

Astonishing DNA complexity demolishes neo-Darwinism

Alex Williams

The traditional understanding of DNA has recently been transformed beyond recognition. DNA does not, as we thought, carry a linear, one-dimensional, one-way, sequential code—like the lines of letters and words on this page. And the 97% in humans that does not carry protein-coding genes is not, as many people thought, fossilized ‘junk’ left over from our evolutionary ancestors. DNA information is overlapping-multi-layered and multi-dimensional; it reads both backwards and forwards; and the ‘junk’ is *far more* functional than the protein code, so there is *no* fossilized history of evolution. No human engineer has ever even imagined, let alone designed an information storage device anything like it. Moreover, the vast majority of its content is *meta-information*—information about how to use information. Meta-information cannot arise by chance because it only makes sense in context of the information it relates to. Finally, 95% of its functional information shows no sign of having been naturally selected; on the contrary, it is rapidly degenerating! That means Darwin was wrong—natural selection of natural variation does not explain the variety of life on Earth. The best explanation is what the Bible tells us: we were created—as evidenced by the marvels of DNA—but then we fell and now endure the curse of ‘bondage to decay’ by mutations.

Neo-Darwinian history of life

The neo-Darwinian theory of evolution says that all life on Earth arose from a common ancestor via random mutations to genes that survived natural selection. Organisms with beneficial mutations produced more offspring, those with deleterious mutations produced less (or no) offspring. Some beneficial mutations provided new adaptations to new or changed environments, thereby producing new kinds of organisms.

In order to get *extra* new information that was needed to build more complex organisms, existing genes must have been duplicated and then mutated into something useful.¹ This error-ridden process was not very efficient and it left behind in our genomes lots of ‘genetic fossil junk’:²

- mutations that were neutral and not selected out,
- duplicated genes that didn’t make it,
- functional genes that mutated beyond their usefulness.

As a result, our [evolutionary] history is woven into the fabric of modern [life] inscribed in its coded characters.³ This code, of course, is on the DNA molecule in the form of a long string of letters consisting of four base molecules, commonly represented by the initials T, A, G and C. The code was thought to be written in a straightforward manner, like the letters and words on this page. Neo-Darwinian molecular taxonomists routinely use this ‘junk DNA’ as a ‘molecular clock’—a silent record of mutations that have been undisturbed by natural selection for millions of years because it does not do anything. They have constructed elaborate evolutionary histories for all different kinds of life from it. How wrong this has proved to be!

The ENCODE Project

When the Human Genome Project published its first draft of the human genome in 2003, they already knew certain things in advance. These included:

- Coding segments (genes that coded for proteins) were a minor component of the total amount of DNA in each cell. It was embarrassing to find that we have only about as many genes as mice (about 25,000) which constitute only about 3% of our entire genome. The remaining 97% was of largely unknown function (probably the ‘junk’ referred to above).
 - Genes were known to be functional segments of DNA (exons) interspersed with non-functional segments (introns) of unknown purpose. When the gene is copied (transcribed into RNA) and then translated into protein the introns are spliced out and the exons are joined up to produce the functional protein-producing gene.
 - Copying (transcription) of the gene began at a specially marked START position, and ended at a special STOP sign.
 - Gene switches (the molecules involved are collectively called *transcription factors*) were located on the chromosome adjacent to the START end of the gene.
 - Transcription proceeds one way, from the START end to the STOP end.
 - Genes were scattered throughout the chromosomes, somewhat like beads on a string, although some areas were gene-rich and others gene-poor.
 - DNA is a double helix molecule, somewhat like a coiled zipper. Each strand of the DNA zipper is the complement of the other—as on a clothing zipper, one side has a lump that fits into a cavity on the other strand. Only one side is called the ‘sense’ strand, and the complementary strand is called the ‘anti-sense’ strand. Protein production usually only comes from copying the sense strand. The anti-sense strand provides a template for copying the sense strand in a way that a photographic negative is used to produce a positive print. Some exceptions to this rule were known (in some cases, anti-sense strands were used to make protein).
- This whole structure of understanding was turned on

its head by a project called ENCODE that recently reported an intensive study of the transcripts (copies of RNA produced from the DNA) of just 1% of the human genome.^{4,5} Their findings include the following inferences:

- About 93% of the genome is transcribed (not 3%, as expected). Further study with more wide-ranging methods may raise this figure to 100%. Because much energy and coordination is required for transcription this means that probably the whole genome is used by the cell and there is no such thing as ‘junk DNA’.
- Exons are not gene-specific but are modules that can be joined to many different RNA transcripts. One exon (i.e. a protein-making portion of one gene) can be used in combination with up to 33 different genes located on as many as 14 different chromosomes. This means that one exon can specify one part shared in common by many different proteins.
- There is no ‘beads on a string’ linear arrangement of genes, but rather an interleaved structure of overlapping segments, with typically five, seven or more transcripts coming from just one segment of code.
- Not just one strand, but *both* strands (sense and anti-sense) of the DNA are *fully* transcribed.
- Transcription proceeds not just one way but both *backwards* and *forwards*.
- Transcription factors can be tens or hundreds of thousands of base-pairs away from the gene that they control, and even on different chromosomes.
- There is not just one START site, but many, in each particular gene region.
- There is not just one transcription triggering (switching) system for each region, but many.

The authors concluded:

‘An interleaved genomic organization poses important mechanistic challenges for the cell. One involves the [use of] the same DNA molecules for multiple functions. The overlap of functionally important sequence motifs must be resolved in time and space for this organization to work properly. Another challenge is the need to compartmentalize RNA or mask RNAs that could potentially form long double-stranded regions, to prevent RNA-RNA interactions that could prompt apoptosis [programmed cell death].’

The problem of using the same code to produce many different functional transcripts means that DNA cannot be endlessly mutable, as neo-Darwinists assume. Most mutations are deleterious, so mutations in such a complex structure would quickly destroy *many* functions at once.

Their concern for the safety of so many RNA molecules in such a small space is also well founded. RNA is a long single-strand molecule not unlike a long piece of sticky-tape—it will stick to any nearby surface, including itself! Unless properly coordinated, it will quickly tie itself up into a sticky mess.

These results are so astonishing, so shocking, that it is going to take an awful lot more work to untangle what is really going on in cells.

Functional junk?

The ENCODE project did confirm that genes still form the primary information needed by the cell—the protein-producing code—even though much greater complexity has now been uncovered. Genes found in the ENCODE project differ only about 2% from the existing catalogue.

The astonishing discovery of multiple overlapping transcripts *in every part of the DNA* was amazing in itself, but the *extent* of the overlaps are *huge* compared to the size of a typical gene. On average, the transcripts are 10 to 50 times the size of a typical gene region, overlapping on both sides. And as many as 20% of transcripts range up to *more than 100 times* the size of a typical gene region. This would be like photocopying a page in a book and having to get information from 10, 50 or even 100 other pages in order to use the information on that one page.

The non-protein-coding regions (previously thought to be junk) are now called *untranslated* regions (UTRs) because while they are *transcribed* into RNA, they are not *translated* into protein. Not only has the ENCODE project elevated UTRs out of the ‘junk’ category, but it now appears that they are *far more active* than the translated regions (the genes), as measured by the number of DNA bases appearing in RNA transcripts. Genic regions are transcribed on average in *five* different overlapping and interleaved ways, while UTRs are transcribed on average in *seven* different overlapping and interleaved ways. Since there are about 33 times as many bases in UTRs than in genic regions, that makes the ‘junk’ about *50 times more active* than the genes.

Transcription activity can best be predicted by just one factor—the way that the DNA is packaged into chromosomes. The DNA is coiled around protein globules called histones, then coiled again into a rope-like structure, then super-coiled in two stages around scaffold proteins to produce the thick chromosomes that we see under the microscope (the resulting DNA/protein complex is called *chromatin*). This suggests that DNA information normally exists in a form similar to a closed book—all the coiling prevents the coded information from coming into contact with the translation machinery. When the cell wants some information it opens a particular page, ‘photocopies’ the information, then closes the book again. Recent other work⁶ shows that this is physically accomplished as follows:

- The chromosomes in each cell are stored in the membrane-bound nucleus. The nuclear membrane has about 2,000 pores in it, through which molecules need ‘permission’ to pass in and out. The required chromosome is brought near to one of these nuclear pores.
- The section of DNA to be transcribed is placed in front of the pore.
- The supercoil is unwound to expose the transcription region.

- The histone coils are twisted to expose the required copying site.
- The double-helix of the DNA is unzipped to expose the coded information.
- The DNA is grasped into a loop by the enzymes that do the copying, and this loop is copied onto an RNA transcript. The transcript is then checked for accuracy (and is corrected or degraded and recycled if it is faulty). Accurate RNA transcripts are then specially tagged for export through the pore and are carried to wherever they are needed in the cell.
- The ‘book’ of DNA information is then closed by a reversal of the coiling process and movement of the chromosome away from the nuclear pore region.

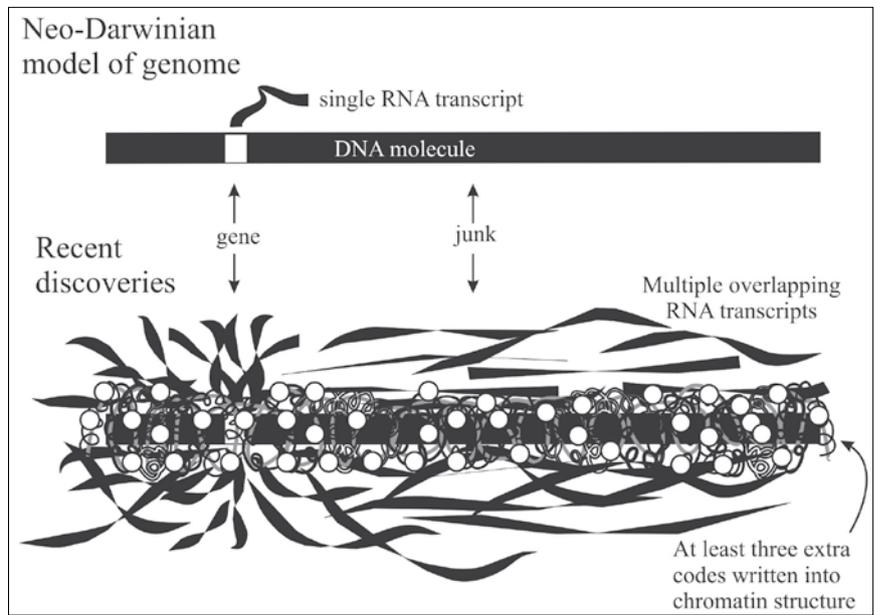
This astonishing discovery that the so-called ‘junk’ regions are *far more* functionally active than the gene regions suggests that probably none of the human genome is inactive junk. Junk is, by definition, useless (or at least, presently unused). But UTRs are being actively used right now. That means they are *not* fossils of bygone evolutionary ages—they are being used right now because they are *needed* right now! If other animals have similar DNA sequences then it means they have similar *needs* that we do. This is sound logic based upon observable biology—as opposed to the fanciful mutational suppositions of neo-Darwinism.⁷

The molecular taxonomists, who have been drawing up evolutionary histories (‘phylogenies’) for nearly every kind of life, are going to have to undo all their years of ‘junk DNA’-based historical reconstructions and wait for the full implications to emerge before they try again. One of the supposedly ‘knock-down’ arguments that humans have a common ancestor with chimpanzees is shared ‘non-functional’ DNA coding. That argument is now out the window.

Multiple Codes

A major outcome of the studies so far is that there are multiple information codes operating in living cells. The protein code is the simplest, and has been studied for half a century. But a number of other codes are now known, at least by inference.

Cell memory code. DNA is a very long, thin molecule. If you unwound the DNA from just one human cell it would be about 2 metres long! To squash this into a tiny cell nucleus, the DNA is wound up in four separate layers of *chromatin* structure (as described earlier). The first level of this chromatin structure carries a ‘histone code’ that contains information about the cell’s history (i.e. it is a cell



Astonishing complexity of DNA. When the genetic code was first discovered, it was thought that only protein information was coded in gene regions. Genes make up only about 3% of the human genome. Francis Crick described the remaining 97% as ‘junk’. But recent discoveries show that so much information is packed into, on and around the DNA molecule that it is the most complex and sophisticated information storage system ever seen by mankind. No one ever imagined such a thing before, and we are still trying to understand the nature and depth of its information content.

memory).^{8,9} The DNA is coiled twice around a group of 8 histone molecules, and a 9th histone pins this structure into place to form what is called a nucleosome. These nucleosomes can carry various chemical modifications that either allow, or prevent, the expression of the DNA wrapped around them. Every time a cell divides into two new cells, its DNA double-helix splits into two single strands, which then each produce a new double-strand. But nucleosomes are not duplicated like the DNA-strands. Rather, they are distributed between either one or the other of the two new DNA double strands, and the empty spaces are filled by new nucleosomes. Cell division is therefore an opportunity for changes in the nucleosomal composition of a specific DNA region. Changes can also happen during the lifetime of a cell due to chemical reactions allowing inter-conversions between the different nucleosome types. The memory effect of these changes can be that a latent capacity that was dormant comes to life, or, conversely, a previously active capacity shuts down.

Differentiation code. In humans, there are about 300 different cell types in our bodies that make up the different tissue types (nerves, blood, muscle, liver, spleen, eyes etc). All of these cells contain the same DNA, so how does each cell know how to become a nerve cell rather than a blood cell? The required information is written in code down the *side* of the DNA double-helix in the form of different molecules attached to the nucleotides that form the ‘rungs’ in the ‘ladder’ of the helix.¹⁰ This code silences developmental genes in embryonic stem cells, but preserves their potential to become activated during embryogenesis.

The embryo itself is largely defined by its DNA sequence, but its subsequent development can be altered in response to lineage-specific transcriptional programs and environmental cues, and is epigenetically maintained.¹¹

Replication Code. The replication code was discovered by addressing the question of how cells maintain their normal metabolic activity (which continually uses the DNA as source information) when it comes time for cell division. The key problem is that a large proportion of the whole genome is required for the normal operation of the cell—probably at least 50% in unspecialized body cells and up to 70–80% in complex liver and brain cells—and, of course, the *whole genome* is required during replication. This creates a *huge logistic problem*—how to avoid clashes between the transcription machinery (which needs to continually copy information for ongoing use in the cell) and the replication machinery (which needs to unzip the whole of the DNA double-helix and replicate a ‘zipped’ copy back onto each of the separated strands).

The cell’s solution to this logistics nightmare is truly astonishing.¹² Replication does not begin at any one point, but at *thousands* of different points. But of these thousands of potential start points, only a subset are used in any one cell cycle—different subsets are used at different times and places. A full understanding is yet to emerge because the system is so complex; however, some progress has been made:

- The large set of potential replication start sites is not essential, but optional. In early embryogenesis, for example, before any transcription begins, the whole genome replicates numerous times without any reference to the special set of potential start sites.
- The pattern of replication in the late embryo and adult is tissue-specific. This suggests that cells in a particular tissue cooperate by coordinating replication so that while part of the DNA in one cell is being replicated, the corresponding part in a neighbouring cell is being transcribed. Transcripts can thus be shared so that normal functions can be maintained throughout the tissue while different parts of the DNA are being replicated.
- DNA that is transcribed early in the cell division cycle is also replicated in the early stage (but the transcription and replication machines are carefully kept apart). The early transcribed DNA is that which is needed most often in cell function. The correlation between transcription and replication in this early phase allows the cell to minimize the ‘downtime’ in transcription of the most urgent supplies while replication takes place.
- There is a ‘pecking order’ of control. Preparation for replication may take place at thousands of different locations, but once replication does begin at a particular site, it suppresses replication at nearby sites so that only one copy of the DNA is made. If transcription happens to occur nearby, replication is suppressed until transcription is completed. This clearly demonstrates

that keeping the cell alive and functioning properly takes precedence over cell division.

- There is a built-in error correction system called the ‘cell-cycle checkpoints’. If replication proceeds without any problems, correction is not needed. However, if too many replication events occur at once the potential for conflict between transcription and regulation increases, and/or it may indicate that some replicators have stalled because of errors. Once the threshold number is exceeded, the checkpoint system is activated, the whole process is slowed down, and errors are corrected. If too much damage occurs, the daughter cells will be mutant, or the cell’s self-destruct mechanism (the apoptosome) will be activated to dismantle the cell and recycle its components.
- An obvious benefit of the pattern of replication initiation being never the same from one cell division to the next is that it prevents accumulation of any errors that are not corrected.

The exact location of the replication code is yet to be pinpointed, but because it involves transcription factors gaining access to transcription sites, and this is known to be controlled by chromatin structure, then the code itself is probably written into the chromatin structure.

Undiscovered codes?

Given that we now have at least four known codes, it seems reasonable to infer that at least three other major activities in cells have a, yet undiscovered, coded basis:

Regulatory code(s). At least some, and perhaps all, of the untranslated regions are involved in gene regulation in one form or another. According to Kirschner and Gerhart’s *facilitated variation* theory,¹³ regulatory information is organized into modules that they liken to Lego® blocks. That is, they have strong internal integrity (hard to break), but they are easily pulled apart (during meiosis) and rearranged into new combinations (at fertilization). This built-in capacity for variation, they claim, is essential for life to persist (i.e. survive stress in any one generation) and evolve (down the generations). It therefore seems reasonable to expect to find some code associated with module structure, the rules by which rearrangements can occur, and the constraints that must apply in order to maintain normal metabolic functions in the face of rearranged regulatory circuits.

Transcription code. The most common activity associated with DNA is transcription. But how does a transcription signal know which version of a transcript is required? For any given gene, there are numerous different transcript-starting sites, a number of different overlap options, and numerous different signal molecules that can trigger a transcription request, but only one transcription machine can operate on a given segment of DNA at any one time.

Nerve code. Nerve cells carry information internally via electrical impulses, but then communicate with one another by converting electrical impulses into neurotransmitter

chemicals that then diffuse across the gap (synapse) between them. There must be a code involved in conversion from electrical to chemical transcription information.

Meta-information: an impossible conundrum for evolution

The astonishing complexity of the dynamic information storage capacity of the DNA/chromosome system is, in itself, a marvel of engineering design. Such a magnificent solution to such a monster logistics problem could surely only come from a Master Designer. But the *nature* of the majority of this information poses an impossible conundrum for neo-Darwinists.

Proteins are the work-horse molecules of biology. But protein-coding genes make up only a *tiny proportion* of all the information that we have been describing above. The vast majority of information in the human genome is not primary code for proteins, but *meta-information*—information about information—the instructions that a cell needs for using the proteins to make, maintain and reproduce functional human beings.

Neo-Darwinists say that all this information arose by random mutations, but this is not possible. Random events are, by definition, independent of one another. But *meta-information* is, by definition, *totally dependent* upon the information to which it relates. It would be quite nonsensical to take the cooking instructions for making a cake and apply them to the assembly of, say, a child's plastic toy (if nothing else, the baking stage would reduce the toy to a mangled mess). Cake-cooking instructions only have meaning when applied to cake-making ingredients. So too, the logistics solution to the cell division problem is

only relevant to the problem of cell division. If we applied the logistics solution to the problem of mate attraction via pheromones (scent) in moths it would not work. All the vast amount of *meta-information* in the human genome only has meaning when applied to the problem of *using* the human genome to make, maintain and reproduce human beings.

Even if we granted that the first biological information came into existence by a random process in an 'RNA-world' scenario, the *meta-information* needed to use that information could not possibly come into existence by the same random (independent) process because meta-information is *inextricably dependent* upon the information that it relates to.

There is thus no possible random (mutation) solution to this conundrum. Can natural selection save the day? No. There are at least 100 (and probably many more) bits of *meta-information* in the human genome for every one bit of primary (protein-coding gene) information. An organism that has to manufacture, maintain and drag around with it a mountain of useless mutations while waiting for a chance correlation of relevance to occur so that something useful can happen, is an organism that natural selection is going to *select against*, not favour! Moreover, an organism that can survive long enough to accumulate a mountain of useless mutations is an organism that does not need useless mutations—it must *already have all the information it needs to survive!*

What kind organism already has all the information it needs to survive? There is only one answer—an organism that was created in the beginning *with* all that it needs to survive.

What kind of information is this?

These results present us with a spectacle never before encountered in science—an information structure so complex that it defies description. How can we possibly understand it?

I believe there are at least two things that we can reasonably conclude about it at this time. First, it is most decidedly *not* the one-dimensional linear sequence of characters that neo-Darwinists need to provide the endlessly mutable source of universal evolution. While it is certainly variable, its extraordinarily complex structure cannot possibly be endlessly mutable because a certain amount of invariance is required to maintain complex structure, otherwise it quickly degrades into error catastrophe. The experimentally verified universally deleterious nature of mutations supports this conclusion.

The second thing we can conclude is that according to Kirschner and Gerhart's theory of *facilitated variation*,¹⁴ DNA contains regulatory-based modules which they liken to Lego® blocks. That is, they have very strong internal coherence

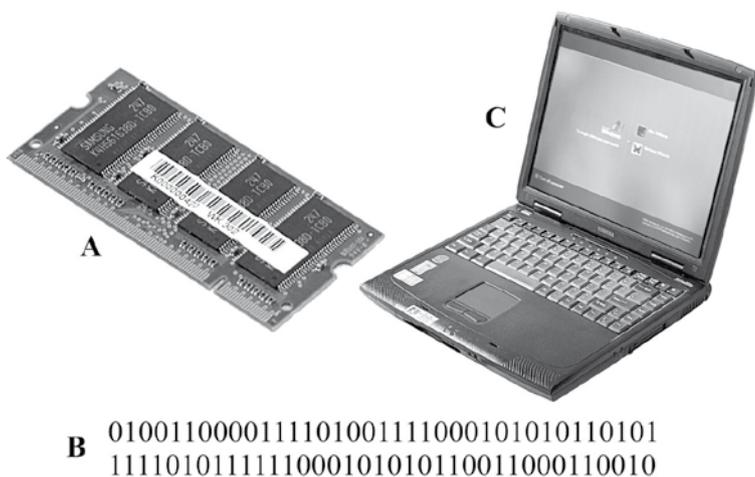


Image by Alex Williams

Meta information. The computer memory chip (A) can hold over 2 billion bits of information in binary code (B) but it requires a computer (C) with its operating system and application software (meta-information) to do anything useful with the information on the chip. Likewise, the protein information coded on the genes in DNA needs the meta-information in the 'junk' regions together with the machinery of the cell to do anything useful with the protein code.

Panel A. Computer binary code

Two symbols, read 8 at a time, and translated into English letter.

A B C D E F
 10000110000101000011100010010001011000110

Panel B. Biological quaternary code

Four symbols, read 3 at a time, and translated into amino acids:

Stop Alanine Serine Leucine Glutamic acid Serine
 TAGCCCGCTAAGTCTGAACTGTGCGAATACAGC

Image by Alex Williams

Information about information. The most basic meta-information needed to use coded information is instructions on how to read the code. In a computer, the binary code (Panel A) is divided into 'bytes' made up of 8 'bits' (0,1), which are then translated into English via the ASCII code. In protein-coding genes (Panel B), the nucleotides (T,A,G,C) are read in triplets, and these triplets are translated into an amino acid sequence via the genetic code.

and integrity (difficult to break) but are easily pulled apart (during meiosis) and reassembled (during fertilization) to produce a built-in capacity for variation. To understand it properly, we need to locate the Lego® blocks and the boundaries between them.

Natural selection—irrelevant!

The most surprising result of the ENCODE project, according to its authors, is that 95% of the functional transcripts (genic and UTR transcripts with at least one known function) show *no sign of selection pressure* (i.e. they are not noticeably conserved and are mutating at the average rate). Why were they surprised? Because man is supposed to have evolved from ape-like ancestors via mutation and natural selection. But if 95% of the human functional information shows no sign of natural selection then it means that natural selection has not been a significant contributor to our ancestry.¹⁵

While this result surprised the neo-Darwinists, it is perfectly in line with current research in human genetics. In his ground-breaking exposé of current knowledge, *Genetic Entropy & The Mystery of the Genome*,¹⁶ geneticist Dr John Sanford of Cornell University showed that deleterious mutations are accumulating at an alarming rate in the human population and that both natural selection and even the worst possible nightmare scenario of eugenics is powerless to stop it. This is because the vast majority are deleterious single nucleotide mutations that have a miniscule effect on fitness so natural selection cannot detect them amongst the 'noise' and thus is unable to delete them. Also, there is so much

other information contributing to fitness, and life always has many different ways of achieving its goals. Natural selection is also powerless to delete numerous mutations simultaneously, because their effects on fitness are complex, interactive and often mutually interfering.

Natural selection only works in simple cases that are easy to explain in textbooks. One organism with one (big) defect will fail to reproduce. Alternately, one big strong male will out-compete his rivals and fertilize more females, thus improving the overall genetic well-being of the species. These kinds of scenarios only have a minor impact on the human population. Most of us are just mediocre, but most of us do find partners and procreate.

If the average number of deleterious mutations per person per generation is much less than

1, then we have some hope that natural selection might favour the strong over the weak and keep our species genetically healthy. But in fact, the average number of deleterious mutations per person per generation is *very much greater than 1*—probably *at least 100* per person per generation, likely about 300 and perhaps much more. This means that *everyone is a mutant* many times over and natural selection is powerless to stop it because deleting the weakest (whatever that might mean) will do nothing to prevent everyone else reproducing, resulting in an inexorable degeneration of the *whole* human population. Even the most horrible eugenics program could not stop it. This problem caused one author to write a paper entitled 'Why are we not dead 100 times over?'

Let's now put this information together with Haldane's dilemma.¹⁷ Famous geneticist J.B.S. Haldane calculated that it would take about 300 generations for a favourable mutation to become fixed in a population (every member having a double copy of it). He calculated that in the approximately 6 million years since our supposed hominid ancestor split from the chimpanzee line, only about 1000 (<2000 according to ReMine¹⁸) such mutations could become fixed. This is certainly not nearly enough to turn an ape into a human. But most importantly, we now know that there are about 125 million single nucleotide differences between humans and chimps, resulting from about 40 million mutational events. This means that somewhere between 39,998,000 and 124,998,000 deleterious changes have occurred since the split with our common ancestor. That means we have *degenerated* from chimps, which makes a mockery of the whole mutation/selection theory of origin.

Conclusion

The discovery that virtually all our DNA is functional right now demolishes the neo-Darwinian argument that it contains mostly junk which constitutes a unique fossilized history of our genetic evolution. The most logical explanation of shared DNA between different kinds of organisms is now shared function, not shared ancestry.

The discovery that DNA contains astonishingly complex information structures, beyond our capacity to even imagine, let alone comprehend, means that neo-Darwinism's total dependence upon random mutations in linear code as a source of new information is in error. We would expect random mutations to extremely complex information structures to be deleterious, and this is exactly the universal result of many years of genetic research. Kirschner and Gerhart's theory that natural variation is largely the result of random rearrangements of specially structured gene modules¹⁴ is a far more reasonable explanation.

The discovery that the vast majority of the information stored in DNA is not primary protein-coding information but secondary meta-information, demolishes the neo-Darwinian argument that it arose by some random (independent) process. Meta-information is inextricably *dependent* upon the information it refers to so an independent origin is impossible.

The discovery that natural selection has been an insignificant factor in our genetic history demolishes Darwin's theory that all life on Earth arose from a common ancestor by means of the natural selection of natural variation. This discovery is also completely consistent with the genetic evidence that the human genome is degenerating at an alarming rate.

Neo-Darwinian theory was a mirage built from ideology upon a few general facts (natural selection, genes and mutations). But now that we have glimpsed just some of the vast molecular detail of how life *actually* works, the mirage has evaporated.

References

1. This theory makes numerous predictions that are all falsified by molecular biology data. The lack of any alternative source of the required new information thus constitutes a fatal weakness in neo-Darwinian theory. See Bergman, J., Does gene duplication provide the engine for evolution? *Journal of Creation* **20**(1):99–104, 2006; <www.creationontheweb.com/images/pdfs/tj/j20_1/j20_1_99-104.pdf>.
2. Carroll, S., *The Making of the Fittest: DNA and the Ultimate Forensic Evidence of Evolution*, Norton, New York, 2006.
3. Dawkins, R., *The Ancestor's Tale: A Pilgrimage to the Dawn of Evolution*, Houghton Mifflin, Boston, MA, 2004, p.20.
4. Birney, E. *et al.*, Identification and analysis of functional elements in 1% of the human genome by the ENCODE pilot project, *Nature* **447**: 799–816, 2007.
5. Kapranov, P., Willingham, A.T. and Gingeras, T.R., Genome-wide transcription and the implications for genomic organization, *Nature Reviews Genetics* **8**: 413–423, 2007.
6. Akhtar, A. and Gasser, S.M., The nuclear envelope and transcriptional control, *Nature Reviews Genetics* **8**:507–517, 2007.
7. Mutations have been intensively researched in medical laboratories over many years now and almost all of them are deleterious. The only exceptions appear to be those that disable an existing mechanism such that some specialized result is beneficial in some specialized conditions (e.g. sickle-cell anemia (a serious disease) reduces the severity of malaria in malaria-prone regions by killing the malaria parasite in the sickled cells).
8. Latent memory of cells comes to life, 17 May 2007, <www.physorg.com/news98623491.html>.
9. Segal, E., Fondufe-Mittendorf, Y., Chen, L., Thåström, A., Field, Y., Moore, I.K., Wang, J.-P.Z. and Widom, J., A genomic code for nucleosome positioning, *Nature* **442**(7104):772–778, 17 August 2006.
10. Mikkelsen, T.S. *et al.*, Genome-wide maps of chromatin state in pluripotent and lineage-committed cells, *Nature* **448**:553–560, 2007.
11. Bernstein, B.E. *et al.*, A bivalent chromatin structure marks key developmental genes in embryonic stem cells, *Cell* **125**:315–326, 2006.
12. Aladjem, M.I., Replication in context: dynamic regulation of DNA replication patterns in metazoans, *Nature Reviews Genetics* **8**:588–60, 2007.
13. Kirschner, M.W. and Gerhart, J.C., *The Plausibility of Life: Resolving Darwin's Dilemma*, Yale University Press, New Haven, CT, 2005.
14. Kirschner and Gerhart, ref. 13. See also, Williams, A.R., Facilitated Variation: A new paradigm emerges in biology, *Journal of Creation*, in press.
15. Natural selection will have played some part in our ancestry because grossly deleterious mutations will have been weeded out, and during times of famine, plague and war, populations are usually depleted of some genetic components more than others (e.g. young soldiers killed in war; the young and the old dying in famine, sickle-cell anemia alleviating the severity of malaria).
16. Sanford, J.C., *Genetic Entropy & The Mystery of the Genome*, Elim Publishing, New York, 2005.
17. Batten, B., Haldane's dilemma has not been solved, *Journal of Creation* **19**(1):20–21, 2005; <www.creationontheweb.com/content/view/4473>.
18. ReMine, W., Haldane's Dilemma, <saintpaulscience.com/Haldane.htm>, 3 August 2007.

Alex Williams received a B.Sc. in botany from the University of New England, an M.Sc.(Hons.) in radioecology from Macquarie University, and is an elected member of the Australian Institute of Biology. He has diplomas in Christian studies from Tabor College and Bible College of South Australia (in missiology), and a Licentiate in Theology (with distinction) from the Australian College of Theology. During 20 years in environmental research, he became the Australian representative to the United Nations in his field. He then spent seven years in mission work and is now an honorary botanist at the Western Australian Herbarium in Perth. He has published in many different areas of science and co-authored *Dismantling the Big Bang*.
