

## **Evolution in the genome**

*If science is to be true to itself, it needs to consider other possibilities than evolution by accident in one corner and the creation of immutable species in the other. This article looks at what a mutation is, and at evidence that the biggest changes in the history of life have been the result of genetic sub-programs being switched on or off by regulatory systems. Biological studies do not support the idea that significant evolution (which has certainly occurred) is undirected.*

The theory of evolution depends on the idea that there is only one kind of evolution. Over time species change, diversifying into new species, and from this it is concluded that every organism is related to every other: bacteria, fungi, animals, plants, plankton in the sea, the lot. The logical leap is as big as the biological: finches on the Galapagos Islands have been evolving different shapes of beak, therefore all organisms are connected to the same tree, rooted in the unseen murk of a 'prebiotic soup'.



There is likewise, we are told, only one alternative to the view that all organisms evolved from a soup of chemicals, and that is the view that all organisms were created just as they are now:

According to creationism, all species present on Earth today have remained unchanged since they were created by God. Darwin's theory of evolution contradicts this belief.

Michael Kent, *Advanced Biology*, 2000.

The belief that the world was created by an Almighty God is called creationism. ... Everything in the world today is still as it was when it was created.

Ernst Mayr, *What Evolution Is*, 2001.

In general the evidence for the fact that evolution has occurred consists of an enormous number of detailed observations which all make sense if we assume the theory of evolution, but which can be explained by the creation theory only if we assume that the creator elaborately set out to deceive us.

Richard Dawkins, *Darwin and Darwinism*, 1998.

No one actually knows the exact number of species on earth. The number already classified is around 2 million. ... Adam was brought by God in Genesis 2:19-20 to name all the animals, but we have a long way to go in finally fulfilling that command!

Denis Alexander, *Creation or Evolution – Do we have to choose?*, 2008.

Given that this is the choice, few would disagree that the theory of evolution is the more reasonable of the two opposites. Darwin presented the argument in much the same way: either evolution of all living species from a single ancestor (possibly from 'a few' simple forms), or creation of all living species by God. Since the evidence of variation and adaptation showed that species could not have been created in their present form, in their present locations, it was

clear that species could not have been created. That implicitly left only Darwin's theory. It was Hobson's choice – the theory of common descent or nothing.

We need to be sure, then, that this really is the kind of change that organisms undergo over time: slow, gradual, random evolution towards forms of ever greater complexity. If that is not the story, then the choice presented may be a false one. We may find that the desire to present it in such terms itself deeply suspicious, belonging more to the world of political knock-about than a concern for truth and honest inquiry. Looking at what nature itself tells us rather than philosophical preconceptions of what it ought to be like, we may find that the real alternative to Darwinian evolution may be creation with the ability to adapt and diversify having been programmed into it, not a Neverland creation which, like Peter Pan, never grows old.

### **The genetic code appears to be optimal**

The biological instructions that make an organism what it is, be it plant or animal, are coded in its DNA. The code consists of the mapping of 64 three-letter codons in the DNA to 20 amino-acids (the building blocks of proteins) and a stop signal. As it happens, this is but one of more than a thousand possible codes for building proteins and orchestrating an individual's development. Nonetheless, the actual code is universal to all organisms and has been shown to be 'very near to (and quite possibly at) a global optimum for error minimization'. All things considered, it is the best of all possible codes (Freeland *et al* 2000, Itzkovitz & Alon 2007).

This is theoretically difficult from a Darwinian point of view, for two reasons. First, in a scenario where the first self-reproducing organism comes into existence accidentally, we cannot assume that the code used would be the one we know now; it could have been one of many. We have to explain why this particular code rather than another non-optimal, but still adequate, code was adopted. Second, the universality of the DNA code cannot be assumed to be evidence that all life descends from a common ancestor. There is an alternative interpretation: that the code is the outcome of choice, being the best for what it is designed to do.

The same applies to the presence of similar, even identical, genes in widely different organisms, for example the *Hox* genes that control the formation of body parts during the development of embryonic mice and fruit-flies. They are generally interpreted as evidence of common descent. However, the 'conservation' of these genes – the fact that they have not changed over time, either within their respective lineages or across the animal kingdom – is something unexpected by the Darwinian paradigm. The alternative view is that the genes are common to animals because they represent optimal design. All embryos need to develop in a controlled way, and evidently *Hox* genes do the job superbly.

### **Variation among individuals**

In general, nature shows a strong preference for diversity rather than uniformity, and genetic systems appear to be designed to promote diversity. Within the same species males and females each have their distinct genetic endowment. When they reproduce, their chromosomes are shuffled to produce an individual that is unique. In addition, part of the uniqueness comes from the fact that genomes (genetic programs) mutationally change over time.

In human beings diversity is most obviously expressed at the individual level. *Homo* is among the most diverse species on the planet, with one individual's genome differing from another's by

an estimated 15 million points of difference. Some of the differences are the result of extensive structural variations, such as additions, deletions, repeat sequences and stretches of ‘backward’ DNA. Some are associated with behavioural traits, others with susceptibility to disease. There are also considerable differences in genome size.

Extreme diversity can also occur in organisms that do not reproduce sexually. The greatest within-species variability occurs in bacteria. Strains of the same species can vary by up to 30% in gene content, raising questions as to whether they should even be regarded as the same species (Konstantinidis & Tiedje 2005). In the case of *E. coli* the number of genes that the nine genomes sampled held in common was 3,050, whereas the number of unique genes exceeded 8,000.

In other organisms diversity is most obviously expressed at the species level and higher. Passerines, for example, which include finches, number 4 suborders, around 120 families and more than five thousand species. Beetles number 4 suborders, 168 families and hundreds of thousands of species.

### Proof-reading and repair mechanisms

Although the code is optimal for minimising the effect of translational errors, mutations of the program in individuals do occur. Potentially harmful errors occur every time a cell divides, but are corrected by elaborate and highly effective proofreading systems during replication and mismatch repair systems thereafter. Further damage to DNA may occur in adult life, for example as a result of exposure to toxins, carcinogens, or ultraviolet light. Again, to deal with such contingencies, genomes can call on a variety of damage-detection and repair mechanisms, including base excision repair, nucleotide excision repair, mismatch repair, non-homologous end-joining and homologous recombination.

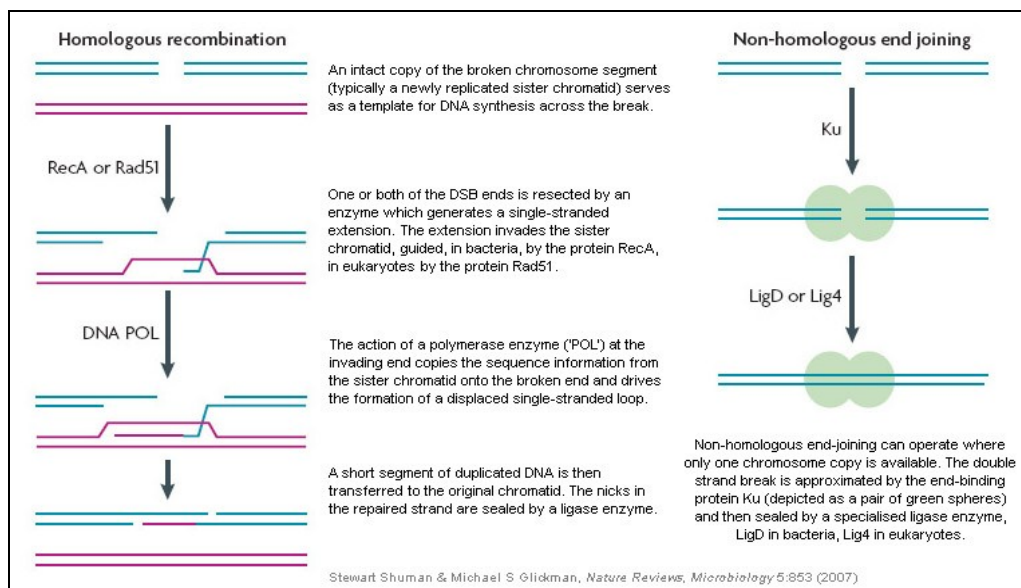


Figure 1. DNA repair mechanism: homologous recombination and non-homologous end-joining.

The detail of how these mechanisms work is not important for the present purpose (the latter two processes are illustrated here). The key point is that chance mutations are not a good thing, and defects in the correcting mechanisms are often implicated in fatal abnormalities such as cancer.

These mechanisms are essential for life. They have been found in representatives of all types of organism, including bacteria. The provision of such machinery is evidence that the genome originated in an intelligence that was well aware of the role of chance in the world and took pains to ensure that life had more than a fighting chance of overcoming it.

### **Mutations – harmful, neutral and beneficial**

According to the modern version of Darwin's theory, all evolutionary change results from random mutation. Despite the best efforts of machinery designed to prevent error, base pairs (the pairs of nucleotides that make up DNA's double helix) occasionally get miscopied, resulting in 'point mutations'. Since the gene is already a fully functioning code, any mutation of it will usually be either harmful or inconsequential. Harmful mutations, such as the miscopying accidents that underlie some diseases, reduce the fitness of an individual and can be lethal. Neutral mutations do not affect fitness and can spread through a population over time through genetic drift. Beneficial mutations are problematic, since 'almost all conceivable beneficial mutations of a population in a stable environment have already been selected in the recent past' (Mayr 2001).

Commonly cited examples of beneficial mutations include those enabling bacteria to resist antibiotics and haemoglobin cells to resist malaria (as in sickle cell anaemia). The mutations appear to be random, and they confer an advantage. However, in the organism's normal environment, where antibiotics or malaria are not present, the cells with the mutation are less fit and the organism is likely to revert to type. In our imperfect world disease is a fact of life. New diseases arise as bacteria mutate and host organisms have to evolve new ways of combating them, in an arms race where both parties have to run just to stand still. The bacteria remain bacteria; the host organisms remain fundamentally the same. Neither party is moving forward, or reaching higher levels of being. Disease may be an example of the 'struggle for existence', but it is not necessarily a good analogy for the kind of evolution that turns bacteria into people.



Figure 2. The bacterium *E.coli*

The accruing of an advantage is not, moreover, the crucial issue. A point mutation simply substitutes one base pair in a sequence for another, and on the rare occasions when this does not incapacitate the gene, the total amount of information remains unchanged. One simply has an allele, or variant, of the same gene. One allele in a flower species may code for proteins that give it a red colour, another for proteins that give it a yellow colour, and over time it could be that many such alleles will arise. There are only four types of base pair, and in a long sequence of such pairs a mutation may result in the code for a closely related protein rather than a code that is nonsensical.

Even here, where accident clearly plays a role, we may not be dealing with uncontrolled accident. In a paper entitled 'Darwinian evolution can follow only very few mutational paths to fitter proteins' Weinreich and colleagues reported (2006) that the joint effect of five point mutations in a bacterial gene was to increase resistance to a certain antibiotic by a factor of ~100,000. In principle, evolution to this high-resistance allele could have followed any of 120 mutational pathways, but 102 of these were found to be inaccessible to Darwinian selection, and

of those remaining many had negligible probabilities of realisation. Despite the paper's title, the conclusion was that protein evolution might not be as random as Darwinian theory predicts. It was constrained to follow pathways that were largely reproducible.

Fitness in the limited sense of resistance to the antibiotic increased 100,000 times, the complexity of the organism not at all. Believers in Darwinian evolution, by contrast, need to demonstrate an increase in complexity, not simply increased fitness. Mutations of existing genes do not exemplify a process whereby life evolved from mud to mankind.

DNA may also undergo more radical changes, such as the excision of a segment from one part of a chromosome and insertion into another. These are also classified in the Darwinian paradigm as random mutations. The genome is assumed to be the work of accident, despite its still unfathomed complexity, and changes to it therefore must be understood as taking place passively – the equivalent, one might say, of the dogma that species are fixed. Nonetheless, from an objective, scientific viewpoint, it is possible that the program is an active one, where non-coding parts of the genome are capable, in some circumstances, of causing the coding parts themselves to change.

### **Evolution through gene loss**

In 1999 Maynard Olson, currently director of the University of Washington Genome Center, wrote an article entitled 'When less is more: gene loss as an engine of evolutionary change'. The paradox hinged on the word 'evolution', as if what was in prospect was an increase in biological complexity. Olson's proposal was that mutations involving the loss of a function could be positively beneficial to an organism.

The idea that genetic loss may be an important engine of evolutionary change is counterintuitive. We like to think that organisms achieve better fitness by having "better" genes, not broken ones. Over the broad sweep of evolutionary time, this principle must be true, but loss and regain of gene function may be common over shorter stretches of a species' history.

Gene loss might be beneficial, but it was not, admittedly, a mechanism for upward evolution. In the long run, evolution 'must' innovate in more fundamental ways than simply by giving up what it had once 'created' (*sic*).

Bacteria provide striking examples of gene loss and reversion. Bacteria that live within the body or cells of another organism ('endosymbionts') generally have small genomes and appear to descend from free-living ancestors that had larger ones (Nilsson *et al* 2005). The reduction of genome size is facilitated by the absence of selection for many bacterial functions as a result of the bacteria utilising the products of metabolism within the host. DNA identified as non-essential can also be lost through genetic machinery that purposely effects its deletion. The most evolved species therefore do not necessarily have the biggest genomes. In the case of the highly specialised *Mycobacterium leprae*, the bacterium that causes leprosy, more than 50% of the ancestral genes have been lost – most of them very recently (Gómez-Valero *et al* 2007). What matters is efficiency, especially efficiency in reproduction. The 'molecular technology' of bacteria optimises efficiency, and optimum genome size can be explained on the same principles of economics as define optimum size in an industrial factory: the optimum is reached when the

genome obtains maximum metabolic complexity (revenue) for minimal regulatory genes (logistic cost) (Ranea *et al* 2005).

The loss of genes in the absence of selective pressure to retain them is a familiar phenomenon. Yeasts, unlike bacteria, are eukaryotes – cells with nuclei – but here too the most evolved species do not necessarily have the biggest genomes. Comparing the genomes of six species, Scannell *et al* (2006) concluded that their common ancestor underwent, as a species, duplication of its entire genome. Subsequently the species diversified into many other species and most of the duplicate genes were silenced or deleted. Gene loss was part of the process whereby the yeasts diversified.

### Evolution through loss or reduction of anatomical structures

Darwin proposed that evolution proceeded through ‘numerous, successive, slight modifications’. In this way, from simple beginnings ‘endless forms most beautiful and most wonderful have been, and are being evolved’. However, as Neil Shubin and Randall Dahn have pointed out (2004), what has been documented by way of evolution is rather different:

Surprisingly, some of the most significant novelties in the history of life are associated not with the evolution of new structures but with the loss or reduction of primitive ones. In vertebrates, for example, the invasion of new ecological niches and the origin of new locomotor adaptations involve either the complete loss or partial reduction of appendages. The complete loss of appendages has been involved in the evolution of new aquatic lifestyles in whales and burrowing niches in snakes, amphisbaenians (worm lizards) and caecilians (rubber eels).

The list is almost endless: loss of eyesight in cave fish and other cave animals, the loss of the sting in certain bees, the loss of venom glands in snakes, the loss of flight in certain insects and birds. A further example is the reduction or loss of the pelvic spine of certain sticklebacks, the occasion for Shubin and Dahn’s commentary.



Figure 3. The three-spined stickleback *Gasterosteus aculeatus*, pelvic spine arrowed.

‘pleiotropic’ gene because it influences many traits, in this case the thymus, the olfactory pits and the tail, as well as the spine. A change in the regulation of the gene causes *Pitx1* to be unexpressed, but only in the pelvic skeleton; the other parts of the body are unaffected. The stickleback genome appears to contain within itself the ability to bring about evolutionary change.

Sticklebacks are also an example of how evolution can occur very rapidly. As Shubin and Dahn mention, the phenomenon of rapid evolution is a puzzle for biology, one on which the research into gene expression is shedding some light. Regulatory changes

are likely to be extraordinarily important components of evolutionary history. Indeed, stratigraphic and geographic analyses suggest that limb loss in sticklebacks has evolved in fewer than 10,000 generations. Extrapolating these results to other taxonomic groups leads to the conclusion that major morphological change can evolve rapidly through regulatory changes in a small number of genes.

The interpretation offered appears to be that changes in the way genes are regulated occur when the genes doing the regulation change. However, the regulatory genes could also be interpreted as initiating the changes in response to environmental stimuli, themselves remaining unchanged, for the same losses can occur independently in different populations and in different species. Pelvic fins, for example, have been lost by independent lineages of fish at least 70 times. As we will discuss, this is strong evidence that the regulatory changes are not fortuitous.

### Convergent evolution

Convergence is the technical term for the appearance of the same feature in two or more related lineages whose common ancestor did not have that feature. The phenomenon occurs throughout the genealogies making up the history of life and it is one of the clearest kinds of evidence that evolutionary novelties are not the result of slight, successive, chance mutations but of the activation of a pre-existing program.



Figure 4. The leaf insect *Phyllium*.

One such example is the incidence of wings amongst stick insects, creatures that bear the testimony of their creator simply by their appearance, without our having to know anything about their phylogeny or biology. Most of them mimic twigs, even to the point of looking as if they have been blotched by lichen, moss or bird droppings. A few mimic leaves, complete with leaf veins, mildew spots and insect-feeding damage. Since insects cannot will into existence a likeness to their surroundings, this fantastic

mimicry must be something they either acquired by accident or were given by design. Design is surely the more plausible alternative. Most remarkable of all is the fact that the twig-like and leaf-like insects are genealogically related, in the single order Euphasmatodea. Somehow, the same order was able to evolve the ability to mimic two totally different forms of vegetation.

Of the 3,000 described species of extant stick insects, 40% are fully winged, the rest being either partially winged or without wings. Although there are wide margins of uncertainty, their relationships can be deduced by analysis of their DNA, and from this it appears that the ancestral condition of the order is winglessness. The six most basal stick-insect lineages are entirely wingless. Species with fully developed wings arose only later, first among leaf insects (*Phyllium*) and then independently in at least three other lineages. In one case (*Lopaphus*) wingless and partially winged occur within the same genus. Still more strikingly, females can be wingless and males fully winged within the same species.

Clearly wings did not evolve again and again from scratch in the course of stick-insect evolution. Indeed ‘entomologists have long assumed that re-evolution of wings in apterous [wingless] lineages was impossible, because functional wings require complex interactions among multiple structures, and the associated genes would be free to accumulate mutations in wingless lineages, effectively blocking the path for any future wing reacquisition’ (Whiting *et al* 2003). Wing development depends on multiple gene systems and interacting gene products, and the origination of such a structure by chance is very difficult to imagine, let alone account for. By contrast, because of the complexity of the developmental process, wing loss could arise from a mutation affecting any one of the genes involved. It could also arise non-randomly, through a regulatory gene switching the whole process off. Bearing in mind the frequency with which winglessness can be reversed and also the fact that both the winged and wingless states can occur within the same species, according to sex, it seems clear that winglessness is primarily controlled through regulation. Even in wingless species, the basic genetic blueprint for wing formation remains intact.

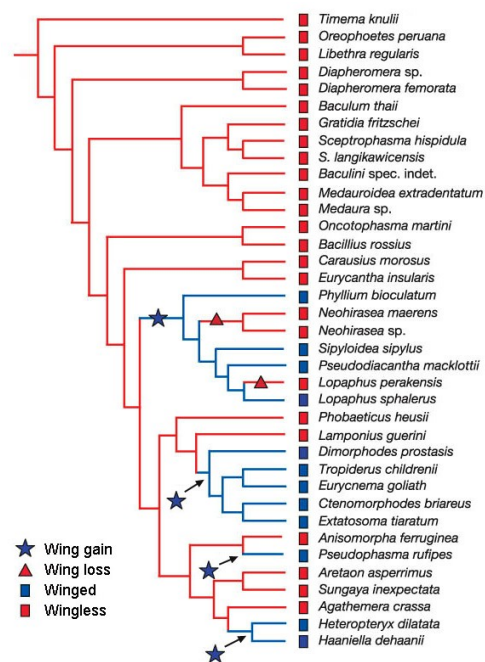


Figure 5. A genealogy of stick insects, winged (blue) and wingless (red).  
Source: *Nature* 421:266.

In the Darwinian narrative one is told that stick insects ‘evolved’ wings. In reality, they did nothing of the sort. Whether or not the first stick insects were wingless, the evidence shows that wings were latent in the genome from the beginning. Similar patterns of recurrence have been suggested for eyes in ostracods, ocelli in cave crickets, wings in water striders and wings in male fig wasps, among other cases (Whiting & Whiting 2004).

### Non-random evolution

Can genomes respond to environmental cues? In the development of an individual organism they clearly can. In addition to genes that code for proteins, genomes hold instructions about where in our body and when in our lives to make the proteins. As the embryo develops, cells with the same genome multiply and, in response to the environmental cues of each other, differentiate into the tissues and structures that make up the body’s different parts. Genes get turned on and off, and the on/off state is passed from mother to daughter cell. The orchestration is nothing if not awe-inspiring. With human beings the process continues into puberty and beyond as the child becomes a sexually mature adult. With stick insects the regulatory gene network underlying wing development can produce winged males but leave females wingless. With ants it produces a winged queen but leaves worker ants wingless. With the Map butterfly it produces wings with speckled orange colouration in the spring and dark wings with white banding in the summer.

What, then, if genomes, maybe in response to some external trigger, are capable not only of repairing but of changing themselves? Or, to phrase the question in the way Darwinians think, what if DNA contains information that makes mutation more likely in some parts and less likely in others? Addressing this question under the title ‘Genomes do not play dice’, Lynn Caporale

says ‘there is clear evidence from organisms as diverse as humans and bacteria that genomes do contain information that can focus mutations in certain areas and direct it away from others’. It’s almost as if genomes are ‘intelligent’, as if they see environmental change coming and in response actively initiate genetic change. Jeffrey Miller (2005) makes similar observations:

The genome should now be envisaged as being in dynamic equilibrium between a multitude of forces for mutational change and forces that counteract such change. In some cases, this is achieved by specific enzymes, and in other cases high mutability is programmed into the sequence of certain genes to help generate diversity.

According to the prevailing dogma, all mutations are random and therefore errors, but in reality organisms appear programmed with an ability to mutate. Cells possess ‘natural genetic engineering functions’ that can initiate evolutionary change by rearranging genomic components and reorganising system architectures (Shapiro 2002).

Perhaps the most striking instance of plasticity is the transposition of whole sequences from one part of a chromosome to another, even from one chromosome to another. As with proof-reading and repair mechanisms, these transposable elements (‘TE’s) have been found in all types of bacteria investigated, from bacteria to human beings. According to Mayr (2001), one of the founders of neo-Darwinism,

Mutations are due to errors of replication during cell division. ... Mutations may also be caused by the insertion of a transposable element in the chromosome. ...No selectively valuable contributions are known for any of the TEs. Rather they seem deleterious, but natural selection seems unable to eliminate them.

In stark contrast is this view from James Shapiro, professor at the University of Chicago, two years earlier:

Cells are capable of sophisticated information processing. Cellular signal transduction networks serve to compute data from multiple inputs and make decisions about cellular behavior. Genomes are organized like integrated computer programs as systems of routines and subroutines, not as a collection of independent genetic ‘units’. DNA sequences which do not code for protein structure determine the system architecture of the genome. Repetitive DNA elements serve as tags to mark and integrate different protein coding sequences into co-ordinately functioning groups, to build up systems for genome replication and distribution to daughter cells, and to organize chromatin. Genomes can be reorganized through the action of cellular systems for cutting, splicing and rearranging DNA molecules. Natural genetic engineering systems (including transposable elements) are capable of acting genome-wide and not just one site at a time. Transposable elements are subject to regulation by cellular signal transduction/computing networks. This regulation acts on both the timing and extent of DNA rearrangements and (in a few documented cases so far) on the location of changes in the genomes. By connecting transcriptional regulatory circuits to the action of natural genetic engineering systems, there is a plausible molecular basis for coordinated changes in the genome subject to biologically meaningful feedback.

The controlled precision with which DNA segments are transferred from one location to another is but an instance of the systems engineering that characterises genome organisation as a whole. Rearrangements, deletions and duplications of genetic modules have been fundamental events in

evolution, and they have occurred under the direction of the genomes themselves, enabling the organism to survive crises or exploit new ecological opportunities that could not be handled using the existing genome. It is not clear that chance plays any constructive role in genome evolution. Apart from generating alleles, its work is only to damage and destroy.

Shapiro (2002) summarises the revolution going on in the way molecular biologists think about the unseen world they investigate with the following table:

Conceptual Category	20th “Century of the Gene”	21st “Century of the Genome”
Dominant scientific perspective	Reductionism	Complex systems
Fundamental mode of biological operation	Mechanical	Cybernetic
Central focus of hereditary theory	Genes as units of inheritance and function	Genomes as interactive information systems
Genome organisation metaphor	Beads on a string	Computer operating system
Sources of inherited novelty	Localised mutations altering one gene at a time due to physico-chemical insults or replication errors	Epigenetic modifications and rearrangement of genomic subsystems by internal natural genetic engineering functions
Evolutionary processes	Background random mutation and natural selection of small increases in fitness; cells passive	Crisis-induced, non-random, genome-wide rearrangements leading to novel genome system architectures; cells actively engineering their DNA

In the wake of Francis and Crick’s discovery of the double helix, the expectation was ‘that molecular biology would confirm the reductionist, mechanical view of life’. Instead, it has revealed a realm of ‘indescribable complexity’.

### Evolution in the fossil record

It remains to be considered whether the fossil record gives any countenance to Darwinian concepts. Apparently, not a lot:

The truth of the matter is that we are still in the dark about the origin of most major groups of organisms. They appear in the fossil record as Athena did from the head of Zeus – full blown and raring to go, in contradiction to Darwin’s depiction of evolution as resulting from the gradual accumulation of countless infinitesimally minute variations.

Jeffrey Schwartz, 1999. *Sudden Origins: Fossils, Genes, and the Emergence of Species*, p. 3.

Paradoxically, the problem for Darwinism is not that there is too little evolution in the fossil record but that there is too much – not in the gaps, in the absences of any record leading up to the first appearance of a major group, but in the lineages once they become visible. The theory

predicts a series of infinitesimally minute variations, linking simple organisms to complex ones in a single tree of life.

Gradualness is of the essence. In the context of the fight against creationism, gradualism is more or less synonymous with evolution itself. If you throw out gradualness you throw out the very thing that makes evolution more plausible than creation.

Richard Dawkins, 1985. *Nature* 316:683.

What we get, however, are discontinuities, the sudden appearance of novelties, long periods of stasis.

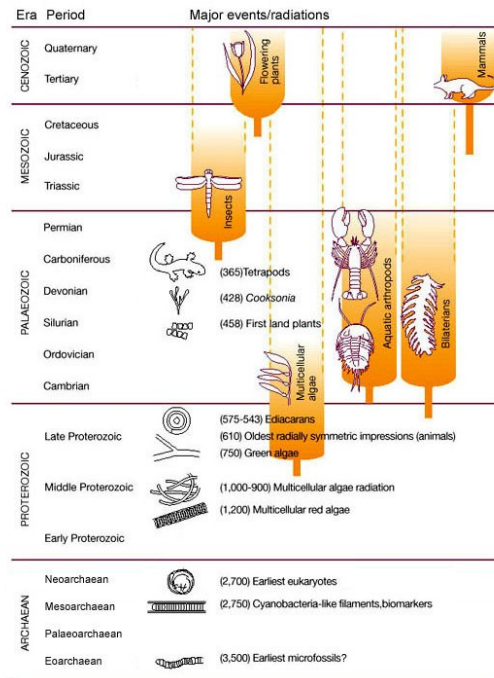


Figure 6. Major events/radiations in the fossil record.  
Source: *Nature* 409:1103.

Progressive increase in knowledge of the fossil record over the past hundred years emphasizes how wrong Darwin was in extrapolating the pattern of long-term evolution from that observed within populations and species. ...

Biologists have long struggled with the conceptual gap between the small-scale modifications that can be seen over the short time scale of human study and major changes in structure and ways of life over millions and tens of millions of years. Palaeontologists in particular have found it difficult to accept that the slow, continuous, and progressive changes postulated by Darwin can adequately explain the major reorganizations that have occurred between dominant groups of plants and animals. Can changes in individual characters, such as the relative frequency of genes for light and dark wing colour in moths adapting to industrial pollution, simply be multiplied over time to account for the origin of moths and butterflies within insects, the origin of insects from primitive arthropods, or the origin of arthropods from among primitive multicellular organisms? How can we explain the gradual evolution of entirely new structures, like the wings of bats, birds, and butterflies, when the function of a partially evolved wing is almost impossible to conceive?

What we observe today by way of evolution is of a relatively trivial kind: varying lengths of beak in Galapagos finches, thicker shells in periwinkles, size and colour variation in North American house sparrows, and so on. The times when more radical transformation took place are more distant, as when the Euphasmatodea became experts in camouflage, some turning into stick insects and others into leaf insects; as when sharks turned into rays, some with electric organs, or lizards turned into snakes, some with heat sensors, or tortoises turned into turtles, some with global positioning systems. Such novelties are problematic for Darwin's theory, and the greater the transformation, the more problematic, for the intricacy of their engineering shows that the evolutionary pathway could not have been random and *ad hoc*, it had to be pre-programmed. A society that has learned to design and make such devices itself should be well able to appreciate the point.

## References

- Caporale, L., 2004. Genomes don't play dice, *New Scientist* issue 2437, pp 42-45.
- Freeland, S. J., Knight, R. D., Landweber, L. F. & Hurst, L. D., 2000. Early fixation of an optimal genetic code, *Molecular Biology and Evolution* 17:511-18.
- Gómez-Valero, L., Rocha, E. P. C., Latorre, A. & Silva, F. J., 2007. Reconstructing the ancestor of *Mycobacterium leprae*: the dynamics of gene loss and genome reduction, *Genome Research* 17:1178-85.
- Konstantinidis, K. T. & Tiedje, J. M., 2005. Genomic insights that advance the species definition for prokaryotes, *PNAS* 102:2567-72.
- Mira, A., Ochman, H. & Moran, N. A., 2001. Deletional bias and the evolution of bacterial genomes, *Trends in Genetics* 17:589-96.
- Nilsson, A. I. *et al.*, 2005. Bacterial genome size reduction by experimental evolution, *PNAS* 102:12112-16.
- Olson, M., 1999. When less is more: gene loss as an engine of evolutionary change, *American Journal of Human Genetics* 64:18-23.
- Pennisi, E. 2007. Human genetic variation, *Science* 318:1842-43.
- Ranea, J. A., Grant, A., Thornton, J. M. & Orengo, C. A., 2005. Microeconomic principles explain an optimal genome size in bacteria, *Trends in Genetics* 21:21-25.
- Shubin, N. H. & Dahn, R. D., 2004. Lost and found, *Nature* 428:703-04.
- Weinreich, D. M., Delaney, N. F., DePristo, M. A. & Hartl, D. L., 2006. Darwinian evolution can follow only very few mutational paths to fitter proteins, *Science* 312:111-14.
- Whiting, M. F., Bradler, S. & Maxwell, T., 2003. Loss and recovery of wings in stick insects, *Nature* 421:264-67.
- Whiting, M. F. & Whiting, A. S., 2004. Is wing recurrence really impossible: a reply to Trueman *et al.*, *Systematic Entomology* 29:1-3.