky of the Rockefeller University admitted that favorable mutations amount to less than 1% of all mutations that occur (as quoted in David-

heiser, 1969, p. 209). Dobzhansky (1995) even remarked: "Most mutants which arise in any organism are more or less disadvantageous to their possessors..." (p. 105). Twenty-one years later, in addressing the rarity of these "good" mutations, Japanese geneticist, Motoo Kimura, commented: "Considering their great importance in evolution, it is perhaps surprising that well-established cases are so scarce" (Kimura, 1976, p. 260). And twenty-five years after that, Harvard's eminent taxonomist, Ernst Mayr, remarked that "…the occurrence of new beneficial mutations is rather rare" (Mayr, 2001, p. 98).

We are told that "nature" has "selected" certain beneficial mutations and incorporated them into various organisms, eventually causing those organisms to change from one kind to another. If "mutations are the ultimate raw materials for evolution," and therefore provide the mechanism for evolution, there are some very serious problems indeed. Considering their rarity and randomness, the good mutations that evolution requires must be rather exceptional. Pierre-Paul Grassé, the preeminent French evolutionist, recognized the error to which so many scientists succumb when interpreting mutations, and commented:

> Some contemporary biologists, as soon as they observe a mutation, talk about evolution. They are implicitly supporting the following syllogism: mutations are the only evolutionary variations, all living beings undergo mutations, therefore all living things evolve. This logical scheme, is, however, unacceptable: first, because its major premise is neither obvious nor general; second, because its conclusion does not agree with the facts. *No matter how numerous they may be, mutations do not produce any kind of evolution* (Grassé, 1977, p. 103).

Figure 3. Image of fly with ectopic eye (used with permission).

However, in order not to rely solely on one evolutionist's admonitions, we add to Grassé's voice the voice of the late American evolutionist, Stephen Jay Gould. In a speech, "Is a New and General Theory of Evolution Emerging?," presented at Hobart College on February 14, 1980, Dr. Gould went on record as stating: "*A mutation doesn't produce major new raw material*. You don't make a new species by mutating the species.... That's a common idea people have; that evolution is due to random mutations. A mutation is not the cause of evolutionary change" (as quoted in Sunderland, 1984, p. 106, emp. added). This brings to bear an excellent point concerning mutations and their supposed ability for macroevolution. Macroevolution, by definition, is the "evolution above the species level; the evolution of higher taxa and the production of evolutionary novelties, such as new structures" (Mayr, 2001, p. 287). Yet, according to Gould, mutation does not make new species. Is mutation a process for macroevolution? Again, according to the definition, macroevolution must "produce evolutionary novelties, such as new structures." However, Gould said that mutations do not make new raw material. Is mutation a process for macroevolution? Not according to Dr. Gould.

In their book, *Acquiring Genomes: A Theory of the Origins of Species*, evolutionists Lynn Margulis and Dorion Sagan boldly stated: "Many ways to induce mutations are known but none lead to new organisms. Mutation accumulation does not lead to new species or even to new organs or new tissues" (Margulis and Sagan, 2002, p. 11). They went on later to conclude: "New mutations generate variations in members of the same species but the accumulation of mutations has never been shown—in laboratory organisms or in the field—to lead to crossing of the species barrier" (p. 72). Creationist David A. DeWitt, professor at Liberty University, also has emphasized the point that, despite their introduction of mutations, "the fact that scientists can significantly alter the body plan does not prove macro-evolution nor does it refute creation. Successful macro-evolution requires the addition of *new* information and *new* genes that produce *new* proteins that are found in *new* organs and systems" (DeWitt, 2002). *These quotes, from both evolutionists and creationists, have emphasized a crucial point: scientists have not produced anything "new" via mutations*. How, then, can evolutionists claim that Hox genes provide the answers to

speciation, when the evidence demonstrates that *new* organisms are not formed? Dewitt (2002) went on to note:

> For example, a single mutation that might prevent legs from forming is much different from a mutation that produces legs in the first place. Making a leg would require a large number of different genes present simultaneously. Moreover, where do the wings come from? Just because an organism loses a few legs doesn't convert a shrimp-like creature into a fly. Since crustaceans don't have wings, where does the information come from to make wings in flies? Having the wings themselves is not even enough. Researchers in another study have found that the subcellular location of metabolic enzymes is important for the functional muscle contraction required for flight. Indeed, the metabolic enzymes must be in very close proximity with the cytoskeletal proteins that are involved in muscle contraction. If the enzymes are not in the exact location in which they are needed within the cell, the flies cannot fly. This study bears out the fact that "the presence of active enzymes in the cell is not sufficient for muscle function; colocalization of the enzymes is required." It also "…requires a highly organized cellular system."

The experiments discussed earlier, although rather impressive in what they have accomplished, have not interjected the addition of new information, genes, or organs. The transplanted genes, whether in the shrimp/fly or the mouse/fly experiments, did not introduce new structures. For example, the ectopic eyes that grew in the uncommon tissues of *Drosophila* were the ordinary compound eyes of a fly, as opposed to the deformable lens of vertebrate eyes. The scientists did not introduce anything new; rather, they rearranged and duplicated the pre-existing structures. *The Hox gene is only an instruction provider and a structure-placement gene; the actual formation of the eye was left up to the "2500 genes from Drosophila…required to assemble an eye"* (Gould, 2002, p. 1124; see also Keller, 2000, p. 96, emp. added).

Further, the eyes were, as Gould proclaimed, not even "wired up," and thus were non-functioning organs that apparently were useless to the fly. Keller (2000) questioned the explanation of the experiment involving these "master control genes," and their morphogenesis of the eye, when she commented:

> [T]here is a sense in which this claim is obviously contradicted by the very experiment that has been taken to corroborate it. If the mouse counterpart to *eyeless* (Pax-6) were truly a 'master control gene,' ought we not to expect that it would induce the formation of a mouse eye and not a *Drosophila* eye? Might one not interpret the fact that the mouse gene is used by the fly to form its own kind of eye as corroborating a claim of a rather different sort—namely, that *eyeless* plays a key role in the formation of an eye, the precise nature of which is determined by the context in which the gene finds itself? (p. 97).

Likewise, in the experiments involving limb growth, which worked with either *Antennapedia* or Ubx, the same holds true. No new structure was added to the organism. In the case of *Antennapedia*, some rearrangement occurred, while in the case of Ubx, there was growth impediment. In the instance of *Antennapedia*, the leg that replaced an antenna was non-functioning and, as such, hardly could be called a "good" mutation. As was the case in the four-winged fly, which did exhibit an additional set of wings, but the wings were of no benefit to the organism. What the scientists formed via mutations were not new and better-equipped organisms, but were organisms encumbered by their misplaced and underdeveloped organs—i.e., laboratory monsters. Are scientists ready to argue that these "unfit" creatures uphold their survival-of-the-fittest theory?

Conservation or Common Designer?

The data obtained thus far clearly have shown that homeobox genes have been found in a variety of specimens, covering many different phyla. However, Morange (2001) cautioned: "We should avoid jumping to conclusions: using the same genes in development does not necessarily indicate the presence of a homology" (p. 99). The similarity of genes and gene development does not require common descent. The late British biologist, Gavin de Beer, remarked: "Characters controlled by identical genes are not necessarily homologous," and conversely, "homologous structures need not be controlled by identical genes" (as quoted in Wells, 2000, p. 73). Both of these statements—which, in the case of an evolutionist, are clearly confessions—obviously restrict the interdependence between genes, structures, and their homology. An example of de Beer's common gene, yet uncommon structural development, is the gene *Distal-less*. This developmental gene is involved in limb formation in the mouse, spiny worm, butterfly, sea urchin, and velvet worm. Each of these creatures is in a different phylum, and each uses the gene to form vastly different structures. In each case, the *Distal-less* gene forms a unique product, with a unique body position, and a unique number of gene expressions—all of which clearly show the separation in the supposed commonality of descent.

Is common ancestry indicated by the fact that both plants and animals contain homeobox genes? Or, could the fact that both possess Hox genes be evidence of a common Designer? The Hox genes are viewed as a remnant from the past. As the earlier definition noted, they are "highly conserved," which means, according to evolutionists, that

Hox genes have been passed down through the phylogeny with little change. Evolutionists allege that a common ancestral descent is proved through the similarity of the genes and physical structures between organisms. Yet, it makes logical sense that these similarities would be present—if life had been designed. Take, for instance, the lung; used by air-breathing organisms, it shows great similarities in many different kinds of creatures. However, this does not demand common descent, but rather shows design intended for survival. Another example is the structures of locomotion, which reveal many similarities, but are complex units that perform the function of transportation. Further, it is logical that if cellular life were designed, many of the same components (i.e., nucleus, ribosomes, DNA, etc.) would be found in all life forms. The idea of the conserved gene has fallen under scrutiny as scientists find differences in homeobox genes of various animals.

Additionally, data suggest that these genes may not be as "highly conserved" as once thought. Galant and Carroll (2002) reported finding a transcriptional repression domain in the carboxy-terminal region of the *Drosophila Ultrabithorax* (Ubx) protein. They found this domain among similar proteins in other insects, but found that it was absent from the Ubx in other arthropods and onychophorans (wormlike carnivorous animals, having characteristics of both arthropods and annelid worms—e.g., the velvet worm). They went on to speculate (Galant and Carroll, 2001):

> The differences between DUbx and OUbx could be due either to the aggregate divergence of sequences along the length of the proteins, or to the presence of one or more discrete functional motifs that arose in the insects or were lost in the onychophorans, some time after the separation of their lineages from a common ancestor more than 520 million years (Myr) ago (p. 910).

The lines connecting such groups to the evolutionary tree of life frequently are found crossing over one another as evolutionists continue to try to harmonize the fossil record with these types of genetic data—all the while keeping in mind that they must be able to account for the loss (or addition) of various Hox genes in different animal species.

Conclusion

Paleoanthropologist Jeffery Schwartz once noted:

> It is mind-boggling to entertain the possibility that, for all intents and purposes, the difference between a fruit fly and a human might have as much (or even more) to do with turning on and off of *homeobox* genes that both animals share (as quoted in Spice, 1999)

With the single stroke of a pen, Schwartz reduced the image of man down to that of a fruit fly. We agree with at least the first part of Schwartz's quote: "It is mind-boggling to entertain" such a possibility, and worse yet—ludicrous. Since 1983, when the homeobox genes were discovered, the scientific world has earnestly sought out their role and function. Numerous experiments continue to be conducted in an effort to manipulate and control the development of a variety of species. Scientists are producing good data in a quest to understand Hox genes. However, it must be stated that it is *not* the scientific data of Hox gene research that are being disputed. What *is* in dispute, though, is the *interpretation*. As in all areas of science, the *motivation* for observations does not affect the validity of correctly gathered data, but most assuredly taints the *explanation*. Despite what the evolutionists are trumpeting, the mutations induced through Hox genes are not introducing *new information*—which is a requirement for macroevolution. As evidenced by their experiments, the mutated creatures are simply deformed, and as Jonathan Wells put it, "In affect, all they've produced is a crippled shrimp" (as quoted in Onion, 2002). Evolutionists also have pointed to the similarity of genes and physical structure as "proof" of common ancestry. However, it has been shown that the similarity of Hox genes does not demand similarity in physical structure; neither does similarity in physical structure require similar genes. As geneticists and biologists continue to probe the physiological elements that define life, there is an abiding sense of awe toward the complexity found. From the studies of heredity and genetics, to the discovery of the double-helix structure of DNA, each new breakthrough has proven life's incredible complexity. Though the Hox gene is a regulatory gene that can direct thousands of other genes, it is still only part of an intricate network. Regulatory genes themselves are directing, being directed by, and interacting with, countless other genes. Simply put, the Hox genes are another example of a design masterpiece.

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