One Engineer's Take on Darwinian Evolution

New Science Challenges Old Ideas

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Change Log

Version	Date	Page	Changes
V1.22	6 January 2021		First version that received some circulation. Numerous changes to readability.
V1.23	26 February 2021	8	Point 5. Added newly discovered synonymous mutation causes cancer.
		24	Codon Degeneracy. Added - changes to protein folding can break function. Added reference to new science paper - see below.
		24	Qualified statement that codon variants are not degenerate.
		24	Added - changed protein function causes kidney cancer. Added reference to science paper
V1.24	14 March 2021		A number of changes to grammar and semantics
		5	Added mention of fruit fly's taste preference with reference Added mention of fruit fly's eye coating with reference Added mention of fruit fly's brain capability with references
		6	New section "*Two Decades That Shook Evolutionary Biology*"
		20	Reference to C Value Paradox removed
		21	Clarified the description of the transcriptome
		28	Moved the section "*Lack of accepted theories*" to appendix 4
		29	Moved the section "*Is the eye badly designed*" to appendix 5
V1.25	14 April 2021		PDF errors fixed
V1.26	20 April 2021	7	Two new references added re discoveries of greater differences between man and evolutionary ancestors
		19	Logical argument expanded
V1.27	24 April 2021	5	Reference to fruit fly flight endurance Reference to new complexity added
V1.28	3 May 2021	13	New sub-section "*High Rates of Speciation*"
		19	Brief discussion of multiverse theory added in "Conclusion - The Qualitative Nature of Evidence"
		26	Fallacy of probability arguments explained in "*Possible number of positions for 10 new base-pairs*"
			Many other improvements throughout document
V1.29	22 October 2021		Division into three main sections to clarify document flow - see 'contents'
			Added Fruit Fly recognise each other The Fruit Fly and the Appeal to Complexity
			Added The bat and the speed of sound in Another Facet of Instinct
			Added Observed Evolution - A New Artery in Human Forearm
			Added Observed Evolution - Elephants Without Tusks
			Added Appendix 6: Is the Neck Badly Designed?
			Added Appendix 7: The Data Inside Us
			Added A Summary of Main Points in Conclusion - The Qualitative Nature of Evidence - Finding Answers
			Expansion of discussion of The Multiverse
V1.30	29 October 2021		Minor change to text error on page 11

Foreword

This document is intended for those with some aptitude in science and engineering. Its aim is to highlight new science relating to the evolution-creation debate. Some of this is game-changing and many are unaware of this.

Views Change in Surprising Ways

I'm a product designer and occasional inventor, working with electronics hardware, firmware and software. My work has taken me into a number of other areas. I've loved science and engineering since a child, and the evolution-creation debate has always fascinated me. I like the idea of a loving creator, but have been drawn by the weight of opinion in the science community. However, things are not always as they appear, especially as portrayed in the popular media.

The testing of predictions is the measuring rod for good science. Many predictions and beliefs have been put forward with great certainty in evolutionary biology. Until recently, many of these were not verifiable, but advances in science have allowed tests to be made using new methods and equipment.

As a result, the landscape has changed in cellular and molecular biology. A number of core beliefs in evolutionary biology have been brought into question, especially in the last decade.

This document looks at the changes in genomics and cellular biology that have brought this situation about. Despite having considerable impact on evolution science, the popular media has not caught up, leaving many unaware of these changes.

Evolutionary biologists can be at war, sometimes with other sciences, most of the time with creationists. There's been an ongoing battle for hearts and minds and, as they say: "Truth is the first casualty of war." This is not to suggest that lies are being told, but truth can be kept a little quiet. When discoveries advance the cause, they are well publicised. When they detract from the cause, the publicity can be less enthusiastic.

The introduction on the next page illustrates how biologists since Darwin have underestimated complexity.

The next sections show how some famous books look outdated in the light of new science. Appendix 1: A Brief History of Time and Unexpected Events in Genomics on page 20, expands on this and highlights new science that changes some fundamental understanding. The section is somewhat technical in order to explain the nature and importance of fascinating new discoveries.

The section on page Target not found!, "Target not found!", is easier to read. It covers some important problems relating to evolution theory, and shows that some things written about probability in this area are questionable.

I've always wondered how scientists can explain the evolution of instinct. The section "No Instinct - No Life" on page 12, shows that they admit to having great difficulty. It shows that this is quite a substantial problem.

It's not unusual for scientists to disagree, but lack of agreement is high in evolution science. This has been the case for decades concerning the balance of genetic drift versus natural selection as driver of evolution[2]. This is core to the theory and highlights that evolution science is more fuzzy than many realise. Despite statements to the contrary, this disagreement has not gone away[3].

In addition, accepted theories are missing in a number of key areas, indicating that evidence can be ambiguous or lacking. See page 13, "Appendix 4: A Lack of Accepted Theories".

A growing list of scientists wish to express their reluctance to accept darwinian evolution[4]. These are not just creationists[4a]. Also, a small minority of scientists have proposed that earth was seeded with life from space, because they don't accept that life could have appeared spontaneously in an earth environment[4b].

Section One

The Complexities

The Fruit Fly and the Appeal to Complexity

The 'appeal to complexity' goes like this; "It couldn't have evolved because it's so complex (or amazing)."

Intuition tells many that accidental design of complex systems is unlikely. Some in science and philosophy consider this reasoning to have little merit, but this can be an error of judgment.

The Perception of Complexity

Biological systems that were once perceived as simple, then later as complex, then extremely complex, are now known to be mega-complex. The search for the complete picture goes on, and new discoveries keep rolling in.

For example, understanding has come a long way since Darwin's day. Pasteur conducted his famous experiment disproving spontaneous generation of life around the same time that Darwin's *On the Origin of Species* was published. Until Pasteur, many thought that flies could emerge from maggots that spontaneously came into existence from rotting meat, so complexity was far from understood in that context. A fly might have been thought of by many as little more than an annoying glob of goo.

Scientists are still trying to get a handle on complexity, and much research has centred around the fly, especially the fruit fly[6][7].

Prof. Michael Dickinson, University of Washington, speaks of the fruit fly's agility. He states: - *"They process this [visual] information so quickly, as anyone who has tried to swat a fly will have noticed, and they can fly like an ace at birth. It's like putting a newborn baby in the cockpit of a fighter aircraft and it knowing what to do."*[8]

Drosophila melanogaster, the 2.5 mm fruit fly, has many sets of muscles for flight and steering[9][10]. It has a heart with a circulation system that can go into reverse flow[11]. It has a navigation system [12], short and long term memory[13] and many behavioural patterns. It has an immune system, and a complex gastrointestinal system[14]. Flight control includes sensor arrays on its wings that return stress measurements reminiscent of advanced avionics, and appendages (halteres) that act as gyroscopes for flight stability[15][16]. It has even been found to suffer from trauma[17]. It has preferences for food flavour and texture[17a]. It sleeps[18]. It can fly more than 12km[17g].

A coating on the eye of the fruit fly has been found to be antimicrobial, anti-reflective and self cleaning. Scientists are currently studying it with a view to copying it for use in medicine and other areas[17b]. They can reliably recognise other fruit flies using their simple eyes and sophisticated brain processing[17h].

In two separate studies, part of the fruit fly brain has been modelled and found to be more efficient than existing computer algorithms for artificial intelligence[17c][17d]. It's thought that machine learning techniques for autonomous vehicles may benefit by copying aspects of the fruit fly's ability to learn and act[17e].

Darwin may have been surprised.

This demonstrates that perception of complexity in biological systems has changed dramatically. In light of this, the question arises, 'Can Darwin's theory still account for this?'

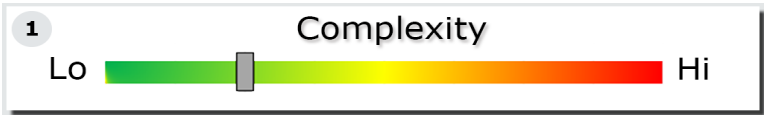
A Simple Thought Experiment in Complexity

'Appeal to Complexity' is viewed by some as a form of 'Argument from Ignorance'.

For example, Dr Bo Bennett (no relation!) on his website logicallyfallacious.com, applies this to someone believing that the eye couldn't evolve. (For more on the eye, see "Appendix 5: Is the Eye Badly Designed?" on page 29)

As more complexity is found in cellular function, does an appeal-to-complexity argument become less ignorant?

As a simple thought experiment, imagine a sliding scale where the pointer indicates the level of complexity.



During time intervals in which many species appeared (e.g. Cambrian Explosion or periods following extinction events), the number of steps that can be attributed to evolution would have to be constrained by the interval time. Each step would require time to emerge, to be selected, and to spread in the population. A limited number of steps could take place within those intervals.

If the pointer keeps moving to the Hi end of the scale, then more steps would have been required to achieve that complexity. At some point, too many steps are proposed to fit the time scale. This, of course, is not rocket science.

At what point on the scale would this happen? Evolutionary biologists have little or no idea. They have an incomplete picture of how things work, so are unable to create a detailed model of speciation. Bearing this in mind, can it be said that only one side argues from ignorance? In reality, we just give it our best shot.

If discovery of new complexity continues, then at some point, the intuitive statement "It couldn't have evolved because it's so complex (or amazing)" will inescapably become correct.

The pointer has been moving inexorably towards the Hi end of the scale, with more complexity coming to light, often every week or less. For example a recent news release has the title: *"Insect evolution was more complex than previously thought."*[17f] Many new discoveries infer the same. So, the question is: Has that point been passed?

There's a Bigger Problem

The knowledge of complexity in organisms has changed very dramatically in the last few years. Evolutionary biologists have had to come to terms with the fact that many more steps of evolution would have been required to evolve new species and each step would require many more mutations. Some think that time would not allow this[18c], but the problem is much greater than this, ase discussed in No Instinct - No Life on page 12.

The next sections and Appendix 1: A Brief History of Time and Unexpected Events in Genomics draw attention to changes in understanding and their impact on evolution theory.

Two Decades That Shook Evolutionary Biology

In the first two decades of the 21st century, new light was shone on the inner workings of the cell and the structure and function of DNA. For more detail, see Appendix 1: A Brief History of Time and Unexpected Events in Genomics.

We are now in another era when it comes to cellular biology and genetics. Functional genomics has become a huge area and involves computer processing of a vastly greater 'data set' that was thought to exist in our DNA when Richard Dawkins wrote his popular books.

To illustrate this: -

> Human DNA has approximately 3.2 billion base pairs. When comparing this with computer code, each of these base pairs is a data bit, but unlike computer's binary code where a bit has two states, DNA's bits are quaternary, having four states. This means that, at face value, our DNA is equivalent to 6.4 Gbits, or 800 MBytes of data.
>
> 800 MBytes of code seems a very small amount of data to describe our metabolism and the location and form of all of our body parts. This amount of data would barely fill a CDROM.
>
> The point is, around thirty years after Dawkins wrote *The Selfish Gene*, evolutionary biologists became agitated when they were told that they were wrong in thinking that 98 percent of this data was junk.
>
> By pursuing the idea that evolution would leave a legacy of cast-offs where mutations could accumulate to produce new genes, they were sure that only 2 percent or less of this data describes the human body, including the brain that has been described as the most complex object in the universe. Two percent would be about 32 megabytes.
>
> This is about the amount of data for a small uncompressed digital image. Some may think this didn't make much sense, but hindsight is a wonderful thing.

So, it's not unreasonable to suggest that books written before this time describing the process of evolution should be questioned. The ongoing ENCODE project continues to expose more function in DNA.
See ENCODE Project Phase One 2013. See, also, Appendix 7: The Data Inside Us.

Genes Not so Simple

In 20th century thinking, genes were thought to be the main functional elements controlling our metabolism by virtue of their composition, location, and the effect they had on the function of other genes by a process called epistasis.

This was a bit like believing that the mechanical parts of a manufacturing robot are all that's required to make complex components for a car. It's true that those parts, like our genes, do the work, but only when controlled by complex electronics that's been programmed for each meticulous operation.

To illustrate this old thinking: -

> In Richard Dawkins' book Climbing Mount Improbable, he describes single genes having major effects on structures like the eye. He points out that the same 'eye making' gene is present in many organisms and is therefore an evidence of relatively simple processes of evolution occurring once in an ancestral organism. Eyes could then benefit many other species that subsequently evolved. He also refers to convergent evolution, where parallel evolutionary processes produce similar results in other species lines.

The thinking now: -

1. Genes do affect each other by epistasis, but they are just the 'machinery' and are controlled by complex networks elsewhere in DNA. For every gene, there are numerous sections of control DNA. This is in the area that evolutionary biologists said was full of evolutionary cast-offs - junk DNA. These are precision mechanisms.
   Genes are not just switched on and off. Very many cellular processes are finely regulated by control sequences that sense our body state and finely adjust gene operation (regulation) to compensate. A well known example is sensing of glucose levels by an array of sensors that send electrical signals to the brain and the fine regulation of insulin production[18d]. Man has tried hard to engineer closed loop systems that attain similar quality of control.
2. As stated by Dawkins, similar genes are found in many organisms, but these are now known to be the 'lego bricks of life'. It's the control networks that really differentiate organisms. So, similarities between organisms are not as previously stated. This in itself has a major impact on evolutionary beliefs.
3. Many genes are unique with no similarity to those in 'ancestral' species. In earlier 20th century thinking this was not thought possible. An article in Royal Society Publishing said in 2015; *"The origin of novel protein-coding genes de novo was once considered so improbable as to be impossible. In less than a decade, and especially in the last five years, this view has been overturned by extensive evidence from diverse eukaryotic lineages."* [29]
4. Seemingly simple attributes such as eye and hair colour are now found to be very much more complex, with many more genes involved than previously thought, along with their regulatory control networks[18e][18f].

Genes Not so Selfish

A strong argument for Richard Dawkins' popular "selfish genes" concept was the existence of transposons. These are also called 'jumping genes' because of their ability to move around our genome, some inserting and some duplicating themselves in our DNA: - natural genetic engineering.

Transposons were previously thought to be harmful parasitic genes. Because of their dynamic nature, transposons can be harmful, but so can any gene when its function is damaged by mutation, or when its regulatory control network is damaged. Several developmental human diseases along with some cancers are now known to be caused by mutations in these regulatory areas[18g].

Transposons were once thought to jump more randomly, but are now known to jump in a highly ordered way in a healthy individual, and specific mechanisms to control them have been identified. New understanding of their function has been appearing recently[18h][18i].

Transposons are not thought of in the same way now and have been found to be involved in important functions such as the development of the brain, the mammary gland, the embryo, and robustness in food crops[18j][18k][18m][18n]. The current trend indicates that many more functions will become apparent in the near future.
Times change and science moves on.

The Changes, Book by Book

In the 21st century, especially from around 2010 onward, research using new methods and equipment has brought revised understanding to biology and overturned long held beliefs. Some of this is summarised below. See Appendix 1: A Brief History of Time and Unexpected Events in Genomics, for more detail and many more references.

On the Origin of Species - Charles Darwin

Published 1859

After discussions with a number of academics, Darwin concluded that the variety of finches found in the Galapagos, and nowhere else, pointed to the transmutation of species. This was based on an idea that had been around for a while, and was later popularised by Darwin's famous book. Darwin's finches became an icon of evolution theory.

After DNA's structure was discovered, and mutation by DNA replication error was known, it was believed that the biological mechanism for the evolution of Darwin's finches was understood. This proved to be incorrect.

New methods and equipment enabled discoveries that surprised many. Evolutionary biologists fought against the idea that inheritance was not necessarily based on change to DNA code sequence. It's now accepted by most academics that epigenetic changes can be inherited, even through several generations.

Simply put, epigenetics refers to molecular tags placed on DNA or its histone support structure that affect the function of genes. In a process that's not understood, external environmental changes can trigger the placement of epigenetic tags. This can cause changes that can even make some species look like new species.

In 2014, a comparison of Darwin's finches revealed that there was little correlation between genetic difference and the various 'species' of the finch[38]. There was a strong correlation between epigenetic changes and these finch pseudo-species (see Epigenetics and Heredity).

Darwin was right in thinking that some factor had brought about change. He didn't realise that this was a built-in mechanism that operated when food supply changed. This mechanism didn't randomly place epigenetic tags on the more than a billion base pairs of the finch's DNA, but did so with a precision that aided survival. The changes are reversible. Had he known this, might history have been different? (see also Speciation and Despeciation).

The Naked Ape - Desmond Morris

Published 1969

Ethologist and Zoologist, Desmond Morris, wrote about the origin and evolution of human behaviour. Some find his views a little off-centre, in the same ilk as some of Sigmund Freud's theories.

His arguments were based on the belief that humans are closely related to primates.

For many years, it was thought that human and chimpanzee DNA differed by less than 2%. There were four main errors at this time: -

1. Contamination of samples with human DNA; common in early days of DNA sequencing.
2. Biased computer models that humanised chimp DNA.
3. The view that our genes were the only functional part of DNA, when there was in fact a complex network of gene regulation in areas previously thought to be junk. See ENCODE Project Phase One 2013.
4. The belief that all genes were inherited from lower species. Many unique genes have since been discovered in man and across many species with no apparent evolutionary ancestry. These are referred to as de-novo genes.

A new comparison in 2018 revealed that the human-chimpanzee difference was, at least, a much larger 17%. The related research paper was critical of previous comparisons.[35]

In 2021, a number of 'Homo' species were found to have a much smaller brain size than previously thought, meaning more ape like than human[35a]. The human scream was thought to have evolved as a cognitive reaction to fear, but has now been found to more effectively communicate excitement[35b]. New evidence keeps undermining old ideas.

There's an equally large problem for those who study animal behaviour, such as Desmond Morris and Richard Dawkins. The evolutionary origin of genetically coded instinct is a mystery. The fact that it closely matches the function of specialised morphological features is a problem of epic proportions. See Climbing Mount Improbable on page 9, and the section No Instinct - No Life on page 12 for an explanation of this, along with references.

Man has a brain prewired to recognise faces, and the framework is in place for other skills. This is instinct, passed on in genetic code. Another example of amazing 'prewired' instinctive behaviour in man was highlighted in a paper published recently (October 2020).[18a]

The Blind Watchmaker - Richard Dawkins

Published 1986

Richard Dawkins wrote a number of best selling books at a time when the understanding of gene expression (how genes build and maintain life) was extremely simplistic.

Since this book was published, the evolutionary biology community has rejected, ridiculed and fought against new findings that include the heredity of epigenetic characteristics, and the debunking of the idea that 98% of our genome is evolutionary baggage - junk DNA. Statements such as the one below appeared in the press in response to the ENCODE project . . .

> *"This is not the work of scientists. This is the work of a group of badly trained technicians."*[18b]

Since then, many of ENCODE's analytical findings have been subjected to test and have been found true. Most of this has happened within the last few years, mainly since 2010, and these areas of new science are now generally accepted.

'The Blind Watchmaker' refers to an analogy made in a 19th century creationist book. The watch was compared with living organisms. If the watch required a maker, then so did living organisms. Dawkins argued otherwise.

There are some very powerful reasons why Dawkins reasoning should be questioned: -

1. When this book was written, it was believed that genes were activated and deactivated by epistasis. This means that the presence and position of genes determined complex metabolic processes. One gene could affect another. Epistasis is known to take place, but the idea that it was the basis of metabolism was incorrect.

   Since the Human Genome Project and its successor, the ENCODE project, it's known that the control of genes is performed by complex regulatory networks with many small molecules modulating genes in a very precise manner, rather than simply switching them on and off. The control elements for this are in DNA previously labelled 'junk' - see ENCODE Project Phase One 2013.

   So, now the picture is very different

   Dawkins' description of the eye being formed by simple changes was soon outdated as gene function was discovered to be much more complex. In addition, knowledge of the eye has increased - see next page and Appendix 5: Is the Eye Badly Designed? on page 29.

2. The description of natural selection given by Dawkins and other evolutionary biologists can be misleading. In principle, if natural selection is given the correct mutations within a sensible time frame, then complexity can increase. However, even if each change benefits survival, that complexity is very unlikely to be coordinated, with new characteristics matching and complementing each other on the large scale necessary for life. This becomes clear when the logic of this is considered - see section two The Probabilities.

3. A 'sensible time frame' as mentioned above, is a problem. There would have to have been many millions of evolutionary transitions for all species to evolve. As an exercise in simple mathematics, try arriving at a rough estimate of the time taken for one transition.

   To do this for one species, you would, at least, need to know the average time between generations, the population size, the average number of mutations passed to each new generation, the size of the genome, and the average number of mutations to achieve a transition.

   You would also need a knowledge of how mutations spread in the population, but as highlighted by the results below, precision is not needed in this calculation.

   I tried this some years ago, and even if the number of mutations per generation is exaggerated by orders of magnitude, the result indicates an excessively long time period for each transition.

   I'm no expert, but an Oxford team published a research paper in November 2020 that arrives at a similar conclusion, stating: -

   " . . . we demonstrate that expected evolutionary transition times likely exceed the lifetime of Earth, perhaps by many orders of magnitude." [18c]

4. Following on from the last point, the chance of a sequence of mutations coming up with anything sensible to build new complexity is normally overstated. Creationists sometimes compare an evolutionary step to winning the lottery, perhaps many times in succession.

   This doesn't come close. See Appendix 2; "Possible number of positions for 10 new base-pairs in a genome of 2 Gbp".

5. Classic evolution theory states that silent mutations build up until a new characteristic appears. This was thought to happen because there were spare codes in DNA that would not alter function. This was dealt a series of blows recently when these 'spare' codes were found to have detailed function in protein production. It has recently been discovered that one of these mutations breaks the function of a protein and can cause kidney cancer. The idea of complex functional codes within code is also very difficult to explain in an evolutionary context.
   See "Codon Degeneracy" in Appendix 1 for more detail and references.

Climbing Mount Improbable - Richard Dawkins

Published 1996

Richard Dawkins writes very well, as many have commented. His writing is engaging, and his love of nature shines through. However, science has moved on considerably since this book was written. Some of the changes are summarised in the previous Blind Watchmaker box and the section Two Decades That Shook Evolutionary Biology.

As a result of changes to understanding, a major evolutionary step is now known to be vastly more complex than portrayed in Richard Dawkins' book, with many more functional areas of DNA required.

Genes don't act alone or just in cooperation with others as in Dawkins' book. Genes are now known to have complex regulatory sense and control mechanisms for the tens of thousands of different molecules that are the basis of our metabolism. Control sequences of DNA code send molecules to finely regulate production of these molecules by specific genes. The genes are sometimes at a distance of many tens of thousands of base-pairs. The 'ease' of producing new characteristics is not as Dawkins stated.

Dawkins uses the illustration of a rock outcrop in Hawaii that looks like one of the faces on Mount Rushmore, but only when viewed from a certain angle. Detail is absent when looking from other angles.

On the other hand, a face on Mount Rushmore is recognisable from many angles, and when observed more closely, obvious signs of chiselling, indicating design, can be discerned. It's a good illustration.

He says that we see things in nature that look like designs, but only when viewed from a certain angle. He refers to these as *designoids* - meaning, having an appearance similar to design

Here are two points from the book where he applies this illustration: -

The Eye is a favourite of creationists who say that its complexity indicates design. When you look from a certain angle it looks like design, says Dawkins, but its features indicate its primitive origins.

As Dawkins says, the retina seems to be back to front so that light has to travel through other cells to reach it. He says, it might have been a better design if it were to be the other way round, but evolution has done a pretty good job with it anyway. It's probably that way because it has evolved from more primitive forms.

In reality, it is now known that the retina benefits from better blood flow in this orientation. This is necessary for function. It's also known that the cells that light travels through to reach the retina form a very impressive 'fibre optic' array with uniquely minimised internal parts and a fascinating graduated index of refraction. The larger end of the cell allows greater light capture and the smaller end delivers light to retinal cells. These light pipe glia cells also perform other functions.

This is described in more detail in the section, Appendix 5: Is the Eye Badly Designed?, along with other areas of complexity and sophistication in the eye that have been discovered.

As with Mount Rushmore when you 'alter your viewing position' you see what some would regard as design, from any angle.

In reality, could the truth be opposite to Dawkins' assertion? Does the eye only look like an evolved organ when viewed from a certain angle? So, is it actually an *evolvoid* rather than a *designoid*?

Flight is a complex attribute of organisms from the fruit fly to the eagle. Dawkins discusses how it developed from jumping and gliding. There's a big problem that he doesn't address, and yet it falls into his area of expertise; animal behaviour. It's a problem that evolutionary biologists don't understand, and it clearly causes concern to some who study this area.

The ability of any organism to fly can be divided into two highly complementary areas; physiology and a closely matched skill-set.

The physics of bird and insect flight has been challenging to understand, but much has been discovered about its complexity in recent years thanks to high speed photography and computer simulations. Fruit flies are now often observed whilst 'strapped' into mini flight simulators whilst brain neuron activity is monitored.

Seemingly authoritative sources still state that birds learn to fly and fledglings can be seen flapping as part of the process. We've probably all got used to hearing this, but when you stop to think about it, it's an illogical cliché. Do we really think that birds can learn the complexities of flight by fluttering their wings at the edge of the nest?

As early as 1875 (Spalding, Nature Magazine) it was known that birds fly instinctively if their wings are constrained and then released at the time they would normally fly. Maturation is the factor, not learning. More recent studies have confirmed this[56]. Birds have extensive code in their DNA that defines their flight apparatus. They also have extensive code in DNA that defines the sophisticated behavioural skills that complement their flight apparatus. So do fruit flies.

A naturalistic explanation for these finely tuned matching codes has not been put forward, and yet this is something that's found throughout the natural world many millions of times. If we were to compare them with the code matching used for encryption in an IT context, how many data bits would be required? Nobody knows, but it might be reasonable to assume that it's very many.

To see the import of this, see "No Instinct - No Life" on page 12, which also has interesting quotations from ethologists. Also "Possible number of positions for 10 new base-pairs" on page 26.

Section Two

The Probabilities

Probability and Evolution

For a moment, forget all the complexity discussed in the previous section. What if Darwinians had been correct in their predictions of vastly lower complexity? Could Darwinian evolution really bring about so many species?

A Google search for "evolution probability" reveals many web pages written by evolutionists and creationists. Creationists say the chance of any biological complexity evolving is very close to zero. Evolutionists point to the frequency of mutations and the power of natural selection which, they say, makes evolution non-random.

There are some deal-breakers that both sides tend to miss.

Non Random Nature Exaggerated

Evolution theory has an elegant simplicity. Survival of the fittest is a truism and natural selection is inherently directional, but there are factors that make natural selection much more random than you may have been told.

a. It relies on events that are totally random; DNA replication error being the main one. If the right mutations don't happen, there will be no natural selection, no evolution.
b. Beneficial mutations would be very rare, but no matter how beneficial they are, many that complement each other would be difficult to achieve. The type of beneficial change would be totally random, so why would they match each other? Evolution of new complex species depends on many millions of these.
c. If mutations occur that cause greater fitness for survival, event variables mean that many are unlikely to be selected. These would include predation, lack of food supply, a failure to inherit changed genes, and other factors. So, however directional natural selection is, it's still dependent on many chance elements.

Evolution Probability is Inversely Proportional to Ordered Complexity

To expand on point b, there's complexity, and there's complexity. In the autumn, the wind can create a scattering of leaves in a park landscape that has great complexity, but it's the groundsman that introduces ordered complexity by sweeping them into neat piles. Evolution is different in that natural selection would often filter out changes that are not beneficial, but is there any rule that would prevent piles of randomised beneficial changes that have no relationship being scattered across life's landscape? If so, what would that rule be?

> When planning a dinner party, would you ask your on-line grocery store to send a random selection of food items to use as ingredients? Would it help if you asked them to select only nutritious food; food items with benefit? It may not work. You'd end up with a selection of beneficial foods that don't complement each other. So, why should randomised evolutionary benefits build sophisticated structure? Being of benefit is not sufficient, and mutation-selection would have a vast array of non-matching benefits; a larger selection than your grocery store.
>
> Another problem is that the store only manages to fulfil one in every ten billion orders.

Complexity is very different from ordered complexity. When evolving ordered complexity, the probability of success decreases as ordered complexity increases.

Evolution is said to have no specific direction, so if mutation-selection causes beneficial mutations to accumulate, is it reasonable to think that these benefits dovetail to complement each other? Natural selection may be kind to a sequence of benefits that create order, but would random mutation be kind enough to provide them? Why should it? At the start, there is latitude to evolve in any direction that benefits the organism, but as a structure builds, subsequent additions need to be more and more specialised in order to fit in. Evolution doesn't do custom jobs.

Life functions by means of very many complex code sequences that complement each other and work in harmony. Scientists observe an incredible degree of pattern matching in genetic code. Millions of species have many complex organs that work together with complementary function. Is pattern matching something that can be reasonably expected from mutation-selection, such as the genetic code defining the eye's data compression that matches the optic nerve, along with the brain's decoding and virtualisation? There's a need to consider this more closely.

Engineers can't get away from the need to reverse engineer in order to fathom out how things work. If you can't make sense of things in reverse, then the chances are it wasn't put together in the way that you thought.

Whales are said to have evolved from a small land mammal. That mammal might have 2 billion base-pairs in its genome. Now to build additional complexity. How many new base pairs for an evolutionary step; ten? Hundreds or many thousands are more likely because gene expression is a complex process involving many complementary sequences of DNA code. A lot more matching code is required to animate and bring this to life, as highlighted on the next page.

Changing only ten base pairs in this genome presents more possible combinations than atoms in the observable universe (see Appendix 2 on page 26). A minute subset of these would be useful and viable. The rest would be nonsense or irrelevant. It seems that the reverse engineered whale would need an additional half billion base pairs in the right places. The whale has very many specialised features related to aquatic life at great depth and pressure.

The problem is simple. If complexity of an evolving feature were to increase, it would become more specialised and the subset of useful mutations that match becomes progressively smaller. Evolution now has to come up with very specific complementary sequences. Consider this illustration: -

> A clever man is invited to give a speech. He randomly chooses a subject.
> The audience is young and impressionable. Many are wondering which path to pursue in life.
> His speech is on the psychological effects of high speed travel.
> When he ends his speech, there is loud applause. Inspired, they go and pursue careers involving high speed.
> The speaker was lucky, but random worked.
>
> Ten years later, he is invited to give a speech to the same audience. He randomly chooses a subject.
> They are now an audience of racing drivers, jet pilots, bobsleigh champions, and stunt men.
> His speech relates to the joys of stamp collecting.
> When he ends his speech, everyone is asleep.

The moral of the story is that when things get specialised, random doesn't work so well, even if it brings complexity with benefits. In theory, stamp collecting would help them relax after a day's racing, but it's just not a good fit for these

thrill-seekers. So even if you ignore the absence of evolutionary wiggle room taken away by changed understanding of junk DNA and codon degeneracy, old school reasoning was always highly optimistic. This is only half of the problem.

No Instinct - No Life

Complementary codes in DNA becomes a much bigger issue when taking instinct into account. Consider an organism nearer the other end of the scale, *lasius niger,* the black ant, suited to urban and country life. For a small insect, it has a complex social life. Epigenetic mechanisms cause the ant to have different body layouts; same DNA, different forms, including queen, males, workers and larvae[50a].

The worker is wingless and around 4 mm in length. The male is of similar length but has wings with protruding flight muscles. The queen is twice the length, initially has wings which are removed and flight muscles absorbed. The queen lives much longer than her minions, even decades, probably owing to DNA repair hormones.

Epigenetic mechanisms put tags on the ant's DNA, causing different body layouts. Even though they all have the same DNA, their body morphologies are very specialised. (See Epigenetics and Heredity)

The big deal is this. Epigenetic mechanisms simultaneously put tags on exactly the right places on the ant's DNA that cause the brain of this small organism to have specialised instinctive behaviour that matches its specialised shape. Workers, males and queens have different behavioural characteristics. Behaviours include walking, flight, mating, feeding and nursing larvae, foraging for food and identifying it, laying down chemical guide paths to food sources, burrowing and maintaining the nest, and a lot more. Unlike the workers, the queen knows how be the centre of attention.

These behaviours are embedded in DNA. They are the equivalent of the computer code that makes a lifeless robot perform tasks. Robotic engineers have spent many years writing and rewriting code just to make robots move and walk in a reasonable manner. If the ant didn't have this extensive coding, it would just be lifeless anty matter.

> *"We don't have a general theory for the mechanics of instinct as we do for learning, and this is something that has troubled me for a very long time,"* says University of Illinois entomologist Gene Robinson[51].

If epigenetic programming switched the queen's instinctive behaviour to that of a male, there would be no ants. As evolutionary biologists tell us, evolution doesn't have a direction. So, how is it that instinct is closely matched to morphology? And how is it that instincts of the different forms of ant complement each other beautifully to create highly organised collectives? This is a paradox of epic proportions that's not understood in evolutionary circles.

To appreciate the import of this, there's a need to bear in mind the complexity of this code matching in light of two factors mentioned on the previous page:-

1) There's a vast number of combinations for only a handful of new single-point mutations. (See Appendix 2)
2) Gene expression is now known to involve much more than genes alone. (See ENCODE Project Phase One 2013)

According to Wikipedia, it's estimated that there are around 1.5 million species of beetle and 5.5 million species of insect. When you extend this problem across all these species, you have a cavernous hole in the middle of evolution theory, with no resolution in sight. Did evolution randomly experiment with complex 'firmware' until the ant colony functioned correctly? The leaf cutter ant takes things to another level with youngsters riding shotgun to protect workers from predators. Then, of course, there's the termite.

In higher animals there is obviously a learning framework. This in itself is instinctive behaviour. Despite learning being a big factor, even humans have instinctive frameworks for speech, facial recognition, and other behaviour.

> *"An animal mind is not born as an empty canvas: Bottlenose dolphins know how to swim and honey bees know how to dance without ever having learned these skills. Little is known about how animals acquire the instincts that enable such innate behavior. Instincts are widely held to be ancestral to learned behavior. Some have been elegantly analyzed at the cellular and molecular levels, but general principles do not exist."*
> Science Magazine, April 2017[52].

There has been discussion for decades on the issue of nature vs nurture. An experiment showed that the characteristic burrowing of two species of mice is an instinctive trait[53]. Again, this means that it is encoded in their DNA in the same way as robotics firmware is encoded in electronic memory.

Scientists say some behaviour that looks like instinct in higher organisms is learned behaviour, sometimes acquired and passed on in herds and other collectives. Evidence supports the idea that some animals have ability to learn complex behaviour. In the case of bird flight, this is a misconception.

An Internet search will turn up many examples of incorrect statements in this regard. On the other hand, one of the foremost reference works, Campbell's Biology, said concerning the learning of flight:-

> *"innate behaviors are not always due to learning. For instance, behavior may change because of ongoing developmental changes in neuromuscular systems, a process called maturation. We commonly speak of birds "learning" to fly, and you may have seen fledgling birds awkwardly fluttering about as if they were practicing. However, young birds have been experimentally reared in restrictive devices so that they could never flap their wings until an age when their normal kin were already flying. Such birds flew immediately and normally when released. Thus, the improvement must have resulted from neuromuscular maturation, not from learning."* [54]

The book Behaviour and Evolution explains that after pigeons were reared so as to restrain their wings, they flew immediately when released at a time when they would normally fly[55][56]. Flight is a feature of maturation, not learning. Many authoritative sources seem not to have caught up with this after many years.

The property of instinctive behaviour obviously extends across the animal kingdom; reptiles, fish, mammals and birds, etc., extinct and extant. The 'software library' of instinct across the natural world is immense. Scientists can spend a lifetime studying the behaviour of just a few species. Even plants have programmed behaviour.

All of these instincts correspond with the specialised physical characteristics of organisms in which they are programmed. Bearing in mind the random nature of evolution, how is this degree of complex code matching explained? This clearly troubles some, especially those closer to the subject of animal behaviour.

> *"It's very difficult for us to come to terms with just how much of our behavior is set in stone."*
> says ethologist Lars Chittka, Queen Mary University of London[51].

Why is it that instinct is so difficult to reconcile, compared with morphological features and cellular function, where incredible pattern matching is also seen? It's probably because we pigeon-hole instinct in different brain space, whereas the reality is that it's 'only' another sequence of matching code in our DNA that causes gene expression.

The psychology of this difference may relate to creepage. Cellular biology has been observed by scientists for several centuries. Wikipedia says this in the topic of Cell Theory:-
"The cell was first discovered by Robert Hooke in 1665 using a microscope. The first cell theory is credited to the work of Theodor Schwann and Matthias Jakob Schleiden in the 1830s."

Scientists have climbed a ladder of knowledge to reach the current understanding of cellular complexity. They have had time to take in gob-smacking new facts, gasp, then get comfortable before the next bombshell arrives. This is creepage.

Instinct isn't physically manifested in the same way, so the process of understanding has not suffered from creepage in a similar manner to morphology and cell function. Could there also be a little burying of heads in sand?

In addition, evolution debate can be divisive and intense. Battle lines have been set, and positions can become very entrenched. The battle for the 'hearts and minds' of the public is ongoing. This doesn't necessarily help any of us to maintain objectivity.

Darwin saw problems with the Cambrian Explosion. You may have been told this has been resolved - if so take a look at Appendix 4: A Lack of Accepted Theories. What would Darwin have thought if he had been told the full extent of cellular complexity, a problem that scientists are still trying to fathom, with its amazing cellular machines, motors and 'roadways', tens of thousands of proteins, and multiple layers of hidden code?

What would Darwin have thought if he had been told that it would require the correct code to accidentally slot into place for instinct to match physical characteristics, and that the possible number of combinations for this accidental code exceeded the number dust particles that make up all of the stars in the sky, and billions of times more.

In virtually every area of the living world, the more that scientists look, the more they find. This shows no sign of slowing down anytime soon.

Another Facet of Instinct

Instinct takes on much greater sophistication in 'higher' organisms. As mentioned in the section *The Fruit Fly and the Appeal to Complexity* on page 5, engineers are learning much by studying the flight control of the fruit fly, but flight control in the natural world can very get much more complex.

It would take a team of expert programmers many man-years to program the equivalent of a bat's echolocation and it's associated virtualisation and flight control, but if evolution is true then echolocation has evolved more than once in bats and has also evolved in oilbirds, cave swifts and cetaceans.

Recent findings indicate that previous thinking lacked appreciation of a very important design parameter.

Up until now, we've been aware of complex behavioural data being programmed in the DNA of every organism. Even slime mould can do things that has amazed scientists, but a fascinating study at Tel Aviv University[77c] found that bats have an environmental constant, the speed of sound in air, programmed into their brains; hence their DNA.

Bats pups raised in a helium-enriched atmosphere in which sound travels faster than usual were unable to accurately locate prey. Also, adult bats were unable to adjust when moved from a normal environment to the helium enriched environment.

In another study, bats were found to shape their mouths to effectively zoom-in their field of view[77d].

High Rates of Speciation

Another problem is the high rate of speciation found in the fossil record. The so called 'Cambrian Explosion' was a very short period in geological terms, in which most types of complex life came into existence. Darwin recognised this as a problem to his theory. Evolutionary paleontologists have minimised the significance of this by saying that it was not as previously thought and that the fossil record was incomplete. The thinking on this has now reverted.

> *"We now know that the sudden appearance of fossils in the Cambrian (541–485 million years ago) is real and not an artefact of an imperfect fossil record."*[63].
> *"Now, new research published in the Proceedings of the National Academy of Sciences (USA) is revealing that this explosion was far shorter than many experts had thought."* Natural History Museum 2019[64]

In addition, more and more complexity is being discovered and, as a result, greater humility is now evident in the related scientific communities.

Use of the term 'evolutionary arms race' has become a popular way of glossing over the problem of speciation.

The users of this expression are referring to periods of time when there was greater environmental stress owing to competition, predation and other factors. For example, if there are only slow moving predators, then there is less need for the prey to move more quickly, so natural selection is less likely to favour mutations that may help in this way. If mutation helps the predator to move more quickly, then selective pressure is greater for mutations that help the prey move more quickly.

If there's some small change to gene expression that does this, then it's possible that such changes could take place. The problem comes when the changes are more complex and sequential. The expression 'evolutionary arms race' is often used to explain events such as the development of the eye, which evolutionary paleontologists tell us happened several times in different species lines.

It's been known for some time that there are very many genes associated with the design of the eye, and the list has been getting longer. More recent findings tell us that each gene has associated control mechanisms that are affected by timing sequences, such as those that trigger growth of the hyaloid artery to nourish the developing fetal lens (the artery then withers in the tenth week of human development), and the levels of the tens of thousands of different molecules that form our metabolism.

As highlighted in this and the previous section, natural selection is not the only driver of evolutionary change. If it were, then there would indeed be rapid change, but these complex attributes require many very specific new sequences of DNA code and it's not a good idea to ignore the mathematical probabilities of this happening. See "Possible number of positions for 10 new base-pairs" for more detail on the probabilities.

An Oxford study suggests that evolutionary transitions would take longer than the time earth had existed[18c].

The next section shows the problems with the term 'speciation', and goes on to give examples of evolution in the real world that comply with the laws of probability.

Section Three

The Realities

Two Flavours of Evolution

It's said that evolution is all around us. If this means organisms with mutations, then it is, but is it the same as darwinian evolution that can bring about millions of new species?

Speciation and Despeciation

There is a problem with the definition of "Species" in that it is not as clear as many might think. A paper on the subject stated:-

> *"To complicate matters, for roughly the past half century, the issue of species delimitation has been confused by a problem involving the concept of species itself. The problem is that currently different subgroups of biologists advocate different and at least partially incompatible species concepts."*[78]

Biologists are keen to find examples of speciation that support evolution. These are often not clear cut.

One of the stronger definitions is "the isolation of groups that can no longer interbreed". Reproductive isolation does appear to occur in groups that are separated by geographical barriers or other causes, but these cases are often unstable, and reintegration can occur. 'Species' of fish are said to have come about through speciation, but then despeciated, challenging the definition of species again[79].

This indicates that a certain amount of genetic ebb and flow occurs, but within the boundaries of 'types' rather than the often vague boundaries of 'species'. There is no evidence for genetic change beyond the boundary of types, apart from an interpretation of the fossil record, which becomes ambiguous if it goes beyond the simple evidence of "I was here".

Geographically isolated sub-species of the African elephant can interbreed and reintegrate. On the other hand, it's been said that species divergence becomes greater over time. However, a recent paper in Nature highlighted the case of two species of ravens that merged after more than of 1.5 million years of separation[80].

Species of Madagascan songbirds that are thought to have diverged around 3.6 million years ago, are now thought to be reintegrating[81].

Speciation is also defined by the emergence of unique phenotypes. Darwin's finches looked good in this regard until it was discovered that, although they are distinct phenotypes, they are genetically the same species[38].

Authoritative sources can lag badly on these things. At time of writing, National Geographic website still states:-

> *"The finches are isolated from one another by the ocean. Over millions of years, each species of finch developed a unique beak that is especially adapted to the kinds of food it eats."*[82]

Other sources are up to date and go further, recognising that Darwin's finches represent a challenge to 'species' definition. An article in Discover Magazine was entitled: "Are Darwin's Finches One Species or Many?": -

> *"The textbooks are wrong, says ornithologist Robert Zink of the University of Minnesota's Bell Museum of Natural History. The ground finches may seem to be different species, at least with superficial comparison, but they're stuck in what he calls Sisyphean evolution. "Species kind of get started, but . . . they never make it to the top of the hill," Zink says.*
>
> *In a recent paper in Biological Reviews, Zink helps make the case. "None of these 'species' are distinct," he says. The various ground finches don't differ significantly in ways that usually differentiate bird species, such as plumage patterns or song. Unlike with discrete species, these features aren't stable and can vary over just a few generations, depending on weather and food availability. Sequences of their nuclear and mitochondrial DNA show little variation and none of the telltale signs that suggest distinct species."*[83]

Epigenetic mechanisms changed the morphology of the finches in the same way that the morphology of colony insects such ants, termites, and bees having queens, workers, drones, etc., with the same DNA is changed. Epigenetic tags have different life times. Some come and go to control metabolism. Others are multi-generational.

The vague speciation of finches has been compared to the case of the European herring and lesser black-backed gulls that, some say, form a ring species. They interbreed around the northern hemisphere through a series of connected hybrid 'species', but don't usually interbreed directly.

Many paleontologists recognise that speciation was often too rapid to be explained by classic darwinian change. A Wikipedia article on Speciation, describes punctuated equilibrium as an explanation:-

> *"Evolution can be extremely rapid, as shown in the creation of domesticated animals and plants in a very short geological space of time, spanning only a few tens of thousands of years. Maize (Zea mays), for instance, was created in Mexico in only a few thousand years, starting about 7,000 to 12,000 years ago."*

This reflects old ideas. Results of the ENCODE project make fast speciation look increasingly far-fetched.

In the case of humans, areas of the human genome seemed to appear very rapidly. 'Human Accelerated Regions' describes areas of our genome that bear no similarity to animals including chimps and other primates.

> *"Human accelerated regions (HARs) are DNA sequences that changed very little throughout mammalian evolution, but then experienced a burst of changes in humans since divergence from chimpanzees. This unexpected evolutionary signature is suggestive of deeply conserved function that was lost or changed on the human lineage*[84]*.*

The close link of humans to chimpanzees has since been discredited and was based on faulty data: -

> *"This bias has effectively "humanized" other ape genome assemblies, minimizing potential structural and transcript differences observed between the species."* [35]

To some, it increasingly looks like there was insufficient time for any major evolutionary transitions[18c].

The Real Nature of Evolution

The overwhelming evidence is that faulty DNA replication causes degeneration. Examples of point mutations are cancer, neurofibromatosis, sickle cell anaemia, tay-sachs disease and colour blindness. Evolutionists tell us that this comes within the bounds of evolution, because evolution doesn't only take things onwards and upwards.

Natural selection is a no-brainer. The fittest generally survive. Examples of mutation followed by natural selection can be found, including sickle cell anaemia, where a carrier of one copy of a specific faulty gene is more resistant to malaria. As a result, natural selection causes a higher incidence of the disease in countries where malaria is prevalent. The disease manifests itself when two copies of the gene are inherited.

Observed Evolution - Crickets in Hawaii

A fascinating example of mutation and natural selection occurred separately on two Hawaiian islands[85]. On both islands, the crickets lost the ability to chirp as a result of separate mutation events. Loss of chirping attracted fewer attacks from parasitic flies, and natural selection caused the mutation to spread across the populations.

A combination of physical components exists to enable the chirping. In addition, instinct is required in the male and female. Instinct is basically firmware, hard coded in DNA.

Losing just a part of any one of these components, instinctive or physical, could cause loss of chirping. On the other hand, evolving many complementary characteristics that combine to give the crickets their chirp is very different.

When errors in DNA replication take random pot-shots at the cricket's DNA sequences, the probability of the loss of the cricket's chirp by a small mutation in one component is relatively great. Randomisation destroys function.

The probability of gaining several components that complement each other to give the cricket its chirp is highly unlikely. The difference between gaining and losing chirping is huge, and would hardly involve just one gene. Several physical components would be required along with separate instinctive behaviour for male and female. Genes require additional resources in ncDNA ('junk' DNA), so many more genetic components are likely to be required than the physical parts that they describe, in addition to the instinctive pathways in the cricket's tiny brain.

Evolution is nature's ratchet mechanism that erodes complexity, and causes disease; a downwards process.

Observed Evolution - A New Artery in Human Forearm

Another example of this is the increasing incidence of the median artery in the human forearm since the late 19th century. Headlines have appeared in the science and popular press such as this: -

"More And More Humans Are Growing an Extra Artery, Showing We're Still Evolving"

This is quite misleading. The artery is present in all human babies and nourishes development of the hand. It normally regresses at around 8 weeks in the pregnancy. It's not the only artery to do this. The hyaloid artery is present in the developing fetus and nourishes the eye's lenses. This also normally ceases to function and disappears before birth. These are part of an amazing cascade of incredibly complex biological events that start with fertilisation and end with a fully formed human baby. When one of these events is lost, it's more accurate to use to term devolution rather than evolution. Changes such as these do not appear to bode well for humanity because what we see is a winding down of our DNA's functionality as more and more mutations are inherited.

Observed Evolution - Elephants Without Tusks

In October 2021, a study was published that led to this headline in Nature Magazine: -

"Ivory hunting drives evolution of tuskless elephants"[85b]

Again, this is quite misleading. Nature magazine is one of the foremost technical journals and is a highly respected source of information for main-stream science. So, a statement like this infers, with little doubt, that this is a case of Darwinian evolution. It certainly involved mutation and also natural selection.

The mutation was found to be to a gene in the X chromosome that is survivable by females, but not by males. It was also discovered that females with this mutation altered their diet, potentially threatening balance of the ecosystem. Poachers have been killing elephants with tusks and, in doing so, have made a rather sordid version of natural selection where mutated tuskless elephants are better suited for survival.

Evolutionary biologists tell us that degradation of the genome in this way is part of Darwinian evolution, but it's obvious that the balance of genomic change should be positive, otherwise new species will not be the dominant trait of evolution. It's clear that if you randomise DNA, the most complex coding system known to man, it will degrade, and reduction of complexity and a lack of new species will be the dominant trait.

In the past, it was thought that gaining a gene or two along with a bit of evolutionary tweaking could bring about large changes. Many readers of Richard Dawkins' books probably still believe this. Since then, the Human Genome Project, and the ENCODE project have changed the landscape, and the full import has not reached many people. A gene-centric view is still put forward on many websites, and few know that we are not closely related to chimps. Few know that new findings of the complexity of genes supports the view that evolution is destructive. See Codon Degeneracy for detail.

The many types of dog did not evolve from a common ancestor as a result of mutation. They were selectively bred for genetic makeup that expressed desired characteristics. An exception being the dachshund and other short legged breeds, where dwarfism was selectively bred. Other deformities have been selectively bred, and do not necessarily improve fitness for natural survival, but rather fitness for purchase.

When dogs were selectively bred, the gene pool became less diverse. How did it get so diverse in the first place? For darwinian evolution to be truly demonstrated, i.e. high rates of complex speciation, a massive expansion of many gene pools must be demonstrated. The same is true for other examples of selective breeding found in books on evolution, such as cabbage, which also demonstrates a method of diminishing the gene pool.

When it comes to complexity, evolution is subtractive, or disruptive, whereas darwinian evolution should be incredibly additive. Picture a small mammal evolving into whale, and evolution of every other species that has lived.

The fossil record merely says, "I was here." Any further conclusion needs to be backed up by additional evidence. See the section Appendix 4: A Lack of Accepted Theories on page 13.

Other Areas that Look Like Evolution

Many inbuilt mechanisms for adaptation, both genetic and epigenetic, have been observed.

When bacteria gain resistance to antibiotics, one main driver can be a complex mechanism to capture external DNA fragments using a tiny arm, a pilus, and splice them into their own DNA[86]. The little blighters use genetic engineering, a process copied by scientists. Some bacteria even have a membrane pump that can clear out poisons whilst they adapt[87]. These are pre-existing mechanisms for bringing about change. Inbuilt mechanisms for change point strongly at a-priori design.

Bacteria reproduce asexually which means that their offspring can receive changes by mutation that would often be diluted by sexual reproduction. Despite vast numbers of generations and high change rates, the changes are to metabolic pathways, not to the overall nature of the organism. This has been indicated when ancient bacteria are discovered. In July 2020 it was reported that bacteria were recovered from deep sea sediment that settled at the time of the dinosaurs, 100 million years ago. These appeared to be close to a stasis condition and also indicated stasis in the genetics of their species as some were genetically identifiable with species currently active across the ocean[88][89].

Bacteria were the first organisms to form a biome on early earth. Obviously, they fed at first on inorganic material[58]. In order for them to survive, they would have to adapt to changing PH values and other chemical balances that they would influence. Life had to have considerable sophistication at day one, or it would not have made it to day two, let alone millennium two. For some, this is evidence of design. Others have spent many decades unsuccessfully trying to model abiogenesis[59].

Directed Evolution

Directed evolution is a fascinating way to develop new organic molecules[90], but it should not be confused with darwinian evolution. It uses a combination of genetic engineering and artificially induced natural selection to steer the process. That should probably be unnatural natural selection. This demonstrates the amazing built-in flexibility of bacteria to adapt to their surroundings.

Conclusion - The Qualitative Nature of Evidence - Finding Answers

A Summary of Main Points

- Biologists from Darwin to Dawkins have written books in which the complexity of life is vastly underestimated. The science has changed, so many statements and conclusions in these books are very inaccurate.
- With the gap closing on the existence of junk DNA, and with codon degeneracy being discredited, the 'wiggle room' that was thought to exist for the evolution of new genes is fast disappearing. It's also now known that genes need to be controlled by complex networks of 'control DNA', so new genes by themselves may achieve little.
- In addition to underestimating the complexity of cellular biology and metabolism, the evolution of the 'other half' of living systems has not been explained. This is the part that animates organisms by giving them skills and purpose; instinct, the programmed behaviour that's found in everything from single celled organisms to plants and animals.
- The chances of the coding required for behaviour complementing the coding for the metabolism to come about by chance even once is incredibly remote and time would not have allowed it[18c].
- Observable examples of evolution demonstrate overwhelmingly that function is lost over time. As we really should expect, randomisation of incredibly complex code degrades it with associated loss of function.

So the Conclusion of the Matter Is . . .

Similar genes are found in different organisms, and similar phenotypes are found in the fossil record. Strictly speaking, their presence only says; "I'm here" or "I was here". Whether you believe in evolution or creation, commonality of components should not be a surprise. A functionally optimised gene found in different organisms could reflect either design success or beneficial natural selection. The same applies to an optimised design in an organism's morphology.

In both cases they are often used to support evolution theory. Whether they actually do or not is dependent on the evolution model being true. If creation were true then they could just as easily fit that model.

Reliance on evidence that can fit either model can reflect circular reasoning. To support evolution, the veracity of evidence must not be contingent on evolution being true. There's a need to look at evidence that is not ambiguous.

There are many paradoxes in evolution. These may be easily solved by applying a creation model, but some may think that's too convenient. On the other hand, if you believe in evolution because you reject the idea of a creator, some might think that it's no less a belief system than creation.

The argument "If all things are designed, who designed the designer?" carries little weight, as it infers that time-dependent cause and effect apply outside the bounds of our time-space universe.

Is 'god' some kind of mystical force? The physics of protein interaction is now known, so cellular biology loses some of its mystery. The nested data arrangement within genes along with the cellular machines that have been discovered, reflect design and logic that we are familiar with in software, hardware, and mechanical engineering. The difference is that the logic is more complex, more deeply layered, more efficient, and more environmentally friendly than anything that man has been able to engineer.

Does this reflect a logical mind of great superiority, or a mystical force that permeates the universe?

The Multiverse. Because the problem of probability is very evident to some evolutionary biologists, they sometimes point to the possibility of a multiverse: - the formation of many or an infinite number of universes as a consequence of expansion after the big bang. In this scenario, many earth-like variations could exist and other universes could have different natural laws to our own. Life could then find the perfect conditions to evolve.

The reality is that we don't know that to be the case, and even if it were, we are getting to know the natural laws on this planet, and they don't lend credence to rapid speciation as a result of random mutation that the theory of evolution proposes. For example, we know how old the earth is and we know the approximate rate of inherited mutation. From this, we know that there has not been enough time[18c]. See also "Possible number of positions for 10 new base-pairs"

Suffering caused by parasites remains a strong reason for many to believe in a naturalistic origin of life. On the other hand, more and more complexity is being discovered, and the time periods that might allow this to evolve are much too short. This has become an overwhelming counter-argument.

Evidence in the fossil record is complicated by the extreme rate of speciation recorded, interspersed with periods of stasis. This could be said to fit the creation model more closely. Metabolism and morphogenesis is being found to be bewilderingly complex, to the extent that some think it reflects a supernatural designer.

There may be a matter of risk assessment here. Which represents to greater risk? To reject God and maybe also great benefit, or to reject popular belief?

Some say that the pleasure we derive from our senses is evidence of a creator; music, fine wine, good food, summer breeze, the touch of a loved one, the colours of nature, art, a high mountain view.

On the other hand, this may seem to be contradicted by greed, pain and suffering, starvation, war, crime, disease, and the effects of old age.

Why does our DNA deteriorate leading to death, if self-preservation and love of life are so deeply embedded?

These contradictions would not be a reason to reject God if they could be logically resolved within the context of a belief in God.

A problem is that religion has often been at the centre of hatred and war, and many religions don't seem to offer a credible resolution to the contradictions.

One suggestion is to look here www.jw.org, then use search terms such as 'suffering', 'war', death, and so on.
You may be quite surprised at the answers.

Appendix 1: A Brief History of Time and Unexpected Events in Genomics

Genomics is study of the entire genome as opposed to the study of individual genes and heredity in genetics.

The last few decades have been very exciting in this area. The greater sensitivity of scientific instruments has transformed observation in many fields of study, from molecules, to gravity waves, to brain function.

Here are some important developments, followed by a summary of their impact on evolution theory. Some discoveries were the culmination of changes in thinking; the growing awareness of genomic function. Others were more in the way of "eureka" events. When researching, I was quite taken aback by the extent of these.

DNA Double Helix 1953

In 1953, Watson and Crick introduced us to the famous double helix model of DNA. This brought knowledge of the binding of base-pair nucleotides, and set the scene for future discoveries.

By the mid 70s, scientists knew that DNA was wrapped around repeating groups of histone proteins, like a string of pearls with gaps. These histones were later shown to play an important part in gene expression.

Gene expression is when a protein or RNA molecule is made by cellular machines using gene data. The main function of DNA is storing data for this purpose. When fully expressed, the data defines body plan and metabolism.

Proteins are at the heart of our metabolism, and perform various functions in and outside the cell. They include hormones, which are inter-cellular messengers, enzymes - powerful agents that help efficient chemical reactions such as digestion, transport mechanisms such as haemoglobin, and structural components.

Knowledge of cellular RNA function was growing. RNA was known to be an intermediate stage of gene expression. Other RNAs were also involved. The ENCODE project would help fill the gaps (see next page).

Late 20th Century

'Epigenetic' means around the gene. Epigenetic tags affect the rate of gene expression and are placed on genes and its histone packaging. Some of them stay a long time, others come and go when modulating metabolism.

By the 90s, awareness was growing of the heritability of changes caused by epigenetic molecular tags on DNA and histones, as opposed to changes to DNA sequence. This proved to be very controversial. Heritability of new characteristics as a result of changes to genetic code was a central dogma of evolution, and surprises were to come in this area.

It was known that DNA contained many genes with introns. Introns are areas of code within genes that are not included in the product of gene expression. They were thought to be evolutionary relics. Ideas were developing on their function, but uncertainty remained. Other code, interspersed with the genes, was also thought to be junk. To recap, these are the components discussed so far: -

- DNA - the molecule that stores the data that defines us
- Histones - a structural element that DNA wraps around
- Genes - the packets of data that are used to express our proteins and some RNAs
- Proteins - a folded chain of amino acids expressed (made) using gene data
- RNA - a molecule built with base pairs similar to DNA but normally with one strand instead of two
- Introns - areas that break the continuity of genes. Intron function is discussed on the next page
- Epigenetic tags - small molecules attached to DNA or its histones that regulate the rate of gene expression

Proteins are the basis of metabolism. It was previously thought that each gene expressed one protein, this had changed in the late 70s with the nobel-winning discovery of intron function. Many still thought that humans might have 100,000 genes to express many protein types to correlate with our complexity.

The Human Genome Project 2004

Many scientists thought that the Project was a waste of resources. Over ten years and three billion dollars later, scientists were presented with a sequence of DNA that many evolutionary biologists considered to be 98% junk.

The project surprised the science community by identifying only 25,000 genes in human DNA. This was considerably less than the 100,000 that many had predicted.

> **Scientists wondered how our DNA could be so simple[19], and why the amount of genes wasn't proportional to the complexity of an organism. They called this the G-value paradox.**
> **The idea that over 98% of our DNA was 'junk' was looking less credible.**

It was known that genes work differently in different cell types. Within those cells is a sophisticated mechanism that places and removes epigenetic molecular tags that control gene expression. All genes can't stay switched on or off all of the time, or we would die, but some tags stay for a lifetime and are passed on to daughter cells. These can silence genes and commit cells to being a certain type; heart, lung, etc., so in effect, they are memory devices. Others stay for a short time to control metabolism. The nature of this mechanism was not understood.

The function of introns was not clear. It was known that some were instrumental in allowing a gene to produce more than one protein. Their junk nature, along with other areas of DNA, was being questioned.

The only functioning part of our DNA was thought to be our 25,000 genes and the epigenetic mechanism that somehow controlled them by an interaction between genes; epistasis. The other 98% of our DNA was considered to be an accumulation of junk during the evolutionary process.

> **Scientists wondered why, as a general rule, the amount of junk DNA was proportional to the complexity of the organism[19a]. When this was understood, scientists would have a fuller understanding of DNA function.**

ENCODE Project Phase One 2013

Scientists had been moving towards a better understanding of gene expression for some time. By 2012, a new awareness of 'junk DNA' function was growing. Ewan Birney, Computational Biologist, made this comment: -

> *"I get this strong feeling that previously I was ignorant of my own ignorance, and now I understand my ignorance. It's slightly depressing as you realize how ignorant you are. But this is progress. The first step in understanding these things is having a list of things that one has to understand, and that's what we've got here."* [20]

Birney's words indicated that monumental changes in understanding were taking place.

Richard Dawkins' famous books were written years earlier. Birney had, no doubt, shared similar views. Dawkins' gene-centric narrative was now looking a lot less relevant.

By 2013, the first phase results of the ENCODE project (Encyclopaedia of DNA Elements) had been released. The purpose of the project was to identify and catalogue the function of sequences in our DNA.

Proteins and RNA molecules in the cell were traced back to point of origin in DNA. Computational Biologists like Birney were needed to assist biologists identify functional elements for the encyclopaedia. The importance of this area to the health sector alone warranted a large expansion of effort.

> **There was shock and disbelief, especially in evolutionary biology, as ENCODE revealed that junk DNA only amounted to 20% of the genome, not 98% as previously thought.**

Understanding of the genome had been simplistic, but now new technology and falling costs brought about more research. Improvements to DNA sequencers, x-ray crystallography, mass spectrometers and liquid chromatography, allowed much greater sensitivity in detecting smaller molecules and their structure. These included microproteins and microRNA, both of which were found to take a part in gene expression. They had previously been a lot less visible. Genetic engineering tools brought new ways to carry out functional testing of DNA sequences. This, in turn, brought greater insight to genomics and cellular biology.

A staggering new level of complexity came into view. In a similar way that our sympathetic and parasympathetic nervous systems achieve balance, a number of RNA molecules are now known to act in opposition to achieve a balance of gene expression. Molecules involved in this process are known as transcription factors and connect with areas of DNA called promoters, enhancers, and silencers. This, along with interaction with epigenetic tags, is very complex and allows for fine tuning.

This replaced the old understanding where our metabolism was governed by genes affecting genes.

It became apparent that introns, which are sometimes larger than the gene regions they interrupt, enable the expression of many more proteins from a single gene than previously thought. They contain gene section markers.

Inside the cell, an exquisitely complex cellular machine, the spliceosome, discovered in the 1970s, is involved in editing gene data for the production of proteins by the ribosome, another exquisite machine. It can splice together many different combinations of gene data. The sections that are spliced reside between introns, and can be edited and spliced to assemble many different proteins. The spliceosome, constructed from over 100 proteins and RNAs, produces an RNA template. The ribosome, constructed from over 300[21], makes proteins from the RNA template.

This is actually a simplification. In a process that's not fully understood, a fruit fly gene has been found to express as many as 38,000 proteins, each with unique function[22]. This may be an extreme example.

> "*Since its discovery, it has become clear that alternative splicing is common and that the phenomenon helps explain how limited numbers of genes can encode organisms of staggering complexity. While fewer than 40 percent of the genes in a fruit fly undergo alternative splicing, more than 90 percent of genes are alternatively spliced in humans.*" The Scientist[22]

Much of the genome that had been termed junk DNA, was found to be functional. How much is still not known. Referred to as non-coding DNA (ncDNA), more function is being found and the ENCODE project is ongoing.

So, now, within the area of Genomics, there are two relatively new terms: - the Transcriptome and the Epigenome. These encompass a description of many previously unknown processes and molecules that represent the underlying mechanisms of our metabolism.

> **It solved the mystery of why our DNA seemed so simple. It wasn't.**

Regulation of our genes controls morphogenesis and metabolism. It's still very far from being fully understood.

Discovery of Other Cellular Machines

Scientists have found biological nanomachines; motors. They were initially discovered in the mid 80s in a large nerve fibre of the squid. Different types with various functions have since been discovered in humans.

Our mitochondria, the power-house organelles in our cells, have many intricate rotary machines, spinning at 6000 RPM under load. They are driven by sub-atomic particles, protons, and produce ATP (adenosine triphosphate), which is essential for many purposes including fuel for muscles and cell division.

Other motors literally walk to perform various functions, using ATP as fuel. Cells have structures called microtubules which protein motors walk along to carry molecular cargo required in another part of the cell. Two types walk in opposite directions and can walk past each other without collision; a two-way transport system[23].

Cell division in eukaryotes (organisms having cells with a nucleus) is carried out by protein motors that walk along tubules in a little understood process in which chromosomes are pulled apart.

Protein motors also walk along tubules in our muscles to provide movement.

These discoveries have amazed scientists and they are wondering what the extent of our complexity will be.

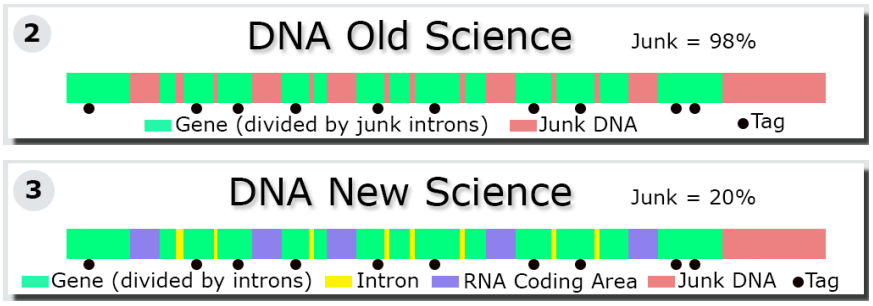
Some Detail of The Impact of New Science on Evolution Theory

Well known author Nessa Carey said in her book, Junk DNA: -

> *"Evolution is the biological discipline where emotions tend to run highest. Normally the bullets are targeted at creationists, but the Gatling guns may also be turned on other scientists. Epigeneticists working on the transmission of acquired characteristics from parent to offspring were probably quite relieved that ENCODE took them out of the firing line for a while."*[24]

As this quote indicates, evolutionary biologists didn't like changes emerging in two related fields; Genomics and Epigenetics. In both cases, old dogmas of evolution theory were under threat. This is not unusual in science. New science can often cause cherished world-views to be challenged and a period of adjustment is needed.

These illustrations are greatly simplified and do not imply scale or relative positions



These illustrations highlight a number of big changes in Genomics over the last few decades.
The game-changer was the discovery that most junk DNA was not junk. This raised two big problems.

Junk DNA No More

Problem #1. A revised explanation for evolution was required.
Previous thinking in popular books taught that life and evolution was centred around genes and gene to gene regulation - epistasis[25]. When ENCODE showed that most of our DNA is not junk, there was vocal resistance from the evolutionist community. Junk DNA had been a central dogma, and gene regulation now required a lot more explanation, because the process was now known to have very much greater complexity.

In 2007 a paper had proposed that removing sections of junk DNA from the yeast genome has little effect[26]. In 2019 another paper showed that those sections allowed the yeast to respond to changes in the supply of nutrients and adjust growth accordingly[27]. Without it, yeast cells could die. Old assumptions of simplicity were wrong.

This, along with the case of Darwin's Finches, mentioned later, strongly indicate that there are many latent mechanisms lurking in 'junk DNA' that spring into action when environmental changes occur. To some people, this might indicate a-priori design.

It was shown that in order for genes to function correctly, there was great interaction with junk DNA. Numerous sequences were involved, so an evolutionary step now had to involve changes to many areas of DNA.

Problem #2. Disrupting the phylogenetic tree.
In the past, the darwinian tree showing evolutionary relationships was based on phenotypes; discernibly different organisms. When it became possible to sequence DNA, the tree had to be modified because some animals that were considered closely related phenotypes, were found to be less similar genotypes.

ENCODE data showed that genes were more like the "Lego bricks" of life, with "junk DNA" controlling them like an operating system. Both differentiate organisms, but it's largely the junk that controls the genes. Our genes have similarity to chimps, rats, mice, fruit flies, some worms, and bananas, but crucial differences were to be found.

Much attention turned to junk DNA. Evolutionists had to adapt their model. From their perspective, junk DNA sequences preserved through evolutionary time must be good if natural selection favoured them. In addition, many areas of junk DNA were shown to adversely affect health when mutations were present.

Attention turned to the unique genes that humans do not share with animals. It turned out that many organisms have unique genes. In humans, these are consistent across different populations.

Importantly, humans have many genes not shared with lower species. Some are completely unique and are called de-novo genes on the basis that they bear no similarity to genes in animals. These, and unique areas of ncDNA (junk DNA) had to be brought into the evolutionary model. Papers on de-novo genes said: -

> *"For many years, it had been considered extremely unlikely, if not impossible, that genes with no detectable homology could emerge. . . About one third of the entire set of genes in baker's yeast has no sequence similarity to genes from other organisms."*[28]

> *"The origin of novel protein-coding genes de novo was once considered so improbable as to be impossible."*[29]

This highlights a problem. Excluding alternatives left no choice but to adopt new ideas that were previously considered virtually impossible. They now had to be regarded as commonplace. This situation became more untenable as new complexities were discovered in genes. See pages 23 and 24.

The Atheist Bus Campaign in 2009 read: "*There's probably no god. Now stop worrying and enjoy your life.*"
Arthur Conan Doyle wrote a pithy line for Sherlock Holmes, later used by Spock in Star Trek:
"*Once you eliminate the impossible, whatever remains, however improbable, must be the truth.*"

The near-to-impossible nature of de-novo gene evolution had been recognised for decades[29]. It was thought that new genes only developed from old ones, by duplication and subsequent mutation.

De-novo genes have been found in many tested species[29], meaning that genes with no known link to other species is

a common trait across the animal kingdom.

There's a paradox here. Because there's much more to gene expression than previously realised, the previous "extremely unlikely, if not impossible" status of de-novo genes now looks much more unlikely, owing to the complexity of gene expression exposed by analysis of ENCODE data. This situation reached another climax when it was realised that complexity of genes was still underestimated, and is discussed on the next page.

In addition to 60 de-novo[30] genes, there are 600+ other unique genes in humans[31]. Different results arise from different software. Some may have more liberal or conservative recognition pass rates, and computer models of DNA structure can only be as good as current understanding, which has often changed.

Unique parts of the human genome are now labelled "HARs" - Human Accelerated Regions. This term was first used in 2006 and applied to genes. It reflects an attempt to rationalise the fact that these genes appeared in a very short time to differentiate humans from primates. It was used to compare differences between humans and apes, particularly the chimpanzee. Professor Robert Sapolsky said, in an article in 2006 in Discover Magazine: -

> *"Scientists have long known that chimps and humans share about 98 percent of their DNA. At last, however, one can sit down with two scrolls of computer printout, march through the two genomes, and see exactly where our 2 percent difference lies."*[32]

These comments are based on old science and highlight how things have changed, as indicated by the Ewan Birney quote on page 21. Scientists thought that human DNA differed from chimp DNA by a very small 2%.

Since ENCODE, HARs have been extended to include unique areas of ncDNA ("junk") in addition to genes. Humans were now looking more unique.

In line with the thinking of the time, Sapolsky thought that genes somehow switched each other on and off. We know that genes do affect each other (epistasis), but the main complexity of regulation is in ncDNA. When this is taken into account, the difference between humans and chimps is much greater. A 2017 paper spoke of this newly discovered uniqueness: -

> *"The review discusses, in a format of a timeline, the studies of different types of genetic variants, present in Homo sapiens, but absent in all other primate, mammalian, or vertebrate species, tested so far."*[33]

And in Scientific American, 2018, the article ""Junk DNA" What Makes Humans Unique?" said: -

> *"About 10 years ago Katherine Pollard, a biostatistician at the Gladstone Institutes and the University of California, San Francisco, compared the two species and identified the parts of the human genome that are unique. Now she is leading a research team that is uncovering how 716 of these human-specific DNA regions work together to create the biological traits that differentiate us from other primates."*[34]

When chimpanzee DNA was sequenced in 2005 it was claimed than humans differed by less than 2%, but in 2018 chimp DNA was re-sequenced. The published results were critical of the 2005 findings, saying: -

> *"This bias has effectively "humanized" other ape genome assemblies, minimizing potential structural and transcript differences observed between the species."*[35]

Another likely problem was contamination. Reanalysis of DNA samples used in early sequencing found that contamination with human DNA was common[36].

> The human-chimpanzee difference was much larger; at least 17%[37], which rules out any close evolutionary relationship. The phylogenetic tree was wrong again. Be aware that many seemingly authoritative sources have not caught up with this.

The trend of discoveries is on a steep curve that diverges from the old-school evolution taught to us. It might be worrying to some that much of this is still taught to our children and grandchildren. Much more was to come.

Epigenetics and Heredity

By the 90s, epigenetics had gained traction as the science of heritable change that didn't involve changes to DNA code. There are interesting examples of epigenetics affecting morphology; same DNA different forms. These include caterpillar/butterfly, tadpole/frog, grasshopper/locust and worker/queen/drone in insect colonies.

A recent shock in evolutionary biology was the discovery that acquired epigenetic tags affecting morphology are not only heritable, but multi-generational. Evolutionists fought against this, as alluded to in the Nessa Carey quote on the previous page. It undermined a basic dogma and discredited at least one classic example of evolution.

Problem #1. It changed the understanding of inheritance in evolution
Clearly, mutations happen and can be selected - see page 16, *Two Flavours of Evolution*. However, a paper published in 2014 showed that Darwin's Finches did not evolve by classic darwinian mutation-selection as previously thought[38]. It was by multi-generational inheritance of acquired epigenetic changes; methyl tags on DNA.

Transgenerational epigenetic inheritance has also been identified in humans[39].

Problem #2. How did latent mechanisms come to exist and continue to exist?
Evidence emerged that latent mechanisms in DNA activate in response to environmental stimuli, as highlighted in the case of Darwin's finches, and the yeast genome, mentioned earlier.

It was thought that the different forms of Darwin's finches, with different beak size and shape, were a result random changes to their DNA, followed by natural selection; classic darwinian change. This was found not to be the case. It turns out that there is a mechanism in the finches' DNA that responded to dietary changes brought about by changing environmental conditions[38]. Natural selection was an important factor that caused the spread of these changes in local populations. This blurs the already fuzzy definition of speciation.

It's not in the nature of evolution to plan ahead. Nor is it in its nature to preserve genetic code that's not used. So the existence of hidden mechanisms that can bring about change when triggered by specific events is a paradox; possibly a double paradox. The potential complexity of these mechanisms raises further questions about their evolution. These re-

versible epigenetic changes infer a structured two-way sense and trigger mechanism.

Robustness of the Genome to Tolerate Mutation - Two Flavours of Degeneracy

The genome needs to be robust if billions of random changes are to survive natural selection. The power of natural selection to bring about new species is proportional to the robustness of the genome. If mutation breaks function, then natural selection will weed out mutated organisms. It becomes the preserver of the genome.

Characteristics of our DNA that were thought to add robustness are not as previously thought. The terminology used is potentially confusing; redundancy and degeneracy. The Plain English Campaign would not be impressed.

Redundancy is a term used in engineering. It describes the use of backup systems to enhance reliability in areas where failure can be costly. This includes life support, safety-critical aircraft systems, and space missions. Anyone following the progress of the Voyager probes may be aware of system failures that were overcome by switching to backup systems. Our DNA provides us with redundant systems in the forms of duplicate genes.

DNA also has degenerate systems. The term degeneracy was borrowed from quantum physics and is also used in mathematics. In our bodies, degeneracy is another kind of redundancy, but instead of duplicate backup systems, it's backup by dissimilar systems. A slightly less confusing expression might be dissimilar redundancy.

To complicate things, there are two types of degeneracy; biological degeneracy and codon degeneracy. Biological degeneracy raises questions about the nature of evolution, as does redundancy. Codon degeneracy is not as previously thought, owing to fascinating new discoveries. For this, there's a need to grasp the basics of codons.

Codon Degeneracy

Codon degeneracy was considered to be a key factor in providing genome robustness when a mutation occurred. The thinking was that it allowed mutation without breaking cell function[40]. This was obviously very important, as mutations were thought to build up harmlessly in genes over millions of years until they provide benefit.

A codon is a the second smallest unit of data in a gene when defining proteins. It's a sequence of three nucleotide base pairs that specify types of amino acids, the building blocks of proteins. Nucleotides and codons could be considered as DNA's equivalent of bits and bytes in computer code. There are four types of nucleotide in DNA, A, T, C and G, but only two combinations of pairs, A-T and C-G. They can be orientated in one of two directions, which gives four functional types. DNA is normally read on one side only, so A-T is different to T-A, and C-G is different to G-C.

Codon degeneracy refers to the way that they code for amino acids. There are 64 possible combinations in a codon (4x4x4), but only 20 amino acids are specified by codons. Additionally, there are three stop codons which tell the cellular machinery that the end of a sequence has been reached.

The main point is that there appeared to be 'spare' codes, some of which code for the same amino acids. It was thought that this provided code degeneracy for coding of proteins. In the event of a mutation, it's possible for the codon sequence to be changed, and for the codon to still specify the same amino acid.

For decades, evolutionary biologists thought this would allow 'silent' mutations to accumulate without disrupting function. Evolution could happen without breaking things on the way. This was not necessarily the case.

A paper published in May 2014, followed up on discoveries of the previous year. In it was this statement: -

> *In doing so, we show why the term "degeneracy" is completely inappropriate. The dual coding functionality of redundancy is anything but "degenerate."*[41]

Complexity Underestimated Again - Codes Within Code

Over the last few years, previously unknown codes have been found within genes. These overlay the already complex DNA code that scientists were exploring. Data from the ENCODE project was revealing new science.

Amazingly, the arrangement and sequence of codons can simultaneously encode separate codes that can be read by sophisticated cellular machinery. This might bring to mind thoughts of Enigma and Bletchley Park.

> **In 2013 a paper described a newly discovered code hidden within genes. The term 'duon' now describes codons that also specify aspects of gene transcription**[42][43][41]**.**

Duons are highly conserved; around for a long time, so in evolution theory, their function must be important.

> **In 2016 a paper described another code in DNA that had been missed for decades, controlling protein production and folding**[44]**.**

A paper published later in 2016 presented a new visualisation of codon complexity as *"The periodic table of genetic code: A new way to look at the code and the decoding process"*[45].

> **In 2018 a paper described another layer of code in codons that has an effect on the rate of gene expression**[46]**.**

This means that the sequences within genes that specify proteins also carry detailed instructions that affect the way that cellular machines produce proteins, including rate and three dimensional molecular construction.

The shape of proteins is an essential part of their function. Many tens of thousands of proteins in our cells are folded in a way that exposes electrostatic, hydrophobic and hydrogen bonding areas; a kind of molecular oregami. The arrangement of these areas allow them to bond with specific complementary molecules and carry out metabolism; an amazing dance. These new codes affected protein folding. If changed, they can damage function, even though their chemical sequence remains the same[91].

The discovery of these sophisticated code areas, overlaying an already incredibly complex code, presented a new set of problems for evolutionary biologists.

Problem #1. Many 'silent' mutations that were thought to be in the genome are not mutations at all.

> Deviations from the old understanding of valid codon arrangements were considered mutations. It turns out now that they can be valid codes that play an important part in gene expression and cell function.

Problem #2. Codon degeneracy was thought to make the genome more robust. This is not entirely true.

Codon variants are not necessarily degenerate, but can be valid codes, so the opportunity for harmless mutation is not as previously thought. The neutral theory of evolution is somewhat reliant on codon degeneracy.

The extreme complexity of the codes within code arrangement means that gene function can be damaged more easily than previously thought. As mentioned on the previous page, damage to protein function has been found to happen as a result of one of these changes. The protein retained the same chemical sequence (of amino acids), but its changed function causes kidney cancer[91].

Problem #3. The structured, nested codes within genes are difficult to explain by random chance.

The sheer complexity and depth of nested structure found in genes and in their control mechanisms is not consistent with the random nature of evolution. Its non-random nature is often considerably overstated. See the section "Probability and Evolution" for the reasoning on this.

Biological Degeneracy

Biological degeneracy is different to the largely discredited area of codon degeneracy. It refers to the ability of functional components in nature to perform the function of a different failed component; backup by means of dissimilar components.

You may read that biological systems improve on engineering's backup systems. They are more extensive and complex, but degenerate systems are also found in engineering[47]. They are referred to by the better descriptive term, dissimilar redundancy. In engineering, this is an advanced form of redundancy that can provide greater reliability if the secondary system is designed to avoid the same failure modes of the primary system.

Biological degeneracy allows for robustness of the organism and has even kept people alive when primary systems fail. Some brain function is degenerate, as experienced by stroke survivors who regain lost muscular control. Another interesting example is an inherited condition causing a lack of albumin in blood plasma. It's been discovered in seemingly healthy people[48], and yet albumin has several important metabolic functions.

Degeneracy is somewhat ubiquitous within organisms, and seemingly in ecosystems too. It increases plasticity, or "bendyness", of the genome when it comes to change by mutation.

Although degeneracy contributes to an organism's robustness, a problem exists from an evolutionary perspective. There's a tendency to write it off as an inherent attribute of complexity. This is likely to indicate a lack of understanding of function, as it was when genes were thought to somehow control each other's expression. As shown previously in this section, the underestimating of complexity has been a prominent feature of evolutionary biology since Darwin.

In light of the existence of these degenerate systems and their frequent occurrence, the question arises: "How could alternate networks evolve that can do this?" It can also be asked: "If these attributes of degeneracy were able to sacrifice themselves to enable evolutionary change, then how is it that they are still so widespread after so much evolutionary change?" These look very much like paradoxes.

It seems likely that sophisticated mechanisms will be found that explain degeneracy. This has been the trend for decades.

Redundancy

Redundancy is backup by similar components. The same question must be asked as with degeneracy: "How do such systems continue to exist?" Some duplicate genes have been found to affect metabolism. Others do not, but provide redundancy. A Wikipedia article on Genetic Redundancy states: -

"Taking these notions into account, the very existence of genetic buffering, and the functional redundancies required for it, presents a paradox in light of the evolutionary concepts. On one hand, for genetic buffering to take place there is a necessity for redundancies of gene function, on the other hand such redundancies are clearly unstable in face of natural selection and are therefore unlikely to be found in evolved genomes."

Function of Other DNA Structures Increase Knowledge of Complexity Again

Some basic aspects of gene regulation have been discussed. It's extremely complex, but in order for it to happen other amazing processes occur.

DNA isn't static. When genes go into action, it's very dynamic. In order for gene regulation to take place, the tightly coiled structure that would normally make genes inaccessible, exposes loops where the genes are located. This is very complex and not fully understood. There are proteins that affect the coiling and exposing of DNA segments, and even proteins that help prevent tangling of DNA. An article in Nature magazine entitled "*DNA's secret weapon against knots and tangles*" is fascinating[48a].

B-DNA is the default structure of DNA, but it has been found to flip into another structure called Z-DNA which has been observed since 1970, function was not known until recently. The Z comes from its zigzag appearance. It was first thought to be associated with disease, but it seems that cause and effect were not clear. It is also known as left-handed DNA and is dynamically formed to perform special cellular function[48b].

G-quadruplex is a four-stranded structure of DNA that has been observed for many decades within DNA and in separate molecules. As with Z-DNA, it was thought to be associated with disease but has recently been found in healthy cells and is now thought to be yet another component involved in gene expression[48c].

More Function Discovered

In 2020, an area of 'junk DNA' that was thought to be an ancient retrovirus has been found to take part in embrionic cell differentiation. This is part of an upward trend in discovering function in areas previously considered junk. It seems likely that areas will remain undiscovered for some time because of the infrequent and dynamic nature of some gene expression[48d].

Appendix 2:
Possible number of positions for 10 new base-pairs in a genome of 2 Gbp

This example is understated to demonstrate that even tiny steps towards major speciation are very unlikely. The calculation applies to single point mutation substitutions. It doesn't account for deletions and insertions which would make the result larger.

- Assume a 25% split of each nucleotide - so for 1bp, 0.5G positions would make no difference
- Positions resulting in change, for one nucleotide = $2x10^9$ less 25% ($5x10^8$) = $1.5x10^9$
- Positions for ten nucleotides = $(1.5x10^9)^{10}$ = approx $5.7x10^{91}$
- The estimated number of atoms in the observable universe is around 10^{78} to 10^{82} - see reference[57]

To reiterate, this calculation is intentionally conservative, and is weighted in evolution's favour.

You'll find rebuttals of this kind of argument that try to convince that probabilities are misapplied.

In some cases websites give the case of the Birthday Paradox, where people normally underestimate the chances of two people in a group having the same birthday. That example is intriguing, but doesn't correspond with the example on this page. In the birthday paradox, many are compared with many. That's the whole point. In this example, one mutation set is compared with a vast number of possibilities, and only some are viable.

An example of how logic can be distorted is found in a few places on the Internet. A deal of a pack of cards is used as an example, with every deal being one of an incredibly large number of possibilities; and yet it happened.

Mathematics Applied with Faulty Logic

> *The statement "Any combination is as likely as any other." is true, but misleading and irrelevant.*

A pack of cards has only 52 elements, each has only one state. The human genome has 3.2 billion elements, each one having four states. The card pack deal is used to support the expression: -

> "Any combination is as likely as any other."

In the case of the first DNA code sequence of a new evolutionary step, this statement is quite relevant, but when another sequence is required to complement the first, any combination won't work. As complexity builds, additional sequences or changed DNA code have to be complementary and very specific or valid functions will not be built. Many finely tuned sequences are required for the functioning of one gene. One tiny mutation can in some cases break function. In turn, each gene must be complementary to others.

Until recently, synonymous codes in our DNA were thought to provide opportunity for mutations to build up without disrupting function. These codes are now known to form a separate coding system that defines how proteins fold; a critical attribute for their function. A protein may have the correct chemical sequence, but may malfunction as a result of changes to these synonymous DNA codes. For more detail and references, see Codon Degeneracy.

So, the statement "Any combination is as likely as any other." is true, but misleading and irrelevant. It would be more to the point to say: -

> "Any *valid combination*, that *complements an existing combination*, is incredibly unlikely."

Significant Evolutionary Transitions - From Here to Eternity

An evolutionary transition may well take an eternity if the build up of mutations leading to change is weeded out by natural selection and never comes to fruition. This scenario seems more likely when taking into account the new findings of hidden genetic code - see Codon Degeneracy on page 24.

Evolution theory depends on millions of genomic changes. Most would need to be be very much larger than the example above, meaning much more unlikely to happen. This is why an Oxford team recently stated: -

> *" . . . we demonstrate that expected evolutionary transition times likely exceed the lifetime of Earth, perhaps by many orders of magnitude."* [18c]

Appendix 3: Vestigial Evolutionary Leftovers?

The Appendix, Tonsils and Adenoid

For a long time, the appendix has been said to be an example of a vestigial organ. Until relatively recently, it was thought to be the remains of an organ that assisted digestion in evolutionary ancestors. It is now known to have important function. It's a reservoir for good gut bacteria that play a part in restoration of the gut biome after illness[73].

It has been discovered relatively recently that the appendix is part of our the immune system[74].

Tonsils and adenoids have also been found to play a part in the immune system[75][76].

Coccyx

The coccyx, commonly called the tailbone, is said to be the remaining part of an ancestral tail.

The function of the coccyx has been known for a long time. It's the anchor point for several muscles and helps provide stability when sitting[77].

Goose bumps

Goose bumps were previously thought to be just an evolutionary relic from ancestral species that raised their hair when threatening or threatened, and having no useful function in humans.

New research has uncovered previously unknown complexity in the skin and shows that this function has a part in stimulating stem cell activation for hair growth[77b].

The Moral of the Story

When evolutionary biologists say that something has no function or is an evolutionary relic, they may mean that they don't understand it. There can be no clearer example than junk DNA which was said to have no function, but turns out to be the main source of control for morphogenesis, metabolism and life.

There are other examples of physiological features that have been said to be vestigial features. Function is normally found in these, so they only appear to be evolutionary relics when viewed from a certain perspective.

Appendix 4: A Lack of Accepted Theories

> *"Today the theory of evolution is about as much open to doubt as the theory that the earth goes round the sun."* Richard Dawkins, The Selfish Gene

From the time we are at school, we are taught that darwinian evolution is a robust theory. This idea is reinforced by media articles and documentaries. This does not necessarily reflect reality.

There are disagreements in most areas of science, but in evolutionary biology these are much more pronounced and run deep into the basic underpinnings of the theory.

- *Abiogenesis* (how the first prokaryotic cell [with no nucleus] came from lifeless rock[58])
  You've probably heard about life starting in hot thermal vents, RNA world and other theories. Some sources have even given the impression that some of these are established theories.

  This is not a part of darwinian evolution, which describes the origin of species from a single ancestor, but if abiogenesis can't be explained by naturalistic processes, then this obviously impacts darwinian theory.

  Status: There is no accepted theory. Some scientists defend pet theories, others think all theories are poor. One peer reviewed paper has the title: -
  "The RNA world hypothesis: the worst theory of the early evolution of life (except for all the others)"[59].
  This was published in 2012, and still remains true.

- *First Eukaryotic Cell* (cell with nucleus)
  All 'higher' forms of life have cells with nuclei. These cells have many large differences from prokaryotic cells. The evolution of prokaryotic to eukaryotic is a key part of evolution theory.

  Status: No accepted theory[60] and disagreement for decades as to how this transition occurred.

- *Sexual Reproduction*
  There is sexual and asexual reproduction. Asexual reproduction allows mutations to be passed on to a greater degree. Sexual reproduction would have been a very major change involving germ cells and very complex processes for their combination. Some of the processes that occur after fertilisation are highly complex and rigidly sequential. In addition, there is instinct in all species to procreate.

  Status: No accepted theory. The evolution of sexual reproduction is a paradox.
  "the paradox of sex remains largely unresolved to date." The Paradox of Sex[61].

- *The Cambrian Explosion*
  Darwin felt that the fossil record was incomplete because it didn't seem to support his theory. This thought continued until relatively recently.

  Some have tried to minimise its importance, a few important fossils have been found in precambrian strata[62], but very few compared with the amount in the Cambrian.

  Status: No accepted theory.
  "We now know that the sudden appearance of fossils in the Cambrian (541–485 million years ago) is real and not an artefact of an imperfect fossil record."[63].
  "Now, new research published in the Proceedings of the National Academy of Sciences (USA) is revealing that this explosion was far shorter than many experts had thought." Natural History Museum 2019[64]

  In addition, life keeps proving to be more complex than previously thought. There's no end in sight to this trend. Each discovery adds to the difficulty in explaining the Cambrian Explosion in evolutionary terms.

- *Basic Mechanism for Evolutionary Change*
  The public-facing side of the science community appears fairly unified in this area.

  Status: The Neutralist-Selectionist debate has been going on for decades[3]. Sometimes, it has been quite heated.

- Also the need for a rethink on evolution has been a source of disagreement for many years. It goes under the heading of EES - Extended Evolutionary Synthesis

Appendix 5: Is the Eye Badly Designed?

Design of the Eye

Genomics and cellular biology have advanced at a fast pace, especially in the last decade (See page 7 and 20). This has resulted in many older web pages and books being past their sell-by date. Some are now very misleading.

The design of the eye has drawn considerable comment, especially because it has been used by creationists as an example of irreducible complexity. Irreducible complexity is not necessarily a good argument.

"The Poor Design of the Human Eye" was Posted in a blog on January 12, 2015 by Prof. Nathan H Lents. His book "Human Errors" also includes this. He makes the following three criticisms of the 'design' of the eye: -

1. Short and long sightedness. Glaucoma. Detached retina. Cataracts.

These represent a departure from design, rather than an error in design. This departure from design is evolution in action. See Two Flavours of Evolution, page 16. They represent the aging process and the degrading effects of 'evolution'. See Conclusion - The Qualitative Nature of Evidence, page 19.

2. Animals have greater visual acuity than humans

There are plenty of artists and photographers who are pretty impressed with the eye's ability. Is it a problem that the eye of the eagle has higher resolution than ours? It flies at altitude whilst hunting small mammals.

The impact of greater visual acuity on the brain is a complex area. This might include the ability to process information, the effect on cooling and oxygenation, the ability to quickly change the focus of our concentration, and many other design issues. As an example, it's recently been discovered that some eye movement is put on hold whilst assessing certain touch stimuli[65a].

3. The vertebrate retina is back to front

This is a popular misconception. Here's the reality.

In the vertebrate eye, light travels through layers of tissue before it reaches retinal neurons. This might seen odd, but we can see quite well.

In 2007, a paper was published with interesting findings about the inverted retina[66]. Specialised cells, called Müller cells, cross the retina. The paper states: -

> *'their parallel array in the retina is reminiscent of fiberoptic plates used for low-distortion image transfer'.*

In engineering, this would indicate great sophistication.

The design of Müller cells goes further. The funnel shaped ends and graded index of refraction are optimised to capture as much light as possible and deliver it with good alignment to the cones. This allows for a greater surface area for light collection, and a smaller area for retinal neurons, providing room for other cells.

Müller cells have frequency-dependent transmission that allows blue light to leak[67]. This light falls on the rods.

The efficiency of light transmission is enhanced by an unusual feature. Cells normally have an array of structures that would interfere with light transmission. Müller cells are unique in that organelles are minimised or even missing. Müller cells also play a part in retinal structure, function, maintenance, and possibly regeneration.

There seems to be a lack of awareness that the retina needs to be inverted in order to have sufficient blood supply for function[68]. The organisation, construction and function of cell types in the eye indicate an amazing system of morphogenesis that goes beyond the outdated view of gene-centric eye evolution popularised in famous books.

The Eye - Some other points

A pulley is an engineered solution with forward thinking design to solve a mechanical problem. A pulley is used in each human eye for movement. It's called the 'trochlea of superior oblique'.

The cornea is the main focussing element. It has exceptional clarity owing to a lack of blood vessels. Uniquely, it receives oxygen and nutrients from the lacrimal gland tears, and nutrients from the aqueous humor. Tear ducts drain excessive tears. Pumps help maintain clarity by keeping the cornea free from excessive fluid.

Tears have several functions, including cleaning, lubricating, providing nutrients, aiding with immune response, and the involuntary reflex of expressing emotions of grief and happiness. The composition of tears is extremely complex[69] and varies with function[70]. The cornea is covered with touch receptors that make it highly sensitive, and cause the reflex of blinking, as you may have noticed.

Data Compression

The retina is equipped with many millions of photodetectors. How does video data get to the brain via an optic nerve with approximately one million nerve fibre 'mini cables' and limited bandwidth?

Amazingly, a data compression algorithm is used with some similarities to MP4[71][72][73]. The brain seamlessly decodes this.

So, we actually observe a highly optimised engineering solution. We could go on to discuss the brain's optimised system of facial recognition that outperforms man-made systems with decades of research and development behind them.

See also, **Argument from poor design** in Appendix 6: Is the Neck Badly Designed?.

Appendix 6: Is the Neck Badly Designed?

Argument from poor design

According to Wikipedia the argument runs that:

- *An omnipotent, omniscient, omnibenevolent creator God would create organisms that have optimal design.*
- *Organisms have features that are suboptimal.*
- *Therefore, God either did not create these organisms or is not omnipotent, omniscient and omnibenevolent.*

It's pretty obvious that there are two options missing from the concluding point, either of which would nullify the argument . . .

1. The argument's proponent does not understand the function of the item in question, as seemingly is the case with the eye (see Appendix 5: Is the Eye Badly Designed?). In which case, we should add the option to the conclusion; "or we have got this plain wrong".
2. There has been loss of function. This has been observed in nature (see The Real Nature of Evolution). In which case, we should add the option; "or there has been a loss of function that could make this look like poor design".

Some have written about the human neck's poor design. This seems to be a favourite this year (2021). Going back 250 years or so, Darwin commented that "every particle of food and drink which we swallow has to pass over the orifice of the trachea, with some risk of falling into the lungs". I'm personally well aware of this because I had a stroke some years ago which caused me to have less control over swallowing.

Sleep apnea is another real problem for many owing to partial blocking of the airways. Is this also poor design?

Reality Check

Apparently, we swallow between 700 and 900 times each day. So over a lifetime of 80 years, we may swallow over 20 million times. Most of us do this without considering it to be a hazardous activity. In fact, one of life's great pleasures is a session of talking, eating and drinking. Dinner dates with my wife are one of my favourite things.

Three stages are involved in swallowing:-

1. The first stage (oral) is very much under our control and involves putting food in the mouth and chewing. To us, this is simple, but in reality it's a complex process that also involves the automatic release of saliva to accommodate shaping of food-balls for swallowing and enzyme modification of starch. Saliva also assists with oral hygiene and taste, and its composition is complex.
2. The second stage (pharyngeal) is autonomic and is controlled by a swallowing centre in the brain that engineers might call a 'finite-state machine'. When food is shaped and pushed to the back by the tongue, it receives electrical signals from an array of sensors at the back of the mouth and responds by starting a timing sequence to operate muscles that close the breathing tube and initiate the swallowing process.
3. The third stage (esophageal) is also autonomic as muscles initiate a peristaltic wave to push the food to the stomach in a similar way to the rotary device observed in a dialysis machine.

Clearly, this is all very complex and involves 26 muscles and a complex control system.

As with other designs in nature, this is a highly optimised process and not poor design. Problems with swallowing are normally attributed to carelessness or loss of function. Many health problems can occur as a result of mutations to genes or 'junk DNA'. This is real world 'evolution' in action. (See The Real Nature of Evolution on page 17)

Sleep apnea is not a problem for the majority of people, but is certainly a big problem for many. So, it also represents a departure from design, not poor design. (See Conclusion - The Qualitative Nature of Evidence - Finding Answers)

Appendix 7: The Data Inside Us - An Analogy for the Computer/IT Engineer

TCP/IP Protocol Looks Simple by Comparison

DNA could be likened to a fascinating 'data-steam' with complex data packets; genes. In addition, it contains instruction packets, previously considered junk (see ENCODE Project Phase One 2013).

Instead of passing between Internet nodes where it's repeated by electronic systems, DNA is repeated from cell to new cell, and to the next generation in germ cells. In a human adult, there are nearly two trillion cell divisions each day.

For data integrity, TCP/IP protocol has checksums. Checksum data is calculated at the transmission and receiving end. A mismatch causes a request for retransmission.

Our cells have a number of data correction mechanisms[92][93][94][95]. New copies of our 6 gbit DNA data are 'proof read' by molecular machines, and errors are usually corrected during and after replication. Mistakes still occur, and DNA variations build up as part of aging. Evolutionists say these mistakes can accumulate over very long periods of time and build new species, but these mutations are more harmful than previously thought (see Codon Degeneracy), and genetic structure is far more complex than previously thought (see ENCODE Project Phase One 2013). Also, bearing in mind the rate of inherited mutations by each generation, nothing of this order seems remotely likely. (see Possible number of positions for 10 new base-pairs and this reference to an Oxford study[18c])

The Internet has packets of data of variable length within the TCP/IP structure. To enable delivery of the data packet, there is metadata. Metadata is data that describes data, including its size, destination, source, and boundaries.

Genes are also highly structured data packets. The data packets are bounded with start code and end markers (stop codons). They carry a data payload of varying length, control data, and astonishingly complex metadata, nested on several levels. Some of these data structures were previously thought to be mutations in degenerate codons (see Codon Degeneracy). Biologists had missed this for decades, and had also thought that gene control data was junk.

A microprocessor with carefully constructed software or firmware is required to separate data from metadata in an incoming TCP/IP data stream. In our cells it's carried out by extremely complex molecular machines. From here on, the limitations of the analogy become more apparent.

Cellular machines identify gene boundaries and *many* data boundaries. One gene can be used to express many proteins, by means of alternative splicing. A fruit fly gene has been found to code as many as 38,000 different protein molecules, each with unique function[22]. For this to happen, different parts of the system have to sense molecular levels, and raise and sense demand signals. Without this, there wouldn't be the correct balance of proteins to support metabolism. There are many tens of thousands of different metabolic and messenger molecules involved.

Extraordinary Data System

Human DNA has c.3.2 billion 'data bits' (3.2Gbp - giga base-pairs). DNA data bits have four states (quaternary) as opposed to computer data's two (binary). Superficially, 3.2 Gbits is equivalent to 6.4 Gbits of computer data. Less than 1 Gbyte to define us is rather humbling. Evolutionists used to think that only 2% was actually data before the ENCODE Project Phase One 2013.

Our eyes and brain even feature a CODEC where video data compression and decompression are performed.
(see Appendix 5: Is the Eye Badly Designed?)

This bewildering array of nested structures, controlled by exquisite precision logic, points to a-priori design.

Epigenetic RAM and ROM Memory

DNA data performs different functions dependent on its location. This location-aware functionality is programmed using something analogous to read-only memory.

When fertilisation of the egg occurs, a series of events happens in a cascade. One event triggers another in an amazingly complex choreographed sequence triggered by the presence of special molecules in the mother's egg. The end result is a child with a biological supercomputer that's said to be the most complex device known to exist in the universe, and which is capable of abstraction so complex that philosophers have difficulty describing it in terms we can grasp.

At first, our DNA is in germ cells where much of the previous ROM data is erased, and then in pluripotent stem cells capable of being transformed into any of the body's many cell types. These cells then differentiate into sub-types and eventually into specialised types such as heart, liver, and so on. At each of these stages of differentiation, epigenetic tags are put on the DNA to identify its cell type. When the cell divides, these 'read-only memory' tags are always copied so that daughter cells are of the same type. In addition, cells actually migrate to the correct location[96].

Epigenetic is a term used to describe the activity of molecules around the genes.

As part of metabolism, genes are modulated (expressed) rather than being switched on and off (see Appendix 1: A Brief History of Time and Unexpected Events in Genomics for more detail). This can happen very rapidly and is controlled by short-term epigenetic tags on genes and their histone support framework. So, the action of these tags could be compared to RAM.

It's interesting to note that the result of epigenetic tags are very noticeable in nature, such as in the various subtypes of finches that Darwin thought were different species (see Epigenetics and Heredity), the stages of metamorphosis of frogs, butterflies and other insects, and in the various forms of ants, bees and termites in colonies.

For a discussion of the programmed memory that defines behaviour, see the section No Instinct - No Life.

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