Wk 4:

**Mutations: evolution’s engine becomes evolution’s end!**

***by***[***Alex Williams***](https://creation.com/alexander-williams)

In neo-Darwinian theory, mutations are uniquely biological events that provide the engine of natural variation for all the diversity of life. However, recent discoveries show that mutation is the purely physical result of the universal mechanical damage that interferes with all molecular machinery. Life’s error correction, avoidance and repair mechanisms themselves suffer the same damage and decay. The consequence is that all multicellular life on earth is undergoing inexorable genome decay. Mutation rates are so high that they are clearly evident within a single human lifetime, and all individuals suffer, so natural selection is powerless to weed them out. The effects are mostly so small that natural selection cannot ‘see’ them anyway, even if it could remove their carriers. Our reproductive cells are not immune, as previously thought, but are just as prone to damage as our body cells. Irrespective of whether creationists or evolutionists do the calculations, somewhere between a few thousand and a few million mutations are enough to drive a human lineage to extinction, and this is likely to occur over a time scale of only tens to hundreds of thousands of years. This is far short of the supposed evolutionary time scales.

**Mutations destroy**

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Ever since Hugo de Vries discovered mutations in the 1890s they have been given a central role in evolutionary theory. De Vries was so enamoured with mutations that he developed an anti-Darwinian saltationist theory of evolution via mutation alone.1 But as more became known, mutations of large effect were found to be universally lethal, so only mutations of small effect could be credibly considered as of value to evolution, and de Vries’ saltationist theory waned. When the Neo-Darwinian Synthesis emerged in the 1930s and 1940s, mutations were said to provide the natural variations that natural selection worked on to produce all new forms of life.

However, directly contradicting mutation’s central role in life’s diversity, we have seen growing experimental evidence that mutations *destroy* life. In medical circles, mutations are universally regarded as deleterious. They are a fundamental cause of ageing,2,3 cancer4,5 and infectious diseases.6

Even among evolutionary apologists who search for examples of mutations that are beneficial, the best they can do is to cite *damaging* mutations that have beneficial *side effects* (e.g. sickle-cell trait,7 a 32-base-pair *deletion* in a human chromosome that confers HIV resistance to homozygotes and delays AIDS onset in heterozygotes,8 *CCR5–delta32* mutation,9 animal melanism,10 and stickleback pelvic spine suppression11). Such results are not at all surprising in the light of the discovery that DNA undergoes up to a million damage and repair events per cell per day.12

**Mutation physics**

Neo-Darwinian theory represents mutations as uniquely biological events that constitute the ‘engine’ of biological variation. However, now that we can see life working in molecular detail, it becomes obvious that mutations are *not* uniquely biological events—they are purely physical events.

All multi-cellular life on earth is undergoing inexorable genome decay because the deleterious mutation rates are so high … and natural selection is ineffective in removing the damage.

Life works via the constant (often lightning-fast) movement of molecular machinery in cells. Cells are totally filled with solids and liquids—there are no free spaces. The molecular machines and the cell architecture and internal structures are made up of long-chain organic polymers (e.g. proteins, DNA, RNA, carbohydrates, lipids) while the liquid is mostly water. All forms of movement are subject to the laws of motion, yet the consequences of this simple physical fact have been almost universally ignored in biology.

Newton’s first law of motion says that a physical body will remain at rest, or continue to move at a constant velocity, unless an external force acts upon it. Think of a message molecule that is sent from one part of a cell to another. Since the cell is full of other molecules, with no empty spaces, the message molecule will soon hit other molecules and either slow down or stop altogether. This is the universal problem known as *friction*.

Friction events can result from many causes, but can be crudely divided into two types: one is referred to as *ploughing* and the other is *shearing*. Ploughing involves the physical displacement of materials to facilitate the motion of an object, while shearing arises from the disruption of adhesive interactions between adjacent surfaces.13

**Figure 1.** A transparent carton of fruit yogurt illustrates how friction in the viscous fluid stopped the motion initiated by mixing the fruit (dark colour) with the yogurt (white colour).

Molecular machines in cells owe a great deal of their structure to hydrogen bonds, but these are rather weak and fairly easily broken. For example, most proteins are long, strongly-bonded chains of amino acids, but these long chains are coiled up into 3-dimensional machine components, and the 3-dimensional structures are held together by hydrogen bonds.14 When such structures suffer mechanical impacts, the transfer of momentum can distort or break the hydrogen bonds and critically damage the molecule’s function.

The inside of a cell has a density and viscosity somewhat similar to yogurt (figure 1). The stewed fruit (dark colour) added to the yogurt during manufacture can be seen swirling out into the white yogurt. The fruit has not continued to disperse throughout the yogurt. It was completely stopped by the initial friction. This is like what happens in a cell—any movement is quickly dampened by friction forces of all kinds coming from all directions.

How do cells cope with this friction? In at least five different ways. First, there are motor proteins available all over the cell that attach to mobile molecules and carry them along the filaments and tubules that make up the cytoskeleton of the cell. Second, these motor proteins are continually re-energized after friction collisions by energy inputs packaged in the form of ATP molecules. Third, there are ‘address labels’ attached to mobile molecules to ensure they are delivered to the correct destination (friction effects continually divert mobile molecules from their course). Fourth, thin films of water cover all the molecular components of cells and provide both a protective layer and a lubricant that reduces the frequency and severity of friction collisions. Fifth, there is a wide range of maintenance and repair mechanisms available to repair the damage that friction causes.

**The friction problem**—and the damage that results from it—is orders of magnitude greater in cells than it is in larger mechanical systems. Biomolecules are very spiky objects with extremely rough and highly adhesive surfaces. They cannot be manufactured and honed to the smoothness that we achieve in our vehicle engine components such as pistons and flywheel pivots, nor can ball-bearings be inserted to reduce the surface contact area, such as we do in wheel axles. As a biological example, consider the rotary motor that drives the bacterial flagellum. The major wear surfaces are on the rotor (attached to the flagellum) and the stator (the housing for the rotor, attached to the cell wall). The stator consists of 22 molecules, set in 11 pairs. The wear rate is so great that the average residence time for a stator molecule in the stator is only about 30 seconds.15 The cell’s maintenance system keeps a pool of about 200 stator molecules in reserve to cope with this huge turnover rate.

Finding suitable lubricants to overcome friction is a major focus in the nanotechnology industry. A special technique called ‘friction force microscopy’ has been developed to quantitatively evaluate potential lubricants.16

This shows that the laws of physics, operating among the viscous components of the cell, both predict and explain the high rate of molecular damage that we observe in DNA. Between 50% and 80% of the DNA in a cell is continually consulted for the information necessary for everyday metabolism. This consultation requires numerous steps that each involve physical deformation of the DNA—moving around within the nucleus, winding and unwinding of the chromatin structures, unzipping the double-helix, binding and unbinding of the transcription machinery, re-zipping the double-helix, rewinding the chromatin structures and shuffling around within the nucleus. Each step of motion is powered by ATP discharges and inevitably causes mechanical damage among the components. While most of this damage is repaired, the repair mechanisms are not 100% perfect because they suffer mechanical damage themselves.17

**Mutations rapidly destroy**

Within neo-Darwinian theory, natural selection is supposed to be the guardian of our genomes because it weeds out unwanted deleterious mutations and favours beneficial ones. Not so, according to genetics expert Professor John Sanford.18 Natural selection can only weed out mutations that have a significant negative effect upon fitness (number of offspring produced). But such ‘fitness’ is affected by a huge variety of factors, and the vast majority of mutations have too small an effect for natural selection to be able to detect and remove them.

Furthermore, if the average mutation rate per person per generation is around 1 or more, then everyone is a mutant and no amount of selection can stop degeneration of the whole population.19 As it turns out, the mutation rate in the human population is *very much greater than 1*. Sanford estimates at least 100, probably about 300, and possibly more.

**All multicellular life suffers**

Two recent reviews of the mutation literature not only confirm Sanford’s claims, but extend them to *all multi-cellular life*.

In a review of the distribution of fitness effects (DFE) of mutations,20 the authors are unable to give any examples of beneficial mutations for humans. In their calculations regarding the rate of deleterious mutations (*MD*) and neutral mutations (*MN*), they use the equalities *MD* = 1 – *MN* and *MN* = 1 – *MD* which both imply that the rate of beneficial mutations is zero. They do give a few non-zero values for beneficial mutation rates in some experimental organisms, but qualify these results by noting the interference of other variables.

In a review of mutation rate variations in eukaryotes,21 the authors admit that all multicellular organisms are undergoing inexorable genome decay from mutations because natural selection cannot remove the damage.22 Their Box 2 and Table 1 list deleterious mutation rates for a wide range of multicellular organisms, noting they are all *underestimates*, with the possible exception of those for the fruit fly *Drosophila melanogaster* with a value of 1.2. The value given for humans is ‘~3’.

Thus, all multicellular life on earth is undergoing inexorable genome decay because the deleterious mutation rates are so high, the effects of the most individual mutations are so small, there are no compensatory beneficial mutations, and natural selection is ineffective in removing the damage.

The wheels have come off the neo-Darwinian juggernaut!

**How long to extinction?**

How long could multicellular life survive in the face of universal genetic degradation? This is a very important question, and I will attempt to answer it by using several different lines of evidence.

**Human ageing and cancer**

We have recently discovered that there is a common biology in cancer and ageing—both are the result of accumulating molecular damage in cells.23 This confirms the arguments outlined above, that for purely physical reasons molecular machinery suffers extremely high damage rates, clearly evident within the lifespan of a single human. Every cell has a built-in time clock to limit this damage and minimize the chance of it becoming cancerous. At every cell division, each *telomere* (the caps on both ends of a chromosome that stop the double-helix from unravelling) is shortened by a small amount, until they reach the *Hayflick Limit*—discovered in 1965 to be a little over 50 cell divisions. The cells then stop dividing and they are dismantled and their parts are recycled.

By adding the enzyme *telomerase*, the telomere shortening problem can be circumvented, but that then exposes the cell to a greater risk of becoming cancerous because of accumulating damage elsewhere in the cell. The overall balance between protection from damage and the need for longevity determines fitness (reproductive success) and life span.24 The body’s normal reaction to increasing genome damage is to kill off the damaged cells via programmed senescence (of which the telomere clock with its Hayflick limit is but one part). But cells become malignant (cancerous) when mutation disables the senescence mechanism itself, which then enables the damaged cells to proliferate without limit.22 The Hayflick limit of around 50 cell divisions for humans seems to provide the optimum balance.

Fifty human generations of 20 years each gives us only 1,000 years as a timescale over which a human lineage would begin to experience a significant mutation load in its genome. This is alarmingly rapid compared with the supposed evolutionary time scale of millions and billions of years.

**Reproductive cells**

**Figure 2.** Schematic representation of human life expectancy (—), male fertility (…), and risk of fetal abnormality with mother’s age (---). Despite the protective Hayflick limit on cell divisions and life expectancy, very significant molecular damage accumulates in humans even during the most productive years of life. Mutations do even more damage than the Hayflick limit and associated cancer rates suggest.

Ever since August Weismann published *The Germ-Plasm: A Theory of Heredity*25 in 1893, a discrete separation has been shown to exist between body cells (the *soma*) and germ-line cells (*germplasm*). Germ-line cells were thought to be more protected from mutation than other body cells. However, another recently discovered cause of ageing is that our stem cells grow old as a result of heritable DNA damage and degeneration of their supporting *niches* (the special ‘nest’ areas in most organs and tissues of the body where stem cells grow and are nurtured and protected). The telomere shortening mechanism—intended to reduce cancer incidence—appears to also induce the unwanted side-effect of a decline in the replicative capacity of certain stem-cell types with advancing age. This decreased regenerative capacity has led to a ‘stem-cell hypothesis’ for human age-associated degenerative conditions.26

Human fertility problems suggest that the decline in niche protection of stem cells also applies to our gametes (eggs and sperm). For males, fertility—as measured by sperm count, sperm vigor and chance of conception—begins to decline significantly by age 40 and the rate of certain paternal-associated birth defects increases rapidly during the 30s (figure 2).27 For females, the chance of birth defects increases rapidly from around the mid-30s, particularly because of chromosome abnormalities (figure 2). In the middle of the most productive part of our lives, our bodies are therefore showing clear evidence of decline through accumulation of molecular damage in our genomes.

**Do germ-line cells really suffer less damage?**

When DNA was discovered to be the carrier of inheritance, Weissman’s germ-plasm theory gave rise to the ‘immortal strand hypothesis.’ When the DNA of an embryonic stem cell replicates itself, it was thought that the ‘old’ strand would remain with the self-renewing ‘mother’ stem cell, while the newly constructed daughter strand proceeds down the path of differentiation into a body cell. In this way, the ‘old’ strand would remain error free—because it has not suffered any copying errors—and thus becomes effectively immortal.

However, a research team at the Howard Hughes Memorial Institute recently tested this theory using the stem cells that produce blood, and found that they segregate their chromosomes randomly.28 That is, the ‘immortal strand hypothesis’ is wrong. If stem cells are not given this kind of preferential treatment then it is reasonable to conclude that germ-line cells are also subject to the same molecular damage as somatic cells. This is confirmed by the observation that human fertility exhibits damage long before age-related diseases take over.

A single human lifetime is enough to show very significant mutation damage, even in our reproductive cells.

**Haldane’s dilemma**

The severe contradictions that these findings pose for neo-Darwinian theory corroborate what has become known as *Haldane’s dilemma*. J.B.S. Haldane was one of the architects of neo-Darwinism who pioneered its application to population biology. He realized that it would take a long time for natural selection to fix an advantageous mutation in a population—fixation is when every member has two copies of an allele, having inherited it from both mother and father. He estimated that for vertebrates, about 300 generations would be required, on average, where the selective advantage is 10%. In humans, with a 20-year generation time and about 6 million years since our last common ancestor with the chimpanzee, only about 1,000 such advantageous mutations could have been fixed. Haldane believed that substitution of about 1,000 alleles would be enough to create a new species, but it is not nearly enough to explain the observed differences between us and our closest supposed relatives.

The measured difference between the human and chimpanzee genomes amounts to about 125 million nucleotides, which are thought to have arisen from about 40 million mutation events.29 If only 1000 of these mutations could have been naturally selected to produce the new (human) species, it means the other 39,999,000 mutations were deleterious, which is completely consistent with the reviews showing that the vast majority of mutations are deleterious. Consequently, we must have *degenerated* from the apes, which is an absurd conclusion.

According to Kirschner and Gerhart’s facilitated variation theory,30 life consists of two main components—*conserved core processes* (the structure and machinery in cells) and *modular regulatory processes* (the signalling circuits and switches that operate the machinery and provide a built-in source of natural variation). The 40 million ‘mutation’ differences between humans and chimps are therefore much more reasonably explained as 40 million *modular differences* between the *design* of chimps and the *design* of humans.

**Quantitative estimates of time to extinction**

There are a number of different ways to estimate the time it would take for relentlessly accumulating mutations to send our species to extinction.

**Binomial estimates**

Some very rough estimates can be derived from the Binomial distribution, which can predict the likelihood of multiple mutations accumulating in an essential genetic functional module. A binomial model of a mutating genome could consist of the cell’s DNA being divided into *N* functional modules, of which *Ne* are essential; that is, the lineage fails to reproduce if any of the essential modules are disabled. For any given mutational event, *p*= 1/*N* is the probability of being ‘hit’, *q* is the probability of being ‘missed’, and *p q* = 1.

What is the likely value of *N*? We can derive two estimates from the knowledge that there are about 25,000 genes, plus the discovery from the pilot study report of the ENCODE project that virtually the whole human genome is functional.31

For the first estimate, the average protein contains a few hundred amino acids and each amino acid requires three nucleotides of code, so the average gene would take up about 1,000 nucleotides of exon space (an *exon* is the protein-coding part of a gene). There are about 3 billion nucleotides in the whole human genome, so if we assume that the average protein represents an average functional unit then *N* = 3 million.

The second estimate comes from the ENCODE report that gene regions produce on average 5 RNA transcripts per nucleotide, and the untranslated regions produce on average 7 RNA transcripts per nucleotide. There are about 33 times as many nucleotides in the untranslated regions as in the genic regions. Assuming that transcript size is approximately equal in each region, then there are 25,000 x 5 = 125,000 gene transcripts and 25,000 x 33 x 7 = 5,775,000 untranslated transcripts, making *N* = 5,900,000 in total. Our two estimates of *N* are therefore 3 to 6 million in round figures.

What is the likely value of *Ne*? Experiments with mice indicate that 85% of genes can be knocked out one at a time without lethal effects.32 This is due to the robustness and failure-tolerance through fallback processes built into the genomic designs. That means any one of those remaining 15% of genes will be fatal if disabled. Multiple mutations occur however, so the likely value of *Ne* when exposed to multiple mutations will be much higher than 15%. The maximum possible value is 100%. In a study of 2,823 human metabolic pathways, 96% produced disease conditions when disrupted by mutation,33 so if we take an average between this value and the minimum 15% then we get about 60% of functional units being essential.

How many random mutations are required on average to disable an essential functional module? In rare cases, a single mutation is enough to disable a person’s ability to reproduce. A two-hit model is common in cancer. In a study of cell signalling networks, these two hits usually knocked out: (i) the programmed death system for dealing with damaged (cancerous) cells, and (ii) the normal controls on cell proliferation—so the damaged cancer cells can proliferate without limit. The proportion of cancer-associated genes was also found to increase with the number of linkages between genes. When a healthy gene is linked to more than 6 mutated genes, ~80% of all genes in the network are cancerous. Extrapolating from this, we find that by the time a normal gene is linked to about 10 mutated genes, then the whole network has become cancerous.34

Almost 70% of known human genes can be causal agents of cancer when mutated.35 Cancers can result from as little as a single mutation in a stem cell, or multiple mutations in somatic cells.36 The minimum possible value of 1 is known to be rare, so the more common occurrence of the 2-hit model makes it a reasonable best-estimate minimum. But it may require 10 modules to receive two hits each for the whole network to become dysfunctional.

The maximum number of hits required to disable a single module may be 100 or more, but if the average functional module only contains 1,000 nucleotides then this figure, at 10% of the whole, seems rather large. An order-of-magnitude average is perhaps more likely to be 10 random mutations per functional module.

To provide some context for these estimates, recent work shows that the cell-cycle checkpoint damage repair system is activated when 10 to 20 double-strand breaks accumulate in a cell undergoing division.37 That is, life will tolerate only 10 to 20 DNA breaks per cell before it starts repair work, whereas we are examining scenarios in which there are thousands and millions of damage events per cell. Our numbers are clearly up in a region where the cell’s repair mechanisms are working at their hardest.

What then is the likelihood of accumulating either 2 hits in 10 modules, or 10 hits in one module, in any one of either 15% or 60% of the 3 to 6 million functional modules? The binomial distribution in Microsoft Excel was used to make the following calculations, making the further assumption that the likelihood of the unit being a critical one must exceed 50% for extinction to be more likely than not in the next generation.

Assuming 60% essentiality, only one functional module needs to be disabled for the probability of its essential status to exceed 50%. For the 2-hit model, about 6,000 to 12,000 mutations are required to disable ten of the 3 to 6 million functional modules. For the 10-hit model, 3 to 6 million mutations are required to disable one functional module.

Assuming 15% essentiality, four modules need to be disabled before the probability of at least one of them being essential exceeds 50%. For the 2-hit model, 250,000 to 500,000 mutations are required to disable ten modules with four mutations each among the 3 to 6 million functional modules. For the 10-hit model, 3.7 to 7.5 million mutations are required to disable four functional modules.

If every individual produces 100 new mutations every generation (assuming a generation time of 20 years) and these mutations are spread among 3 to 6 million functional modules across the whole genome, then the average time to extinction is:

* 1,200 to 2,400 years for the 2-hits in 10 modules model and 60% essentiality
* 50,000 to 100,000 years for the 2-hits in 10 modules model and 15% essentiality
* 600,000 to 1,200,000 years for the 10-hit model and 60% essentiality
* 740,000 to 1,500,000 years for the 10-hit model and 15% essentiality.

**Truncation selection**

Evolutionary geneticist Dr James Crow argued that humans are probably protected by ‘truncation selection’.27 Truncation occurs when natural selection preferentially deletes individuals with the highest mutation loads. Plant geneticist John Sanford put Crow’s claims to the test by developing a computer simulation of truncation. His assumptions were: 100 individuals in the population, 100 mutations per person per generation, 4 offspring per female, 25% non-genetic random deaths per generation, and 50% selection against the most mutant offspring per generation. He assumed an average fitness loss per mutation of 1 in 10,000. His species became extinct in only 300 generations. With a generation time of 20 years this corresponds to 6,000 years.38

Sanford’s assumptions are somewhat unrealistic, but there are other ways to approach the problem. Mutations are pure chance events that follow a Poisson distribution, and this behaves like the normal curve when the average expected value is greater than about 30.39 In a Poisson distribution, the variance is equal to the average expected value, and the standard deviation is the square root of the variance. When the expected average value is 100, the standard deviation will be 10. The normal curve now tells us the following:

* Half the people will suffer about 100 mutations or more, and half the people will suffer about 100 mutations or less.
* About 84% of people will suffer 110 mutations or less, and so the remaining 16% of people will suffer 110 or more mutations. Alternatively, about 16% of people will suffer 90 or less.
* About 97.7% of the population will experience 120 mutations or less, and the remaining 2.3% will suffer 120 mutations or more. Alternatively, 2.3% will suffer 80 or less.
* About 99.9% of the population will suffer 130 mutations or less, and the remaining 0.1% will suffer 130 or more mutations. Alternatively, 0.1% will suffer 70 or less.

If we remove the most mutant—those above 130 mutations per person per generation—then we will only remove 0.1% of the population and it will make virtually no difference. If we removed the most mutant 50% of the population that would not solve the problem either, for two reasons. First, the great majority of the remaining people still suffer between 70 and 100 mutations per person per generation, far above the value of 1 that ensures inexorable decline. Second, removing half the population each generation would send it extinct in a few dozen generations.

**Table 1.** Estimated number of generations and years to extinction for populations of various sizes, when fitness declines by 1.5% in each generation.

**Synergistic epistasis and population size**

None of the above models include the effect of *synergistic epistasis* (if one gene is mutated, its impact is ameliorated by the coordinated activity of other genes) or of population size. We can include these by using Crow’s estimate that the fitness of the human race is currently degenerating at a rate of about 1 to 2% per generation. If we use an average value of 1.5% then only 98.5% of the next generation will produce reproductively viable offspring. The next generation after that will only have 98.5% of those survivors able to produce reproductively viable offspring, and so on.

For any given stable population size *N*, the size of the next generation that can produce reproductively viable offspring will be 98.5% of *N*, and for any given number of generations *G*, the number of survivors able to produce reproductively viable offspring will be (98.5%)*G* of *N*.

Table 1 shows the approximate numbers of generations after which the population degenerates to extinction (only one individual is left, so breeding cannot continue). No population can sustain a continual loss of viability of 1.5%.

Like rust eating away the steel in a bridge, mutations are eating away our genomes and there is nothing we can do to stop them.

The above model assumes that right from the beginning there will be 1.5% loss of fitness each generation. However, the binomial simulations earlier showed that individuals can tolerate somewhere between a few thousand to a few million mutations before the damage critically interferes with their ability to reproduce. This means that *synergistic epistasis* is a real phenomenon—life is robust in the face of mutational assault. Instead of the immediate loss of 1.5% every generation, the general population would remain apparently healthy for a much longer time before the damage became apparent.

However, the rate at which mutations accumulate will remain the same because the cause remains the same—mechanical damage. This means that most people will be apparently healthy, but then approach the threshold of dysfunction over a much shorter period, creating a population crash rather than a slow decline.

Either way, however, the time scales will be approximately the same because the rate of damage accumulation remains approximately the same.

**Summary**

Mutations are not uniquely biological events that provide an engine of natural variation for natural selection to work upon and produce all the variety of life. Mutation is the purely physical result of the all-pervading mechanical damage that accompanies all molecular machinery. As a consequence, *all multicellular life on earth* is undergoing inexorable genome decay because the deleterious mutation rates are so high, the effects of the individual mutations are so small, there are no compensatory beneficial mutations and natural selection is ineffective in removing the damage.

So much damage occurs that it is clearly evident within a single human lifetime. Our reproductive cells are *not* immune, as previously thought, but are just as prone to mechanical damage as our body cells. Somewhere between a few thousand and a few million mutations are enough to drive a human lineage to extinction, and this is likely to occur over a time scale of only tens to hundreds of thousands of years. This is far short of the supposed evolutionary time scales. Like rust eating away the steel in a bridge, mutations are eating away our genomes and there is nothing we can do to stop them.

Evolution’s engine, when properly understood, becomes evolution’s end.

**Isn’t it *obvious*? Natural selection can *eliminate*, but *never create*!**

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Many proponents of evolution repeatedly cite examples of natural selection as evidence of evolution—i.e. evidence of the sorts of processes that could have turned microbes into man, given enough time. Often the terms ‘natural selection’ and ‘evolution’ are used interchangeably, as if they were synonymous.1

Occasionally an evolutionist will speak out against such error. One notable example was John Endler, who in his 1986 book *Natural Selection in the Wild*2 warned that “natural selection must not be equated with evolution”, and he also said:

Natural selection is common enough in natural populations to have been detected in a wide variety of organisms … However, natural selection does not explain the origin of new variants, only the process of changes in their frequency.

Endler had seen this himself in his own previous research on guppies in mountain streams in Trinidad, Tobago and Venezuela.3 He observed that populations of guppies there include drab-coloured males as well as brightly coloured ones, and the relative frequency of each goes up or down in line with predation pressure. If predators are few or absent, brightly coloured males predominate, as female guppies prefer them as mates; so gaudy males are more likely to pass their genes to the next generation. But when predators are numerous, the better camouflaged drab male guppies are less likely to be eaten than the gaudy ones, and so the females have to be content to mate with the survivors, thus drabness becomes predominant in the population.

“Natural selection eliminates and maybe maintains, but it doesn’t create.”—Dr Lynn Margulis (1938–2011)

The guppy population dynamics are indeed a terrific example of natural selection. Ironically, however, Endler’s warning in his 1986 book hasn’t stopped others from incorrectly misrepresenting his guppies as demonstrating evolution. E.g. Richard Dawkins proclaimed it to be “a spectacular example of evolution before our very eyes”4 but it most definitely is *not*, as there is *no new genetic information* in evidence anywhere here. There is not a shred of any evidence from Endler’s creditable guppy research that fish could have turned into *fishermen, fishmongers and fish physiologists*(yet that is what the evolution story would have us believe).5

As *Creation* magazine has pointed out many times, natural selection has been amply observed happening in many populations of insects, animals, fish, and plants, but in all instances it is *not* evolution.6 Whether by differential reproduction or differential survival, natural selection results in the *culling* or *loss* of genetic information, not its creation.

That last point was certainly obvious to another noted evolutionist who spoke out against the natural-selection-equals-evolution sham, *viz.*, the late Lynn Margulis.7 Just before her death in 2011, she said in an interview:

Natural selection eliminates and maybe maintains, but it doesn’t create.8

That’s telling it like it is: natural selection by itself generates no new genetic information. It can *eliminate* genes that already exist, but *never create*.

**Evolution evidence evades evolutionists (and everyone else!)**

“I was taught over and over again that the accumulation of random mutations led to evolutionary change— … I believed it until I looked for evidence.”—Dr Lynn Margulis(1938–2011)

According to standard evolutionary theory today, evolutionists look to *mutations* as being the process responsible for generating the new genetic information evolution requires, which is then sorted by natural selection. But where is the *evidence* of this happening? Margulis had a very blunt, and bleak, assessment of this. Note again that she was no creationist, being absolutely committed to Darwin’s ideas of evolution, which she happily defined as, “Darwinism says that there has been change through time, since all life comes from a common ancestor”.8 But her biological experience and observations of nature had made her contemptuous of the neo-Darwinists’ faith in mutations as being the engine9 of evolution:

[N]eo-Darwinists say that [evolutionary change occurs] when mutations occur and modify an organism. I was taught over and over again that the accumulation of random mutations led to evolutionary change— … I believed it until I looked for evidence.8

Indeed, when other evolutionists over the years have been pressed to give specific evidence of mutations that increase the information in the genome, they are unable to give coherent answers.10 That’s because mutations are overwhelmingly a *downhill* process—see box p. 40 “Mutations can’t climb mountains!”.

Is it any wonder that Margulis went looking for some other mechanism that might provide a means of evolutionary change, latching on to her own pet theory of *endosymbiosis*?8 Not that there’s any evidence of any evolution there, either.11 But Margulis was certainly right to point out the dearth of evidence for mutations and natural selection ever being able to create the evolutionary change required for all living things to have arisen from a common ancestor.

**Examples of natural selection and mutations galore—but no evolution**

As with Endler’s guppies, when one looks at the evidence in the real world, stand-out examples of natural selection and mutations show a consistent *loss* or mere *maintenance* of genetic information, not the gains that microbes-to-man evolution requires:

Evolution needs to invent eyesight, not destroy it!

* **Dance-or-die lizards:** Fire ants invading parts of the USA quickly cull out fence lizards that don’t do the ‘twitch dance’ to shake off biting ants. Only the twitch-dancing lizards survive—a characteristic which was *already present* in the lizard population. In the face of such natural selection, the lizard population is now better *adapted* to maintain a presence in fire ant areas.12 Note: no new genetic information, therefore no evolution.
* **Kauai’s silent crickets:** A deadly parasitic fly on the island of Kauai acoustically tracks down chirping male crickets—but crickets with an X-chromosome mutation that silences them evade detection by the killer fly. Somehow the mutant mute crickets have been able to find mates, and pass their mutated genes to the next generation. Note that genetic information (for chirping) has been *eliminated*. In this classic example of natural selection and genome degradation, Kauai’s crickets are now silent, but at least they survive.13
* **Not-so-dark deer mice:**In Nebraska’s sand hills, deer mice with a mutation conferring pale colouration are better camouflaged against bird predators than normal deer mice. But the mutation is a *downhill* change, not gain-of-function—no justification whatsoever for those trying to claim this as the latest ‘icon of evolution’.14
* **Shorter-winged swallows:** Cliff swallows live in mud nests they build on highway bridges and road culverts. But when flying out from the nest they are vulnerable to being killed by passing vehicles—particularly those with longer wings, less able to take off vertically than birds with shorter wings. The genes for longer wings are thus being progressively *eliminated*, leaving behind a population with most birds having shorter wings.15
* **Wingless beetles**: In the absence of their usual predators found on the mainland, the mutant flightless offspring of beetles on windswept islands are favoured by natural selection over their winged prone-to-being-blown-out-to-sea siblings. But there’s no gain-of-function evolution here—the genes for flight have been *lost*.16
* **Smaller fish to fry**: It’s getting harder to catch fish of even minimum legal size—in some commercial fishery populations (e.g. Canadian cod) the genes for large size have been *completely eliminated*.17,18
* **Shorter-tusked elephants:** By selectively killing elephants with the largest tusks, poachers for the ivory trade have left behind elephant populations having short, or even no, tusks. The genes for large tusks are being *eliminated*.19

This observed genetic change is in the wrong direction for primordial ooze to have ever turned into plants and animals.

* **Blind cave fish:** Living in dark, underwater caves, natural selection favours eyeless mutant fish over their sighted kin. Eyes in such an environment are a disadvantage as the delicate tissue is prone to injury as fish bump against sharp rocks in the darkness, becoming an entry point for potentially lethal bacteria. Eyelessness clearly represents a *loss*, not a gain,20 yet leading evolutionists bizarrely have claimed it as supporting their case.21 But evolution needs to *invent* eyesight, not destroy it!
* **Not-so-tall snow lotus:** Highly prized for traditional Chinese medicine, the Tibetan snow lotus has halved in height during the past century, because every year at flowering time people scour the alpine slopes for the taller plants considered more potent. Only the smaller plants have been left behind to produce seed, and thus the genes for snow lotus tallness are being progressively *eliminated* from the Himalayas.22 Far from being one of the top seven examples of ‘evolution in action’ as some claim,23 this observed genetic change is in the *wrong direction* for primordial ooze to have ever turned into plants and animals.

From a straightforward view of the evidence from these and other real-world examples, surely it’s obvious: natural selection can *eliminate*, but *never create*. And we see that mutations are no help to the microbes-to-man evolutionary storyline either. The facts instead fit the Bible’s historical account of our post-Fall world having originally been created “very good” ([Genesis 1:31](https://biblia.com/bible/esv/Gen%201.31)) but now being “in bondage to decay” ([Romans 8:19–22](https://biblia.com/bible/esv/Rom%208.19%E2%80%9322)). The evidence is there for all to see—and the wise to comprehend.

**Mutations can’t climb mountains**



Mutations overwhelmingly degrade genetic information, a *downhill* process. Whereas for microbes-to-man evolution to be true, evolutionists should be able to point to thousands of examples of information-gaining mutations, an *uphill* process, but they can’t. Note that sometimes diehard anti-creationists quibble over the definition of ‘information’. As information is foundationally an argument from probability, we might expect a few cases of trivial information increase (see the CMI DVD *Understanding the Law of Decay*, and [creation.com/edge-evolution](https://creation.com/edge-evolution)). But evolution requires *encyclopedic amounts of new information*. Some evolutionists have pointed to nylon-eating bacteria as being a lead candidate, but unfortunately for proponents of evolutionary theory, it turns out not to be new information. Rather, the new ‘ability’ comes from two ‘typos’ in an existing enzyme finely-tuned to break bonds in certain chemicals. The mutated enzyme is less tuned for its current task, but can digest other chemicals, including nylon, with the same bond ([creation.com/evoquest#nylonase](https://creation.com/evoquest#nylonase), [creation.com/infoloss](https://creation.com/infoloss); see also [creation.com/new-info](https://creation.com/new-info)). Such mutations are therefore evidence of downhill change, not uphill. They are thus of no help to evolutionists in the climbing of the ‘Evolution Mountain’—representing evolution’s mooted uphill journey from microbes to *marlin, macaws, magnolias, and man*—no matter how many millions or billions of years are invoked.

***Refuting Evolution*—Chapter 2**

A handbook for students, parents, and teachers countering the latest arguments for evolution

***by***[***Jonathan Sarfati***](https://creation.com/dr-jonathan-d-sarfati)***, Ph.D., F.M.***

Variation and natural selection versus evolution

**First published in**[***Refuting Evolution***](https://creation.com/refuting-evolution-index)**, Chapter 2**

This chapter contrasts the evolution and creation models, and refutes faulty understandings of both. A major point is the common practice of *Teaching about Evolution and the Nature of Science*to call all change in organisms ‘evolution.’ This enables *Teaching about Evolution*to claim that evolution is happening today. However, creationists have never disputed that organisms change; the difference is the *type* of change. A key difference between the two models is whether observed changes are the type to turn particles into people.

**Evolution**

Evolution, of the fish-to-philosopher type, requires that non-living chemicals organize themselves into a self-reproducing organism. All types of life are alleged to have descended, by natural, ongoing processes, from this ‘simple’ life form. For this to have worked, there must be some process which can generate the genetic information in living things today. Chapter 9 on ‘Design’ shows how encyclopedic this information is.

So how do evolutionists propose that this information arose? The first self-reproducing organism would have made copies of itself. Evolution also requires that the copying is not always completely accurate—errors (mutations) occur. Any mutations which enable an organism to leave more self-reproducing offspring will be passed on through the generations. This ‘differential reproduction’ is called *natural selection*. In summary, evolutionists believe that the source of new genetic information is mutations sorted by natural selection—the neo-Darwinian theory.

**Creation**

In contrast, creationists, starting from the Bible, believe that God created different kinds of organisms, which reproduced ‘after their kinds’ ([Gen. 1:11–12](https://biblia.com/bible/esv/Gen.%201.11%E2%80%9312), [21](https://biblia.com/bible/esv/Gen%201.21), [24–25](https://biblia.com/bible/esv/Gen%201.24%E2%80%9325)). Each of these kinds was created with a vast amount of information. There was enough variety in the information in the original creatures so their descendants could adapt to a wide variety of environments.

All (sexually reproducing) organisms contain their genetic information in *paired* form. Each offspring inherits half its genetic information from its mother, and half from its father. So there are two genes at a given position (*locus*, plural *loci*) coding for a particular characteristic. An organism can be heterozygous at a given locus, meaning it carries different forms (*alleles*) of this gene. For example, one allele can code for blue eyes, while the other one can code for brown eyes; or one can code for the A blood type and the other for the B type. Sometimes two alleles have a combined effect, while at other times only one allele (called *dominant*) has any effect on the organism, while the other does not (*recessive*). With humans, both the mother’s and father’s halves have [20,687 protein-coding genes](http://www.sciencedaily.com/releases/2012/09/120905134912.htm), while [97% of the rest of the DNA has an important role in coding for RNA, for control of gene expression](https://creation.com/astonishing-dna-complexity-update). Overall, the information equivalent to a thousand 500-page books (3 billion base pairs, as *Teaching about Evolution* correctly states on page 42). The ardent neo-Darwinist Francisco Ayala points out that humans today have an ‘average heterozygosity of 6.7 percent.’1 This means that for every thousand gene pairs coding for any trait, 67 of the pairs have different alleles. If we consider only the protein-coding genes, this would mean 1,340 heterozygous loci overall. Thus, any single human could produce a vast number of different possible sperm or egg cells 2¹³⁴⁰ or 2.4 × 10⁴⁰³. The number of atoms in the whole known universe is ‘only’ 10⁸⁰, extremely tiny by comparison. So there is no problem for creationists explaining that the original created kinds could each give rise to many different varieties. In fact, the original created kinds would have had much more heterozygosity than their modern, more specialized descendants. No wonder Ayala pointed out that most of the variation in populations arises from reshuffling of previously existing genes, not from mutations. Many varieties can arise simply by two previously hidden recessive alleles coming together. However, Ayala believes the genetic information came ultimately from mutations, not creation. His belief is contrary to [information theory](https://creation.com/information-theory-questions-and-answers), as shown in [chapter 9 on ‘Design’](https://creation.com/refuting-evolution-chapter-9-is-the-design-explanation-legitimate).

**Deterioration from perfection**

An important aspect of the creationist model is often overlooked, but it is essential for a proper understanding of the issues. This aspect is the *deterioration* of a once-perfect creation. Creationists believe this because the Bible states that the world was created perfect ([Gen. 1:31](https://biblia.com/bible/esv/Gen.%201.31)), and that death and deterioration came into the world because the first human couple sinned ([Gen. 3:19](https://biblia.com/bible/esv/Gen.%203.19), [Rom. 5:12](https://biblia.com/bible/esv/Rom.%205.12), [8:20–22](https://biblia.com/bible/esv/Rom%208.20%E2%80%9322), [1 Cor. 15:21–22](https://biblia.com/bible/esv/1%20Cor.%2015.21%E2%80%9322), [26](https://biblia.com/bible/esv/1%20Cor%2015.26)) [see [The Fall: a cosmic catastrophe](https://creation.com/the-fall-a-cosmic-catastrophe-journal-of-creation-tj)]

As the previous chapter showed, all scientists interpret facts according to their assumptions. From this premise of perfection followed by deterioration, it follows that mutations, as would be expected from copying errors, destroyed some of the original genetic information. Many evolutionists point to allegedly imperfect structures as ‘proof’ of evolution, although this is really an argument against perfect design rather than for evolution. But many allegedly imperfect structures can also be interpreted as a deterioration of once-perfect structures, for example, eyes of blind creatures in caves. However, this fails to explain how sight could have arisen in the first place.2

**Adaptation and natural selection**

Also, the once-perfect environments have deteriorated into harsher ones. Creatures adapted to these new environments, and this adaptation took the form of *weeding out* some genetic information. This is certainly natural selection—evolutionists don’t have a monopoly on this. In fact, a creationist, Edward Blyth, thought of the concept 25 years before Darwin’s *Origin of Species* was published. But unlike evolutionists, Blyth regarded it as a *conservative* process that would remove defective organisms, thus conserving the health of the population as a whole. Only when coupled with hypothetical information-gaining mutations could natural selection be creative.

For example, the original dog/wolf kind probably had the information for a wide variety of fur lengths. The first animals probably had medium-length fur. In the simplified example illustrated below,3 a single gene pair is shown under each dog as coming in two possible forms. One form of the gene (L) carries instructions for long fur, the other (S) for short fur.

In row 1, we start with medium-furred animals (LS) interbreeding. Each of the offspring of these dogs can get one of either gene from each parent to make up their two genes.



In row 2, we see that the resultant offspring can have either short (SS), medium (LS) or long (LL) fur. Now imagine the climate cooling drastically (as in the Ice Age). Only those with long fur survive to give rise to the next generation (line 3). So from then on, all the dogs will be a new, long-furred variety. Note that:

1. They are now *adapted* to their environment.
2. They are now more *specialized* than their ancestors on row 1.
3. This has occurred through *natural selection.*
4. There have been *no new genes* added.
5. In fact, genes have been lost from the population—i.e., there has been *a loss of genetic information*, the opposite of what microbe-to-man evolution needs in order to be credible.
6. Now the population is less able to adapt to future environmental changes—were the climate to become hot, there is no genetic information for short fur, so the dogs would probably overheat.

Another information-losing process occurs in sexually reproducing organisms—remember, each organism inherits only half the information carried by each parent. For example, consider a human couple with only one child, where the mother had the AB blood group (meaning that she has both A and B alleles) and the father had the O blood group (both alleles are O and recessive). So the child would have either AO or BO alleles, so either the A or the B allele *must* be missing from the child’s genetic information. Thus, the child could not have the AB blood group, but would have either the A or the B blood group respectively.4

A large population as a whole is less likely to lose established genes because there are usually many copies of the genes of both parents (for example, in their siblings and cousins). But in a small, isolated population, there is a good chance that information can be lost by random sampling. This is called *genetic drift*. Since new mutant genes would start off in small numbers, they are quite likely to be eliminated by genetic drift, even if they were beneficial.5

In an extreme case, where a single pregnant animal or a single pair is isolated, e.g., by being blown or washed onto a desert island, it may lack a number of genes of the original population. So when its descendants fill the island, this new population would be different from the old one, with less information. This is called the *founder effect*.

Loss of information through mutations, natural selection, and genetic drift can sometimes result in different small populations losing such different information that they will no longer interbreed. For example, changes in song or color might result in birds no longer recognizing a mate, so they no longer interbreed. Thus a new ‘species’ is formed.

**The Flood**

Another aspect of the creationist model is the Bible’s teaching in Genesis chapters 6 to 8 that the whole world was flooded, and that a male and female of every kind of land vertebrate (animals with biblical life in the Hebrew נֶפֶשׁ חַיָּה (*nephesh chayyāh* sense) were saved on Noah’s ark. A few ‘clean’ animals were represented by seven individuals ([Gen. 7:2](https://biblia.com/bible/esv/Gen.%207.2)). The Bible also teaches that this ark landed on the mountains of Ararat. From these assumptions, creationists conclude that these kinds multiplied and their descendants spread out over the earth. ‘Founder effects’ would have been common, so many ‘kinds’ would each have given rise to several of today’s ‘species.’

**Contrasting the Models**

Once biblical creation is properly understood, it is possible to analyze the ‘evidence’ for ‘evolution as a contemporary process’ presented by *Teaching about Evolution* on pages 16–19. The three diagrams below should help:

|  |
| --- |
| tree diagram**Figure 1:** The evolutionary ‘tree’ which postulates that all today’s species are descended from the one common ancestor (which itself evolved from non-living chemicals). This is what evolution is really all about. |
| Lawn diagram**Figure 2:** The alleged creationist ‘lawn’ this represents the caricature of creationism presented by *Teaching about Evolution* —the Genesis ‘kinds’ were the same as today’s species. |
| Orchard diagram**Figure 3:** The true creationist ‘orchard’ diversity has occurred with time within the original Genesis ‘kinds’ (creationists often call them *baramin*, from Hebrew *bara* = create, and *min* = kind). Much of the evidence of variation presented by *Teaching about Evolution* refutes only the straw-man version of creationism in Figure 2, but fits the true creationist ‘orchard’ model perfectly well. |

**The alleged evidence for evolution in action**

This section will deal with some of the examples used by*Teaching about Evolution*, and show that they fit the creationist model better.

**Antibiotic and pesticide resistance**

*Teaching about Evolution* claims on pages 16–17:

The continual evolution of human pathogens has come to pose one of the most serious health problems facing human societies. Many strains of bacteria have become increasingly resistant to antibiotics as natural selection has amplified resistant strains that arose through naturally occurring genetic variation.

Similar episodes of rapid evolution are occurring in many different organisms. Rats have developed resistance to the poison warfarin. Many hundreds of insect species and other agricultural pests have evolved resistance to the pesticides used to combat them—even to chemical defenses genetically engineered into plants.

However, what has this to do with the evolution of *new kinds* with *new genetic information*? Precisely nothing. What has happened in many cases is that some bacteria *already* had the genes for resistance to the antibiotics. In fact, some bacteria obtained by thawing sources which had been frozen before man developed antibiotics have shown to be antibiotic-resistant. When antibiotics are applied to a population of bacteria, those lacking resistance are killed, and any genetic information they carry is eliminated. The survivors carry less information, but they are all resistant. The same principle applies to rats and insects ‘evolving’ resistance to pesticides. Again, the resistance was already there, and creatures without resistance are eliminated.

In other cases, antibiotic resistance is the result of a mutation, but in all known cases, this mutation has destroyed information. It may seem surprising that destruction of information can sometimes help. But one example is resistance to the antibiotic penicillin. Bacteria normally produce an enzyme, penicillinase, which destroys penicillin. The amount of penicillinase is controlled by a gene. There is normally enough produced to handle any penicillin encountered in the wild, but the bacterium is overwhelmed by the amount given to patients. A mutation disabling this controlling gene results in much more penicillinase being produced. This enables the bacterium to resist the antibiotic. But normally, this mutant would be less fit, as it wastes resources by producing unnecessary penicillinase.

Another example of acquired antibiotic resistance is the transfer of pieces of genetic material (called *plasmids*) between bacteria, even between those of different species. But this is still using *pre-existing* information, and doesn’t explain its *origin*.

More information on antibiotic resistance can be found in the article [Superbugs not super after all](https://creation.com/superbugs-not-super-after-all-creation-magazine).6

**Lacewing species**

Another example of ‘evolution’ is given on page 17, where *Teaching about Evolution* states:

The North American lacewing species *Chrysoperla carnea* and *Chrysoperla downesi* separated from a common ancestor species recently in evolutionary time and are very similar. But they are different in color, reflecting their different habitats, and they breed at different times of year.

This statement is basically correct, but an evolutionary interpretation of this statement is not the only one possible. A creationist interpretation is that an original *Chrysoperla*kind was created with genes for a wide variety of colors and mating behavior. This has given rise to more specialized descendants. The specialization means that each has lost the information for certain colors and behaviors. The formation of new species (*speciation*) without information gain is no problem for creationists.7 Adaptation/variation within *Chrysoperla*, which involves no addition of complex new genetic information, says nothing about the origin of lacewings themselves, which is what evolution is supposed to explain.

**Darwin’s finches**

On page 19, *Teaching about Evolution*claims:

A particularly interesting example of contemporary evolution involves the 13 species of finches studied by Darwin on the Galápagos Islands, now known as Darwin’s finches … . Drought diminishes supplies of easily cracked nuts but permits the survival of plants that produce larger, tougher nuts. Drought thus favors birds with strong, wide beaks that can break these tougher seeds, producing populations of birds with these traits. [Peter and Rosemary Grant of Princeton University] have estimated that if droughts occur about every 10 years on the islands, then a new species of finch might arise in only about 200 years.

However, again, an original population of finches had a wide variety of beak sizes. When a drought occurs, the birds with insufficiently strong and wide beaks can’t crack the nuts, so they are eliminated, along with their genetic information. Again, no new information has arisen, so this does not support molecules-to-man evolution.

Also, the rapid speciation (200 years) is good evidence for the biblical creation model. Critics doubt that all of today’s species could have fitted on the ark. However, the ark would have needed only about 8,000 kinds of land vertebrate animals, which would be sufficient to produce the wide variety of species we have today.8 Darwin’s finches show that it need not take very long for new species to arise.9

**Breeding versus evolution**

On pages 37–38, *Teaching about Evolution*compares the artificial breeding of pigeons and dogs with evolution. However, all the breeders do is select from the information *already present*. For example, Chihuahuas were bred by selecting the smallest dogs to breed from over many generations. But this process eliminates the genes for large size.

The opposite process would have bred Great Danes from the same ancestral dog population, by eliminating the genes for small size. So the breeding has *sorted out* the information mixture into separate lines. All the breeds have less information than the original dog/wolf kind.

Many breeds are also the victims of hereditary conditions due to mutations, for example the ‘squashed’ snout of the bulldog and pug. But their loss of genetic information and their inherited defects mean that purebred dogs are less ‘fit’ in the wild than mongrels, and veterinarians can confirm that purebreds suffer from more diseases.

Actually, breeds of dogs are interfertile, even Great Danes and Chihuahuas, so they are still the same species. Not that speciation is a problem for creationists—see the section on lacewings above. But if Great Danes and Chihuahuas were only known from the fossil record, they would probably have been classified as different species or even different genera. Indeed, without human intervention, Great Danes and Chihuahuas could probably not breed together (hybridize), so they could be considered different species in the wild. Creationists regard the breeds of dogs as showing that God programmed much variability into the original dog/wolf created kind.

**Darwin versus a faulty creation model**

On pages 35–36, *Teaching about Evolution*discusses some of Darwin’s observations. For example, living and fossil armadillos are found only in South America. Also, animals on the Galápagos Islands are similar to those in Ecuador, while creatures on islands off Africa’s coast are related to those in Africa. The book then states:

Darwin could not see how these observations could be explained by the prevailing view of his time: that each species had been independently created, with the species that were best suited to each location being created at each particular site.

Scale image of the ark, shown next to a large truck, and people (bottom right corner)

Actually, this is setting up a straw man, as this is not what biblical creationists believe, because it completely ignores the global flood as stated in Genesis chapters 6–9. The flood wiped out all land vertebrates outside the ark and would have totally re-arranged the earth’s surface. So, there’s no way that anything was created in its present location.



Also, all modern land vertebrates would be descended from those which disembarked from the ark in the mountains of Ararat—over generations, they migrated to their present locations. It should therefore be no surprise to biblical creationists that animals on islands off Africa’s coast should be similar to those in Africa—they migrated to the islands via Africa.

Darwin’s observations were thus easily explainable by the biblical creation/flood model. However, by Darwin’s time, most of his opponents did not believe the biblical creation model, but had ‘re-interpreted’ it to fit into the old-earth beliefs of the day.

A prevalent belief was a series of global floods followed by re-creations, rather than a single flood followed by migration. Darwin found observations which didn’t fit this non-biblical model. This then allowed him to discredit creation and the Bible itself, although it wasn’t actually the true biblical belief he had disproved!

An interesting experiment by Darwin, cited by *Teaching about Evolution*on page 38, also supports the creation-flood model.

By floating snails on salt water for prolonged periods, Darwin convinced himself that, on rare occasions, snails might have ‘floated in chunks of drifted timber across moderately wide arms of the sea.’ … Prior to Darwin, the existence of land snails and bats, but not typical terrestrial mammals, on the oceanic islands was simply noted and catalogued as a fact. It is unlikely that anyone would have thought to test the snails for their ability to survive for prolonged periods in salt water. Even if they had, such an experiment would have had little impact.

Thus, Darwin helped answer a problem raised by skeptics of the Bible and its account of the flood and ark: ‘How did the animals get to faraway places?’ This also showed that some invertebrates could have survived the flood outside the ark,10 possibly on rafts of pumice or tangled vegetation, or on driftwood as Darwin suggested. Other experiments by Darwin showed that garden seeds could still sprout after 42 days’ immersion in salt water, so they could have traveled 1,400 miles (2,240 km) on a typical ocean current.11 This shows how plants could have survived without being on the ark—again by floating on driftwood, pumice, or vegetation rafts even if they were often soaked. Therefore, the creation-flood-dispersion model could also have led to such experiments, despite what *Teaching about Evolution* implies.12

**The fact of natural selection**

From time to time, we receive requests to reject natural selection, and adopt the alternative theories of Dr Randy Guliuzza of ICR. The main one is “continuous environmental tracking” (CET), that is, God endowed the created kinds to detect and respond to environmental stimuli. How they do so is by a process he calls “programmed filling”, where God programmed organisms with the ability to adapt to various environments. But Guliuzza rejects natural selection, or any idea that the environment has a role in what organisms thrive or die. As will be seen, CMI doesn't deny that God programmed creatures in marvellous ways. But we also affirm natural selection as part of the biblical creationist model—as creationists before Darwin understood!

One sample letter follows (slightly modified), then [Dr Jonathan Sarfati](https://creation.com/dr-jonathan-sarfati) explains why creationists should not be afraid of natural selection and thus abandon the concept to evolutionists. We have expanded and updated this article considerably beyond the initial response, to keep up with some of Dr Guliuzza’s claims as well as counter-claims from more conventional creationists.

Wikipedia.orgTexas blind salamander. Photograph by Joe N. Fries. Public domain

From B.R., USA:

The term ‘natural selection’ does not make sense either, it is not only circular, worse; it is misleading in that it personifies nature giving the impression nature has the cognitive ability to engage in a selection process. The environment does not nor can it ‘select’ and equating the statement that it ‘can select’ with ‘just as human breeders select’ is to ascribe intelligence to nature that it simply does not possess. In the process, it takes from the Glory of God, who is lovingly sovereign over all His creation.

The creatures themselves by their own God given instincts and with His guidance determine responses to changes in various environments and they do it with the adaptive ability with which the Good Merciful Creator created them. The idea of ‘natural selection’ would have it the other way around. Whether the term was first coined by a creationist is not relevant, when I read such statements from creationist organizations I sense a real danger in adopting the common language of evolutionists.

“If the incorrect usage leads, then a misinterpreted deification of Nature follows.” Randy J. Guliuzza

Mr. Herbert Spencer has well expressed the same idea [natural selection] by the Survival of the Fittest. The term ‘natural selection’ is in some respects a bad one, as it seems to imply conscious choice; but this will be disregarded after a little familiarity.—Darwin

Dear B.R.

Thank you for writing to CMI.

To be blunt, we think Dr Guliuzza of ICR is just wrong about natural selection. I discussed this with a couple of his colleagues when he started talking about natural selection in public and was not impressed. Our own Dr Robert Carter attended a weekend-long workshop hosted at ICR to discuss Guliuzza’s then-new ideas. That was in 2010. He left entirely unconvinced and has since written articles like [Natural Selection in Paradise](https://creation.com/natural-selection-in-paradise) that not only claim Guliuzza is wrong but that natural selection itself would have been in operation prior to the Fall (in other words, before suffering and death entered the world). Another ICR scientist (now with AiG), geneticist Dr Nathaniel Jeanson, has written a powerful critique of Dr Guliuzza’s idea,1 as has independent creationist scientist Dr Jason Lisle.2

CMI scientists are unanimous that natural selection is a fact, and part of this fallen creation where unfit creatures die and sometimes even become extinct. [Creationists proposed it before Darwin](https://creation.com/charles-darwins-illegitimate-brainchild), so why should we be fearful of the term, and let Darwinists monopolize this phenomenon? So our major books like [*The Greatest Hoax on Earth*](https://creation.com/store_redirect.php?sku=10-2-555)? and [*Evolution’s Achilles’ Heels*](https://creation.com/store_redirect.php?sku=10-2-640) each have a whole chapter explaining this.

We also include our [natural selection is a tautology [circular]](https://creation.com/arguments-we-think-creationists-should-not-use#tautology)in our important page [Arguments we think creationists should NOT use](https://creation.com/arguments-we-think-creationists-should-not-use). Note also, critics can logically pick only one of “Natural selection is false” and “Natural selection is a tautology”, because a tautology is an analytically true statement such as *A implies A.*

What, then, about the criticisms summarized by the above letter?

**What evolutionists mean by natural selection**

The ‘personification of nature’ claim is simply a hyperliteralistic misunderstanding of a phrase, and a failure to understand that language is defined by *usage*. The creationists who first proposed NS and Darwinists who followed them never *intended* it to mean anything cognitive by nature. So we can’t blame the evolutionists for any misunderstandings. This is easy to document.

Natural selection is a culling force, not a creative force. It is really death of the unfittest more than survival of the fittest, and doesn’t explain the arrival of the fittest.

First, Darwin himself explained that he used the term to mean the preservation of individuals best adapted to their conditions:

But if variations useful to any organic being do occur, assuredly individuals thus characterised will have the best chance of being preserved in the struggle for life; and from the strong principle of inheritance they will tend to produce offspring similarly characterised. This principle of preservation, I have called, for the sake of brevity, Natural Selection.3

Actually, Darwin’s co-discoverer, [Alfred Russel Wallace](https://creation.com/alfred-russel-wallace-co-inventor-of-darwinism), expressed concerns to him that Darwin’s critics hyper-literalized it just the way Dr Guliuzza thinks it was intended. These critics, said Wallace, charged Darwin with “blindness” for not seeing an intelligent “chooser” at work as in artificial selection. Another anti-Darwinian, according to Wallace, claimed that Darwin’s “weak point” was that he didn’t see that “thought & direction are essential to the action of ‘Nat. Selection’.” Wallace laid the blame on Darwin’s term “Natural selection”, but here it was concern that *anti-evolutionists* have misunderstood a term that should have been clear:

Now I think this arises almost entirely from your choice of the term “Nat. Selection” & so constantly comparing it in its effects, to Man’s selection, and also to your so frequently personifying Nature as “selecting” as “preferring” as “seeking only the good of the species” &c. &c. To the few, this is as clear as daylight, & beautifully suggestive, but to many it is evidently a stumbling block. I wish therefore to suggest to you the possibility of entirely avoiding this source of misconception in your great work, (if not now too late) & also in any future editions of the *Origin*, and I think it may be done without difficulty & very effectually by adopting Spencer’s term (which he generally uses in preference to Nat. Selection) viz. “Survival of the fittest”.

This term is the plain expression of the *facts*,—*Nat. selection* is a metaphorical expression of it—and to a certain degree *indirect* & *incorrect*, since, even personifying Nature, she does not so much *select* special variations, as *exterminate* the most unfavourable ones.4

Actually, this is what informed creationists point out: in this fallen world, *natural selection is a culling force, not a creative force*. It is really *death of the unfittest* more than survival of the fittest, and doesn’t explain the *arrival* of the fittest. I.e. it [*removes* genetic information](https://creation.com/3-rs-of-evolution) whereas evolution needs to *add* information.

Darwin responded to Wallace basically agreeing with him, and evidently took his advice to heart. Two years later, in another book, Darwin explained in the introduction:

Renaming natural selection to ‘programmed filling’ is a distinction without a difference. From a strategic viewpoint, it would give the impression that creationists have no answer to a known process, and concede important scientific territory to evolutionists.

This preservation, during the battle for life, of varieties which possess any advantage in structure, constitution, or instinct, I have called Natural Selection; and Mr. Herbert Spencer has well expressed the same idea by the Survival of the Fittest. The term “natural selection” is in some respects a bad one, as it seems to imply conscious choice; but this will be disregarded after a little familiarity.5

In the 20th century, one of their greatest disciples, Theodosius Dobzhansky (1900–1975), made it clear what evolutionists mean, at least as the result of the process—that some organisms have more surviving offspring. That is, natural selection is really not so much survival of the fittest but *reproduction* of the fittest, or as Dobzhansky put it:

*Natural selection is differential reproduction, organism perpetuation.*In order to have natural selection, you have to have self-reproduction or self-replication and at least two distinct self-replicating units or entities.6

Thus Dr Robert Carter has even defended the idea that [natural selection would have been in existence in Eden](https://creation.com/natural-selection-fact-contra-guliuzza), prior to the time when death started.

Then Darwin’s most famous disciple, Richard Dawkins, famously made it clear that natural selection had nothing to do with personifying nature, but rather, it was:

Natural selection, the blind, unconscious, automatic process which Darwin discovered, and which we now know is the explanation for the existence and apparently purposeful form of all life, has no purpose in mind. It has no vision, no foresight, no sight at all. If it can be said to play the role of watchmaker in nature, it is the blind watchmaker.7

Therefore, the leading evolutionists have always made it clear that they had no intention of attributing to nature a literal ability to select. Further, they explicitly contradicted misunderstandings to that effect.

**Natural selection: creationist discovery before Darwin**

The predictions of opposing scientific theories often overlap. Any argument that is true for both sides (zone II) cannot be used as proof for one side. Yet, we often hear arguments like, “Natural selection proves Darwinian evolution!” But since natural selection has been part of the biblical creationist model even before Darwin’s time, this cannot be “proof” of evolution and disproof of biblical creation. Diagram from [Why CMI rejects ‘conspiracy’ theorizing.](https://creation.com/cmi-rejects-conspiracy-theory)

You claim, “Whether the term was first coined by a creationist is not relevant.” It is most definitely relevant. The NS-detractors claim things like “creationists have been playing Darwin’s game, on his field, by his rules.” But if natural selection was proposed by creationists before Darwin, then who is playing whose game?

Indeed, we have thoroughly documented that [creationists thought of NS before Darwin](https://creation.com/charles-darwins-illegitimate-brainchild). The famous evolutionist [Stephen Jay Gould](https://creation.com/leading-evolutionist-stephen-jay-gould-dies) (1941–2002) pointed out, “Natural selection ranked as a standard item in biological discourse” among the pre-Darwinian creationists.8

Creationists long recognized natural selection as a conservative force, removing unfit organisms and thus hindering the effects of the Curse. According to Gould, Darwin’s contribution was not natural selection per se, but *natural selection as a creative force.*

So *that* is “Darwin’s game”—hijacking the creationist-discovered phenomenon of NS to try to transform it into a creative force. CMI instead sticks to NS as understood by its pre-Darwinian creationist discoverers. Therefore, NS is an example of *non-discriminating information,* as per the diagram (left).

A helpful analogy (borrowed from Dr Jason Lisle): creationists accept the law of gravity, discovered by the creationist Sir Isaac Newton. Evolutionists believe that stars formed by gravitational collapse of gas clouds. Creationists reject this (see [Solar system origin: Nebular hypothesis](https://creation.com/nebular-hypothesis) and [Stars](https://creation.com/stars)). But just because we reject evolutionary claims about what gravity can *do,* it doesn’t follow that we should reject the observable creationist-discovered Law of Gravity *itself.* Similarly, we reject evolutionary claims about what NS can do—turn protists into people—but it doesn’t mean we should reject the creationist-discovered phenomenon itself.

**Personification of nature?**

This is a major objection by Guliuzza to the term ‘natural selection’. However, as seen, the leading evolutionists didn’t intend this literally, and *explicitly disclaimed* this type of meaning. So they can’t be blamed for trying to mislead the public by choosing the term, when they actually go out of their way to exclude anthropomorphic meanings.

However, despite the evolutionists’ disclaimers, should we be concerned by a term that even figuratively personifies nature? Actually, scientists do that all the time without any problems. For example, pharmacists might warn about ‘light-sensitive’ medicine that should be stored away from light. Is this really claiming that medicine is literally sentient? According to Guliuzza and some of his colleagues, we must logically say yes. But here I must agree with Darwin, who pointed out

No one objects to chemists speaking of ‘elective affinity’ and certainly an acid has no more choice in combining with a base, than the conditions of life have in determining whether or not a new form be selected or preserved.9

The Bible contains passages like “the mountains and hills will burst into song before you, and all the trees of the field will clap their hands” ([Isaiah 55:12](https://biblia.com/bible/esv/Isa%2055.12)), i.e. personifying nature.

As a final observation, it’s rather strange to claim that personification of nature is anti-biblical, when the Bible contains passages like “the mountains and hills will burst into song before you, and all the trees of the field will clap their hands” ([Isaiah 55:12](https://biblia.com/bible/esv/Isa%2055.12)), unless of course the Bible is anti-biblical.

**Continuous Environmental Tracking / Programmed filling?**

We also think it’s futile to rename phenomena adequately explained by ‘natural selection’ as ‘programmed filling’ as Guliuzza advocates. At best, this renaming is a distinction without a difference, when it comes to matters of practical, [operational science](https://creation.com/Whos-really-pushing-bad-science-rebuttal-to-Lawrence-S-Lerner). E.g. is anything changed if we said that salt dissolving in water was “programmed dissociation” as opposed to following the laws of chemistry that God uses to sustain His creation? And there are serious disadvantages. Also, from a strategic viewpoint, it would give the impression that creationists have no answer to a known process, and would concede important scientific territory to evolutionists.

The aspects of the CET model that deal with genetic change are more ambiguous. That’s because there is nothing known in the genome that directs specific, reversable genetic changes under different environmental conditions.

It also fails to account for the fact that we live in a [*fallen world*](https://creation.com/the-fall-a-cosmic-catastrophe), so creatures not adapted appropriately for some environmental pressure, for instance, will die off.

**Where defects can be an advantage**

In this fallen world, clear *destructions* of programming can be beneficial, such as [eyeless creatures in caves](https://creation.com/christopher-hitchens-blind-to-salamander-reality), [wingless creatures on windswept islands](https://creation.com/beetle-bloopers), [sickle-cell anemia in malarial areas](https://creation.com/exposing-evolutions-icon), [loss of skin pigmentation in snowy areas](https://creation.com/white-deer), and more. But in most cases, these changes are regarded as harmful. It was no accident that Jesus healed the blind, lame, dumb, and sick. We also *expect* downhill changes, simply because there are many ways to break something, but not many ways to make something in the first place. So it’s not surprising that it would be relatively easy for a mutation, or copying mistake in the genes, to ruin the eyes, wings, hemoglobin, or melanin production, as in the above examples, but hard to make them right. [Genetic entropy](https://creation.com/genetic-entropy-defense) is real, and is a [powerful argument against evolution](https://creation.com/genetic-entropy-vs-evolution)!

**Case study: blindness in people and cave creatures**

E.g. when it comes to blindness in humans, it could be caused by damage to the eyeball, but also a perfectly formed eyeball could be blind because the optic nerve is underdeveloped (optic nerve hypoplasia) or destroyed by when the finely tuned pressure controls fail and allow excess pressure (glaucoma). And all those could be fine, but a mutation in any of the proteins in the visual cycle could block vision as a whole, e.g. Leber’s Congenital Amaurosis (LCA) or Retinitis Pigmentosa (RP). Also, the eye doesn’t really see; the brain sees by interpreting the signals from the eye, and this takes training. Some people who have their eyes fixed after a lifetime of blindness have trouble seeing because their brains are not trained to handle the new information. But whatever the cause of blindness in people who asked Jesus for healing, Jesus healed them, suggesting that blindness really is the result of breaking something, not a programmed filling (see [Walking trees … Modern science helps us understand a puzzling miracle](https://creation.com/walking-trees)).

This must be taken into account for one of Dr Guliuzza’s examples: supposed programmed filling to explain blind fish in caves. In most cases of blind creatures, the best explanation is what CMI has stated, e.g. [in 2008](https://creation.com/christopher-hitchens-blind-to-salamander-reality), consistent with the above:

This is easily explainable: there are many ways to break something, but not many ways to make something in the first place. So it’s not surprising that it would be relatively easy for a mutation, or copying mistake in the genes, to ruin the eyes. In the light, natural selection would eliminate such mutations, since blind creatures could see neither prey nor predators.

But in a pitch-black cave, there would be no natural selection against blind creatures, so they could proliferate. They might even have an advantage, because a shrivelled eye is less likely to be damaged.

But this article also noted an explanation in *one* case with a cave fish, and in my 2010 book *The Greatest Hoax on Earth?*, written before I had ever heard of Dr Guliuzza or his anti-NS arguments, which shows that we were already well aware of pre-programming:

**Pleiotropy**

Dawkins continues:

Of course, the story of the cave-dweller’s eye isn’t only a negative one: positive selection comes in too, to favour the growth of protective skin over the vulnerable sockets of the optically deteriorating eyes.” (p. 353)

There is another positive selective effect Dawkins doesn’t mention here, proposed by evolutionists but also compatible with the biblical creation model. As [I pointed out](https://creation.com/christopher-hitchens-blind-to-salamander-reality):

In one of the best known blind cave fish, *Astyanax mexicanus,* there is another reason why the blind fish can have an advantage in caves. This is pleiotropy, where a single gene has more than one effect on an organism. It turns out that a control gene, hedgehog, which affects a number of processes including development of the jaws and tastebuds, also inhibits another control gene, pax6, which controls development of the eyes. A fish with bigger jaws and more sensitive tastebuds would have an advantage in finding food, but this must be traded off with the loss of eye development. In the light, loss of eyes is a big disadvantage, so natural selection would eliminate a fish that over-expresses hedgehog, despite its better jaws and taste. But in the dark caves, a fish with highly expressed hedgehog would have a big advantage, since the loss of eyes would be irrelevant.10

Unless Dawkins can show that the growth of skin over the eye sockets of eyeless fish involves the creation of new genes by natural processes, it does not qualify as ‘proving evolution’. It is likely a pre-programmed response to having an unfilled hole in the skeleton of the fish, or a pleiotropic effect resulting from the damaging of the genes for eye formation—the genes for normal eyes some way or other prevent the skin from growing over the eyes, so damaging those genes would likely allow the growth of the skin over the eye sockets.

The conventional biblical creationist model not only includes geniune pre-programming, but also all the cases best explained by deleterious mutations and natural selection. A model that explains more observations is generally more useful. Furthermore, the conventional creationist explanation provides a good young-earth argument that Guliuzza’s theory undermines, as per *Greatest Hoax:*

Also, to underscore the point that there are many ways to break things, there are actually a number of ways to produce blindness, even in *Astyanax.* This is shown by breeding different populations of blind fish, and resulting in a number of sighted progeny. This is explained because the sight loss in the different populations is caused by different mutations, so ‘when you cross them, the genetic deficiencies in one lineage are compensated for by strengths in the other, and vice-versa.

But if the sight loss had occurred millions of years ago, then other genes involved with sight would have had time to be “cratered” by mutations, not removed by natural selection. Then crossing such mutated genes would not restore sight. … exactly the same principles apply to blind salamanders and other blind troglobionts.

When it comes to breaking something, it need not take very long either. Breaking is often quicker than making, just as it’s often much quicker to fall off a mountain than to climb it. We can see this in humans, when sighted parents have blind children due to a genetic defect—this can happen in only one generation. …

Indeed, the fact that sight can be regained in one generation shows that there has been little time for mutations to further degenerate the genes—note that natural selection would not preserve genes connected to eyes and the visual parts of the brain if there were no selection for eyesight. [I.e. to use Dawkins’ helpful analogy, these genes would be heavily cratered.]

**Epigenetics**

Epigenetics is another important concept that Guliuzza raises. The term is derived from adding the Greek prefix epi-/ἐπι-, meaning = over, outside of, around to *genetics*. That is, epigenetic changes are heritable changes of the phenotype (observable features) of an organism that are ‘over’ the genes coded in the DNA. That is, we not only have the genes, but also controls *over* these genes that turn them on and off, and control the rate of expression. A well known example is why identical twins are not identical even though they have identical DNA. The reason is different epigenetic codes. Some scientists have even said that the genes are like puppets, while the epigenetics is like a genetic puppeteer. Epigenetics is vital for the growth of organisms from a fertlized egg, as explained in *Greatest Hoax:*

It’s notable that the development of every individual multi-celled creature involves a programmed switching off of genetic information. Each individual begins as a single cell—a zygote or an ovum fertilized by a spermatozoon. This fertilized ovum has all the instructions coded in the DNA to make us what we are physically (given the right environmental conditions).

But as the embryo grows, different cells in different places have to specialize, so that only certain instructions are executed—the cells become differentiated. The instructions are there, but turned off somehow. There are complicated genetic switches involved, and also a process called methylation—attaching methyl groups to the chemical ‘letters’ of DNA that code for instructions that need to be ‘turned off’.

All the on/off switching must occur in the right sequence; the information for this sequencing is partly encoded in the DNA, but there are also controls outside the genes, hence the term epigenetic. This is why it would be impossible to clone dinosaurs and mammoths even if we found intact DNA—we would need the ovum (mother’s egg) too.

The result of these elaborately designed switching sequences is that bone cells execute only instructions pertaining to bone—the instructions for blood, nerves, skin, etc. are still in the cells’ DNA, but turned off. Similarly for blood, skin and other types of cells.

Thus if one can believe that this switching-off information was programmed by a Master Genetic Programmer, it is plausible that this Programmer could also have switched on information at the Fall.

CMI has long pointed out that epigenetics is a problem for neo-Darwinian evolution. For example, our 2008, pre-Guliuzza article [The genetic puppeteer](https://creation.com/the-genetic-puppeteer) explains two types of genetic coding above the well known genetic code

1. Methylation code, whereby –CH₃ groups are added to some of the DNA ‘letters’ to switch genes off.
2. Histone code: DNA is spooled around ball-shaped proteins called *histones*, and the code controls how tightly the histones are packed. If they are packed loosely, then the information is easily accessible, while tight histones restrict gene expression on the DNA wrapped around them.

In 2010, Dr Carter [wrote about the *splicing code*](https://creation.com/splicing-and-dicing-the-human-genome). The ‘splicing code’ controls how different parts of DNA are chopped out and spliced together. This editing process enables a single gene to encode multiple proteins. It explains why humans have only about 21,000 genes, yet can make perhaps up to a million proteins—a surprise to those who decoded the human genome.11

For example, thanks to studies of the splicing code, researchers found that “three neurexin genes can generate over 3,000 genetic messages that help control the wiring of the brain,” according to co-discoverer Brendan Frey. This splicing also involves a complex machine called a spliceosome. One paper was tellingly entitled, “Mechanical devices of the spliceosome: motors, clocks, springs, and things.”12

These are all aspects of the epigenetic code, the code above the genes, which is heritable. It is also dynamic, not static like the DNA it controls. It can change throughout development, and can respond to the environment.

Therefore Guliuzza is right to point out epigenetics as an important part of the biblical creation model. CMI agrees, as above, and in the instructive article [The four dimensional human genome defies naturalistic explanations](https://creation.com/four-dimensional-genome). But Guliuzza errs by implying that this is incompatible with the conventional biblical Creation/Fall that includes mutation + selection *but is not confined to this!*The conventional creation model is superior to *both* the neo-Darwinian model *and* the Programmed Filling model:

* It is superior to evolution, because it includes both the epigenetic (and pleiotropy) phenomena out of reach of neoDarwinian evolution.
* It is superior to programmed filling, because it includes clear cases of deleterious mutations and elimination by natural selection.

**We live in a fallen world**

Much of what Dr Guliuzza says applies in the unfallen world only. There, with no suffering or death, organisms could choose among whatever options they were given. If a bacterium preferred (note: this is not ‘personifying’ bacteria!) to live in a particular environment, it could easily swim in one direction or another, following whatever chemical cue it was programmed to detect. Alternatively, if a rabbit preferred to live in an area with dense sagebrush, it could easily avoid the less preferable pine forests.

However, once the Curse was applied to the natural world, all bets are off. Mice could not choose to not live in Siberia when the Ice Age ended and that vast landscape suddenly changed from a temperate grassland to a tundra and pine-covered plain. The mice that were there died in droves, along with the caribou, beavers, foxes, and mammoths, to name just a few animals that no longer live in the area. The ability of these animals to choose was overwhelmed by the unforgiving environment.

We also must ask the question why so many organisms have gone extinct. Clearly, [creatures that are now extinct, such as dinosaurs](https://creation.com/dinosaurs-almost-certainly-extinct), were unable to adapt to the post-Flood world. In one sense, the environment exceeded their design specifications. No amount of ‘programming’ enabled them to survive. The same thing goes for trilobites, elephant birds, the dodo, and thousands of other species. The world is a harsh place and the organisms that don’t make it take their genes with them to the grave. When only some individuals in a species are a good match for the environment, they have a higher likelihood to survive, along with the genes that they carry. This is called natural selection. It is not a big deal.

In fact, suffering and death are intrinsic parts of the biblical model. If death reigns, as the Bible says, life-and-death situations happen constantly. It’s nice to think that organisms choose their own fate, but it just isn’t so.

Hope this helps.
In Christ
[Jonathan Sarfati](https://creation.com/dr-jonathan-sarfati) (with assistance from [Robert Carter](https://creation.com/dr-robert-carter)).

**The limits of Neo-Darwinism**

Jared from Zimbabwe asks for clarification over just what mutations and natural selection are capable of doing and not capable of doing. CMI’s [Dr Don Batten](https://creation.com/dr-don-batten) responds.

Wikimedia: H. ZellMutations and natural selection only effect change *within* a created kind.

Hi,

I am curious about the few, seemingly at odds, articles that I have read recently. The topic is that of genetic mutations and natural selection. It seems in some ways this is shown to be insignificant and unable to affect any real changes, then in other ways it seems to be very significant.

Many articles have suggested how the great diversity we have in the biological world is from mutations and selections. The suggestion is that this process is pretty solid and consistent giving us many diverse species from just a few. The point has also been made that some of these mutations are actually more severe and occur more rapidly than previously thought. A couple of examples that come to mind is the interbreeding of certain cave dwelling blind fish that regain sight in just a single generation, and tunnel mosquitoes that have developed so independently that they are unable to interbreed with above ground species. This would seem to make sense.

But then I have also read a few other articles that suggest otherwise, mostly summarised by the following points made by John Sanford:

“The bottom line is that Darwinian theory fails on every level. It fails because: 1) mutations arise faster than selection can eliminate them; 2) mutations are overwhelmingly too subtle to be “selectable”; 3) “biological noise” and “survival of the luckiest” overwhelm selection; 4) bad mutations are physically linked to good mutations,2 so that they cannot be separated in inheritance (to get rid of the bad and keep the good). The result is that all higher genomes must clearly degenerate.”

He suggests that mutations get overwhelmed by “noise” and are too subtle to be “selectable”. But does this not work against the very arguments that creationists use to support rapid speciation and variation? He says mutations are too subtle, but surely losing or gaining eyes is not subtle at all and would definitely affect selection? Surely that’s the whole basis for our creation arguments?

So are mutations and natural selection minor and insignificant in the scheme of things, or are they effectual and foundational for biological diversity? Do mutations make a difference or don’t they? It sometimes seems a little confusing.

Genetic entropy should be responsible for dwindling numbers and eventually extinction, but yet there are many examples of massive propagation and survival from just a few, the exact opposite. We would like to believe that both prove creation, but arguing it that way with an evolutionist is very hard. How can the process be used for both upward and downward explanations? Surely that leaves us in a pickle of not adequately explaining anything? It’s not falsifiable? I’m presuming that I am missing something somewhere that will make it all make sense.

Clarification on this would be much appreciated by myself, and I’m sure many others.
Thanks!

Dear Jared,

**Mutations and natural selection only effect change within a created kind**

Thanks for asking. I can see why you could be a bit confused and probably others as well.

We are looking at different things here.

Mutations and [natural selection](https://creation.com/muddy-waters) only effect change *within*a created kind; for example the mosquitoes and blind cave fish you mention (they are still mosquitoes and the same type of fish).1 Mutations only modify *existing* genes to create different coat colours, for example, in cattle and dogs. [Dr Jean Lightner has written about this](https://creation.com/colourful-creature-coats). [Mutations won’t create](https://creation.com/mutations-new-information) the genes that allow an animal to produce colour where it could not before, but they can damage an existing gene so that the animal produces less brown pigment, resulting in a light colour (such as fawn or white). On the other hand, a mutation can damage the control system for pigment production causing it to run ‘full speed’ and produce much more of the pigment so the animal is black rather than brown.

These sorts of changes can give us varieties (even ‘species’) of wolves (dogs, arctic wolves, African wolves, dingoes, jackals, foxes, etc.) but they won’t change a reptile into a wolf to give rise to wolves in the first place, which requires the invention of brand new genes. We wrote about the mosquitoes you mentioned as an example of the sorts of changes that can cause [rapid speciation *within a created kind*](https://creation.com/brisk-biters).

[Dr Sanford](https://creation.com/geneticist-evolution-impossible) is talking about the number of mutations that are slightly harmful in organisms with large genomes, like humans (and wolves). There are many slightly harmful mutations and because there are many and they are only slightly harmful, natural selection cannot get rid of them; they are effectively invisible to natural selection. Think about a mutation that resulted in a wolf being born blind. Natural selection would normally eliminate the individual with this mutation (that is, it would not survive; that’s all we mean by natural selection). Such large-effect mutations can be/are eliminated. However, we acquire something like 100 new mutations per person. Many of these are only slightly deleterious (no obvious defect). Because such mutations have only a small effect, with no obvious effect on survival, natural selection is powerless to eliminate them. Also, all offspring are born with more mutations than their parents; none have fewer. That means that these slightly harmful mutations are accumulating, generation by generation, relentlessly causing genetic deterioration because of the summed effect of all of them. It is in this context that Dr Sanford makes his conclusions about Darwinian evolution being ‘dead in the water’. The degeneration he speaks of is happening in all higher organisms. It is a pattern of deterioration that overlays everything and prevents any possibility of upward evolutionary ‘progress’ (“climbing mount improbable”) that is supposed to have changed a microbe into a microbiologist.

Within this general pattern, adaptation (blind cave fish) can occur, but notice that this ‘adaptation’ is still, when it involves mutations, via loss of function.

Within this general pattern, adaptation (blind cave fish) can occur, but notice that this ‘adaptation’ is still, when it involves mutations, via loss of function (messing up the genes that say how to make eyes).

Another classic example is [beetles on a windy island](https://creation.com/beetle-bloopers) where mutants with defective wings did not get blown into the sea and quickly became the dominant population on that island. Note again that the mutation has been adaptive, causing diversity in the beetles, but it is a *downhill* change. The Darwinian paradigm needs a mechanism for creating new genes, not modifying and degrading existing genes, which is what we overwhelmingly see.

We have talked of this downhill change also in terms of natural selection eliminating genes in individuals that are not adapted to their environment. This also causes an impoverishment of the genetic information. For example, the beetles with normal wing-making genes are eliminated on the windy island and so the genes for normal wings are lost.

Here is another illustration of this principle. Let’s say that five of the ~20,000 gene pairs of a breeding wolf male and female pair were both AaBbCcDdEe. Because of the way sexual reproduction recombines the genes (variants of a gene are called “alleles”), offspring could be AABBCCDDEE or aabbccddee, or any other combination, thus producing lots of potential varieties of offspring. Now let’s say that ones with the genes aabbccddee were adapted to cold conditions and were the only ones that survived the Ice Age in northern Europe after Noah’s Flood. These wolves have lost the genes A,B,C,D and E. When they breed, they can only produce more wolves with a,b,c,d and e, none with the variety of genes in the original population. From this you can see that if the wolves adapted to hot conditions were AABBCCDDEE, for example, you could never get this wolf from the ones adapted to the cold. The genes for adaptation to hot conditions have been lost.

123rf.com/bedolaga

Artificial breeding/selection is a good analogy. The original mongrel dog/wolf population had more genetic variety than any of the many individual breeds of dog today, but because of continual mixing up of the genes/alleles due to free inter-breeding (out-breeding), the outward appearance of the dogs/wolves would not generally have shown extreme variety. But selecting rare dogs with Chihuahua-like features and breeding them together (inbreeding) for many generations results in the concentration of alleles that give Chihuahua-likeness and the elimination of a lot of the other alleles—ones for ‘large dog’ for example. So you could never breed a Great Dane from a Chihuahua; the genetic information required has been depleted by the very process that has generated the extreme features.

In genetics, variety of alleles is known as *heterozygosity* (the degree to which pairs of genes differ); lack of variety is *homozygosity* (the degree to which pairs of genes are the same). So in genetic terms, we believe that God created organisms with a higher degree of heterozygosity than we generally see today. The variety/diversity seen today is largely due to the sorting of the originally created genes into more specialized sets of genes (as in the example of wolves adapting to the cold above). Where mutations have contributed, it is almost always via degradation of an existing gene or gene control. It might be that God even created organisms with ‘hot spots’ for mutation that would enable adaptation.2 Of course such hot spots negate the idea of mutations being random (purely chance) occurrences.

When you breed together several different breeds of dogs, you can end up with a “mongrel” with a more comprehensive set of alleles more like the original dog/wolf. This mongrel dog loses the extreme features of the dog breeds and looks more like a regular dog/wolf. If you took all the descendants of an original created kind, such as the [cattle kind](https://creation.com/identification-of-species-within-the-cattle-monobaramin-kind), and bred them together, you could end up with an animal closer to what God created in the beginning (except for the deleterious mutations that have accumulated).

The big picture is that God created various kinds of organisms with a wealth of genetic information that has allowed for adaptation/speciation within each kind. But both mutations and natural selection are downhill processes. Even as these natural processes produce greater variety in daughter populations, each of those populations becomes genetically depleted—more specialized, but therefore with less variety within each population, and thus less able to adapt to future environmental challenges. Overarching this we find the relentless accumulation of slightly harmful mutations, which Dr Sanford has highlighted. Extended forwards in time, all these natural processes head towards extinction. Since the Fall ([Genesis 3](https://biblia.com/bible/esv/Gen%203)) everything has been wearing out, like a piece of clothing wears out ([Hebrews 1:11](https://biblia.com/bible/esv/Heb%201.11)). Evolution (microbes-to-man) via mutations and natural selection is a bankrupt idea, scientifically and biblically.

Every blessing,

Don Batten

**Evolution’s well-kept secret:**

Mutations are not random!

***by***[***Paul Price***](https://creation.com/paul-price)

For nearly a hundred years, evolutionists have been operating under the paradigm that is known as the “Neo-Darwinian Synthesis”, also known as the “Modern Synthesis”. This view has repeatedly been summarized as ‘natural selection working upon *random*mutations’. I have taken the liberty of adding emphasis in the quotes below to show you how common this language is.

This goes back decades. A biology textbook from the 1960s says:

“With the development of the gene theory the term ‘mutation’ has come to refer to sudden, discontinuous, **random changes** in the genes and chromosomes, although it is still used to some extent to refer to the new type of plant or animal.”1

To give an example from a somewhat newer biology textbook (from 1989), one study question asks:

“Based on your knowledge of DNA structure, the genetic code, and protein structure, what sorts of **random** mutations would you expect to persist in a lineage of organisms, generation after generation, unaffected by natural selection?”2

A Google search for “biology textbook” comes up with a free option through ‘openstax’ called Biology 2e. Here is what this online book (published in 2018) says:

“The diversity of life on Earth is a result of mutations, or **random** changes in hereditary material over time. These mutations allow the possibility for organisms to adapt to a changing environment. An organism that evolves characteristics fit for the environment will have greater reproductive success, subject to the forces of natural selection.”3

Think for a moment about randomness. Anything could happen, right? Is it not a limitless sea of opportunity? If you think of it like that, and if you apply the magic of seemingly limitless time, life evolving by chance can start to seem plausible. After all, that’s a lot of time for nature to gradually work with random variations and sort out only the best ones. But this has been a fairy tale all along!

According to famous evolutionist Dr. Stephen J. Gould, who said before he died:

How convenient that this “sloppy” (and ongoing) use of language just so happens to gloss over a major fundamental problem facing evolutionary theory!

“Textbooks of evolution still often refer to variation as ‘**random**’. We all recognize this designation as a misnomer, but continue to use the phrase by force of habit. Darwinians have never argued for ‘random’ mutation in the restricted and technical sense of ‘equally likely in all directions,’ as in tossing a die. But our sloppy use of ‘random’ … does capture, at least in a vernacular sense, the essence of the important claim that we do wish to convey—namely, that variation must be *unrelated to the direction of evolutionary change*; or, more strongly, that nothing about the process of creating new raw material biases the pathway of subsequent change in adaptive directions.”4

Wow, Dr. Gould, what a stunning admission! How convenient that this “sloppy” (and ongoing) use of language just so happens to gloss over a major fundamental problem facing evolutionary theory. If mutations are not random, like throwing a die, then that means certain outcomes are more likely. And if certain outcomes are more likely, then how could that *not* bias the direction of evolutionary change in the long term?

Apparently, this point is not lost on everyone. In 2014, one science writer, Kevin Kelly, came out with an article calling for the ‘retirement’ of this idea of random mutations:

“While we can’t say mutations are random, we can say there is a large chaotic component, just as there is in the throw of a loaded dice [*sic*]. But loaded dice should not be confused with randomness because over the long run—which is the time frame of evolution—the weighted bias will have noticeable consequences.”5

Why, then, do so many continue using this misleading word? Kelly has some shockingly honest answers for us:

“There are several related reasons why this unsubstantiated idea continues to be repeated without evidence [and actually, as I will show, *against* the evidence]. The first is fear that non-random mutations would be misunderstood and **twisted by creationists** to wrongly deny the reality and importance of evolution by natural selection. The second is that if mutations are not random and have some pattern, than [*sic*] that pattern creates a micro-direction in evolution. And since biological evolution is nothing but micro actions accumulating into macro actions, these micro-patterns leave open the possibility of macro directions in evolution**.**That raises all kinds of red flags. If there are evolutionary macro-directions, where do they originate? And what are the directions? To date, there is little consensus about evidence for macro-directions in evolution beyond an increase in complexity, but the very notion of evolution with any direction is so contrary to current dogma in modern evolution theory that it continues to embrace the assumption of **randomness**.”

There we have it! The use of the word “random” is (at least for some) a deliberate ploy to deceive people about the theory of evolution. Is that not what our science writer above has just admitted in writing? They don’t want us evil creationists to take the opportunity to point out all the problems that are inherent with this idea of non-random mutations. Well, too late, because now the cat is out of the bag. I’m going to sound the alarm about this major fundamental problem in evolution.

**What is ‘mutational bias’?**

If mutations are more likely to produce some results over others, then what is that tendency, exactly? The prevailing view as of now is that GC → AT mutations are more likely. To explain what that means, we need a quick refresher on basic DNA structure.

**Basics of DNA composition**

DNA is composed of 4 nucleotides, or bases, which function as the basic elements of the code (like letters). They are represented by the letters A, T, C and G. But DNA is a double helix, meaning that each base is paired on the other side of the helix with a corresponding base, and these correspond in a set way. G with C, and A with T. Thus, if you know the string of bases on one side of the double helix, you can predict the other side by simply exchanging G and C, and A and T. Scientists can look at the total sequence of DNA and compile statistics about that data. One such statistic, GC content, refers to the percentage of Gs and Cs as opposed to As and Ts.

**Figure 1**: The A-T pair is connected by two hydrogen bonds, while the C-G pair has three bonds. The hydrogen bonds between complementary bases connect the two helices of DNA together.

The base bias

There is substantial evidence that a general bias exists in all mutations toward AT (GC nucleotides are more likely to mutate into AT).6,7 As one paper by Hildebrand *et al.* states:

“It has been suggested that there is a universal mutational bias in both prokaryotes and eukaryotes towards AT… Our analysis provides some limited support for this hypothesis.”7

The exact reasons for this bias have to do with things such as the basic laws of chemistry as well as the actions of various enzymes such as DNA polymerase, and there is still much speculation and debate swirling about in this area—which goes beyond the scope of this article to address.

But if this mutational bias against GC content is going on across the board, and mutations are the “raw material” for evolution, why do we have any GC-rich genomes (or sections of genomes) at all? Some hypothetical mechanisms of selection in favour of GC content have been proposed,6 but these all seem to ignore the problem that most mutations are so small as to have negligible effects in isolation (in accordance with ‘neutral theory’).8 The authors of the Hildebrand study admit the problem and rather sheepishly write:

“Such a general decrease in GC-content across GC-rich species is clearly unsustainable … This therefore suggests that selection, **or some other force**, is maintaining high GC49 in many bacteria.”

Considering this is a peer-reviewed scientific paper, that certainly doesn’t sound very scientific, does it? “Some other force?” Another paper by Couce *et al*., in which they analysed data from Lenski’s famous Long Term Evolution Experiment (LTEE) with *E. Coli*, is similarly speculative:

Why do we have any GC-rich genomes (or sections of genomes) at all?

“Despite these mutational pressures, large GC-rich genomes are widespread across bacterial phyla, which points to strong forces driving genomes away from their mutational equilibrium. Many adaptive explanations have been suggested, including biosynthetic costs and the greater stability of GC-rich sequences under high-temperature, oxidizing, and UV irradiation conditions. **Whatever the particular selection pressures**…”6 [references omitted]

Much like in the Hildebrand paper, this amounts to an admission of ignorance, and completely ignores the problem that most mutations are too small to be selected in the first place. The idea of ‘strong forces’ at work preserving and building GC content is completely at odds with nearly neutral theory.

**Can selection overcome this bias?**

The speculation that selection may be responsible for maintaining GC content fails because most mutations are too small to be selected at all. They should have known that, given that one of the authors here (Dr Eyre-Walker) also authored a different paper where he stated, “ … particularly for multicellular organisms … most mutations, even if they are deleterious, have such small effects that one cannot measure their fitness consequences.”10

But if most mutations are that small, how can selection act on them? For a mutation to be ‘seen’ by natural selection it has to affect the organism’s ability to reproduce. Because of this it is understood, even by the secular evolutionary experts, that very small mutations are not subject to natural selection. This makes sense because natural selection is just another term for ‘differential reproduction’. If a mutation is too small to affect reproduction in any noticeable way, then selection cannot, even in principle, act on it:

“In terms of evolutionary dynamics, however, mutations whose effects are very small … are expected to be dominated by drift rather than selection.”11

Indeed, according to the commonly accepted ‘neutral theory’ of evolution, there is a limit at which mutations become too small to be selected.12 If most mutations are too small to make any detectable difference for reproduction, then it follows that most mutations are actually not being operated on by natural selection:

“Mutagenesis and mutation accumulation experiments can give us detailed information about the DFE [distribution of fitness effects] of mutations only if they have a moderately large effect, as these are the mutations that have detectable effects in laboratory assays. However, it seems likely that many and possibly the majority of mutations have effects that are too small to be detected in the laboratory.”10

**Figure 2**. Relative percentage changes in the four nucleotides in the human H1N1 virus from 1918 to 2009. Years are adjusted for the reintroduction of a strain from approximately 1955 in 1976, giving a total sampling period of 70 years. The break in the data around year 55 does not represent the 1957 extinction but missing data from 1990–1994.

[Dr John Sanford](https://creation.com/john-sanford), who developed the idea of Genetic Entropy using nothing but the assumptions of neutral theory, has done much work in recent years to test and confirm this hypothesis. A lot of this has been done with a biologically-realistic simulation program which he helped develop: ‘Mendel’s Accountant’. 13 It’s quite telling that the most realistic “evolution simulator” in the world was made by *creationists*. This program has been used to show that evolution is impossible due to the expected accumulation of damaging mutations in all evolutionary scenarios. Mendel’s Accountant shows that even with strong selection at work, fitness declines continuously over time. Some have tried to argue against it, [unsuccessfully](https://creation.com/genetic-entropy) I might add, but after more than 10 years, nobody in the secular community has ventured to challenge these results by producing a simulation of their own.

Additionally, in 2012 Drs Sanford and Carter did their own independent peer-reviewed research on the trajectory over time of the H1N1 (human strain) virus, starting from its outbreak in 1917 all the way through its final apparent demise in 2009.14 They showed that, just as Mendel’s Accountant predicted,15 mutations accumulated continuously in the population of flu viruses over time. But not only that—importantly, they also showed that the mutations accumulated “according to the laws of chemistry”. In other words, the mutations were [not really being filtered](https://creation.com/evidence-for-genetic-entropy) or guided by anything (like selection). GC content went down over time.

**The GC-Conundrum**

The question stands unanswered: why is the GC content of many genomes (and, for that matter, sections of genomes) so much higher than the mutational bias would generate? The evolutionary process that supposedly built life—mutations—is biased *against* GC and *for* AT. After hundreds of millions of years of accumulating mutations, and acting upon the assumption that mutations are the source of our genetic information, we would predict to find a level of GC content in line with the overall mutational bias. But that is not what we find.

**Conclusion: Evolution has no mechanism!**

I have been studying creation apologetics for many years (most of my life, in fact), and I was stunned when I discovered this well-kept evolutionary secret. Most people, including those educated in biological science, have absolutely no idea this major issue exists. As I explored in some of the quotes above, it appears this general ignorance is no accident; those in the know about this deliberately decide not to bring it up, so as to avoid embarrassment for the sacred Primary Axiom of evolution. It is time for us creationists to break the silence in a big way! The fact that mutations are biased in a particular direction due to the laws of chemistry means that we have powerful evidence that *mutations are not the original source of the information* in DNA. This is by no means the only problem with Darwinian evolution, but this problem is particularly devastating because it shows a deep insufficiency in evolutionary theory at the most fundamental level. Evolution is like a blindfolded person trying to build the Notre Dame cathedral out of Legos**®** one haphazardly-placed block at a time. The more we learn, the more Darwinism is revealed to be a primitive myth, while the Bible’s account that life was authored by God is shown true.

**Refutation of *New Scientist’s* Evolution: 24 myths and misconceptions**

Evolution v natural selection

***by***[***Jonathan Sarfati***](https://creation.com/dr-jonathan-d-sarfati)

Ed. Note: this is the third instalment of a detailed critique of a major *New Scientist* anti-creationist diatribe (see [introduction and index page](https://creation.com/refutation-of-new-scientists-evolution-24-myths-and-misconceptions-index)). This one deals the widespread confusion between evolution and natural selection, actually a process discovered by creationists and an important part of the creation model.

**Evolution: 24 myths and misconceptions**

It will soon be 200 years since the birth of Charles Darwin and 150 years since the publication of *On the Origin of Species*, arguably the most important book ever written. In it, Darwin outlined an idea that many still find shocking – that all life on Earth, including human life, evolved through natural selection.

Yet even many evolutionists admit that his book actually didn’t *demonstrate* what the title indicated: the origin of species. One of Darwin’s highly qualified contemporaries, [Professor Johann H. Blasius, director of the Duke’s Natural History Museum of Braunschweig](https://creation.com/natural-history-museum-director-of-darwins-day-denounces-his-theory) (Brunswick), Germany, was highly critical:

‘I have also seldom read a scientific book which makes such wide-ranging conclusions with so few facts supporting them. … Darwin wants to show that *Arten* [types, kinds, species] come from other *Arten*. I regard this as somewhat of a highhanded hypothesis, because he argues using unproven possibilities, without even naming a single example of the origin of a particular species.’ 1

If truth be told, evolution hasn’t yielded many practical or commercial benefits. … Evolution cannot help us predict what new vaccines to manufacture because microbes evolve unpredictably. But hasn’t evolution helped guide animal and plant breeding? Not very much. Most improvement in crop plants and animals occurred long before we knew anything about evolution, and came about by people following the genetic principle of ‘like begets like’. Even now, as its practitioners admit, the field of quantitative genetics has been of little value in helping improve varieties.—Antitheistic evolutionist Jerry Coyne

And despite its hyped up ‘importance’, evolution provides no practical benefit to biology—see the detailed discussion in [Does science need evolution?](https://creation.com/science-creation-and-evolutionism-refutation-of-nas) A modern evolutionist—and ardent misotheist—Jerry Coyne, argues that evolution is important as his (atheistic) theory of ‘How did we get here?’, but had to admit:

[I]f truth be told, evolution hasn’t yielded many practical or commercial benefits. Yes, bacteria evolve drug resistance, and yes, we must take countermeasures, but beyond that there is not much to say. Evolution cannot help us predict what new vaccines to manufacture because microbes evolve unpredictably. But hasn’t evolution helped guide animal and plant breeding? Not very much. Most improvement in crop plants and animals occurred long before we knew anything about evolution, and came about by people following the genetic principle of ‘like begets like’. Even now, as its practitioners admit, the field of quantitative genetics has been of little value in helping improve varieties. Future advances will almost certainly come from transgenics, which is not based on evolution at all.2

And even the claim about bacteria ‘evolving’ drug resistance is overstated, because this took evolutionists by surprise when it first occurred, and the changes involved are not those that would evolve bacteria into biologists. See the discussion in [Anthrax and antibiotics: Is evolution relevant?](https://creation.com/anthrax-and-antibiotics-is-evolution-relevant)

Darwin presented compelling evidence for evolution in *On the Origin* and, since his time, the case has become overwhelming.

Often those who declare the evidence to be ‘overwhelming’ or that ‘the debate is over’ say this to avoid debate. That’s why opposition is censored by ‘[peer review](https://creation.com/creationism-science-and-peer-review)‘ and opponents often [demonized](https://creation.com/article/2730/31/) or [discriminated against](https://creation.com/darwinian-thought-police-strike-again), as documented in the new film [Expelled](https://creation.com/store_redirect.php?sku=30-9-555). As Thomas Sowell (1930– ) pointed out in another context (in his book *Race and Culture*about politically correct theories on race):

‘No belief can be refuted if it cannot be discussed.’

Countless fossil discoveries allow us to trace the evolution of today’s organisms from earlier forms.

Yet experts point out that it’s impossible to tell from fossils whether one creature was an ancestor of another, such as the late Colin Patterson. Furthermore, the fossil record should show gradual change from one kind of creature to another, millions of times over and it does not. Evolutionist Stephen Jay Gould called the scarcity of transitional fossils the ‘[trade secret of paleontology](https://creation.com/that-quote-about-the-missing-transitional-fossils)’ (see [this analysis](https://creation.com/that-quote-about-the-missing-transitional-fossils) as well as [The Links Are Missing](https://creation.com/chapter-3-the-links-are-missing)). For example, one evolutionist admitted:

‘The oldest bat fossils, belonging to an extinct lineage, were unearthed from rocks about 54 million years old, but the creatures that they represent aren’t dramatically different from living bats, says Mark S. Springer, an evolutionary biologist at the University of California, Riverside.

Hallmark features of these creatures [the ‘earliest’ fossil bats] include the elongated fingers that support the wing membranes and the extensive coiling of bony structures in the inner ears, a sign that they were capable of detecting the high-frequency chirps used in echolocation

‘Hallmark features of these creatures include the elongated fingers that support the wing membranes and the extensive coiling of bony structures in the inner ears, a sign that they were capable of detecting the high-frequency chirps used in echolocation.’3

DNA sequencing has confirmed beyond any doubt that all living creatures share a common origin.

*Ipse dixit*(a dogmatic assertion without supporting evidence)*.* All it can show are *similarities*; common origin as opposed to common design is an *interpretation*, and [one fraught with problems](https://creation.com/chapter-6-argument-common-design-points-to-common-ancestry). Rather, they support the *biotic message theory*, as proposed by Walter ReMine in [The Biotic Message](https://creation.com/store_redirect.php?sku=10-3-007). That is, the evidence from nature points to a *single* designer, but with a pattern which thwarts evolutionary explanations because of the many similarities that cannot be explained by any theory of common ancestry—such as the incredible similarities between many marsupials and their placental counterparts (e.g. flying squirrels and flying phalangers—see [Are look-alikes related?](https://creation.com/are-look-alikes-related-creation-magazine)). Also, in most cultures that have ever existed, a consistent unifying pattern ‘brought honour to the Creator and would also indicate the Creator’s authority over and mastery of His creation.’4

Innumerable examples of evolution in action can be seen all around us, from the pollution-matching pepper moth to fast-changing viruses such as HIV and H5N1 bird flu.

Is this the best they can offer? These are examples of [a theory invented by the creationist, Edward Blyth](https://creation.com/charles-darwins-illegitimate-brainchild), wrongly claimed as Darwin’s invention, and today is an important part of the creation model: [natural selection](https://creation.com/natural-selection-questions-and-answers) (see also [Darwin and the search for an evolutionary mechanism](https://creation.com/darwin-and-the-search-for-an-evolutionary-mechanism), which shows the historical and philosophical influences on Darwin’s ostensibly scientific theory). They have nothing to do with turning moths into motorists or viruses into vets, because the [changes are in the wrong direction](https://creation.com/the-evolution-trains-a-comin), i.e. *removing* information instead of *adding* it as goo-to-you evolution requires.

Conflating natural selection and evolution is a staple of evolutionary propaganda. Recognizing this alone would almost be enough to see through the dogma. I’ll address these specific examples in later instalments when Le Page, the *New Scientist* author, cites them in more detail.

Evolution is as firmly established a scientific fact as the roundness of the Earth.

‘Evolution has been observed. It’s just that it hasn’t been observed while it’s happening.’—leading misotheist Richard Dawkins

The roundness of the earth can be *observed* (see also [these articles refuting the flat earth myth](https://creation.com/countering-the-critics-questions-and-answers)); but evolution can’t be. Or in the words of Dawkins:

‘Evolution has been observed. It’s just that it hasn’t been observed while it’s happening.’5

And yet despite an ever-growing mountain of evidence, most people around the world are not taught the truth about evolution, if they are taught about it at all.

That’s true: the government schools and MMM ([Mendacious Mainstream Media](https://creation.com/time-and-newsweek-blatantly-attack-christian-doctrine)) teach evolution as fact, which is not the truth about it!

Even in the UK , the birthplace of Darwin with an educated and increasingly secular population, one recent poll suggests less than half the population accepts evolution.

So, despite the huge amount of evolutionary indoctrination, the [indoctrinators](https://creation.com/the-indoctrinator) are unhappy that it’s not working on everyone. And this includes even [deliberately misleading students as long as it convinces them that evolution is true](http://www.evolutionnews.org/2008/08/lying_in_the_name_of_indoctrin.html), since they believe, ‘Education is a subversive activity that is implicitly in place in order to counter the prevailing … deeply conservative religious culture.’

For those who have never had the opportunity to find out about biology or science, claims made by those who believe in supernatural alternatives to evolutionary theory can appear convincing. Meanwhile, even among those who accept evolution, misconceptions abound.

Yes, we have already encountered some propounded by Le Page, arguing that examples of an observable process (natural selection) is equivalent to proving the historical goo-to-you claim.

Most of us are happy to admit that we do not understand, say, string theory in physics,

True enough. Indeed, *New Scientist* itself has documented that even experts are confused by it,6,7 and reported the joke about why our universe is unique: it’s the only one string theory can’t explain. See also [String theory unstrung](https://creation.com/evolutionists-puzzle-over-the-origin-of-the-universe).

yet we are all convinced we understand evolution. In fact, as biologists are discovering, its consequences can be stranger than we ever imagined. Evolution must be the best-known yet worst-understood of all scientific theories.

Then blame the propaganda pieces — like this one — that are more interested in point-scoring and word games than educating.

So here is *New Scientist’s* guide to some of the most common myths and misconceptions about evolution.

So here is *New Scientist’s* admission of ownership of this shoddy drivel, so they deserve all they get as a result. The gossip on the skeptics’ own websites suggested that *Scientific American (SciAm)* had suffered a financial downturn as a result of their ‘mistake’ in producing their anti-creationist article. A [rebuttal on this site to a National Geographic anti-creationist tirade](https://creation.com/national-geographic-is-wrong-and-so-was-darwin) also resulted in people [switching subscriptions](https://creation.com/national-geographic-readers-switch-to-tj-dont-spout-off-your-faith-as-science) to what is now our [Journal of Creation](https://creation.com/journal-of-creation).

There are already several good and comprehensive guides out there. But there can’t be too many.

However, one of the allegedly good guides was the [SciAm article that we demolished](https://creation.com/15-ways-to-refute-materialistic-bigotry)! Others were on [skeptics websites, which so often prove not be at all objective and reliable](https://creation.com/countering-the-critics-questions-and-answers).

**Shared misconceptions:**

**Everything is an adaptation produced by natural selection**

*We tend to assume that all characteristics of plants and animals are adaptations that have arisen through natural selection. Many are neither adaptations nor the result of selection at all.*

The 20-nanometer motor (height), ATP synthase (one nanometer is one thousand-millionth of a metre). These rotary motors in the membranes of mitochondria (the cell’s power houses) turn in response to proton flow (a positive electric current). Rotation of the motor converts ADP molecules plus phosphate into the cell’s fuel, ATP.

This is all true. But when it comes to the complex machinery of life, such as [ATP synthase](https://creation.com/design-in-living-organisms-motors-atp-synthase) and the [DNA winding](https://creation.com/viral-motor-helps-it-pack-long-strand-of-dna) motors, natural selection is the only game in town to try to avoid the overwhelming improbability of these machines arriving by chance (that is avoiding the abundantly clear implication that a super-intelligent Creator designed them).

Why do so many of us plonk ourselves down in front of the telly with a microwave meal after a tiring day? Because it’s convenient? Or because TV meals are ‘[the natural consequence of hundreds of thousands of years of human evolution](http://www.admin.cam.ac.uk/news/press/dpp/2007041302)‘?

Stop laughing. You’ve probably made similar assumptions.

Hence our article [Evolution made me do it!](https://creation.com/evolution-made-me-do-it-creation-magazine)

For just about every aspect of our bodies and behaviour, it’s easy to invent evolutionary [Just So stories](http://www.newscientist.com/article/mg16322056.800-just-so-stories.html) to explain how they came to be that way.

Don’t blame the public; blame the *evolutionary establishment* that tolerates such [Just So stories](http://www.newscientist.com/article/mg16322056.800-just-so-stories.html) and then feed them to the public. And why the tolerance of the scientific community for [Just So stories](http://www.newscientist.com/article/mg16322056.800-just-so-stories.html)? An *a priori* commitment to materialism, [according to atheist genetics professor Richard Lewontin](https://creation.com/amazing-admission-lewontin).

We tend to assume that [everything has a purpose](http://www.newscientist.com/article/mg18324635.600-ia-reason-for-everything-natural-selection-and-the-english-imaginationi-by-marek-kohn-2004.html), but often we are wrong.

This is a criticism of prominent evolutionary ‘adaptationism’. Gould and Lewontin invented the term ‘spandrel’ for features that supposedly arose not because of any direct adaptation, but as a by-product. This comes from cathedral architecture, a spandrel being the space between a rectangular corner and an inside curve such as an arch. It is also used of the space under a staircase. In cathedrals, this arch could be filled with a richly decorated panel or stained glass, and space under stairs is often used for a cabinet. But although the spaces could be put to use, the spaces were not intended per se, but were merely a consequence of something designed for another purpose (structural strength). Since artists use spandrels as a ‘canvas’ on which to paint their decorations, Gould argued that organisms could likewise use ‘functionless’ artefacts of anatomy for some new purpose.8

Take male nipples. Male mammals clearly don’t need them: they have them because females do and because it doesn’t cost much to grow a nipple. So there has been no pressure for the sexes to evolve separate developmental pathways and ‘switch off’ nipple growth in males.

We agree, except that it has nothing to do with ‘evolution’ switching or not switching as shown in [Male nipples prove evolution?](https://creation.com/male-nipples-prove-evolution)

…

Then there’s our sense of smell. Do you find the scent of roses overwhelming or do you struggle to detect it? Can you detect the distinctive odour that most people’s urine acquires after eating asparagus? People [vary greatly](http://dx.doi.org/10.1038/nature06162) when it comes to smell, [largely due](http://dx.doi.org/10.1038/79829) to [chance mutations](http://www.newscientist.com/channel/being-human/mg19526225.600-sniffers-genes-dictate-if-sweat-smells-sweet.html) in the genes that code for the smell receptors rather than for adaptive reasons.

Certainly. So this has nothing to do with evolution, and it could be a *built-in* high-mutation system that can scan a wide range of chemicals. The [elaborate design of the olfactory system, likely based on the principles of vibrational spectroscopy](https://creation.com/olfactory-design-smell-and-spectroscopy-journal-of-creation-tj), would make this very easy, because the mutations could cause small changes in the quantum energy levels of the receptors. The elaborate olfactory system speaks of incredible design, not evolution.

Yet other features are the result of selection, but not for the trait in question. For instance, the [short stature of pygmies](http://www.newscientist.com/channel/being-human/mg19626343.900-modern-times-causing-human-evolution-to-accelerate.html) could be a side effect of selection for early childbearing in populations where mortality is high, rather than an adaptation in itself.

That’s reasonable. But once again, nothing here is incompatible with the biblical [Creation/Fall/Flood/Dispersion model](https://creation.com/episode-1-darwins-dangerous-idea), of which variation, natural selection and speciation are important parts.

**Multiskilled genes**

Another reason why apparent adaptations can be side effects of selection for other traits is that genes can have different roles at different times of development or in different parts of the body. So selection for one variant can have all sorts of seemingly unrelated effects.

This is called pleiotropy, and is a huge problem for evolution directed by natural selection. I.e. natural selection may not be able to improve one characteristic so straightforwardly without deleterious effects on other characteristics. The fact that [on average each human gene codes for 4 or 5 proteins underlines the problem](https://creation.com/message-mania-creation-magazine). One well known example of pleiotropy is one form of blindness in cave fish—see [Christopher Hitchens blind to salamander reality: A well-known atheist’s eureka moment shows the desperation of evolutionists](https://creation.com/christopher-hitchens-blind-to-salamander-reality).

[Male homosexuality](http://www.newscientist.com/article/dn6519-survival-of-genetic-homosexual-traits-explained.html) might be linked to gene variants that increase fertility in females, for instance.

This presupposes a genetic basis for homosexual behaviour in the first place (see for example [Homosexual animals](https://creation.com/homosexual-animals)). This will be discussed in later instalments, but meanwhile see the articles under [Homosexuality: What are the biblical and scientific issues?](https://creation.com/morality-and-ethics-questions-and-answers)

A non-adaptive or detrimental gene variant can also spread rapidly through a population if it is on the [same DNA strand](http://dx.doi.org/10.1073/pnas.212277199) as a highly beneficial variant. This is one reason why sex matters: when bits of DNA are swapped between chromosomes during sexual reproduction, good and bad variants can be split up.

Indeed, there is no dispute that sexual reproduction has its advantages. But explaining *how this arose in the first place* is a problem for evolutionists—see [Evolution of sex?](https://creation.com/refuting-evolution-2-chapter-11-argument-evolution-of-sex)

Other features of plants and animals, such as the wings of ostriches, may once have been adaptations but are no longer needed for their original purpose.

As we have argued in [Vestigial Organs: What do they prove?](https://creation.com/vestigial-organs-what-do-they-prove):

There are at least three possibilities as to why ostriches, emus, etc have wings:

a) They derived from smaller birds that once could fly. This is possible in the creationist model. Loss of features is relatively easy by natural processes; acquisition of new characters, requiring new DNA information, is impossible.

b) The wings have a function. Some possible functions, depending on the species of flightless bird, are: balance while running, cooling in hot weather, warmth in cold weather, protection of the rib-cage in falls, mating rituals, scaring predators (I’ve seen emus run at perceived enemies of their chicks, mouth open and wings flapping), sheltering of chicks, etc. If the wings are useless, why are the muscles functional that allow these birds to move their wings?

c) It is a result of ‘design economy’ by the Creator. Humans use this with automobiles, for example. All models might have mounting points for air conditioning, power steering, etc. although not all have them. Likewise, all models tend to use the same wiring harness, although not all features are necessarily implemented in any one model. In using the same embryological blueprint for all birds, all birds will have wings.

Such ‘[vestigial traits](http://www.newscientist.com/article/mg15821315.200-a-waste-of-space.html)’ can persist because they are neutral, because they have taken on another function or because there hasn’t been enough evolution to eliminate them even though they have become disadvantageous.

If they have another function, then it is compatible with being created that way. Often the ‘vestigial organ’ argument is an appeal to ignorance: we don’t know a function, therefore it has none. There are so many times when organs thought to be useless turn out to have important functions, e.g. [short muscle fibres in horse legs that turn out to have a vital role in dampening vibrations](https://creation.com/useless-horse-body-parts-no-way-creation-magazine), as well as [the important thyroid and thymus glands](https://creation.com/performing-surgery-upon-evolutionary-thinking).

Take the [appendix](http://www.talkorigins.org/faqs/vestiges/appendix.html). There are plenty of claims that it has this or that function but the evidence is clear: you are more likely to survive without an appendix than with one.

This is outdated. The appendix has long been known to be [rich in lymphatic tissue](https://creation.com/the-human-vermiform-appendix-journal-of-creation-tj), and is now thought to be a ‘safe-house’ for bacteria, to ‘reboot’ the colon flora in case of an infection that clears them out.9 See [Appendix: a bacterial safe house : New research suggests function for appendix in maintaining good digestive bacteria populations](https://creation.com/appendix-a-bacterial-safe-house). The problem of appendicitis seems to result from a fibre-poor Western diet and perhaps even ‘[another case of an overly hygienic society triggering an overreaction by the body’s immune system.](http://web.archive.org/web/20071011011949/http%3A/edition.cnn.com/2007/HEALTH/10/05/appendix.purpose.ap/index.html?iref=mpstoryview)

Also, if this vestigial idea were true, then we would expect that more ‘primitive’ primates would have a more developed appendix, but this is not so. Rather, the appendix is *more* prominent in humans and gorillas. So one evolutionist researcher claimed that it gradually *progressed* to the current ‘fully developed organ’, so it ‘should not be regarded, in the anthropoid apes and man, as a purely degenerative structure’.10 See also [More musings on our useless appendix: A not-so-recent study on the pattern of the appendix among our alleged primate cousins showed that, even using evolutionary assumptions, it cannot be a degenerate evolutionary structure](https://creation.com/more-musings-on-our-useless-appendix).

So why hasn’t it disappeared? Because [evolution](http://www.newscientist.com/channel/life/evolution) is a numbers game. The worldwide human population was tiny until a few thousand years ago, and people have few children with long periods between each generation. That means fewer chances for evolution to throw up mutations that would reduce the size of the appendix or eliminate it altogether — and fewer chances for those mutations to spread through populations by natural selection. Another possibility is that we are stuck in an evolutionary Catch-22 where, as the appendix shrinks, appendicitis becomes more likely, favouring its retention.

Yet in this supposed numbers game, there were enough mutations to cause [bipedalism](https://creation.com/the-lucy-child) and development of a large brain enabling [language](https://creation.com/linguistics-questions-and-answers) development.

Wisdom teeth are another vestigial remnant. A smaller, weaker jaw allowed our ancestors to grow larger brains, but left less room for molars. Yet many of us still grow teeth for which there is no room, with potentially fatal consequences. One possible reason why wisdom teeth persist is that they usually appear after people reach reproductive age, meaning selection against them is weak.

This is also outdated. Wisdom teeth are rarely a problem for people, except those enjoying a modern western diet with soft, processed foods. This means less hard chewing during childhood jaw development, which causes a reduction in jaw size, and less stimulation of natural forward tooth movement in the jaw that would normally leave room for the third molar. Also, many dental surgeons caution against removal of these teeth unless they cause actual problems, not just as a preventive measure. See also [Are wisdom teeth (third molars) vestiges of human evolution?](https://creation.com/are-wisdom-teeth-third-molars-vestiges-of-human-evolution)

For all these reasons and more, we need to be sceptical of headline-grabbing claims about evolutionary explanations for different behaviours. [Evolutionary psychology](http://www.newscientist.com/channel/opinion/mg18725161.700-the-glamour-of-evolutionary-psychology.html) in particular is notorious for attempting to explain [every aspect](http://www.newscientist.com/channel/being-human/mg19426104.400-yawning-may-boost-brains-alertness.html) of [behaviour](http://www.newscientist.com/article/mg15821336.100-genes-in-the-family.html), from gardening to [rape](http://www.newscientist.com/article/mg16522264.500-crimes-of-passion.html), as an adaptation that arose when our ancestors lived on the African savannah.

Yes, see for example [Rape and evolution: Evolution shows its true colours](https://creation.com/rape-and-evolution) and [Evolution of mankind](https://creation.com/chapter-12-argument-evolution-of-mankind). This cites Jerry Coyne, a strong opponent of evolutionary psychology, as saying that [memes](https://creation.com/chapter-12-argument-evolution-of-mankind) are ‘but a flashy new wrapping around a parcel of old and conventional ideas.’

…

**Natural selection is the only means of evolution**

*Much change is due to random genetic drift rather than positive selection. It could be called the survival of the luckiest.*

Take a look in the mirror. The face you see is rather different to that of a Neanderthal. Why? The unflattering answer could be for no other reason than random genetic drift. With features that can vary somewhat in form without greatly affecting function, such as the [shape of the skull](http://www.newscientist.com/channel/being-human/mg19526135.700-chance-and-isolation-gave-humans-elegant-skulls.html), [chance](http://dx.doi.org/10.1073/pnas.0709079105) [might](http://johnhawks.net/weblog/topics/evolution/neutral/neutral-neandertals-weaver-2008.html) play a [bigger role](http://www.newscientist.com/article/dn2908-gene-technique-reveals-human-evolution.html) in their evolution than natural selection.

Random genetic drift happens, but it has nothing to do with explaining how some reptiles changed into birds, for example. Such random changes do not explain the origin of the complex, integrated DNA coding necessary to specify how to make new features such as feathers. [Neandertals were likely post-Babel humans](https://creation.com/symbolic-items-really-show-that-neandertals-had-human-cognition-and-symbolic-thinking) [adapted](https://creation.com/chapter-2-variation-and-natural-selection-versus-evolution) for the [post-Flood Ice Age](https://creation.com/ice-age-questions-and-answers).

The DNA in all organisms is under [constant attack from highly reactive chemicals and radiation](http://en.wikipedia.org/wiki/Mutation), and errors are often made when it is copied. As a result, there are [at least 100](http://sandwalk.blogspot.com/2007/07/mutation-rates.html) new mutations in each human embryo, possibly far more. Some are harmful and are likely to be eliminated by natural selection — by death of the embryo, for instance. Most make no difference to our bodies, because most of our DNA is [useless junk](http://www.newscientist.com/channel/life/mg19526121.500-genomics-junking-the-junk-dna.html) anyway.

More outdated nonsense, and largely derived from the evolutionary assumption that we have been around for millions of years. I.e. if much of our genome were functional, [such a high rate of mutation would lead to error catastrophe unless most were non-functional](https://creation.com/review-of-the-biotic-message). However, at least 97% of our DNA is now known to be transcribed, but much of it into regulatory RNA molecules rather than proteins. See [Astonishing DNA complexity uncovered](https://creation.com/new-research-debunks-alsquojunk-dnaarsquo-and-creates-a-giant-headache-for-materialists) and [update](https://creation.com/more-astonishing-dna-complexity). This is further evidence that the evolutionary timescale is false because if we had been here for millions of years we would be extinct from the damage that mutations cause.

Dr John Sanford inventor of the gene gun, explains this in his new book [Genetic Entropy and the mystery of the genome](https://creation.com/store_redirect.php?sku=10-3-513) (see also [Plant geneticist: Darwinian evolution is impossible](https://creation.com/geneticist-evolution-impossible), and his research papers published in secular journals.

A future instalment will discuss ‘junk DNA’ further, but meanwhile see the articles under [What about Vestigial ( junk ) DNA that evolutionists claim is a useless leftover of evolution?](https://creation.com/vestigial-organs-questions-and-answers)

A few cause minor changes that are neither particularly harmful nor beneficial.

You might think that [largely neutral mutations](http://mbe.oxfordjournals.org/cgi/content/abstract/22/12/2318) would remain restricted to a few individuals. In fact, while the vast majority of neutral mutations die out, a few spread throughout a population and thus become ‘fixed’. It is pure chance — some just happen to be passed on to more and more individuals in each generation.

Although the likelihood of any neutral mutation spreading by chance is tiny, the enormous number of mutations in each generation makes [genetic drift](http://www.biology.arizona.edu/evolution/act/drift/about.html) a significant force. It’s a little like a lottery: the chance of winning is minuscule but because millions buy a ticket every week there is usually a winner.

And this drift has a good chance of eliminating even the rare *beneficial* mutations. This is a big problem for the gradualistic theories of evolution: the smaller the effect of a mutation, the more likely that drift will swamp its selective advantage. See [this discussion in a review of Dawkins Climbing Mount Improbable](https://creation.com/book-review-of-dawkins-climbing-mount-improbable).

As a result, most changes in the DNA of complex organisms over time are due to drift rather than selection, which is why biologists focus on sequences that are similar, or conserved, when they compare genomes. Natural selection will preserve sequences with vital functions, but the rest of the genome will change because of drift.

The actual *evidence* says the opposite. Most mutations have a small effect, so are immune from selection pressure. And genetic drift can often eliminate *beneficial* mutations.

See diagram (right) from Dr Sanford’s book (below)

Far more mutations are deleterious than advantageous. Individually, most have too small an effect to be acted upon by natural selection.

**Drifting through bottlenecks**

Genetic drift can even counteract natural selection. Many slightly [beneficial mutations](http://dx.doi.org/10.1038/sj.hdy.6801077) can be lost by chance, while mildly deleterious ones can spread and become fixed in a population. The smaller a population, the greater the role of genetic drift.

This is true. But then there is a lower supply of mutations, so it will take much longer for mutations to throw up anything useful.

Population bottlenecks can have the same effect. Imagine an island where most mice are plain but a few have stripes. If a volcanic eruption wipes out all of the plain mice, the island will be repopulated by striped mice. It’s a case of survival not of the fittest, but of the [luckiest](http://www.newscientist.com/article/mg17323294.100-law-of-the-jungle.html).

And nothing to do with evolution, because the disaster merely *removed* some information from the gene pool by chance.

Random genetic drift has certainly played a [big role in human evolution](http://www.newscientist.com/channel/being-human/dn6920-hominid-inbreeding-left-humans-vulnerable-to-disease.html). Human populations were tiny until around 10,000 years ago, and went through a [major bottleneck](http://mbe.oxfordjournals.org/cgi/reprint/17/1/2) around 2 million years ago. [Other bottlenecks occurred](http://www.newscientist.com/article/dn721-founding-fathers.html) when a few individuals migrated out of Africa around 60,000 years ago and colonised other regions.

The evidence for bottlenecks is *consistent with* a biblical model of creation (e.g. [mitochondrial Eve](https://creation.com/cmi-responds-to-sceptics-criticism-of-mitochondrial-eve-article)) and the Flood. See also [Out of Africa theory going out of style?](https://creation.com/out-of-africa-theory-going-out-of-style)

There is no doubt that most of the genetic differences between humans and other apes — and between different human populations — are due to genetic drift. However, most of these mutations are in the nine-tenths of our genome that is junk, so they make no difference. The interesting question is [which mutations](http://hmg.oxfordjournals.org/cgi/content/abstract/16/19/2281) affecting our bodies or behaviour have spread because of [drift rather than selection](http://www.newscientist.com/channel/being-human/mg18925421.300-are-we-still-evolving.html), but this is far from clear.

Since at least nine-tenths of our genome is known to be functional, this argument collapses. But see also [Decoding the dogma of DNA similarity](https://creation.com/decoding-the-dogma-of-dna-similarity) and [Greater than 98% Chimp/human DNA similarity? Not any more: A common evolutionary argument gets reevaluated by evolutionists themselves](https://creation.com/greater-than-98-chimphuman-dna-similarity-not-any-more-journal-of-creation-tj).

**Natural selection leads to ever-greater complexity**

*In fact, natural selection often leads to ever greater simplicity. And, in many cases, complexity may initially arise when selection is weak or absent*.

If you don’t use it, you tend to lose it. Evolution often takes away rather than adding. For instance, [cave fish](http://en.wikipedia.org/wiki/Cave_fish) lose their eyes, while parasites like [tapeworms](http://en.wikipedia.org/wiki/Tapeworm) lose their guts.

Sure, there are many natural ways to *destroy* information, but evolution needs a viable, believable way to generate it. See [Let the blind see Breeding blind fish with blind fish restores sight](https://creation.com/let-the-blind-see).

Such [simplification](http://www.newscientist.com/article/mg16121694.700-making-life-simple.html) might be [much more widespread](http://www.newscientist.com/channel/life/mg18925441.400-half-virus-half-beast%E2%80%93its-a-20sided-freak.html) than realised. Some [apparently primitive creatures](http://www.newscientist.com/channel/life/mg19426083.100-evolution-hacking-back-the-tree-of-life.html) are turning out to be the descendants of more complex creatures rather than their ancestors. For instance, it appears the ancestor of brainless starfish and sea urchins had a brain.

Which is compatible with the [Fall](https://creation.com/the-fall-a-cosmic-catastrophe-journal-of-creation-tj) which was [cosmic in scope](https://creation.com/cosmic-and-universal-death-from-adamarsquos-fall).

Nevertheless, there is no doubt that evolution has produced more complex life-forms over the past four billion years. The tough question is: why? It is usually simply assumed to be the result of natural selection, but recently a few [biologists](http://www.bio.indiana.edu/facultyresearch/faculty/Lynch.html) studying our own [bizarre and bloated genomes](http://www.newscientist.com/channel/life/mg19526121.500-genomics-junking-the-junk-dna.html) have [challenged this idea](http://dx.doi.org/10.1126/science.1089370).

No doubt? Well of course there is no doubt if you are a true believer and have decided that you don’t want to believe in a Creator; then evolution is the only game in town, by definition. See [A tale of two fleas.](https://creation.com/a-tale-of-two-fleas) And once again *New Scientist* promotes the outmoded idea that our genomes are full of junk (‘bloated’).

Rather than being driven by selection, they propose that complexity initially arises [when selection is weak](http://www.newscientist.com/article/dn4817-early-humans-swapped-bite-for-brain.html) or absent. How could this be? Suppose an animal has a gene that carries out two different functions. If mutation results in some offspring getting [two copies of this gene](http://www.newscientist.com/channel/life/mg19025461.300-genomics-we-are-all-numbers.html), these offspring won’t be any fitter as a result. In fact, they might be slightly less fit due to a double dose of the gene. In a large population where the selective pressure is strong, such mutations are likely to be eliminated. In smaller populations, where selective pressure is much weaker, these mutations could spread as a result of random genetic drift (see *Natural selection is the only means of evolution*) despite being slightly disadvantageous.

[Gene or chromosome duplication](https://creation.com/does-gene-duplication-provide-the-engine-for-evolution) is hardly the answer. In plants, but not in animals (possibly with rare exceptions), the doubling of all the chromosomes may result in an individual which can no longer interbreed with the parent type—this is called *polyploidy*. Although this may technically be called a new species, because of the reproductive isolation, no new information has been produced, just repetitious doubling of *existing* information. If a malfunction in a printing press caused a book to be printed with every page doubled, it would not be more informative than the proper book. (Brave students of evolutionary professors might like to ask whether they would get extra marks for handing in two copies of the same assignment.)

Duplication of a single chromosome (which contains many genes) is normally harmful, as in Down’s syndrome. Insertions are a very efficient way of completely destroying the functionality of existing genes, so if a duplicated gene is inserted randomly, it would likely cause damage to other functioning genes.

The evolutionist’s ‘gene duplication idea’ is that an existing gene may be doubled, and one copy does its normal work while the other copy is non-expressed. Therefore, it is free to mutate free of selection pressure (to get rid of it). However, such ‘neutral’ mutations are powerless to produce new genuine information. Dawkins and others point out that natural selection is the only possible naturalistic explanation for the immense design in nature (not a good one, as Spetner and others have shown). Dawkins and others propose that random changes produce a new function, then this redundant gene becomes expressed somehow and is fine-tuned under the natural selective process.

This ‘idea’ is just a lot of hand-waving. It relies on a chance copying event, genes somehow being switched off, randomly mutating to something approximating a new function, then being switched on again (how?) so natural selection can tune it.

Furthermore, mutations do not occur in just the duplicated gene; they occur throughout the genome. Consequently, all the deleterious mutations in the rest of the genome have to be eliminated by the death of the unfit. Selective mutations in the target duplicate gene are extremely rare—it might represent only 1 part in 30,000 of the genome of an animal. The larger the genome, the bigger the problem, because the larger the genome, the lower the mutation rate that the creature can sustain without error catastrophe; as a result, it takes even longer for *any* mutation to occur, let alone a desirable one, in the duplicated gene. There just has not been enough time, even with mythical evolutionary time, for such a naturalistic process to account for the amount of genetic information that we see in living things.

Two geneticists [argue](https://creation.com/do-new-functions-arise-by-gene-duplication):

1. gene duplications are aberrations of cell division processes and are more likely to cause malformation or diseases rather than selective advantage
2. duplicated genes are usually silenced (no longer produce proteins) and subjected to degenerative mutations
3. regulation of supposedly duplicated gene clusters and gene families is irreducibly complex, and demands simultaneous development of fully functional multiple genes and switching networks, contrary to Darwinian gradualism.11

The more widely the [duplicated genes](http://www.newscientist.com/article/mg18124355.300-the-great-inventors.html) spread in a population, the faster they will acquire mutations. A mutation in one copy might destroy its ability to carry out the first of the original gene’s two functions. Then the other copy might lose the ability to perform the second of the two functions. As before, these mutations won’t make the animals any fitter – such animals would still look and behave exactly the same – so they will not be selected for, but they could nevertheless spread by genetic drift.

This is another problem for the gene duplication idea.

**Use your mutations**

In this way, a species [can go](http://www.genetics.org/cgi/content/abstract/151/4/1531?ijkey=c4605368fe5edabead24a0d1e8196c6e363af1a2%26keytype2=tf_ipsecsha) from having one gene with two functions to two genes that each carry out one function. This increase in complexity occurs not because of selection but despite it.

Once the genome is more complex, however, further mutations can make a creature’s body or behaviour [more complex](http://www.newscientist.com/channel/life/mg19225831.100-festive-special-the-brewers-tale.html). For instance, having two separate genes means each can be switched on or off at different time or in different tissues. As soon as any beneficial mutations arise, natural selection will [favour its spread](http://dx.doi.org/10.1186/1745-6150-2-17).

If this picture is correct, it means that there are opposing forces at the heart of evolution. Complex structures and behaviour such as eyes and [language](http://www.newscientist.com/article/dn2678-gene-study-gives-language-lesson.html) are undoubtedly the product of natural selection.

Undoubtedly? Once again the philosophy of materialism reigns over rational thought and proper scepticism. Also, evolution does not explain the origin of [eyes](https://creation.com/book-review-of-dawkins-climbing-mount-improbable) or [language](https://creation.com/chapter-12-argument-evolution-of-mankind).

But when selection is strong—as in large populations—it blocks the random genomic changes that throw up this greater complexity in the first place.

Yet natural selection is the only real materialistic solution to the origin of complexity. That’s why atheists such as Dawkins defend(ed) it so strongly (see [A Who’s Who of evolutionists](https://creation.com/a-whos-who-of-evolutionists)**)**.

This idea might even explain why [evolution appears to speed up](http://www.landesbioscience.com/journals/cc/article/kooninCC3-3.pdf) after environmental catastrophes such as asteroid impacts. Such events would slash the population size of species that survive, weakening selection and increasing the chances of greater genomic complexity arising through non-adaptive processes, paving the way for greater physical or behavioural complexity to arise through adaptive processes.

Sure, so lets improve the human race by exposing it to nuclear radiation, or chemical carcinogens that will speed up the mutation rate. This is the sort of fact-free story telling that the author has supposedly eschewed.

Evolution produces creatures perfectly adapted to their environment

*You don’t have to be perfectly adapted to survive, you just have to be as well adapted as your competitors. The apparent perfection of plants and animals may be more a reflection of our poor imaginations than of reality.*

It’s a theme repeated endlessly in wildlife documentaries. Again and again we are told how perfectly animals are adapted to their environment. It is, however, seldom true.

Take the UK ‘s [red squirrel](http://www.newscientist.com/article/mg14920134.100-red-or-dead.html). It appeared perfectly well adapted to its environment. Until the grey squirrel arrived, that is, and proved itself rather better adapted to broadleaf forests thanks, in part, to its ability to digest acorns.

Certainly there is a gradation in complexity and a variety of features that enable plants and animals to adapt to different environments. But one would not claim that the [Wright brother’s](https://creation.com/the-wright-brothers-pioneers-of-the-skies) first man made (but [not first overall](https://creation.com/100-years-of-airplanesbut-these-werent-the-first-flying-machines-creation-magazine)) heavier-than-air flying machine was not designed, simply because there are far more complex planes now. A future instalment will discuss alleged design flaws further. But remember that the Bible tells us that we live in a fallen creation where things are no longer perfect.

There are many reasons why evolution does not produce ‘designs’ that are as good as they could be. Natural selection’s only criterion is that something works, not that it works as well as it might. [Botched jobs](http://www.newscientist.com/channel/life/mg19526161.800-evolutions-greatest-mistakes.html) are common, in fact. The classic example is the [panda’s thumb](http://pandasthumb.org/about.html), which it uses to grasp bamboo. ‘The panda’s true thumb is committed to another role. [So the panda must… settle](http://www.stephenjaygould.org/library/gould_panda%E2%80%99s-thumb.html) for an enlarged wrist bone and a somewhat clumsy, but quite workable, solution,’ wrote [Stephen Jay Gould](http://www.newscientist.com/article/dn2306-stephen-jay-gould-biologist-and-writer-dies-.html) in 1978.

On closer inspection, however, there is nothing clumsy at all about the panda’s design.12 Instead, the ‘thumb’ is part of an elaborate and efficient grasping structure that enables the panda to quickly strip leaves from bamboo shoots.13

Claims that the panda’s thumb is some kind of non-designed ‘contraption’ is a smokescreen to distract from the real question—that evolution simply does not explain how life could start in a pond and finish with a panda.14

As this example shows, evolution is far more likely to reshape existing structures than to throw up novel ones. The lobed fins of early fish have turned into structures as diverse as wings, fins, hoofs and hands.

See illustration and discussion in [The horse shows that similarities are due to creation!](https://creation.com/the-non-evolution-of-the-horse)

[We have five fingers](http://www.sjgarchive.org/library/text/b16/p0405.htm) because [our amphibian ancestors](http://www.newscientist.com/article/mg16722524.200-one-small-step-for-fish-one-giant-leap-for-us.html) had five digits, not because five is necessarily the optimal number of fingers for the human hand.

Yet the creatures they claim to be possible common tetrapod ancestors *did not have five digits! Acanthostega*had eight, while*Ichthyostega*had seven.

Credit Vij Sodera: *One Small Step to Man* (see below)Difference between reptile’s bellows lung and bird’s one-way lung. Click [here](https://dl0.creation.com/articles/p062/c06207/6207reptilevlung600.jpg) to view larger image.

Many groups simply never evolve features that might have made them even more successful. Sharks lack the gas bladder that allows bony fish to control their buoyancy precisely, for example, and instead have to rely on swimming, buoyant fatty livers and, occasionally, a gulp of air. Similarly, mammals’ two-way lungs are far [less efficient](http://www.newscientist.com/channel/life/mg19526161.800-evolutions-greatest-mistakes.html) than birds’ one-way lungs.

But still good enough. On evolutionists’ dating, sharks have thrived for 300 million years without the gas bladder. One could ask why fish evolved such a thing that was clearly not necessary! And the transition from a bellows lung (as reptiles also possess) to an avian flow-through lung (see [Blown away by design](https://creation.com/blown-away-by-design-michael-denton-and-birds-lungs)) has not been explained by evolutionists. One evolutionary expert in lungs explains the problem:

‘The earliest stages in the derivation of the avian abdominal airsac system from a diaphragmatic-ventilating ancestor would have necessitated selection for a diaphragmatic hernia [i.e. hole] in taxa transitional between theropods and birds.

‘Such a debilitating condition would have immediately compromised the entire pulmonary ventilatory apparatus and seems unlikely to have been of any selective advantage.’ 15

And sometimes creatures evolve features that actually [reduce their overall fitness](http://edge.org/q2008/q08_15.html) rather than increase it, such as the peacock’s tail.

Vij Sodera: *One Small Step to Man* (see below)Hypothetical stage of evolution of bird’s lung.

The [complex design of the peacock tail](https://creation.com/the-beauty-of-the-peacock-tail-and-the-problems-with-the-theory-of-sexual-selection) is indeed a problem for evolutionists, especially as the sexual selection theory, which did not explain its intricate design anyway, has been disproven for this case—the very thing that Darwin invoked sexual selection to explain! See [Peacock tail tale failure](https://creation.com/research-bombshell-females-unimpressed-by-peacock-tail).

**Use it or lose it**

Continual mutation also means that if you don’t use it, you lose it. For instance, many primates [cannot make vitamin C](http://dx.doi.org/10.1016/j.ygeno.2003.08.018), because of a gene mutation. This mutation makes no difference to animals that get plenty of vitamin C in their diet. However, when the environment changes, such loss of function can make a big difference, as one primate discovered on long sea voyages.

This may well be true. The Creation/Fall model predicts some deterioration in design. But this doesn’t help evolution, because guinea pigs likewise are unable to produce vitamin C, but share some of the apparent degrading errors seen in the human DNA. Here is a case where the [shared mistakes are not due to common ancestry](https://creation.com/potentially-decisive-evidence-against-pseudogene-shared-mistakes). For a detailed treatment that shows that this evolutionary story fails the test see: [Why the shared mutations in the Hominidae exon X GULO pseudogene are not evidence for common descent](https://dl0.creation.com/articles/p062/c06207/j21_3_118-127.pdf)

Evolution’s lack of foresight can produce inherently flawed designs. The vertebrate eye – with its [back-to-front wiring](http://www.newscientist.com/channel/life/mg19526161.800-evolutions-greatest-mistakes.html) and blind spot where the wiring goes through the retina – is one example.

Not this boring old canard again! It would be nice if the propagandists for evolution actually researched the reason for the [backwardly wired retina](https://creation.com/is-our-inverted-retina-really-bad-design-journal-of-creation-tj): the need for a [blood supply behind the photoreceptors](https://creation.com/an-eye-for-creation-george-marshall-interview-creation-magazine). They should also learn how the [Müller glial cells act as a fibre optic plate to guide the light through the nerve network without distortion](https://creation.com/fibre-optics-in-the-eye). So the vertebrate eye is superbly designed, as the eyesight of eagles testifies!

…

**Evolution’s peak?**

Humans are not running fast enough. Evolving through natural selection is about time and numbers. The number of new mutations that appear, and the number of chances that natural selection has to eliminate the harmful and favour the beneficial ones, depends on the size of a population, the number of offspring each individual has and on the number of generations, among other things.

We might like to think of ourselves as the most ‘[highly evolved](http://genomicron.blogspot.com/2007/04/chimps-are-not-more-evolved-than-humans.html)‘ species but, in terms of how many rounds of mutation and selection we’ve undergone, we are one of the [least evolved species](http://dx.doi.org/10.1371/journal.pbio.0030042).

Around [10 billion new viral particles](http://science-education.nih.gov/supplements/nih1/diseases/activities/activity5_aids-database2.htm) can be produced every day in the body of a person infected with [HIV](http://www.newscientist.com/article/dn9948-instant-expert-hiv%E2%80%93aids.html). By contrast, the total human population on Earth was no more than a few million until a few thousand years ago.

Indeed, Dr John Sanford (see [above](https://creation.com/refutation-of-new-scientists-evolution-24-myths-and-misconceptions-natural-selection#sanford)), shows that the known rate of harmful mutations accumulation really would have resulted in error catastrophe (i.e., extinction!) if we had really been around for millions of years16 (see his research papers published in secular journals17,18).

Furthermore, in a decade bacteria can produce 200,000 generations — about the number of generations of humans there have been since our lineage split from that of chimpanzees. So it’s hardly surprising that in less than a human lifespan we’ve seen the evolution of new diseases such as [HIV](http://endogenousretrovirus.blogspot.com/2007/08/michael-behe-please-allow-me-to.html) and numerous antibiotic-resistant bacteria.

Behe’s second book, *The Edge of Evolution*,19 covers the issue of beneficial mutations and the limits of Darwinian processes. As his Ph.D. research involved malaria, he applies his expertise to the malarial parasite (*Plasmodium falciparum*) and the mutations humans have to deal with it, and the parasite’s counter-measures to human-made drugs.

One of the most effective anti-malarial drugs is chloroquine, because the parasite took longer to develop resistance to this. Behe shows that chloroquine resistance likely involves *two* specific mutations occurring together in the one gene. This explains why resistance to chloroquine took a long time to develop, whereas resistance to other anti-malarial drugs, which only needs *one* mutation, occurs within weeks. Behe works out the probability of this double mutation occurring in the same gene, using other scientists’ figures for the parasite’s population, etc.

If it took so much time for a double mutation to occur in an organism that has a huge population and short life cycle (and therefore huge opportunity for all manner of mutations to occur), then how long would it take for a double mutation to occur in an organism like a human, with a long generation time and small population? Behe showed that it would never occur even with evolutionary time assumed. And this is just one double mutation in a gene. So, any adaptation that requires two specific mutations in one gene to work, will *never* evolve in a human, and yet such must have happened *numerous* times if humans arose through evolutionary processes.

Behe also points out that the chloroquine-resistant parasites do *worse* than the non-resistant ones where there is no chloroquine. This suggests that the double mutation is informationally downhill, as usual. It seems that the reason that the parasite is resistant to chloroquine is that concentration in the parasite’s vacuole is reduced, and one mechanism is *impaired uptake*. According to one paper:

‘Chloroquine-resistant parasite isolates consistently have an import mechanism with a lower transport activity and a reduced affinity for chloroquine.’

This is the same *principle* that explains some antibiotic-resistant bacteria, where a mutation confers resistance by impairing a cell pump so the germ pumps in less of its would-be executioner.20

[Antibiotic resistance] is not so much an arms race as trench warfare or a scorched earth policy. Many of the changes are destroying machinery that the enemy could otherwise use.

This leads to another of Behe’s major points: *there is not so much an arms race as trench warfare or a****scorched earth policy***. Many of the changes are *destroying machinery that the enemy could otherwise use*. E.g. defenders will destroy their own bridges to prevent an enemy crossing, sabotage their own factories if the enemy is using them to churn out armaments, burn their own crops so the enemy will run out of food … This is why the [world-class expert on sickle cell anemia, Dr Felix Konotey-Ahulu, rejects this icon of evolution](https://creation.com/exposing-evolutions-icon).

Behe further reinforced the point by citing microbiologist Barry Hall on carbapeneme antibiotics:

‘Instead of assuming that [the chief kind of enzyme that might destroy these antibiotics] will evolve rapidly, it would be highly desirable to accurately predict their evolution in response to carbapeneme selection.’21

Hall showed that most antibiotics failed, but one (‘iminepen’) did not, simply because neither single nor double point mutations would suffice, but it would require more than two simultaneous mutations. Hall wrote that this was beyond the reach of mutation + selection:

‘The results predict, with >99.9% confidence, that even under intense selection the [enzyme] will not evolve to confer resistance to imipenem.’

See also the explanations of [nylon-eating bacteria](https://creation.com/the-adaptation-of-bacteria-to-feeding-on-nylon-waste), [Lenski’s citrate-eating bacteria](https://creation.com/bacteria-evolving-in-the-lab-lenski-citrate-digesting-e-coli), and [B-cell hypermutation](https://creation.com/web-cast-questions-and-answers-2002), showing why these cases are irrelevant to goo-to-you evolution.

Biophysicist Dr Lee Spetner in his book [Not By Chance](https://creation.com/store_redirect.php?sku=10-3-085) analyzes examples of mutational changes that evolutionists have claimed to have been increases in information, and shows that they are actually examples of *loss of specificity*, which means they involved loss of information (which is to be expected from information theory). See also this discussion, [Is antibiotic resistance really due to increase in information?](https://creation.com/is-antibiotic-resistance-really-due-to-increase-in-information)

**The 3 Rs of Evolution: Rearrange, Remove, Ruin—in other words, *no* evolution!**

The genetic changes observed in living things today could not have turned bacteria into basset hounds—*ever*

Credit: ©iStockphoto.com/fotojagodka|©iStockphoto.com/GlobalP

***by***[***David Catchpoole***](https://creation.com/david-catchpoole-cv)

Evolution textbooks cite variation as being something upon which ‘evolution depends’.1 However, when one examines closely the claimed ‘demonstrable examples’ of ‘evolution’, they actually fall into three categories, which we can label here as the ‘3 Rs’.

Let’s look at each of these in turn.

‘R’#1:**Rearrange** existing genes

Careful examination of many purported instances of ‘evolution in action’ shows that such ‘variation’ actually *already exists*, conferred by genes that *already exist*.

Here’s a simplified example that shows this, and also how such genetic variety might be misconstrued as ‘evidence of evolution’. The two dogs in the top row of Figure 1 are a male and a female. They each have a gene that codes for short hair (inherited from its mother or father) and a gene that codes for long hair (inherited from the other parent). In combination, this gene pair for fur length results in medium length hair.2

**Figure1.** The red bars represent the genes for hair length (short and long hair). One of each gives medium length hair. By re-arranging (recombining) the parents’ genes (top) in reproduction, variety is generated in the appearance of the offspring, but no new genes are involved.

Now when these two dogs are crossed, what new combinations of the genes are possible in the resulting offspring? The second row of Figure 1 shows this:

There’s no new information here of the kind needed to have turned pond scum into poodles, Pekingese, pointers and papillons

The dog at the far left has inherited its father’s short-hair gene and its mother’s short-hair gene. Result: short hair.

The two dogs in the middle have each inherited a long-hair gene from one parent and a short-hair gene from the other parent. Result: medium-length hair (just like the mother and father).

The dog at the far right has inherited its mother’s long-hair gene and its father’s long-hair gene. Result: long hair.

A casual observer, looking only at the outward appearance, i.e. unaware of what is happening at the genetic level, might think: “There were no long-hair dogs in the parents’ generation. This long hair is a new characteristic—evolution is true!”

But such a view is incorrect. The only thing this ‘evolution’ has done is to *rearrange existing genes*. There’s simply been a sorting out of *pre-existing* genetic information. There’s no new information here of the kind needed to have turned pond scum into poodles, Pekingese, pointers and papillons.

‘R’#2: **Remove** genetic information

What about natural selection, adaptation and speciation?

None of these represent the generation of any *new* microbes-to-mastiff genetic information either. In our ‘hairy dog’ example, if we were to send our new population of dogs, some with short hair, others with medium or long hair, to an icy, very cold location, we wouldn’t be at all surprised to see natural selection at work, killing off any dog that didn’t have long hair (Figure 2, Line 1). When the survivors reproduce, the only fur-length genes passed on to the offspring are those that code for long hair (Figure 2, Line 2).



Thus we now have a population of dogs beautifully *adapted*to its environment. Biologists encountering our ice-bound population of dogs, observing them to be isolated3 from other populations of dogs, could argue that they be given a new *species* name.

So here we see *natural selection*, *adaptation, and possibly even speciation*—but *no new genes* have been added. In fact, there’s been a *loss* of genes (the genetic information for short-and medium-length hair has been removed from the population).

… crop and livestock breeders are scouring the world for the original genes created during Creation Week.

Note that such examples of natural selection, adaptation and speciation are often portrayed as evidence for evolution, but the only thing this ‘evolution’ has done is to *remove* existing genes. If this population of exclusively long-hair dogs were now forcibly relocated to a steamy tropical island, the population could not ‘adapt’ to the hot climate unless someone re-introduced the short-hair gene to the population again, by ‘back-crossing’ a short-or medium-length hair dog from elsewhere. This is exactly the sort of thing that our crop and livestock breeders are doing. They are scouring the world for the original genes created during Creation Week4 but which have subsequently been ‘bred out’ (lost) from our domestic varieties/breeds of plants and animals because of breeders artificially selecting certain characteristics, which means other features are de-selected (lost).

‘R’#3: **Ruin** genetic information

In the above examples, we see that natural selection, adaptation and speciation are real and observable. And that these simply demonstrate the *rearranging* and/or *removing* of dog genes that were *originally present at Creation*. (I.e. by the end of Day 6, when God *completed* Creation, declaring it ‘very good’—[Genesis 1:31](https://biblia.com/bible/esv/Gen%201.31).)

©iStockphoto.com/Humonia**Figure 3:** Dogs with the floppy ear mutation, such as bassets, are much more prone to ear infections (e.g. from food scraps) than dogs with erect ears (they clearly can’t hear as well either!)

However, there are forms of dog genes today which were *not* present at Creation but have arisen since. But those have not arisen by any *creative* process, but by *mutations*, which are copying mistakes (typos, we might say) as genes are passed from parents to offspring. You would expect such accidental changes to *wreck* the existing genes, and that’s what happens. For example, the dog pictured in Figure 3 has just such a mutated gene, resulting in ‘floppy ear syndrome’.5

Dogs with this genetic mutation have weaker cartilage and cannot lift up their ears. So they just hang, floppy before dinner, and sloppy after it—unless their owners are diligent in cleaning them. Such regular attention to ear hygiene is necessary, as dogs with floppy ears are prone to serious ear infections, which can even lead to hearing loss.6 Not that their hearing was especially good anyway. As you might expect, dogs with erect ears are far superior to floppy-eared dogs at detecting prey by sound.7

I can remember reflecting on this when I was an atheist/evolutionist, and wondering how such floppy-eared dogs could have ever evolved and survived in the wild. I now know that they *didn’t*. Instead this mutation in the genes has arisen since the original “very good” world ([Genesis 1:31](https://biblia.com/bible/esv/Gen%201.31)) was cursed as a result of Adam’s sin ([Genesis 3:17–19](https://biblia.com/bible/esv/Gen%203.17%E2%80%9319)). The floppy-eared mutation in dogs is but one example of how a post-Fall world is very much “in bondage to decay” ([Romans 8:19–22](https://biblia.com/bible/esv/Rom%208.19%E2%80%9322)). So common is this mutational defect in modern domestic dogs that many people have naïvely come to think of floppy-eared dogs as ‘normal’. But Adam and Eve, if they were alive today, would no doubt be shocked to see such deformity. The original dogs, probably something like today’s gray wolves, would have had *erect*, superbly functional, ears.

Why is this so important to consider, in the context of evolutionary claims that no Creator was necessary?

Evolutionary biologists, when pressed with the *facts* about natural selection, will concede that natural selection by itself can only remove existing genetic information. However, they argue that in tandem with mutations, natural selection would be a creative process.

But the floppy-ear mutation, for one, is a classic example of the widespread *degradation* of the genome—a *downhill* process. For microbes-to-man evolution to be true, evolutionists should be able to point to thousands of examples of information-gaining mutations, an *uphill* process, but they can’t.8 Mutations overwhelmingly *ruin* genetic information. Therefore evolutionists looking to mutations as being evolution’s ‘engine’ do so in vain.9 Thus they are left with no known mechanism capable of *ever* turning microbes into mutts—i.e. no way of ‘climbing’ up the supposed evolutionary ‘tree’.

Note that while mutations degrade genetic information, sometimes an advantage arising from such degradation can outweigh the disadvantage vis-à-vis survival. While a floppy-eared mutant mutt might not last long in the wild, under human care—i.e. with regular ear cleaning—the equation changes. And what about the key moment when a buyer is looking for the ‘cutest’, friendliest pup in the pet shop window? Indeed, there is increasing evidence that the floppy-eared characteristic is strongly associated with tameness.10,11 Little wonder then, that floppy-eared dogs are so common today.12

Conclusion: 3 Rs = no **new**information = no evolution

The above examples of changes in fur length and ear structure of dogs are not evolutionary changes, though they are often claimed as such. ***R***earranging genes, ***R***emoving genes, and ***R***uining genes are not the sort of genetic changes that could have turned bacteria into basset hounds—*ever*. These ‘3 Rs’ are repeatedly cited as evolution in a host of other settings, too, e.g. in antibiotic and pesticide resistance, and in sticklebacks, beetles, mosquitoes, worms, sheep, and codfish.13 But none of these are evidence of evolution. The ‘3 Rs’ could never add up to mosquitoes, mesquite, mutts and man from microbes (let alone from molecules!).

The evidence instead fits with the biblical account of God having created a multiplicity of ‘kinds’, each programmed to reproduce according to its kind. Geneticists recognize that the diversity of dog breeds we have today could have arisen *quickly*, in recent history.14 As we’ve seen in our fur length example, long hair and short hair can appear in *just one generation*, arising from the in-built canine genetic variation—variation that was built-in to dogs at Creation. So Noah didn’t need to take on board the Ark multiple pairs of dingoes, Dalmatians, and dachshunds; or coyotes, corgis, and cocker spaniels; or jackals, jack russells, and jackadoodles. He only needed *two* dogs—just as the Bible suggests ([Genesis 6:19–20](https://biblia.com/bible/esv/Gen%206.19%E2%80%9320)).

**Natural selection ≠ evolution**

***by***[***Marc Ambler***](https://creation.com/marc-ambler)



This is an important ‘equation’1 that all people should be aware of, namely ‘Natural Selection does not equal (**≠**) Evolution’.2 Christians should know it so they do not get conned, and evolutionists should know it as a reminder that they still have lots of work to do to be able to claim that they have a mechanism for evolution.

How often we hear an example of natural selection being used as proof of evolution. Changing sizes, colours, skin patterns and shapes are often paraded as evolution’s honour roll. This bait-and-switch tactic has been so often exposed for what it is, it’s a wonder that it is still used, or that people are still taken in by it.

The very term should put people on their guard that something is missing. If we think of the word ‘selection’, in our common, daily experience, we select from something pre-existing. Think of being asked to select cards from a pack. You could select cards from a pack every second for the rest of your life and all you would only ever produce is different groups of the same cards. You would not have created anything new—only re-arranged cards, or removed cards or added cards from another pack.

If we think of the word ‘selection’, in our common, daily experience, we select from something pre-existing.

If an illusionist asks you to select a card from a pack, and surprises you with something new, you know it is an illusion, a sleight of hand. We need to learn to see the evolutionists’ sleight of hand when they claim to have pulled something ‘new’ out of the pack. Selection is always from a pre-existing series or range; it creates nothing new.

This illustration applies equally to ‘selection’ in the biological context. The all-wise Creator knew the different environments that His creatures would have to adapt to after the Fall and Curse, and particularly after the Flood of Noah, in order to survive. He included in the genetic information of each ‘kind’ of creature He created, a smorgasbord of variety in their makeup. This includes those features that would interact with the environment: the overall size of a plant, animal or person; the size of individual organs or limbs such as beaks and noses, leaf sizes, skin colours, hair and feather lengths, textures and colours. All of these and many more variations were programmed into the DNA of His creatures in order that as populations of the various kinds moved into new environments, expression of those variations enabled individuals to survive those environments. Individuals with those variations then passed them on to their young. When these variations and the habitat of the population expressing that variation are distinct enough, we might distinguish different ‘species’. In all of this selection process, new information is never added. It can be conserved or lost, but never gained.



The creationist chemist/zoologist Edward Blyth (1810–1873) wrote about natural selection about 25 years before Darwin misappropriated it to support his theory of evolution. Blyth clearly saw this remarkable phenomenon as arising from the providence of the all-wise, all-knowing, ingenious Creator God.

Knowing God’s love for beauty (reflected in men and women who are made in His image), God probably also had in mind the spectacular array of birds, fish, dogs and cats that we have varied by ‘artificial selection’ purely for the sake of ‘beauty’ rather than survival.3

But whether variation is selected naturally by the environment, or artificially by breeders for a particular trait, it remains just that, ‘selection’ from existing genetic information. Nothing new is created.

**Evolution desperately needs ‘Natural Invention’, ‘Natural Novelty’ and ‘Natural Creation’.**

Patent law calls for a product to have an ‘inventive step’ in order for it to be patented. Mere changes in design of an existing product cannot be patented. Many legal battles over patent rights have been waged over this point. Evolution requires the same thing—an ‘inventive step’, a novel organ or body part, facilitated by new information in the DNA that wasn’t there before. Despite the huge resources thrown at evolution in universities and research institutions, natural selection has never been shown to bring about this type of ‘inventive step’.

Today’s Darwinists point to mutations as the mechanism which provides this novelty from which ‘Natural Selection’ selects. Evolutionists should then focus on mutations to defend their theory, instead of ‘Natural Selection’. When pressed for examples of novel genetic information or body organs created by mutation, they typically point to instances such as wingless beetles4 on islands, or the flightless cormorant on the Galapagos islands.5 The problem with these examples is obvious. While they may confer a benefit to the creatures in a specific, very unusual environment, nothing ‘new’ is added to the DNA or creatures’ body parts. They actually involve a *loss* or *corruption* of existing genetic information.6

Evolution desperately needs ‘Natural Invention’, ‘Natural Novelty’ and ‘Natural Creation’. ‘Natural Selection’ just does not pass muster as exhibit A for evolution. Rather, it is a wonderful tribute to God’s design, and His providence for a fallen world. **Natural Selection ≠ Evolution.**